Cardiac System

PHYSIOLOGY OF THE CARDIAC SYSTEM

Structure of the Heart
A. The heart is located in the mediastinal space of the thoracic cavity.
B. The apex of the heart points downward and to the left; the apex comes in contact with the chest wall at about the fifth to sixth intercostal space. In the healthy individual, the point of maximum impulse (PMI) may be palpated here; this is also the area to auscultate when evaluating the apical heart rate.
C. The heart is contained in a loose sac called the pericardium.
   1. Fibrous pericardium: the outer surface.
   2. Parietal layer: lines the fibrous pericardium.
   3. Epicardial (visceral layer): vascular and adherent to the heart.
   4. There is a potential space between the visceral and parietal layers of the pericardium. This area contains about 5 to 20 mL of pericardial fluid to lubricate the sac and prevent friction from cardiac movement.
D. Myocardial wall.
   1. Epicardium: the outer surface.
   2. Myocardium: the middle layer of cardiac muscle.
   3. Endocardium: the lining of the inner surface of the cardiac chambers.
E. Cardiac chambers (Figure 17-1).
   1. Four chambers are located within the heart; these chambers represent two pumps.
   2. Both atria are the receiving chambers; both ventricles are the ejecting chambers.
   3. The right side of the heart has a thinner myocardium than the left side and is a low-pressure system.
   4. The left ventricle is composed of a thicker muscle, is a high-pressure system, and is capable of generating enough force to eject blood through the aortic valve and through the systemic circulation.
F. Cardiac valves: maintain the directional flow of blood through the heart chambers.
   1. Atrioventricular valves are controlled and supported by papillary muscle connected to the ventricular muscle and chordae tendineae extending from papillary muscle to valve leaflets.
   a. The tricuspid valve lies between the right atrium and the right ventricle.
   b. The mitral valve lies between the left atrium and the left ventricle.
   c. Both valves prevent backflow of blood from the ventricles into the atria during systole.
   2. Semilunar valves (cusp valves) are controlled by the backward pressure of blood flow at the end of systole.
   a. Pulmonic valve: the outflow valve of the right ventricle into the pulmonary circulation.
   b. Aortic valve: the outflow valve of the left ventricle into the aorta.
   c. Both valves prevent the backflow of blood from the pulmonary artery and the aortic arch into the ventricle during diastole.
G. Direction of blood flow through the heart structure (see Figure 17-1).
   1. From the venous system, the blood enters the right atrium via the superior and inferior vena cavae; it flows through the tricuspid valve into the right ventricle; it is ejected through the pulmonic valve into the pulmonary artery; and it flows to the lungs for oxygenation.
   2. Oxygenated blood returns to the left atrium via the pulmonary veins; it flows through the mitral valve into the left ventricle; it is ejected through the aortic valve into the aortic arch; and it flows into the systemic circulation.
   3. The pulmonary artery is the only artery in the circulatory system to carry deoxygenated blood; the pulmonary vein is the only vein in the circulatory system to carry oxygenated blood.

Cardiac Function
A. One complete cardiac cycle consists of contraction of the myocardium (systole) and subsequent relaxation of the myocardium (diastole).
B. The amount of blood ejected with ventricular contraction is the stroke volume.
C. Starling’s law of the heart: the greater the cardiac muscles are stretched, the more forceful the contraction. If an increased amount of blood flows into the heart, the heart will increase the force of contraction and eject a larger amount of blood.
D. Cardiac output (CO = SV × HR).
1. The cardiac output can be determined by multiplying the stroke volume (SV) by the heart rate (HR) in beats per minute (CO = SV × HR).
2. The heart pumps approximately 5 L of blood every minute.
3. The heart rate increases with exercise; therefore, cardiac output increases.
4. The cardiac output will vary according to the amount of venous return (preload).
5. Factors regulating stroke volume.
   a. Degree of stretch of the cardiac muscle before contraction (Starling’s law): determined by the volume of blood in the ventricle at the end of diastole or diastolic filling.
   b. Contractility: ability of the myocardium to contract; contractility is increased by circulating catecholamines and medications such as digitalis.
   c. Preload: the filling of the ventricles at the end of diastole. The more the ventricles fill, the more the cardiac muscles are stretched, and the greater the force of the contraction during systole (Starling’s law). If there is a decrease in the preload, there is also a decrease in contractility and in cardiac output.
   d. Afterload: the pressure in the aorta that the ventricles must overcome to pump blood into the systemic circulation. A decrease in the afterload causes a decrease in the workload of the ventricles; this in turn helps increase the stroke volume and the cardiac output (Figure 17-2).
E. Innervation of the myocardium (autonomic nervous system).
1. Parasympathetic (vagus nerve) stimulation.
   a. Slows rate of impulse generation at the sinoatrial (SA) node.
   b. Slows transmission of the impulse through the atrioventricular (AV) node.
   c. Atropine blocks vagal stimulation to the heart.
2. Sympathetic stimulation.
   a. Increases heart rate.
   b. Increases force of contraction.
F. Factors that increase myocardial oxygen demands.
1. Increased heart rate.
2. Increased force of contractions.
3. Increased afterload.
G. Cardiac compensatory mechanisms: when the normal compensatory mechanisms cannot maintain cardiac output to meet body needs, the client is in a state of cardiac decompensation.
1. Acute.
   a. Sympathetic nervous system receptors initiate an increase in the release of epinephrine and norepinephrine to increase the heart rate and myocardial contractility.
   b. Increased diastolic filling (preload) increases cardiac output by increasing the stretch of the myocardial muscle fibers, thus increasing the contractility (Starling’s law). In a healthy heart, this is the mechanism that functions when there is a need for increased cardiac output, as in exercise. The increase in the stretch of the myocardial fibers and the increase in the contractile force also require an increase in oxygen consumption. If the myocardial fibers have a decreased oxygen supply and/or the demand on the myocardial muscle is increased for a prolonged period, decompensation will occur.
2. Chronic: ventricular hypertrophy increases cardiac output by increasing the size of the myocardial muscle. This also increases the myocardial need for
oxygen, and in the diseased myocardium, the hypertrophy will eventually lead to a decompensated state.

**Myocardial Blood Supply**
A. Coronary arteries.
1. Originate at the coronary sinus, just outside the aortic valve.
2. Provide the only source of oxygenated blood for the myocardium.
3. Arteries fill during diastole; a diastolic pressure of 60 mm Hg is required to adequately perfuse the coronary arteries.
B. Collateral circulation.
1. There are no direct connections between the large coronary arteries.
2. When gradual occlusion of large coronary vessels occurs as a result of arteriosclerotic heart disease (ASHD), the smaller vessels increase in size and provide alternative blood flow.
3. Because of the development of collateral circulation, coronary artery disease may be well advanced before the client experiences symptoms.

**Conduction System**
A. Controls the rate and rhythm of the heart.
B. Located in the myocardium are pathways for conduction of an electrical impulse that initiates contraction of the heart muscle.
C. Characteristics of cells in the conduction system.
1. Automaticity: the ability of certain conductive pathway cells to initiate an impulse spontaneously and consistently.
2. Excitability: the ability of a cell to respond to an impulse.
3. Conductivity: the ability of a cell to conduct an electrical impulse.
4. Refractoriness: the inability of a cell to respond to incoming stimuli.
D. Impulse generation.
1. Resting state: cell is ready to receive an impulse.
2. Depolarization: flow of electrical current along cardiac membrane, initiating muscle contraction.
3. Repolarization: cells regain the electrical charge and are returned to a resting state.
E. Relationship of conducting pathways to the electrocardiogram (ECG) (Figure 17-3).
1. P wave: indicative of the impulse generated from the sinoatrial node; initiates atrial depolarization.
2. PR interval: delay of the impulse at the atrioventricular node and bundle of His to promote ventricular filling.
3. QRS complex: passage of the impulse through the bundle of His, down the bundle branches, through the Purkinje fibers; depolarization of the ventricle occurs.
4. T wave: ventricular repolarization and return to the resting state.
5. S-T segment: above the baseline in cardiac injury and below the baseline with ischemia.

![FIGURE 17-3 Normal electrocardiogram (ECG). (From Hockenberry MJ, Wilson D: Wong’s nursing care of infants and children, ed 8, St. Louis, 2007, Mosby.)](image)

**System Assessment**
A. Health history.
1. Identify presence of risk factors for the development of arteriosclerotic disease.
2. Coping strategies.
3. Respiratory.
   a. History of difficulty breathing.
   b. Medications taken for respiratory problems.
   c. Determine normal activity level.
   a. History of chest discomfort (Table 17-1).
   b. History of edema, weight gain.
   c. History of syncope.
   d. Medications taken for the heart or for high blood pressure.
B. Physical assessment.
1. What is the general appearance of the client: Is there any evidence of distress? What is the client’s level of orientation and ability to think clearly?
2. Evaluate blood pressure.
   a. Pulse pressure: the difference between systolic and diastolic pressure.
   b. Assess for postural hypotension (decrease in blood pressure when client stands).
   c. Take blood pressure sitting, standing, and lying if client is having problems with pressure changes (see Chapter 16 for accurate blood pressure measurement).
   d. Paradoxical blood pressure (paradoxical pulse): a decrease in systolic blood pressure of at least 10 mm Hg that occurs during inspiration.
3. Evaluate quality and rate of pulse; assess for dysrhythmias (see Appendix 17-7 for determining dysrhythmias).
   a. Pulse deficit: the radial pulse rate is less than the apical pulse rate; occurs in atrial fibrillation.
b. Pulsus alternans: regular rhythm but quality of pulse alternates with strong beats and weak beats.
c. Thready pulse: weak and rapid; difficult to count.
5. Auscultation of the heart (Figure 17-4).
   a. Heart sounds heard during the cardiac cycle.
      (1) S1: closure of the mitral and tricuspid valves.
      (2) S2: closure of the aortic and pulmonic valves.
      (3) S3: represents rapid ventricular filling; normal in children and young adults; in adults older than 30 years, it may be an indication of volume overload, ventricular dysfunction secondary to hypertension.
      (4) S4: extra sounds heard during atrial contraction are abnormal; may indicate a forceful atrial contraction due to increased resistance.
   b. Presence of murmurs created by turbulent blood flow: graded on a scale of loudness.

(1) Abnormal flow through diseased valves: stenosis and insufficiency.
(2) Abnormal flow of blood between cardiac chambers (congenital heart disease).
   c. Presence of a friction rub: usually heard over the apex during S1 and S2; can be heard best with client sitting and leaning forward.

**ALERT** Identify common abnormal heart sounds (e.g., S3, S4).

6. Evaluate adequacy of peripheral vascular circulation and check for presence of peripheral edema.
7. Evaluate for presence of chest discomfort (see Table 17-1).
   a. Location.
   b. Intensity of pain.
   c. Precipitating causes.

**ALERT** Perform focused assessment or reassessment; interpret data that need to be reported immediately (see Box 17-1).

### DISORDERS OF THE CARDIAC SYSTEM

**Angina Pectoris**

Arteriosclerotic heart disease (ASHD), also called coronary artery disease (CAD), occurs as a result of the atherosclerotic process (see Chapter 16) in the coronary arteries. Angina pectoris is caused by myocardial ischemia due to narrowed or blocked coronary arteries. The buildup of plaque or fatty material in the coronary artery causes a narrowing of the lumen of the artery and precipitates myocardial ischemia that causes chest pain.

A. Pain (angina) occurs when the oxygen demands of the heart muscle exceed the ability of the coronary arteries to deliver it.

#### Table 17-1 ASSESSING CHEST PAIN (PQRST)

<table>
<thead>
<tr>
<th><strong>P—Precipitating Factors</strong></th>
<th><strong>Q—Quality</strong></th>
<th><strong>R—Region and Radiation</strong></th>
<th><strong>S—Symptoms and Signs (associated with chest pain)</strong></th>
<th><strong>T—Timing and Response to Treatment</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>May occur without precipitators</td>
<td>Pressure</td>
<td>Substernal or retrosternal</td>
<td>Diaphoresis, cold clammy skin</td>
<td>Sudden onset</td>
</tr>
<tr>
<td>Physical exertion</td>
<td>Squeezing</td>
<td>Spreads across the chest</td>
<td>Nausea, vomiting</td>
<td>Constant</td>
</tr>
<tr>
<td>Emotional stress</td>
<td>Heaviness</td>
<td>Radiates to the inside of either or both arms, the neck, jaw, back, upper abdomen</td>
<td>Dyspnea</td>
<td>Duration &gt;30 min</td>
</tr>
<tr>
<td>Eating a large meal</td>
<td>Smother</td>
<td></td>
<td>Orthopnea</td>
<td>Not relieved with</td>
</tr>
<tr>
<td></td>
<td>Burning</td>
<td></td>
<td>Syncope</td>
<td>nitrates or rest</td>
</tr>
<tr>
<td></td>
<td>Severe pain</td>
<td></td>
<td>Apprehension</td>
<td>Relief with narcotics</td>
</tr>
<tr>
<td></td>
<td>Increases with movement</td>
<td></td>
<td>Dysrhythmias</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Palpitations</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Auscultation of extra heart sounds</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Auscultation of crackles</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Weakness</td>
<td></td>
</tr>
</tbody>
</table>
B. Temporary ischemia does not cause permanent damage to the myocardium. Pain frequently subsides when the precipitating factor is removed.

C. Types of angina.
1. Chronic stable angina: predictable with level of stress or exertion; consistently responds well to medication; pain rarely occurs at rest.
2. Unstable angina (acute coronary syndrome).
   a. Unstable angina pain occurs spontaneously at rest (usually between midnight and 8 a.m.); progressive in frequency of attacks; unpredictable, not relieved with sublingual nitroglycerin.
   b. Variant angina (Prinzmetal angina) pain that tends to be cyclic caused by coronary spasms.
3. New onset angina: first symptoms of angina that most frequently occur after exertion.

**Assessment**

A. Risk factors/etiology.
1. ASHD (Box 17-1).
2. Cardiac ischemia.
3. Aortic valve disease (impedes filling of the coronary arteries).
4. Increased cardiac demands.
   a. Exercise, emotional stress.
   b. Heavy meals, thyrotoxicosis.
   c. Exposure to cold temperature.

B. Clinical manifestations—chronic stable angina.
1. Pain in varying levels of severity (see Table 17-1).
2. Pain most often is located behind or just to the left of the sternum.
3. Pain may radiate to neck, jaw, and shoulders.

4. Client may describe pain as squeezing, choking, or constricting or as a vague feeling of pressure and indigestion.

5. Client will frequently deny seriousness of the pain.

6. Most clients correlate pain with activity and increased cardiac demands.

7. Pain is of short duration, generally lasting about 5 minutes; may be longer if associated with anger or heavy meals.

8. Accompanying symptoms may include diaphoresis, increased anxiety, pallor, and dyspnea.

C. Diagnostics—chronic stable angina (see Appendix 17-1).

**Treatment—Chronic Stable Angina**

A. Primary goal of treatment is to relieve pain and prevent future attacks.

B. Medication.
   2. Beta-adrenergic blockers (Appendix 17-3).
   3. Calcium-channel blockers (Appendix 17-3).
   4. Antiplatelet medications (see Appendix 16-4).

C. Procedures/surgical intervention.
   1. Percutaneous coronary intervention (PCI): a balloon is threaded from an artery in the groin to the affected coronary artery. The balloon is then inflated in an effort to compress the plaque and dilate the narrowed artery reestablishing blood flow to the myocardium.
   2. Laser angioplasty: a special catheter with a laser tip is inserted into coronary artery. When confronted with a blockage, the laser emits pulsating beams of light that vaporize the plaque.
   3. Atherectomy: technique in which the catheter has a rotating shaver on the tip that cuts away the plaque.
   4. An intracoronary stent is an expandable wire mesh that can be inserted during any of the above procedures. A stent serves as a scaffold to maintain patency of the coronary artery.
   5. Cardiac revascularization: coronary artery bypass graft (CABG) surgery, open heart surgery.

D. Restricted activity.

E. Supplemental oxygen.

F. Control of the modifiable risk factors (see Box 17-1).

**Acute Coronary Syndrome: Unstable Angina Pectoris**

According to the American Heart Association, acute coronary syndrome (ACS) includes the various degrees of coronary artery occlusion that can develop in individuals with coronary atherosclerosis.

A. Includes unstable angina, non-ST-segment elevation myocardial infarction (MI), and ST-segment elevation MI, all of which can lead to sudden cardiac death.

B. Unstable angina pectoris occurs when a thrombus partially occludes a coronary artery causing prolonged symptoms of ischemia which can occur at rest.
Assessment
A. Risk factors/etiology.
   1. Family history of coronary artery disease.
   2. Hypertension, hypercholesterolemia.
   3. Diabetes, smoking.
   4. Average age for 1st MI - men over 64.5 years, women over 70.5 years.
   5. Women are at increased risk after menopause.
B. Clinical manifestations.
   1. Two or more anginal events within the past 24 hours.
   2. Prolonged chest pain (greater than 20 minutes).
   3. Presenting symptoms in women: indigestion, pain between the shoulders, shortness of breath, and anxiety.
   4. Hypotension, bradycardia, or tachycardia.
   5. New onset S3.
C. Diagnostics (Appendix 17-1).
   1. 12-lead ECG.
      a. ST segment elevation (STEMI), traditional.
      b. Non-ST elevation MI (NSTEMI), common in women.
   2. Elevated cardiac troponin 1.
   3. Elevated CK-MB.
D. Treatment (initial).
   1. Bed rest.
   2. Monitor vital signs, including oxygen saturation level.
   3. Supplemental oxygen at 4 L/min via nasal cannula (maintain O2 saturation above 90%).
   4. Reduce coronary reocclusion with antiplatelet medications (Appendix 16-4).
   5. Reduce and control ischemic pain: vasodilators (nitroglycerin, sublingual, spray or IV, see Appendix 17-2), narcotics (morphine sulfate IV—infusion or by the nitroglycerin).
   6. Beta-adrenergic receptor blockers (Appendix 17-3) to decrease cardiac demand for oxygen; anticoagulants (Appendix 16-3) to prevent emboli.
   7. Complete fibrinolytic checklist and, if appropriate, initiate fibrinolytic therapy (Appendix 17-5).
   8. Transmyocardial laser revascularization (TMR): laser probe is inserted into the wall of the left ventricle; channels are created to promote the development of revascularization.

Complications
A. Dysrhythmias (see Appendix 17-7).
B. Myocardial infarction (MI).

Nursing Interventions for Angina and Acute Coronary Syndrome

**ALERT** Intervene to improve client’s cardiovascular status; assess client for decreased cardiac output; meet client’s pain management needs; use critical thinking when addressing pain management.

**Goal:** To decrease pain and increase myocardial oxygenation.

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**NURSING PRIORITY** To relieve chest pain and to decrease cardiac damage resulting from an inadequate blood and oxygen supply to the myocardium, there must be an immediate reduction in the workload of the heart that results in a decrease in oxygen consumption: rest, nitroglycerin, oxygen therapy.

**Goal:** To evaluate characteristics of anginal pain and client’s overall response.

A. Does pain increase with breathing? (Anginal pain is generally not affected by breathing or changes of position.)
B. Assess activity tolerance or precipitating factor.
C. Assess changes in characteristics of pain (see Table 17-1).
D. Evaluate response of pain to treatment or progression to more severe level.
E. Obtain a 12-lead ECG.
F. Evaluate vital signs.
   1. Presence of S3 gallop, which may indicate heart failure.
   2. Presence of jugular vein distention; peripheral edema.
   3. Presence of dyspnea or wet breath sounds.
   4. Adequacy of cardiac output: peripheral pulses, urinary output, level of consciousness.
G. Continuous ECG monitoring; assess for presence of dysrhythmia and impact on cardiac output.
H. Monitor troponin levels (Appendix 17-1).
I. Assess client’s psychosocial response: denial is common; anger, fear and depression occur in both client and family.

**ALERT** Intervene to improve client’s cardiovascular status; provide client with strategies to manage decreased cardiac output.

**Goal:** To provide care after percutaneous coronary intervention (with or without stent).

A. Monitor for chest pain and hypotension; reocclusion is a primary complication.
B. Assess for bleeding or hematoma formation.
C. Frequently assess status of circulation distal to area of cannulation.
D. A sheath may be left in place; monitor area for bleeding; if bleeding occurs, put manual pressure on the area and notify the physician.
E. Prevent flexion of affected extremity and maintain bed rest for 6 to 8 hours.
F. Client is to avoid heavy lifting; may return to work in 1 to 2 weeks.
G. Notify the doctor of any chest pain, syncope, or bleeding at the site.
H. Assess ECG for evidence of ST-segment changes.

**Home Care**

A. Education regarding ASHD.
   1. Client will be able to identify personal risk factors and appropriate health practices to decrease risk factors.
   2. Client will be able to identify factors precipitating pain.
B. Determine that client understands his or her medication regimen (Box 17-2).
C. Assess understanding of diet and exercise regimen.
D. Assess understanding of seeking medical assistance if pain persists and is not relieved by medication.
E. Help client identify resources for counseling to decrease stress.
F. Advise client not to take erectile dysfunction drugs (Appendix 22-1) if on nitrates for chest pain.

**Acute Coronary Syndrome: Myocardial Infarction**

A myocardial infarction (MI), also called coronary occlusion or heart attack, is a total occlusion of a portion of a coronary artery. After the occlusion, myocardial ischemia, injury, or death occurs.

A. Infarction most often occurs in the area of the left ventricle.
B. The severity of the situation depends on the area of the heart involved, as well as the size of the infarction.
C. Healing process.
   1. In the first 24 hours, the inflammatory process is well established; leukocytes invade the area, and cardiac enzymes are released from the damaged cells.
   2. In 4 to 10 days: necrotic zone is well defined.
   3. In 10 to 14 days: the formation of scar tissue foundation begins.
D. The presence of pre-established collateral circulation will assist in decreasing the size of the necrotic area.

**NURSING PRIORITY** Danger of death from an MI is greatest during the first 2 hours.

**Assessment**

A. Risk factors/etiology (see unstable angina).
B. Clinical manifestations.
   1. Typical pain is severe, substernal, crushing, and unrelieved by nitroglycerin.
   2. Denial of the seriousness of the pain; clients with MI frequently wait more than 2 hours to seek assistance.
   3. Dyspnea, nausea, vomiting, indigestion.
   4. Pale, dusky skin.
   5. Pain may radiate down arm or up the jaw.
   6. Onset is usually sudden.
   7. Diaphoresis; extreme weakness.
   8. Decrease in blood pressure, tachycardia, syncope.
C. Diagnostics: see acute angina (see Appendix 17-1).

**Treatment** (Figure 17-5)

A. Bed rest.
B. Monitor vital signs, oxygen saturation, and ECG.
C. Supplemental oxygen at 4 L/min via nasal cannula (maintain O₂ sat above 90%).
D. Pain relief (morphine sulfate IV).
E. Beta-adrenergic receptor blockers (Appendix 17-3); antiplatelets (Appendix 16-4); anticoagulants (Appendix 16-3).
F. Anti dysrhythmic agents (Appendix 17-4).
G. Reperfusion (fibrinolytic) therapy (Appendix 17-5).
H. Percutaneous coronary intervention (PCI): “door-to-balloon” goal of 90 minutes for optimum response.
I. Cardiac surgery if appropriate.

**Complications**

A. Dysrhythmias (see Appendix 17-7).
B. Cardiogenic shock.
C. Heart failure (congestive heart failure).
Immediate Treatment of an MI

**NURSING PRIORITY** As long as chest pain persists, cardiac ischemia continues. If client experiences tachycardia, decrease activity whether client has chest pain or not.

**Goal:** To decrease pain and increase myocardial oxygenation (see Nursing Interventions for Angina and Acute Coronary Syndrome).

**Alert** Identify potential/actual stressors for the client; implement measures to reduce environmental stress; promote methods to reduce stress.

**Goal:** To evaluate characteristics of cardiac pain and client’s overall response (also see Nursing Interventions for Angina and Acute Coronary Syndrome).

- A. Continuous cardiac monitoring to identify and treat dysrythmias affecting cardiac output.
- B. Frequent assessment for dysrythmias, murmurs, and presence of S3, S4.
- C. Maintain IV access.
- D. Maintain bed rest for the first 24 hours.

**Alert** Determine changes in client’s cardiovascular system. Monitor progress of client with cardiac disease and evaluate tolerance to changes in activity.

**Immediate Treatment of an MI**

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**Alert** Determine changes in client’s cardiovascular system. Monitor progress of client with cardiac disease and evaluate tolerance to changes in activity.

**Home Care**

- A. Participate in organized cardiac rehabilitation program.
- B. Understand medication regimen.
- C. Teach client how to take his or her pulse and check for rate and regularity.
- D. Teach client how to evaluate response to exercise (dyspnea, tachycardia, chest pain, etc.).
- E. Call the doctor for pain not controlled by nitroglycerin, significant changes in pulse rate, decreased tolerance to activity, syncope, or an increase in dyspnea.

- F. Evaluate urinary output and renal response.
- G. Assess respiratory system for pulmonary congestion.
- H. Evaluate peripheral system; assess for dependent edema.
- I. Maintain NPO (nothing by mouth) status initially; then allow clear liquids; progress to light meals that are low in sodium and cholesterol.

- J. Promote normal bowel pattern.
  1. Stool softeners.
  2. Bedside commode.
  3. Caution against stimulation of the vagus nerve (Valsalva maneuver).
  4. Increase fiber in diet.

**Nursing Interventions**

**Alert** Identify potential/actual stressors for the client; implement measures to reduce environmental stress; promote methods to reduce stress.

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- J. Promote normal bowel pattern.
  1. Stool softeners.
  2. Bedside commode.
  3. Caution against stimulation of the vagus nerve (Valsalva maneuver).
  4. Increase fiber in diet.
F. Sexual intercourse can generally be resumed in 4 to 6 weeks after MI or when the client can walk one block or climb two flights of stairs without difficulty.
1. Do not drink any alcohol before sexual activity.
2. Take nitroglycerin before sexual activity.
3. Do not have sex after a heavy meal.
4. Position for intercourse does not influence cardiac workload.
5. Do not take erectile dysfunction medications (Appendix 22-1) if taking nitrates.

**ALERT** Assess client’s ability to perform self-care. Determine whether the client with cardiac disease understands the illness and whether he or she can demonstrate knowledge of care.

**Heart Failure (HF)**

Heart failure (also referred to as congestive heart failure [CHF] and chronic heart failure) includes cardiac decompensation, cardiac insufficiency, ventricular failure, and ultimately results in the inability of the heart to pump adequate amounts of blood into the systemic circulation to meet tissue metabolic demands.

**Physiology of Heart Failure**

A. Left-sided heart failure.
1. Results from failure of the left ventricle to maintain adequate cardiac output.
2. Blood backs up into the left atrium and into the pulmonary veins.
3. Increasing pressure in the pulmonary capillary bed causes lungs to become congested, resulting in impaired gas exchange.
4. Precipitating factors.
   a. MI (left ventricular infarct).
   b. Hypertension.
   c. Aortic and mitral value disease.
B. Right-sided heart failure.
1. The right ventricle is unable to maintain adequate output.
2. Blood backs up into the systemic circulation and causes peripheral edema.
3. Precipitating factors.
   a. Left-sided heart failure.
   b. Chronic pulmonary disease.
C. Each side of the heart is dependent on the other for adequate function.
1. Left-sided failure results in pulmonary congestion; this causes an increase in pulmonary pressure, which further increases the workload and pressure in the right side of the heart and precipitates failure.
2. The majority of clinical situations involve failure of both sides of the heart.
D. High-output versus low-output failure.
1. High-output failure occurs when the body’s needs for oxygen are excessively increased; the heart increases output but is still unable to meet the body’s needs (hyperthyroid and anemia).
2. Low-output failure occurs when the myocardium is so damaged that it cannot maintain adequate cardiac output; it is the failure of the heart as a pump.

E. Cardiac compensatory mechanisms will attempt to maintain the body requirements for cardiac output; when these mechanisms become ineffective, cardiac decompensation or failure will occur.

F. Edema development in heart failure.
1. Decreased cardiac output leads to decrease in renal perfusion, the kidneys respond by stimulating the adrenal cortex to increase the secretion of aldosterone, thus increasing the retention of sodium and water.
2. With an increase in the venous pressure from the increased circulating volume, there is an increase in the capillary pressure; and dependent, pitting edema occurs.

G. In children, HF occurs most often as the result of a structural problem of the heart. Ventricular function may not be impaired, but symptoms occur because of increased pulmonary artery pressure and pulmonary venous congestion.

**ALERT** If a test question states that a client is in heart failure, assume that both sides are in failure unless indicated otherwise.

**Assessment**

A. Risk factors/etiology.
1. Myocardial disease, valvular disease.
2. Hypertension.
4. Fluid overload.
B. Clinical manifestations (Box 17-3).
1. Impaired cardiac function.
   a. Tachycardia evaluated according to age level (see Table 15-2).
   b. Cardiomegaly from dilation and hypertrophy; PMI is displaced.
   c. S₃ or S₄ (heart gallop) caused by impaired ventricular function.

**ALERT** Reevaluate the client with abnormal heart sounds.

d. In infants, failure to thrive and failure to gain adequate weight.
e. Poor perfusion: cool extremities, weak pulses, poor capillary refill.
2. Pulmonary congestion.
   a. Dyspnea, dyspnea on exertion, tachypnea.
   b. Orthopnea: infants and adults.
   c. Paroxysmal nocturnal dyspnea occurs while client is asleep.
   d. Symptoms of respiratory distress and hypoxia (see Table 15-2).
e. Congested breath sounds, cough.
f. Feeding difficulties in infants, caused by dyspnea.
g. Increase in pulmonary artery pressure (PAP) and pulmonary arterial wedge pressure (PAWP) (see Appendix 17-9).
### ASSESSMENT FINDINGS OF HEART FAILURE

#### Right-Sided (Systemic Symptoms)
- Neck vein distention
- Generalized edema (anasarca)
- Dependent pitting edema
- Nausea, vomiting
- Anorexia
- Altered GI function
- Weight gain
- Ascites
- Decreased urinary output
- Liver engorgement
- Electrolyte imbalances
- Dysrhythmias
- Elevated CVP

#### Left-Sided (Pulmonary Symptoms)
- Dusky skin and nail beds
- Chest pain
- Tachycardia
- Decreased systolic BP
- Ventricular ectopic beats
- Presence of S3 and S4
- Shift of the PMI to the left
- Irritability, restlessness
- Decreased cardiac output
- Increased PAWP
- Wheezing crackles
- Dyspnea
- Productive cough

\(BP\): Blood pressure; \(CVP\): central venous pressure; \(GI\): gastrointestinal; \(PMI\): point of maximum impulse; \(PAWP\): pulmonary arterial wedge pressure.

   a. Hepatomegaly: may be an early sign in children.
   b. Dependent edema generalized in infants; evaluate by weight gain.
   c. Peripheral pitting edema.
   d. Ascites.
   e. Increase in central venous pressure (CVP) (see Appendix 17-9).

**ALERT** Determine changes in client’s cardiovascular status as related to the client’s HF; interpret what data need to be reported immediately.

C. Diagnostics (see Appendix 17-1).

### Treatment

A. Treatment of the underlying problem.
B. Prevention.
   1. Administration of prophylactic antibiotics to clients with rheumatic heart disease before medical procedures to prevent mitral valve damage.
   2. Effective early treatment of hypertension.
   3. Early treatment of dysrhythmia.
C. Oxygen.

D. Activity limitations.
E. Medications: treatment for HF includes reduction of the preload and afterload, as well as improvement of the contractility of the myocardium.
   1. Cardiac glycosides (Appendix 17-6).
   2. Diuretics (see Appendix 16-6).
   3. Aldosterone antagonists (see Appendix 16-6).
   4. Vasodilators (see Appendix 16-5).
   5. Angiotension-converting enzyme (ACE) inhibitors (see Appendix 16-5).
   7. Electrolyte replacement, carefully monitor serum potassium levels.
F. Decreased sodium diet, as well as fluid restrictions, for adults and older children.
G. Sodium and fluids may not be restricted for infants and children; infants seldom need fluid restriction because of difficulty feeding.

### Complications

A. Pulmonary edema (Chapter 15).
B. Cardiogenic shock.
C. Dysrhythmias.

#### Nursing Priority

The goals for therapeutic intervention in clients with HF are to:
- Improve cardiac output: digitalis and oxygen.
- Decrease cardiac afterload: decrease activity, administer vasodilator.
- Decrease cardiac preload: diuretics; decrease sodium and fluid intake; place client in semi-Fowler’s position.

### Nursing Interventions

**Goal:** To decrease cardiac demands and improve cardiac output.

A. Assess vital signs and compare with other physical assessment data.
B. Conserve energy.
   1. Encourage rest alternated with activity.
   2. Monitor pulse, respiratory rate, and dysrhythmias during periods of activity.
C. Avoid chilling; it increases oxygen consumption, especially in infants.
D. Supplemental oxygen, especially when needed with increased activity.
E. Provide uninterrupted sleep, when possible.
F. Minimize crying in children and infants.
G. Decrease stress and anxiety; encourage parents to remain with child.

**ALERT** Check for interactions among client’s drugs, foods, and fluids. For clients receiving digitalis and diuretics, it is very important to monitor the serum potassium level. A significant decrease in the serum potassium level may affect cardiac rhythm and can precipitate digitalis toxicity.

**Goal:** To decrease circulating volume.

A. Assess breath sounds; check for distended neck veins and peripheral edema.
B. Low-sodium diet and fluid restriction in adults and older children.
C. Evaluate fluid retention by determining accurate daily weight (1 kg or 2.2 lb weight gain = 1 L of fluid loss or retention).
D. Accurate intake and output; monitor electrolyte balance and therapeutic effect of fluid loss.

**Goal:** To reduce respiratory distress.

A. Position.
   1. Adult may be placed in Fowler’s or semi-Fowler’s position or may sit in an armchair.
   2. When in semi-Fowler’s position, do not elevate client’s legs; this increases venous return (preload).
   3. An infant or small child may breathe better in a sidelying position with the knees drawn up to the chest.
   4. An infant may be placed in an infant seat.
   5. Make sure diapers are loosely pinned and safety restraints do not hinder maximum expansion of the chest.
   6. Hold infant upright over the shoulder with knees flexed (knee–chest position).
B. Administer oxygen: use oxygen hood for infants and nasal cannula for adults and older children; supplemental oxygen to keep saturation levels at or above 90%.
C. Evaluate breath sounds, evaluate adventitious breath sounds and presence of congestion.
D. Do not allow infants to cry for extend periods.

**PEDIATRIC NURSING PRIORITY** Infants: if cyanosis decreases with crying, the problem is usually pulmonary; if cyanosis increases with crying, the problem is usually cardiac.

**Goal:** To monitor for development of hypoxia (see Chapter 15).

**Goal:** To maintain nutrition.

A. Provide small, frequent meals of easily digestible foods; allow client adequate time to eat.
B. Assist client with cultural implications in dietary management.
C. An infant will require increased calories due to increased metabolic rate.
   1. May require tube feedings.
   2. An infant does not generally require fluid restriction, because of decreased fluid intake secondary to the dyspnea.
   3. Do not prop the bottle; burp the infant frequently.
   4. Decreased calorie intake will result in decreased strength, decreased weight gain, and failure to meet developmental motor skills.

**Home Care**

A. Client should begin walking short distances, 250 to 300 feet, at least three to four times per week; distance can be increased as tolerated (no shortness of breath, dizziness, chest pain, or tachycardia).
B. Teach client how to count his or her pulse.
C. Teach client to do daily weights every morning, before breakfast and with similar clothes on (nightgown, pajamas, etc.).

D. Discuss use of and safety factors for home oxygen.
E. Contact health care provider for:
   1. Weight gain of 3 to 5 pounds over a week or 1 to 2 pounds overnight.
   2. Increase in dyspnea or angina, especially with decreased activity or at rest.
   3. Decrease in activity tolerance that exceeds 3 or 4 days.
   4. Increased urination at night; presence or increase in peripheral edema.
   5. Cough or respiratory congestion that lasts longer than 3 or 4 days.
F. Provide written instructions for medications, especially if they are on digitalis.
G. Assess client’s home situation and ability of caregivers.

**Rheumatic Heart Disease**

Rheumatic heart disease is an inflammatory disease that is usually self-limiting. The primary concern is the development of rheumatic heart disease involving the endocardium and cardiac valves.

A. Usually preceded by a group A beta-hemolytic streptococcal infection.

**NURSING PRIORITY** Prevention and adequate treatment of streptococcal infections prevent the development of rheumatic heart disease.

B. Myocardial involvement is characterized by the development of valvulitis, pericarditis, and myocarditis.
   1. Valvulitis produces scarring of the cardiac valves.
   2. Rheumatic carditis is the only symptom that produces permanent damage, most often involves damage to the endocardium and primarily to the mitral valve.
   3. Rheumatic fever usually occurs during childhood, but manifestations of cardiac damage may not be evident for years.

**Assessment**

A. Risk factors/etiology: previous infection by group A beta-hemolytic streptococcus.
B. Clinical manifestations: symptoms vary; no specific symptom or lab test is diagnostic of rheumatic fever. Criteria for the diagnosis require a combination of symptoms to be present.
   1. Carditis.
      a. Tachycardia out of proportion to fever.
      b. Long high-pitched apical systolic murmur beginning with S1 and continuing throughout cycle.
      c. Pericarditis, pericardial friction rub, and complaints of chest pain.
   2. Migratory polyarthritis.
   3. Chorea.
   4. Erythema marginatum.
   5. Subcutaneous nodules over bony prominences.
   6. History of a recent streptococcal infection.

**Treatment**

A. Adequate treatment of initial streptococcal infection.
B. Rest and decreased activity until tachycardia subsides.
C. Salicylates to control inflammatory process.
D. Prophylactic treatment: client is susceptible to recurrence of rheumatic fever.
   1. Begin after immediate therapy is complete.
   2. Monthly administration of penicillin over extended period of time, depending on extent of cardiac involvement.
   3. Administration of additional prophylactic antibiotics when invasive procedures are necessary (genitourinary procedures, dental work, etc.).

**Complications**
Severe valvular damage precipitates the development of HF and may require open heart surgery for replacement of diseased valve.

**Nursing Interventions**
Child is generally cared for in the home environment.

**Goal:** To assist parents and family to provide home environment conducive to healing and recovery.

A. Decrease activity if pulse rate is increased or if child is febrile.
B. Friends may visit for short periods; child is not contagious.
C. Maintain adequate nutrition.
   1. May be anorexic during the febrile phase.
   2. Provide soft or liquid foods as tolerated.
   3. Assist child with feeding if choreic movements are severe.
   4. Maintain adequate hydration.
D. Salicylates to control inflammatory process and as analgesics for arthralgia.
E. Reassure child that chorea and joint involvement are only temporary and there will be no residual damage.

**Goal:** To help parents understand need for long-term prophylactic antibiotic therapy.
A. Importance of preventing recurring infections.
B. Include child in planning, especially when numerous injections are involved.
C. Importance of prophylactic therapy before invasive procedures.
D. Continued medical follow-up for the development of valvular problems as child grows.
E. Follow-up required with females; cardiac problems may not be manifested until woman is pregnant.

**Endocarditis (Bacterial, Infective)**
Endocarditis is an infection of the valves and inner lining or endocardium of the heart.
A. Organisms tend to grow on the endocardium in an area of increased turbulence of blood flow or in areas of previous cardiac damage (rheumatic heart disease, congenital malformations).
B. Bacteria may enter from any site of localized infection.
C. Organisms grow on the endocardium and produce a characteristic vegetation consisting of fibrin deposits, leukocytes, and microbes; the vegetation may then invade adjacent valves.
D. Vegetation is fragile and may break off, resulting in emboli.

**Assessment**
A. Risk factors/etiology.
   1. Most often bacterial, but may be fungal or viral.
   2. History of endocarditis, prosthetic valves, or acquired valvular disease.
   3. IV drug abuse.
   4. History of recent invasive procedures.
B. Clinical manifestations.
   1. Symptoms of systemic infection: low-grade fever, chills, weakness, malaise.
   2. Murmur develops; or changes in previous murmurs occur.
   4. Vascular symptoms.
      a. Petechiae in conjunctiva, lips, buccal mucosa, on the ankle, and in the antecubital and popliteal areas.
      b. Osler’s nodes on fingertips.
      c. Janeway lesions: small, flat, painless red spots on the palm and soles.
   5. Symptoms associated with emboli.
      c. Brain: hemiplegia, decreased level of consciousness, visual changes.
C. Diagnostics.
   1. Echocardiography.
   2. Blood cultures, drawn 30 minutes apart.

**Treatment**
A. IV antibiotic therapy for 4 to 6 weeks (see Appendix 6-9).
B. Bed rest if high fever or evidence of heart failure is present.
C. Prophylactic antibiotics for 3 to 5 years in children with history of rheumatic carditis.
D. Surgical intervention for severe valvular damage.

**Complications**
A. HF secondary to valve damage.
B. General systemic emboli.

**Nursing Interventions**

**Goal:** To help parents understand the need for long-term prophylactic therapy (see Nursing Interventions under Rheumatic Heart Disease).
A. IV antibiotic medications.
B. Assess activity tolerance; activities may be restricted until:
   1. Temperature is normal.
   2. Resting pulse is below 100 beats/min.
   3. ECG is stable.
C. Evaluate for occurrence of emboli and heart failure.
**Home Care**

A. Good oral hygiene: daily care and regular dental visits.
B. Avoid excessive fatigue; plan rest periods and activity.
C. Client and family must understand the need for continued antibiotics.
D. Report temperature elevations, fever, chills, anorexia, weight loss, and increased fatigue.
E. Advise all health care providers of history of endocarditis.

**Pericarditis**

Pericarditis is an inflammation of the pericardium. The pericardial space is a cavity between the inner and the outer layers of the pericardium.

A. Acute pericarditis: may be dry or may cause excessive fluid accumulation in the pericardial space.
B. Chronic constrictive pericarditis: results from scarring and causes a loss of elasticity; the scarring and thickening of the pericardium prevent adequate cardiac filling during diastole.

**Assessment**

A. Risk factors/etiology.
   1. Acute pericarditis.
      a. Infection.
      b. Myocardial infarction.
         (1) Acute pericarditis may occur within 48-72 hours after an MI.
         (2) Dressler’s syndrome occurs about 4-6 weeks after an MI.
   2. Chronic constrictive pericarditis: usually begins with acute episode; fluid is gradually absorbed with scarring and thickening.
B. Clinical manifestations.
   1. Acute.
      a. Pericardial friction rub caused by myocardium rubbing against inflamed pericardium.
      b. Pain increases with deep inspiration and lying supine; sitting may relieve pain; pain may radiate, making it difficult to differentiate from angina.
   2. Chronic: symptoms are characteristic of gradually occurring HF and decreased cardiac output; chest pain is not a prominent symptom.
C. Diagnostics: acute and chronic (see Appendix 17-1).
   1. Increased leukocytes.
   2. Inflammation: increased C-reactive protein (CRP), increased erythrocyte sedimentation rate (ESR).

**Complications**

A. Pericardial effusion (fluid in pericardial space) can result in cardiac tamponade.

**Treatment**

A. Acute episode.
   1. Treat underlying problem.
   2. Bed rest.

3. Antiinflammatory medications (NSAIDs).
4. If pleural effusion and tamponade occur, pericardiocentesis (aspiration of fluid from the pericardial sac) is performed.

**Nursing Interventions**

**Goal:** To maintain homeostasis and promote comfort.

A. Assess characteristics of pain; administer appropriate analgesics.
B. Upright position, with client leaning forward, may relieve the pain.
C. Decrease anxiety, because client often associates problem with an MI; help client distinguish the difference.
D. Observe for symptoms of cardiac tamponade.
   1. Paradoxical blood pressure: precipitous decrease in systolic blood pressure on inspiration.
   2. CVP increased; presence of jugular venous distension.
   3. Heart sounds are muffled or distant.
   4. Narrowing pulse pressure.
   5. Tachypnea, tachycardia, decrease in cardiac output.
E. In a client with chronic pericarditis, evaluate for symptoms of HF and initiate appropriate nursing intervention.

**Cardiac Valve Disorders**

A. Causes of valve disease.
   2. Rheumatic heart disease.
   3. Bacterial endocarditis.
   4. Ischemia caused by ASHD.
B. Mitral valve is the most common area of involvement, followed by the aortic valve (see Figure 17-1).
C. Valvular stenosis: a narrowing of the valve opening and progressive obstruction to blood flow; increase in workload of the cardiac chamber pumping through the stenosed valve.
D. Valvular insufficiency (incompetency, regurgitation): impaired closure of the valve allows blood to flow back into the cardiac chamber, thereby increasing the workload of the heart.
E. Mitral valve.
   1. Stenosis: increases workload on the left atrium as it attempts to force blood through the narrowed valve.
   2. Mitral insufficiency (regurgitation): with each cardiac contraction, the left ventricle forces blood back into the left atrium.
   3. With both conditions, the left atrium dilates and hypertrophies because of an increase in workload. This causes an increase in pulmonary pressure and the subsequent development of HF.
F. Aortic valve.
   1. Aortic stenosis: increased (afterload) work of the left ventricle as it attempts to propel blood through the narrowed valve.
2. Aortic insufficiency: increased (afterload) work of the left ventricle as blood leaks back into the left ventricle after contraction.
3. With both conditions, the left ventricle dilates and hypertrophies as a result of increased pressure; this precipitates left ventricular failure.

Assessment
A. Risk factors: associated with history of rheumatic fever, endocarditis, cardiovascular disease.
B. Clinical manifestations—mitral valve disorders.
   1. Exertional dyspnea progressing to orthopnea.
   2. Progressive fatigue caused by decrease in cardiac output.
   3. Cardiac murmur (diastolic), palpitations.
   4. Systemic embolization.
   5. Atrial fibrillation.
C. Clinical manifestations—aortic valve disorders.
   1. Syncope and vertigo.
   2. Angina (condition interferes with coronary artery filling).
   3. Dysrhythmia, systolic murmur in stenosis.
   4. Dyspnea and increasing fatigue, heart failure.
D. Diagnostics (see Appendix 17-1).

Treatment
A. Prevention of heart failure (HF, CHF), pulmonary edema, thromboembolism, and endocarditis.
   1. Digitalis (see Appendix 17-6).
   2. Diuretics.
   3. Beta-blockers to decrease cardiac rate.
B. Prophylactic anticoagulation to prevent thrombus formation.
C. Prophylactic antibiotics.
D. Open heart surgery for valve replacement when there is evidence of progressive cardiac failure.
E. Percutaneous transluminal balloon valvuloplasty (PTBV): performed in cardiac catheterization lab; balloon is threaded through the affected valve in an attempt to separate the valve leaflets.

Nursing Interventions
Goal: To prevent development of rheumatic heart disease and provide prophylactic treatment to individuals with history of rheumatic heart disease.
Goal: To prevent and/or to identify early development of HF (see Heart Failure, Nursing Interventions).

Nursing Priority
Primary care for the client with cardiac valve disease consists of maintaining homeostasis and preventing the development of HF. The client should be advised to avoid excessive fatigue and should be assessed according to the level of activity tolerance.

Cardiovascular Disease in Pregnancy
Rheumatic heart disease (mitral valve problems) and congenital heart defects account for the greatest incidence of cardiac disease in pregnancy.

- Normal physiologic alterations of pregnancy that increase cardiovascular stress.
  1. Increase in oxygen requirements.
  2. 30% to 50% increase in cardiac output; peaks at 20 to 26 weeks’ gestation.
  3. Increase in plasma volume.
  4. Weight gain.
  5. Hemodynamic changes during delivery.
B. As normal pregnancy advances, the cardiovascular system is unable to maintain adequate output to meet increasing demands. Classification of the severity of cardiac disease in pregnancy (including relation to prescription of physical activity) is presented in Table 17-2.

Assessment
Clinical manifestations indicative of cardiac decompensation are those of impending CHF.
A. Frequent cough, progressive dyspnea.
B. Progressive general edema, jugular venous distension (JVD).
C. Palpitations.
D. Excessive fatigue for level of activity.
E. Dysrhythmia, atrial fibrillation, and tachycardia.
F. Congested breath sounds.
G. Cardiac decompensation increases with length of gestation; highest incidence of HF is observed at 28 to 32 weeks’ gestation.

Treatment
A. Management of the pregnant client.
   1. Balanced nutritional intake; iron supplements.
   2. Limited physical activity; stop any activity that increases shortness of breath.

<table>
<thead>
<tr>
<th>Table 17-2</th>
<th>CLASSIFICATION OF CARDIOVASCULAR DISEASE</th>
</tr>
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<tbody>
<tr>
<td>Class</td>
<td>Description</td>
</tr>
<tr>
<td>I</td>
<td>Clients with no limitations on activities; they suffer no symptoms from ordinary activities</td>
</tr>
<tr>
<td>II</td>
<td>Clients with slight, mild limitation of activity; they are comfortable at rest or with mild exertion</td>
</tr>
<tr>
<td>III</td>
<td>Clients with marked limitation of activity; they are comfortable only at rest</td>
</tr>
<tr>
<td>IV</td>
<td>Clients who should be at complete rest, confined to a bed or chair; any physical activity brings on discomfort, and symptoms occur even at rest</td>
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</table>

Nursing Priority
The functional classification of the disease is determined at 3 months’ gestation and again at 7 to 8 months’ gestation. Pregnant women may progress from class I or II to III or IV during pregnancy. Women with cyanotic congenital heart disease do not fit into the New York Heart Association (NYHA) classifications because the causes of their exercise-induced symptoms are not related to heart failure.

Adapted from the Criteria Committee, New York Heart Association (NYHA), Boston, 2005.
3. Diuretics, digitalis, anticoagulants, and antidyssrhythmics may be given.
4. May be hospitalized at 28 to 32 weeks’ gestation because of impending HF.
5. If coagulation problems occur, heparin is usually used because it does not cross the placenta.

**B. Management of the client during labor and delivery.**
1. Supplemental oxygen.
2. Epidural regional anesthesia is generally used for delivery.

**C. Management of the client during the postpartum period: treated symptomatically according to status of cardiovascular system; the first 24 to 48 hours postpartum is period of highest risk for HF in the mother.**

### Nursing Interventions

**Goal:** To assist client to maintain homeostasis during pregnancy.

A. Written information regarding nutritional needs.
B. Nursing assessment and client education regarding early symptoms of HF.
C. Frequent rest periods; activity may be severely restricted during the last trimester.

**D. Decrease stress by keeping client informed of progress.**

**Goal:** To help client maintain homeostasis during labor and delivery.

A. Position client on side with head and shoulders elevated.
B. Observe for increasing dyspnea, cough, or adventitious breath sounds during labor.
C. Evaluate information from continuous fetal and maternal monitoring.
D. Encourage open glottis pushing during labor; prevent Valsalva maneuver.
E. Prepare for vaginal delivery.
F. Provide pain relief as indicated.
   1. Pain increases cardiac work.
   2. Evaluate effects of analgesia on fetus.

**ALERT** Interpret what data from a client need to be reported immediately.

**Goal:** To maintain homeostasis in the postpartum period.

A. Assessment of cardiac adaptation to changes in hemodynamics.
   1. Increased blood flow due to decreased abdominal pressure may precipitate reflux bradycardia.
   2. Assess for chest pain and adequacy of cardiac output.
B. Maintain semi-Fowler’s position or side lying position with the head elevated.
C. Gradual progression of activities (depending on cardiac status) as indicated by:
   1. Pulse rate.
   2. Respiratory status.
   3. Activity tolerance.
D. Progressive ambulation as soon as tolerated to prevent venous thrombosis.
E. Assist mother and family to prepare for discharge.

### Congenital Heart Disease

A. Clinical manifestations depend on the severity of the defect and the adequacy of pulmonary blood flow.
B. Normal pressure in the right side of the heart is lower than pressure in the left side; there is an increased blood flow from an area of high pressure to an area of low pressure.

1. When there is an opening between the right and left side of the heart, oxygenated blood will shunt from the left side of the heart to the right side (right-to-left shunt).
2. When the pressures on the right side of the heart exceed the pressure on the left side of the heart, unoxygenated blood from the right side will flow into the left side and unoxygenated blood will flow into the systemic circulation (left-to-right shunt).
C. Physical consequences of congenital heart defects.
   1. Delayed physical development.
      a. Failure to gain weight, caused by inability to maintain adequate caloric intake to meet increased metabolic demands.
      b. Tachycardia and tachypnea precipitate increase in caloric requirements.
   2. Excessive fatigue, especially during feedings.
   3. Frequent upper respiratory tract infections.
   4. Dyspnea, tachycardia, tachypnea.
   5. Hypercyanotic spells (called “blue” spells or “tet” spells): infant suddenly becomes acutely cyanotic and hyperpneic; occur most often in children 2 months to 1 year of age.
D. Diagnostics (see Appendix 17-1).

### Defects With Increased Pulmonary Artery Blood Flow

Increased blood volume on the right side of the heart increases the flow of blood through the pulmonary artery. This may decrease the systemic blood flow.

A. Atrial septal defect (ASD): opening in the septum between the atrium; blood shunts from left to the right; often caused by failure of foramen ovale to close (Figure 17-6).
B. Ventricular septal defect (VSD): opening in the septum between the ventricles; blood shunts from left side to right side (Figure 17-7).
C. Patent ductus arteriosus (PDA): failure of the fetal ductus arteriosus to close after birth; blood is shunted from the left side to the right side; may be treated with indomethacin or ibuprofen (Advil), nonsteroidal antiinflammatory drugs that change the oxygen concentration of
the tissue and enhance tissue changes that close the defect (Figure 17-8).

D. Clinical manifestations.
   1. May be asymptomatic.
   2. Signs of CHF are common.
   3. Characteristics murmurs.

Obstructive Defects With Decreased Pulmonary Artery Blood Flow

Problems occur when the normal blood flow through the heart meets an obstruction. The pressure in the ventricle and in the artery prior to the obstruction is increased; pressure beyond the obstruction is decreased.

A. Coarctation of the aorta: a narrowing of the aorta; specific symptoms depend on the location of the coarcta-

tion in relation to arteries coming off the aortic arch (Figure 17-9).

1. Clinical manifestations.
   a. Marked differences in blood pressure in the upper and lower extremities; area proximal to the defect has a high pressure and a bounding pulse.
      (1) Epistaxis.
      (2) Headaches.
      (3) Bounding radial and temporal pulses.
      (4) Dizziness and fainting.
   b. Area distal to defect has decreased blood pressure and weaker pulse.
      (1) Lower extremities are cooler; mottling is present.
      (2) Weak peripheral and femoral pulses.
   c. May develop CHF.
2. Increased risk for hypertension, ruptured aorta, aortic aneurysm, and stroke.

B. Aortic stenosis: a narrowing or constriction of the aortic valve that increases resistance to blood flow from the left ventricle into systemic circulation; increases pulmonary vascular congestion.

1. Clinical manifestations.
   a. Decreased cardiac output, faint pulses, hypotension, tachycardia.
   b. Children have exercise intolerance and may have chest pain.
   c. Characteristic murmur.

2. At risk for bacterial endocarditis and coronary insufficiency.

**Defects With Decreased Pulmonary Blood Flow**

An anatomic defect is present between the right and left sides of the heart (ASD or VSD). There is increased pulmonary artery resistance to blood flow; therefore pressure increases in the right side of the heart. As the resistance in the pulmonary circulation increases, pressure in the right ventricle increases. This pressure increases until it is greater than pressure on the left side of the heart and unoxygenated (unsaturated) blood moves into the left side of the heart (right-to-left shunt).

A. Tetralogy of Fallot: consists of four defects—ventricular septal defect (VSD), pulmonic stenosis, overriding aorta, and right ventricular hypertrophy (Figure 17-10).

1. Clinical manifestations depend on the size of the VSD and the pressures in the heart.
   a. May be acutely cyanotic at birth or may have minimal cyanosis due to patent ductus arteriosus.
   b. Child may have mild cyanosis that progresses.
   c. Infant may experience acute episodes of hypoxia (blue spells, tet spells), may occur during agitation or crying.

B. Tricuspid atresia: tricuspid valve does not develop, and there is no opening from the right atrium to the right ventricle; an ASD, or patent foramen ovale, is present and provides mixing of oxygenated and unoxygenated blood.

1. Clinical manifestations.
   a. Cyanosis usually occurs during newborn period.
   b. Tachycardia, dyspnea.
   c. Older children have chronic hypoxemia and clubbing.

2. Continuous infusion of prostaglandin E₁ may be used to maintain a patent ductus arteriosus.

**Mixed Defects**

Mixed defects are present when the survival of the infant depends on the mixing of oxygenated and unoxygenated blood for survival. Pulmonary congestion occurs as a result of the increased flow of blood into pulmonary vasculature.

A. Transposition of great vessels: the pulmonary artery receives blood from the left ventricle, and the aorta receives blood from the right ventricle. A communicating defect—ASD, VSD, or patent ductus arteriosus—must be present to provide for mixing of oxygenated and unoxygenated blood (Figure 17-11).

1. Clinical manifestations depend on size of associated defects and mixing of blood.
   a. Newborns with minimal communication defects are cyanotic at birth.

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**FIGURE 17-10** Tetralogy of Fallot. (From Hockenberry MJ, Wilson D: *Wong’s nursing care of infants and children*, ed 8, St. Louis, 2007, Mosby.)

**FIGURE 17-11** Transposition of the great vessels. (From Hockenberry MJ, Wilson D: *Wong’s nursing care of infants and children*, ed 8, St. Louis, 2007, Mosby.)
b. Symptoms of CHF.
c. Cardiomegaly develops shortly after birth.
2. Prostaglandins may be given to maintain patency of ductus arteriosus to promote mixing of blood.
B. Truncus arteriosus: the pulmonary artery and aorta fail to separate; results in a single vessel that receives blood from both ventricles (Figure 17-12).
1. Clinical manifestations.
   a. Cyanosis and CHF develop in infancy.
   b. Characteristic murmur.
2. Multiple surgical repairs are necessary to replace conduits as child grows.

**Nursing Interventions**

**Goal:** To evaluate infant’s response to cardiac defect.
A. Evaluate infant’s Apgar scores at birth.
B. Evaluate adequacy of weight gain in first few months.
C. Assess feeding problems.
   1. Poor sucking reflex.
   2. Poor coordination of sucking, swallowing, and breathing.
   3. Fatigues easily during feeding.
D. Frequency of upper respiratory tract infections.
E. Determine whether cyanosis occurs at rest or is precipitated by activity.
F. Presence and quality of pulses in extremities.
G. Bacterial endocarditis is a primary concern before and after correction of a congenital defect; all fevers should be reported.

**Goal:** To promote oxygenation.
A. Effectively cope with hypercyanotic spells (blue spells, tet spells).
   1. Occurs suddenly, often when the infant is agitated.
   2. Hold the infant upright in a knee chest position; this reduces the venous return and increases systemic vascular resistance, which increases pressure on the right side of the heart and diverts more blood flow through the pulmonary artery.
3. Administer 100% O₂ via a face mask until respirations are improved.
4. Maintain good hydration and carefully monitor hemoglobin levels.
5. Older child may assume squatting position to increase peripheral resistance and decrease right-to-left shunting.

B. Supplemental oxygen, good pulmonary hygiene, and chest physiotherapy to maintain oxygen saturation levels.
C. Prevent overexertion, decrease excessive crying, and promote rest in order to conserve energy and caloric expenditure.
D. Maintain hydration for pulmonary hygiene and to prevent embolic problems and stroke.
E. Infants may require gavage feeding if respiratory distress limits oral feeding.

**Goal:** To assist parents in adjusting to diagnosis.
A. Allow family to grieve over loss of perfect infant.
B. Evaluate parents’ level of understanding of the child’s problem.
C. Foster early parent-infant attachment; encourage touching, holding, feeding, and general physical contact.
D. Help the family develop a relationship that fosters optimum growth and development of all family members. (See Chapter 3 for psychosocial aspects of caring for chronically ill children.)

**Alert** Provide emotional support to family; assist family members in managing care of a child with a chronic illness.

**Goal:** To detect, prevent, and treat CHF.

**Goal:** To provide appropriate nursing interventions for the client undergoing open heart surgery for repair of a defect.

**Cardiac Surgery**

A. Coronary artery bypass graft (CABG), aortic and mitral valve replacements: open heart surgery is performed with the use of the cardiopulmonary bypass machine; this machine allows for full visualization of the heart while maintaining perfusion and oxygenation.
B. Closed heart surgery is performed without the use of the bypass machine.
   1. Minimally invasive direct coronary artery bypass graft (MIDCABG); one of the most common procedures are grafts to the left anterior descending artery.
   2. Aortic and mitral balloon valvuloplasty.

**Nursing Interventions**

For preoperative and postoperative care, see Chapter 3.

**Goal:** To prepare the client psychologically and physiologically for surgery.
A. Evaluate client’s history for other chronic health problems.
B. Establish baseline data for postoperative comparison.
C. Provide appropriate preoperative teaching according to age level; frequently includes a visit to the intensive care unit (parents should accompany the child on the visit).
D. Discuss immediate postoperative nursing care and anticipated nursing procedures.
E. Correct metabolic and electrolyte imbalances.
F. Establish and maintain adequate hydration.
G. Anticipate adjustments in medication schedule before surgery.
1. Digitalis dosage is usually decreased.
2. Diuretics may be discontinued 48 hours before surgery.
3. Long-acting insulin will be changed to regular insulin.
4. Antihypertensive medication dosage may be modified.
H. Eliminate possible sources of infection.
**Goal:** To evaluate and promote cardiovascular function after surgery.
A. Maintain adequate blood pressure.
B. Evaluate for dysrhythmia; document and treat accordingly (see Appendixes 17-4 and 17-7).
C. Maintain client in semi-Fowler's position.
D. Evaluate fluid and electrolyte balances.
1. Record daily weight and compare with previous weight.
2. Evaluate electrolyte balance, especially potassium levels.
3. Assess for adequate hemoglobin and hematocrit values.
4. Evaluate hemodynamic levels for adequate volume (see Appendix 17-9).
5. Measure urine output hourly to evaluate adequacy of renal perfusion.
6. Administer IV fluids, as indicated.
E. Observe for hemorrhage: most often identified by increase in chest drainage.
F. Evaluate adequacy of peripheral circulation.
G. Mediastinal chest tubes are frequently placed in the pericardial sac to prevent tamponade. These tubes are cared for in the same manner as pulmonary chest tubes (Appendix 15-4).
H. Bedside hemodynamic monitoring as indicated (see Appendix 17-9).
**Goal:** To evaluate and promote respiratory function.
A. Mechanical ventilation via an endotracheal tube may be used for approximately 12 to 24 hours after surgery. Generally, children are extubated sooner than adults.
B. Maintain water-sealed drainage for mediastinal/chest tubes (see Appendix 15-4).
C. Pulmonary hygiene via endotracheal tube; carefully assess pulse oximetry during suctioning.
D. Promote good respiratory hygiene after extubation (fluids, incentive spirometry, activity).
E. Evaluate arterial blood gases for adequate oxygenation, as well as acid-base balance.
F. Encourage activity as soon as possible; client should be out of bed on the first postoperative day.
G. Monitor pulse oximetry levels.
**Goal:** To evaluate neurologic status for complications after surgery.
A. Recovery from anesthesia within appropriate time frame.
B. Able to move all extremities equally.
C. Appropriate response to verbal command.
D. Assess for changes in neurologic status.
**Goal:** To prevent complications of immobility (see Chapter 3) after surgery.
**Goal:** To decrease anxiety and promote comfort after surgery.
A. Frequent orientation to surroundings.
B. Careful explanation of all procedures.
C. Prevent sensory overload.
D. Promote uninterrupted sleep.
E. Administer pain medications as appropriate.

**Common Complications After Cardiac Surgery**
A. Dysrhythmia (see Appendix 17-7).
1. Premature ventricular contractions.
2. Ventricular tachycardia.
3. Atrial dysrhythmias are common with mitral valve replacement.
B. Hypovolemia.
1. Low cardiac output may be due to hypovolemia.
2. Frequently, vasoconstriction is present immediately after surgery. Vasodilation occurs as client's body temperature increases and precipitates hypovolemia.
3. Evaluate hemodynamic parameters for adequacy of cardiac output.
4. Observe client closely for response to fluid replacement, because fluid overload occurs very rapidly, especially in children.
C. Emboli.
1. Pulmonary emboli.
   a. Occur most frequently after valvular surgery.
   b. Client may be receiving prophylactic anticoagulants.
   c. Evaluate neurologic system for evidence of cerebral emboli.
D. Cardiac tamponade.
1. Pressure on the heart caused by a collection of fluid in pericardial sac.
2. May be caused by fluid collecting postoperatively or from pericarditis.
3. Pressure prevents adequate filling of ventricles, thereby decreasing cardiac output.
4. Paradoxical blood pressure: precipitous decrease in systolic blood pressure on inspiration; diagnostic for tamponade.
5. CVP is frequently increased; blood pressure is decreased.
6. Heart sounds are described as distant.
7. A decrease in drainage may occur in the mediastinal tubes.
8. Treatment involves reestablishing patency of mediastinal tube, surgical exploration of bleeding areas or pericardiocentesis with pericarditis.
E. Psychosis (most common in adults).
   1. Characteristics.
      a. Inappropriate verbal response.
      b. Disorientation to time and place.
      a. Maintain orientation to time and place.
      b. Maintain continuity in nursing staff.
      c. Carefully explain all procedures.
      d. Encourage ventilation of feelings.

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### Appendix 17-1 CARDIAC DIAGNOSTICS

#### Serum Laboratory Studies

**Cardiac Enzymes**

Creatinine kinase (CK): CK isoenzyme MB (CK-MB). Increases greater than 5% of total creatine kinase are highly indicative of MI. Increases occur within 4 to 6 hours after an MI, peak in 12 to 24 hours, and return to normal in 24-36 hours.

Cardiac troponin T and cardiac troponin I: Normal level is <0.2 ng/mL (for T) and <0.03 ng/dL (for I). Levels are elevated within 4 to 6 hours after an MI and peak within 10 to 24 hours. Elevated levels are highly indicative of myocardial damage.

**Nursing Implications**

1. Enzymes must be drawn on admission and obtained in a serial manner thereafter. There is a characteristic pattern to the increases and decreases of enzyme levels in a client with an MI.
2. The larger the infarction, the larger the enzyme response.
3. Evaluate serial levels of the enzymes; increased levels of troponin are the most significant and diagnostic of myocardial damage.
4. Cardiac-specific troponin helps discriminate from other tissue injury.

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**Hypokalemia**

1. Premature ventricular contractions (PVCs).
2. Ventricular tachycardia.
3. Ventricular fibrillation.

**Erythrocyte Sedimentation Rate**

Used as a gauge for evaluating progression of inflammatory conditions; inflammatory process precipitates a rapid settling of the red blood cells. Normal values vary greatly, depending on the laboratory method used.

**B-Type Natriuretic Peptide (BNP)**

First whole blood marker for identifying and treating heart failure. Serial BNP values may be determined to evaluate left ventricular function; assists to identify heart failure versus respiratory failure as cause of dyspnea. Normal is less than 100 pg/mL.

#### Noninvasive Diagnostics

**Electrocardiogram (ECG)**

A graphic representation of the electrical activity of the heart, generally conducted by using a 12-lead format. Test to identify conduction in rhythm disorders and the occurrence of ischemia, injury, or death of myocardial tissue.

**Holter Monitor**

Client is connected to a small portable ECG unit with recorder that records the client’s heart activity for approximately 24 to 48 hours. The client keeps a log of activity, pain, and palpitations. Client should not remove leads but is encouraged to maintain normal activity; client should not shower or bathe while the monitor is in place. The recording is analyzed and compared with the client’s activity log.

**Event Monitor (Transtelephonic Event Recorder)**

Client is connected to portable ECG unit with a recorder. The client can activate the recorder whenever he or she feels any type of dizziness or palpitations. Monitor may be worn for extended periods of time. Monitor leads and battery are removed for showering but should be reconnected immediately. Recordings are transmitted over the phone.

**Exercise Stress Test**

This test involves the client exercising, usually on a treadmill, which increases speed and incline to increase the heart rate. ECG leads are attached to the client, and the response of the heart to exercise is evaluated.
Appendix 17-1  CARDIAC DIAGNOSTICS—cont’d

Nursing Implications
1. Provide appropriate pretest preparation; establish baseline vital signs and cardiac rhythm.
2. Client should:
   a. Avoid smoking, eating, or drinking any caffeine beverages for 3 hours before the test.
   b. Avoid stimulants, eating, and extreme temperature changes immediately after the test.
3. Cardiac monitoring is done constantly during test.
4. Reasons for terminating an exercise stress test: chest pain, significant increase or decrease in blood pressure, or significant ECG changes.
5. If any of the above changes occur, the test is terminated and the stress test result is said to be positive.

Echocardiogram
An ultrasound procedure to evaluate valvular function, cardiac chamber size, ventricular muscle, and septal motion. The ultrasound waves are displayed on a graph and interpreted.

Transesophageal Echocardiography
Provides a higher-quality picture than regular echocardiogram. The throat is anesthetized, and a flexible endoscope is passed into the esophagus to the level of the heart. Sedation is used during the procedure.

Nursing Implications
1. Maintain NPO for 4 to 8 hours before test.
2. Check for gag and swallow reflex before client resumes oral intake of fluids.

Invasive Diagnostics
Cardiac Catheterization
An invasive procedure in which a catheter is passed through the brachial or femoral artery for left-side cardiac catheterization; the brachial or femoral vein for right-side cardiac catheterization will provide data regarding status of the coronary arteries, as well as cardiac muscle function, valvular functions, and left ventricular function (ejection fraction).

Nursing Implications
   a. Maintain NPO status for 6 hours before test.
   b. Check for dye allergy, especially allergy to iodine and contrast media.
   c. Record quality of distal pulses for comparison after test.
   d. Determine whether any medications need to be withheld.
   e. Appropriate client education (feeling of warmth when dye is injected; will be awake during procedure; report any chest pain; need to lie still).
2. After the test.
   a. Evaluate catheterization entry site (most often femoral) for hematoma formation. Notify physician immediately for excessive bleeding at the site, dramatic changes in blood pressure.
   b. Evaluate circulatory status (pulses distal to catheterization site, color, sensation of the extremity). Notify physician immediately for a decrease in peripheral circulation or neurovascular changes in affected extremity.
   c. Assess for dysrhythmias.
   d. Maintain bed rest; avoid flexion; keep extremity straight for 3 to 6 hours.
   e. Keep head of bed elevated at 30° or less.
   f. Encourage oral intake of fluids; IV access may be maintained.

Positron Emission Tomography (PET)
Very sensitive in identifying viable and nonviable cardiac tissue. The procedure takes about 2 to 3 hours, and a radioactive dye is injected intravenously, followed by glucose. A client’s glucose must be between 60-140 mg/dL before the test.

Appendix 17-2  NITRATES

Nitrates Increase blood supply to the heart by dilating the coronary arteries; cardiac workload is reduced due to decrease in venous return because of peripheral vasodilation.

<table>
<thead>
<tr>
<th>MEDICATIONS</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin (NTG, Nitrostat): sublingual</td>
<td>Headaches (will diminish with therapy), postural hypotension, syncope, blurred vision, dry mouth, reflex tachycardia</td>
<td>1. Advise client that alcohol will potentiate postural hypotension.</td>
</tr>
<tr>
<td>Nitroglycerin (Nitro-BID, Nitrol): topical (patch)</td>
<td></td>
<td>2. Educate client regarding self-medication (see Box 17-2).</td>
</tr>
<tr>
<td>Nitroglycerin ointment (Nitropaste): topical, by the inch</td>
<td></td>
<td>3. Do not take with erectile dysfunction drugs.</td>
</tr>
<tr>
<td>Nitroglycerin translingual spray</td>
<td></td>
<td>4. Topical or transdermal application is used for sustained protection against anginal attacks.</td>
</tr>
<tr>
<td>Isosorbide dinitrate (Isordil, Sorbitrate): PO</td>
<td></td>
<td>5. Avoid skin contact with topical form; remove all previous applications when applying topical form.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6. Sublingual tablets and translingual spray given for an immediate response.</td>
</tr>
</tbody>
</table>
### Appendix 17-3 CALCIUM CHANNEL BLOCKERS

**MEDICATIONS**

**Calcium Channel Blockers** Blockade of calcium channel receptors in the heart causes decreased cardiac contractility and a decreased rate of sinus and AV node conduction. Used to treat chronic stable angina, hypertension, and supraventricular dysrhythmias.

- Diltiazem (Cardizem): PO, IV
- Nifedipine (Procardia): PO
- Verapamil (Calan, Isoptin): IV, PO

**SIDE EFFECTS**

- Constipation, exacerbation of CHF, hypotension, bradycardia, peripheral edema

**NURSING IMPLICATIONS**

1. Nifedipine is less likely to exacerbate preexisting cardiac conditions; is not effective in treating dysrhythmias.
2. Intensifies cardiosuppressant effects of beta-blocker medications.

**Beta-Adrenergic Blocking Agents (adrenergic antagonists)** Blockade of beta, receptors in the heart causes decreased heart rate, and decreased rate of AV conduction. Used to treat hypertension as well as angina. Beta blockers should be administered to all clients experiencing unstable angina or having an MI unless contraindicated. Give with caution to clients with history of bronchospasm.

- ▲ Labetalol (Trandate): PO, IV
- Metoprolol (Lopressor, Toprol XL): PO, IV
- Propranolol (Inderal): PO, IV
- Atenolol (Tenormin): PO, IV
- ▲ High-Alert Medications: all IV preparations.
- Carvedilol (Coreg): PO

**NURSING IMPLICATIONS**

1. High-alert medications.
2. Closely monitor cardiac client—may precipitate heart failure, but is also used to treat heart failure.
3. Teach client how to decrease effects of postural hypotension.
4. Teach client not to stop taking medication when he or she feels better.
5. Bradycardia is common adverse effect.
6. If client has diabetes, blood glucose control may be impaired.

Note: Carvedilol, bisoprolol, and metoprolol sustained release are used to treat HF.

### Appendix 17-4 ANTIDYSRHYTHMIC DRUGS

**Antidysrhythmics** Decrease cardiac excitability; delay cardiac conduction in either the atrium or ventricle. Atropine is cardiac stimulant for bradycardia.

**General Nursing Implications**

- Assess client for changes in cardiac rhythm and impact on cardiac output.
- Evaluate effect of medication on dysrhythmia and resulting effects on cardiac output.
- Client should be on bedrest and have a cardiac monitor attached during IV administration.
- Have atropine available for cardiac depression resulting in symptomatic bradycardia.
- All cardiac depressant medications are contraindicated in clients with sinus node or AV node blocks.
- Digitalis will enhance cardiac depressant effects.
- Closely monitor for dysrhythmias that are precipitated by the treatment.
- See Appendix 17-7: Cardiac Dysrhythmias.

**MEDICATIONS**

- Quinidine sulfate (Quinidine Sulfate, Quinidex): PO

**SIDE EFFECTS**

- Hypotension
- Diarrhea, nausea, vomiting

**NURSING IMPLICATIONS**

1. Administer with food.
2. Monitor for ECG changes for toxicity—widened QRS and prolonged QT.
3. Use: supraventricular tachycardia and ventricular dysrhythmias.

**Adenosine (Adenocard, Adenoscan): IV**

**NURSING IMPLICATIONS**

2. Has a rapid half-life; therefore must be given rapidly undiluted over 1 to 3 sec followed by 20 mL of normal saline.

**SIDE EFFECTS**

- Tachycardia
- Dry mouth, blurred vision, dilated pupils

**MEDICATIONS**

- Atropine: subQ, IV

**NURSING IMPLICATIONS**

1. Use: block cardiac stimulation, primarily for bradycardias that are hemodynamically significant or for PVCs related to slow heart rate.
2. Assess client’s cardiac output in response to the bradycardic episode.
## Appendix 17-4  ANTIDYSRHYTHMIC DRUGS—cont’d

<table>
<thead>
<tr>
<th>MEDICATIONS</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone hydrochloride (Cordarone): PO, IV</td>
<td>AV heart block, hypertension</td>
<td>1. <strong>Use:</strong> life-threatening ventricular arrhythmias, atrial fibrillation.</td>
</tr>
<tr>
<td><strong>High-Alert Medication:</strong> IV route</td>
<td>Toxicity—lung and visual problems</td>
<td>2. Do not administer with other drugs that prolong the QT interval.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Continually monitor ECG during IV infusion.</td>
</tr>
<tr>
<td>Lidocaine hydrochloride (Xylocaine): IV</td>
<td>Drowsiness, confusion, seizures, severe depression of cardiac conduction</td>
<td>1. <strong>Use:</strong> ventricular dysrhythmias.</td>
</tr>
<tr>
<td><strong>High-Alert Medication:</strong> IV route</td>
<td></td>
<td>2. Must use IV preparation of <strong>Lidocaine</strong> for IV infusion.</td>
</tr>
<tr>
<td>Mexiletine (Mexitil): PO</td>
<td>GI disturbances—nausea/vomiting, diarrhea; CNS disturbances—tremor, dizziness</td>
<td>1. <strong>Use:</strong> prevent/control ventricular dysrhythmias.</td>
</tr>
<tr>
<td>Procainamide (Pronestyl): PO, IV</td>
<td>Abdominal pain, cramping, hypotension, prolonged QT interval</td>
<td>1. <strong>Use:</strong> short- and long-term control of ventricular and supraventricular dysrhythmias.</td>
</tr>
<tr>
<td></td>
<td>Blood dyscrasias</td>
<td>3. Closely monitor for bradycardia and hypotension.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Do not take OTC cold preparations.</td>
</tr>
<tr>
<td>Propranolol hydrochloride (Inderal): PO, IV</td>
<td>See previous discussion of beta-adrenergic blockers</td>
<td>1. <strong>Use:</strong> long- and short-term treatment and prevention of tachycardia.</td>
</tr>
<tr>
<td><strong>High-Alert Medication:</strong> IV route</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

## Appendix 17-5  FIBRINOLYTIC MEDICATIONS

**Fibrinolitics** work by different actions to initiate fibrinolysis of a clot. Medications are not clot specific; they will break up a clot on a surgical incision as well as a clot from an MI (see Fibrinolytic Therapy below).

### General Nursing Implications
- Therapy should begin as soon as the MI is diagnosed or when there is a history of prolonged angina.
- For best results, from admission in the ED until medication is administered is 30 min (door-to-needle), or within 60 min of onset of symptoms.
- Bleeding precautions (see Table 9-1).

<table>
<thead>
<tr>
<th>MEDICATIONS</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alteplase (tPA, Activase): IV</td>
<td>Bleeding and hypotension</td>
<td>1. Obtain base vital signs and coagulation studies.</td>
</tr>
<tr>
<td>Streptokinase (Streptase, Kabikinase): IV</td>
<td></td>
<td>3. Avoid venipunctures during and after infusion.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. <strong>Use:</strong> MI, PE, DVT; contraindicated in clients with active bleeding.</td>
</tr>
</tbody>
</table>

### Fibrinolytic Therapy

“Door-to-needle goal” of 30 min for most optimum results

If a client has any of the following, fibrinolytic therapy may be contraindicated:
- Systolic BP >180 mm Hg or diastolic BP >110 mm Hg
- Significant closed head or facial trauma within the past 3 months
- History of intracranial hemorrhage
- Currently on anticoagulants
- CPR >10 min
- Pregnancy
- Serious systemic disease (liver, kidney)
Appendix 17-6 CARDIAC GLYCOSIDES

▲ Cardiac Glycosides (e.g., digitalis) Increase myocardial contractility, thereby increasing cardiac output. Decrease heart rate by slowing conduction of impulses through the AV node. Enhance diuresis by increasing renal perfusion.

General Nursing Implications
— Take the apical pulse for a full minute; if the rate is below 60 beats/min in an adult or below 90 to 110 beats/min in infants and young children, or below 70 in a child, hold the medication and notify the physician.
— Evaluate for tachycardia, bradycardia, and irregular pulse. If there is significant change in rate and rhythm, hold the medication and notify the physician.
— Evaluate serum potassium levels and response to diuretics; hypokalemia potentiates action of digitalis.
— Gastrointestinal symptoms are frequently the first indication of digitalis toxicity.
— Teach client not to increase or double a dose in the case of a missed dose; if client vomits, do not give an additional dose.
— Quinidine, verapamil, and ACE inhibitors increase plasma levels of digitalis.
— To achieve maximum results rapidly, an initial loading dose is administered; then dose is reduced to a maintenance dose.
— Digibind is a digitalis antagonist and may be used for digitalis toxicity; watch for decreased potassium levels and client's response to decreased digitalis levels.

MEDICATION
▲ Digoxin (Lanoxin): PO, IV

SIDE EFFECTS
Most common: anorexia, nausea, vomiting
Most serious: drug-induced dysrhythmias
Visual disturbances, fatigue
Children/infants: frequent vomiting, poor feeding, or slow heart rate may indicate toxicity

NURSING IMPLICATIONS
1. Therapeutic plasma levels of digoxin are 0.5-2.0 ng/mL.
2. First sign of toxicity is usually GI symptoms.
3. Use: supraventricular tachycardia, CHF.

1 OLDER ADULT PRIORITY Older adults are more sensitive to digitalis and are more likely to experience toxicity.

Appendix 17-7 CARDIAC DYSRHYTHMIAS

A dysrhythmia is defined as an interruption in the normal conduction of the heart, either in the rate or the rhythm.

▲ ALERT Initiate, maintain, and/or evaluate telemetry monitoring; identify abnormal cardiac rhythms; and initiate protocols to manage cardiac arrhythmias.

Characteristics of Normal Sinus Rhythm
Rate: 60 to 100 beats/min
Rhythm: regular
P waves: present, precede each QRS complex
P-R interval: 0.12 to 0.20 sec
QRS complex: present and under 0.10 sec
Dysrhythmias may be classified according to rate—either brady- or tachycardia. They are also classified according to their origin—atrial or ventricular. Ventricular dysrhythmias are more life-threatening than atrial dysrhythmias. (See Common Dysrhythmias below.)

Nursing Implications
1. Identify dysrhythmia and evaluate client's tolerance.
2. Monitor adequacy of cardiac output, keep on bed rest, and oxygen.
3. Convert abnormal rhythm to normal sinus rhythm—medica- tions or cardioversion.
4. Prevent complications.

Interpretation of Electrocardiogram
1. Determine heart rate. Count the P waves in a 6-sec strip to determine the atrial rate and count the R or Q waves to determine the ventricular rate.
2. Are the QRS complexes occurring at regular intervals?
3. Are P waves present for each QRS complex?
4. Measure the P-R interval.
5. Measure the QRS interval.
6. Identify the T wave and S-T segment.
7. Analyze any abnormalities.
8. Correlate findings with characteristics of normal sinus rhythm.

▲ ALERT Identify cardiac rhythm strip abnormalities (sinus tachycardia, premature ventricular contractions, V-tach, atrial, and ventricular fibrillation); initiate, maintain, and/or evaluate telemetry monitoring; intervene to improve client cardiovascular status; initiate protocol to manage cardiac dysrhythmias (see Appendix 17-4).
### Common Dysrhythmias

<table>
<thead>
<tr>
<th>RHYTHM</th>
<th>CHARACTERISTICS</th>
<th>INTERVENTIONS/IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sinus bradycardia</strong></td>
<td>Sinus rhythm below a rate of 60 beats/min</td>
<td>Assess client for tolerance of the rate. If hypotension or decreasing LOC occurs, the rhythm is treated. <em>Treatment:</em> atropine.</td>
</tr>
<tr>
<td><strong>Sinus tachycardia</strong></td>
<td>Regular rhythm; P waves present Rate: 100 to 150 beats/min</td>
<td>Assess client for tolerance of the rate. Most often caused by caffeine, alcohol, or physiologic response to stimuli. <em>Treatment:</em> based on underlying cause.</td>
</tr>
<tr>
<td><strong>Paroxysmal atrial (supraventricular) tachycardia</strong></td>
<td>Rhythm is intermittent and is initiated suddenly; terminates suddenly with or without treatment</td>
<td></td>
</tr>
<tr>
<td><strong>Atrial fibrillation</strong></td>
<td>Grossly irregular rate; cannot identify P waves or P-R interval on ECG Controlled rate: 60 to 100 beats/min Uncontrolled rate: 140 to 180 beats/min</td>
<td>Assess client for tolerance to rapid cardiac rate. <em>Most common dysrhythmia.</em> Evaluate client for systemic emboli (pulmonary emboli or an embolic stroke) from clots that tend to form in fibrillating atrium; a decrease in cardiac output will occur with tachycardia; maintain bedrest until rate is controlled. Pulse deficit will occur with rapid rate. Anticoagulation before cardioversion if client is in AF for longer than 48 hours. <em>Treatment:</em> digitalis, calcium channel blockers, beta-blockers, cardioversion.</td>
</tr>
<tr>
<td><strong>AV Block</strong></td>
<td>Prolonged P-R interval; all P waves are followed by a QRS complex May result from digitalis toxicity, electrolyte imbalance, or myocardial ischemia</td>
<td>Identify and treat the underlying cause. Must have an ECG tracing to identify.</td>
</tr>
</tbody>
</table>
CHAPTER 17  Cardiac System

Appendix 17-7  CARDIAC DYSRHYTHMIAS—cont’d

<table>
<thead>
<tr>
<th>RHYTHM</th>
<th>CHARACTERISTICS</th>
<th>INTERVENTIONS/IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Third-degree or complete block</td>
<td>No correlation between atrial impulses and ventricular response</td>
<td>May occur after an MI; symptoms depend on client’s tolerance to rhythm; very unstable and may lead to asystole. Treatment: atropine, isoproterenol, dopamine until pacing can be established with a pacemaker.</td>
</tr>
<tr>
<td>Premature ventricular contractions or beats (PVCs, PVBs)</td>
<td>Premature ectopic beats; occur within a basic rhythm; are of ventricular origin; no P waves; wide, bizarre QRS complex</td>
<td>Indicative of ventricular irritability; considered to be significant and should be treated if they: 1. Occur in excess of 6 beats/min. 2. Occur in a consecutive manner or in pairs. 3. Occur on a T wave of a preceding complex. 4. Cause a pulse deficit. Treatment: oxygen, electrolyte replacement, lidocaine, procainamide; PVCs frequently precede ventricular tachycardia.</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>Looks like a row of PVCs in configuration, wide and bizarre; very rapid rate of 125 to 200 beats/min</td>
<td>Severe decrease in cardiac output; potentially life-threatening situation. May have a pulse or be pulseless. If pulseless initiate CPR. Treatment: lidocaine, amiodarone, procainamide, cardioversion.</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>Very rapid, disorganized rate; cannot identify QRS complexes; erratic conduction</td>
<td>Client is unresponsive, with no pulse; initiate a code and begin CPR. Treatment: defibrillation, lidocaine.</td>
</tr>
<tr>
<td>Asystole</td>
<td>No electrical activity “Straight line”</td>
<td>No cardiac output. Initiate CPR. Treatment: external pacemaker, epinephrine, atropine.</td>
</tr>
</tbody>
</table>


AV, Atrioventricular; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; LOC, level of consciousness; MI, myocardial infarction; PVCs, premature ventricular contractions.
Temporary Pacing

Noninvasive: application of two large external electrodes attached to an external pulse generator.

Invasive: wire electrodes are in contact with the heart and are attached to a battery operated pulse generator. Wires may be placed on the epicardium during heart surgery; leads are passed through the chest wall. Wires may also be placed via a catheter inserted in the antecubital vein and lodged in the right atrium and/or ventricle. Used in emergency situations and in severe cases of bradycardia. Anticipate placement of permanent pacemaker.

Permanent: An internal generator is inserted into soft tissue of upper chest or abdomen, and electrodes are positioned in the atrium and ventricles. PM may synchronize pacing between atrium and ventricle. (Figure 17-13). Procedure is planned and conducted under optimal conditions; used in persistent chronic heart block, dysrhythmias, and/or severe bradycardia.

Pacing Modes

Synchronous demand: The heart is stimulated to beat when the client’s pulse rate falls below a set value or rate (majority of pacemakers have a set rate between 60 and 72 beats/min; if client’s pulse rate falls below the set value, the pacemaker initiates a heartbeat). The pacemaker “senses” the normal heartbeat and the following conduction. If a normal cardiac beat is initiated and conducted, the pacemaker does not initiate an impulse.

Atrioventricular: The ventricle is sensed, and the atrium is paced. If the ventricle does not depolarize, then it is also paced.

Universal atrioventricular: Both the atrial and ventricular circuits sense and pace in the respective chambers. This most closely resembles the normal conduction system.

Pacemaker Failure A cardiac monitor or an electrocardiogram (ECG) must be available to verify pacemaker failure.

Failure to sense: The pacemaker fails to recognize spontaneous heart beats and will fire inappropriately. This is dangerous because the pacer impulse may be discharged during a critical time in the cardiac cycle.

Failure to pace: A malfunction in the pacemaker generator; it may also be due to dislodgment of the leads.

Failure to capture: May occur with a low battery or poor connection. The pacemaker is discharging the impulse; however, there is no responding cardiac contraction.

Nursing Implications

1. Assess for dysrhythmias; rate should not fall below preset level.
2. Maintain bed rest if pulse rate drops below preset level.
3. Hypotension, syncope, and bradycardia should not occur.

Client Education

1. Notify all health care providers of pacemaker placement and parameters; wear a medical alert identification.
2. Avoid constrictive clothing that puts pressure on or irritates the site; report any signs of infection.
3. Safe environment: Avoid areas of high voltage, magnetic force fields (MRI).
4. Avoid activity that requires vigorous movement of arms and shoulders or any direct blows to PM site.
5. Teach client to check his pulse and report if pulse rate is below 60 and client is experiencing symptoms; if this occurs, the pacemaker is not functioning properly, and this needs to be reported immediately.
6. Advise client to immediately report episodes of syncope.
7. Follow-up care and monitoring of pacemaker are important.
8. Client may travel without restrictions.

ALERT Connect and maintain pacing devices; monitor pacemaker and implantable cardioverter defibrillator functions.
Central Venous Pressure Monitoring

Central venous pressure (CVP) is an indication of the pressure within the right atrium or within the large veins in the thoracic cavity. An intravenous (IV) catheter is placed in the superior vena cava or in the right atrium via the subclavian vein or jugular vein. A CVP reading may also be obtained from the proximal lumen of the pulmonary artery catheter. (Figure 17-14)

Interpretation of Pressure Readings
Normal response may range from 4 to 10 mm of H2O pressure. CVP is a dynamic measurement; it must be correlated with client’s overall clinical status for correct interpretation.

Factors That Affect CVP Reading
1. Venous return to the heart (venous tone and vascular volume)
2. Intrathoracic pressure
3. Function of the left side of the heart
4. Coughing or straining (increase CVP)

Nursing Considerations
1. Each CVP reading should be measured from the same zero level point (level of the right atrium or the phlebostatic axis).

The zero level on the manometer or the transducer should be placed at this level to obtain the reading. Client should be supine when the measurements are obtained; head may be slightly elevated, but the position should be consistent for all measurements.

2. If the CVP catheter is patent within the thoracic cavity, the fluid in the manometer or the digital readout will fluctuate with respirations or changes in thoracic pressure when the reading is obtained.
3. Patency of the CVP line is maintained via IV infusion drip.
4. Catheter should be stabilized on the skin at the insertion site, and an occlusive sterile dressing should be applied.

Complications
Air emboli and infections

NURSING PRIORITY To obtain hemodynamic measurements, the zero point of the transducer must be at the level of the right atrium, which is the midaxillary line at the fourth or fifth intercostal space.

Pulmonary Artery Pressure Monitoring (Swan-Ganz Catheter)

A multilumen catheter (Figure 17-15) is inserted and advanced through the right atrium and right ventricle into the pulmonary artery. The flow of blood carries the catheter into the pulmonary artery. Heparinized saline solution is infused under pressure to maintain the patency of the line.

FIGURE 17-14 Pulmonary artery catheter. (From Monahan FD et al: Phipps’ medical-surgical nursing: health and illness perspectives, ed 8, St. Louis, 2007, Mosby.)

Interpretation of Pressure Readings
Pressure readings are obtained via a transducer; for readings to be accurate, the zero level of the transducer must be at the level of the right atrium or the phlebostatic axis (see CVP).
Right atrial (RA) pressure: Normal is 1-8 mm Hg; a direct measurement of the pressure in the right atrium.
Pulmonary artery pressure (PAP): Normal is 15-25 mm Hg systolic and 5-15 mm Hg diastolic, with a mean pressure around 12-15 mm Hg. Mean PAP is increased in clients with chronic pulmonary diseases and those with CHF.
Pulmonary capillary wedge pressure (PAWP or PCWP): Normal is 4-12 mm Hg. It is determined as a mean pressure and is obtained by inflating or wedging a small balloon on the catheter tip in a distal branch of the pulmonary artery. The wedge pressure is indicative of pressure in the left cardiac chambers and reflects the function of the left atrium and ventricle. An upward trend is indicative of increases in pressure and/or fluid, whereas a downward trend indicates fluid, dehydration, or severe vasodilation.

Nursing Implications
1. Maintain patency of line via a heparinized flush solution administered under pressure.
2. The transducer should be at the level of the right atrium to ensure accurate measurements. Catheter should be secured to the skin and covered with an occlusive dressing. Each time the PCWP is evaluated, it is imperative that the balloon or the wedge be deflated to prevent pulmonary infarction, which is validated on the pressure monitor.

Alert Evaluate invasive monitoring data obtained with a pulmonary artery pressure.

Complications
Infections, sepsis, ventricular dysrhythmias, pulmonary infarction, air emboli

Appendix 17-10 CARDIOPULMONARY RESUSCITATION (CPR) FOR HEALTH CARE PROVIDERS

The American Heart Association (AHA) has established standards for cardiopulmonary resuscitation for the health care provider. For further delineation of the procedure, consult the American Heart Association Cardiopulmonary Resuscitation Guidelines. For health care providers, the American Heart Association uses the term infant to refer to individuals between birth and 1 year of age; child is used to refer to those who are between 1 year of age and the onset of puberty.

1. Airway.
   - Identify that the victim is unconscious.
   - Activate the emergency medical services (EMS) system.
   - Obtain automated external defibrillator (AED). Use AED as soon as it is available for all adults and children for sudden, witnessed collapse and for in-hospital clients.
   - Place victim in position to open airway.
   - Open the airway: head-tilt/chin-lift maneuver.
   - Check for adequate breathing (look, listen, and feel for breaths).
   - If victim is breathing, place in recovery position.
   - If victim is not breathing, give 2 rescue breaths using mouth to mouth, or a pocket mask or bag mask.

2. Breathing.
   - Maintain the open airway.
   - Pinch nostrils closed.
   - Give two breaths that make the chest rise, at 1 sec per breath, using mouth-to-mouth technique (mouth-to-nose and mouth technique may be used for small children and infants).
   - Do not give “extra” breaths; do not give large, forceful breaths.
   - When an advanced airway is present, ventilate at the rate of 8-10/min; do not pause for compressions.

3. Check the pulse for no more than 10 sec.
   - Adult and child: Check the carotid pulse.
   - Infant: Check the brachial or femoral pulse.
   - If the pulse is absent, begin chest compressions—cycles of 30 compressions and 2 breaths until ACLS or PALS standards are initiated.

4. External cardiac compression.
   - If the pulse is present, continue rescue breathing, and recheck pulse every 2 min. If, despite adequate ventilation, the heart rate of an infant or child remains under 60 bpm, chest compressions should be started.
   - Adult: 1 breath every 5-6 sec, 10-12 per min.
   - Child and infant: 1 breath every 3-5 sec, 12-20 breaths per min.
   - Advanced airway present (laryngeal mask airway, endotracheal tube): 1 breath every 6-8 sec without trying to synchronize breaths with compressions.

Pediatric Priority Be careful not to hyperextend the infant’s head; this may block the airway. Don’t pinch the infant’s nose shut—cover the nose with your mouth instead. Breathe slowly, just enough to make the chest rise.

4. External cardiac compression.
   - Place the victim on a firm surface. If the client is in a bed, put a cardiac board behind him or her. DO NOT attempt to remove the victim from the bed.
   - Locate the lower half of the sternum in the adult. For the adult, place one hand over the lower sternum; place the other hand on top of the previous hand. For a child (age 1 year to puberty), use the heel of one hand, or two hands based on the size of the child, and press on the center of the chest at the nipple line. For an infant, locate the nipple line; the area for compression is one finger’s width below the line.
   - Depress the sternum 1½ to 2 inches in the adult; in children and infants depress approximately one-third to one-half of depth of chest.
   - Push hard and fast; allow the chest to recoil completely between compressions and as few interruptions as possible.

Adult and child: One rescuer and two rescuers: 30 compressions (rate of 100 per min) to 2 ventilations.
Appendix 17-10  CARDIOPULMONARY RESUSCITATION (CPR) FOR HEALTH CARE PROVIDERS—cont’d

Infant:
One rescuer: 30 compressions (rate of 100 per min) to 2 ventilations.
Two rescuers: 15 compressions to 2 ventilations.
Defibrillation: Use automatic electronic defibrillator (AED) as soon as available for sudden collapse and for in-hospital clients.
Steps for using an automated external defibrillator (AED):
1. Provide CPR until the AED arrives.
2. On arrival, open the case and turn the power on.
3. Select the correct pads for the size and age of the victim (only use the child pads/system for children less than 8 years old).
4. Attach the adhesive electrodes—one on the upper-right side of the chest directly below the clavicle, the other below the left nipple below the left arm pit.
5. Attach the connecting cable to the AED.
6. Compressions will need to be discontinued while the rhythm is analyzed.
7. If shock is indicated, loudly state “Clear!” and visually check to ensure that no one is touching the client.
8. Press the SHOCK button.
9. Resume CPR immediately with cycles of 30 compressions to 2 breaths.
After 2 min, the AED will prompt you to repeat steps 6 through 9.
5. Termination of CPR. A rescuer who is not a physician should continue resuscitation efforts until one of the following occurs:
   • Spontaneous circulation and ventilation have been restored.
   • Resuscitation efforts are transferred to another equally responsible person who continues the resuscitation procedure.
   • A physician, or physician-directed person, assumes responsibility for resuscitation procedure.
   • The victim is transferred to an emergency medical service (e.g., paramedics and ambulance).
   • The rescuer is exhausted and unable to continue resuscitation.


Appendix 17-11  NONDRUG TREATMENT OF DYSRHYTHMIAS

Defibrillation is depolarization of the electrical impulses in the heart to allow the heart’s own intrinsic pacemaker to regain control of cardiac rhythm.

Defibrillation (Unsynchronized)
Depolarization of cardiac cells in an emergency situation with a life-threatening dysrhythmia:
1. Always begin cardiopulmonary resuscitation (CPR) if client has no pulse and is unconscious; do not wait for a defibrillator.
2. Make sure everyone is clear of the bed and the client is not lying in fluids.
3. Make sure everyone is clear from touching the bed or client.
4. Synchronization is turned off.
5. Defibrillation is started at 200 J and progresses to a higher energy level (300–360 J).
7. Remain with client and closely assess for adequacy and stability of cardiac output and cardiac rhythm.

Synchronized Cardioversion
Depolarization of cardiac cells to allow the sinus node to regain control of cardiac rhythm:
1. An elective, planned procedure or used when client has a tachycardic rhythm with a pulse rate greater than 150.
2. Requires client teaching and informed consent when it is an elective procedure.
3. Document rhythm before synchronized cardioversion and immediately afterward.
4. The defibrillator is set (synchronized) to deliver the electrical discharge during the QRS complex; this prevents the discharge from occurring on the T wave. Look for the sync markers on the R wave.
5. Verbalize “Clear!” loudly and validate that no one is touching the client or bed before administering the shock.
6. Cardioversion is started at 50 J to 100 J.
7. Remain with client and closely assess for adequacy and stability of cardiac output and cardiac rhythm.

Implantable Cardioverter Defibrillators (ICD)
1. Used in clients who have survived one or more episodes of sudden death from ventricular dysrhythmias or from refractory ventricular dysrythmias not associated with a myocardial infarction.
2. Implantable device can also be used as a pacemaker.
3. Inserted in a subcutaneous pocket in the subclavicular or abdominal area.
4. Client should avoid contact sports, increase activity gradually, and carry ICD identification information.
5. Client should avoid working over large motors and should move away from devices if dizziness occurs; client should avoid magnets directly over area of ICD.
6. Common response to device is anxiety and fear.
7. Assess for fluid hazards; intravenous fluids and urine may conduct voltage.
8. Client should sit or lie down if they become dizzy or if they feel the ICD discharge.

Radiofrequency Catheter Ablation
1. Used in clients who experience atrial tachycardia or AV nodal reentrant tachycardia. Electrophysiologic testing identifies the small region of the myocardium generating the dysrhythmia. A radiofrequency catheter is placed at the site, and the catheter is activated to generate RF energy. This energy destroys the tissue eliminating the dysrhythmia.
2. Complications are minimal but can include AV blocks and myocardial perforation.
Study Questions  Cardiac System

1. A client with a diagnosis of acute coronary syndrome is on a cardiac monitor. The nurse interprets the monitor rhythm to be ventricular tachycardia at a rate of 160 beats/min. The client is awake and coherent, and oxygen is being administered at a rate of 6 L/min via a nasal cannula. What is the first nursing action?
   1. Immediately defibrillate.
   2. Administer lidocaine IV push.
   3. Initiate transvenous pacing.
   4. Administer adenosine IV push.

2. The nurse is caring for a client with cor pulmonale, or right-side heart failure. What nursing assessment information correlates with an increase in venous pressure?
   1. Jugular vein distention with client sitting at a 45-degree angle
   2. Crackling sounds over the lower lobes with client in an upright position
   3. Bradycardia, restlessness, and an increase in respiratory rate
   4. Decreased pulsations at the point of maximum impulse

3. The nurse is administering nitroglycerin intravenously to relieve chest pain. What is the therapeutic action of this medication?
   1. Increases glomerular filtration, which results in decreased venous return
   2. Enhances contractility of the myocardium, which increases oxygen delivery
   3. Produces an immediate analgesic effect and relieves chest pain
   4. Increases the coronary blood supply and decreases the afterload

4. In discharge planning for the client with heart failure, the nurse discusses the importance of adequate rest. What information is most important?
   1. A warm, quiet room is necessary.
   2. Bed rest promotes venous return.
   3. A hospital bed is necessary.
   4. Adequate rest decreases cardiac workload.

5. The nurse is assessing a client whose condition is being stabilized after experiencing an acute coronary syndrome (myocardial infarction). What finding on the nursing assessment would indicate inadequate renal perfusion?
   1. Decreasing serum blood urea nitrogen (BUN) level
   2. Urine specific gravity of less than 1.010
   3. Urine output of less than 30 mL/hr
   4. Low urine osmolality and creatinine clearance

6. The nurse applies a Nitro-Dur patch on a client who has undergone cardiac surgery. What nursing observation indicates that a Nitro-Dur patch is achieving the desired effect?
   1. Chest pain is completely relieved.
   2. Client performs activities of daily living without chest pain.
   3. Pain is controlled with frequent changes of patch.
   4. Client tolerates increased activity without pain.

7. The nurse has been assigned a group of cardiac clients. What would be the most important information for the nurse to check on the initial evaluation of each client? Select all that apply:
   _____ 1. Presence of cardiac pain
   _____ 2. Medications taken prior to hospitalization
   _____ 3. Presence of jugular vein distention
   _____ 4. Heart sounds and apical rate
   _____ 5. Presence of diaphoresis
   _____ 6. History of difficulty breathing

8. During the night, a client with a diagnosis of acute coronary syndrome (myocardial infarction) is found to be restless and diaphoretic. What is the best nursing action?
   1. Check his temperature and determine his serum blood glucose level.
   2. Turn the alarms low and promote sleep by decreasing the number of interruptions.
   3. Check the monitor to determine his cardiac rhythm and evaluate vital signs.
   4. Call the physician to obtain an order for sedation.

9. The nurse is caring for a client who begins to exhibit the cardiac rhythm shown in the illustration below. As the nurse observes, the rhythm remains the same. What is the best nursing action?
   1. Call an emergency code and begin resuscitation.
   2. Turn client on his left side and take his blood pressure.
   3. Prepare to administer lidocaine IV push.
   4. Take the apical pulse for a full minute.

![Cardiac Rhythm Illustration]
10. The nurse is providing preoperative care for a client who is scheduled for cardiac surgery. During the preoperative preparation, what is an important nursing action?
   1. Perform a thorough nursing assessment to provide an accurate baseline for evaluation after surgery.
   2. Discuss with the client the steps of myocardial cellular metabolism and the anticipated surgical response.
   3. Provide preoperative education regarding the mechanics of the cardiopulmonary bypass machine.
   4. Discuss with the client and family the anticipated amount of postoperative chest tube drainage.

11. An older adult client is taking digitalis, 0.025 mg twice a day, and Lasix (furosemide), 40 mg daily, for CHF. She has complained of increasing lethargy and nausea over the past 2 days; however, she is still able to take her medication. Her blood pressure is 150/98 mm Hg; pulse is 110 beats/min and irregular; respiratory rate is 18 breaths/min. What laboratory information is most important for the nurse to evaluate?
   1. Hemoglobin, hematocrit, and white blood cell count
   2. Arterial blood gases and acid-base balance
   3. Blood urea nitrogen (BUN) and serum creatinine levels
   4. Serum electrolyte levels, particularly potassium

12. During the shift hand-off report, the nurse learns that one of the assigned clients is in first-degree heart block. What is a nursing measure to assess the status of this dysrhythmia?
   1. Count the radial pulse for 1 full minute.
   2. Determine the cardiac rate at the PMI.
   3. Evaluate an ECG or monitor strip.
   4. Hourly pulse checks and correlate with blood pressure.

13. The nurse is preparing a client for a cardiac catheterization. What is the best explanation regarding the purpose of a cardiac catheterization with coronary angiography?
   1. Evaluate the exercise tolerance.
   2. Study the conduction system.
   3. Evaluate coronary artery blood flow.
   4. Measure the myocardial contractility.

14. A client is admitted for evaluation of his permanent pacemaker. What data would confirm that the pacemaker is not working correctly?
   1. Pulse rate of 96 beats/min with regular rate and rhythm
   2. Irregular pulse rate with premature ventricular beats
   3. Atrial premature beats shown on the monitor
   4. Pulse rate of 48 beats/min with premature ventricular beats

15. The nurse is assessing the pulse of a client in atrial fibrillation. Based on the graphic below, where should the stethoscope be placed to correctly auscultate this client’s pulse?

16. The vital signs of a client with cardiac disease are as follows: blood pressure of 100/78 mm Hg, pulse of 46 beats/min, and respiratory rate of 18 breaths/min. Atropine (atropine sulfate) is administered IV push. What nursing assessment indicates a therapeutic response to the medication?
   1. Pulse rate has increased to 70 beats/min.
   2. Systolic blood pressure has increased by 20 mm Hg.
   3. Pupils are dilated.
   4. Oral secretions have decreased.

17. The nurse is taking the history of a client with heart failure (congestive heart failure) caused by chronic hypertension. The nurse identifies what data as supportive of the client's medical diagnosis?
   1. Dyspnea after walking about half a block
   2. Weight loss of 15 pounds over last 3 months
   3. Lower extremity edema in the evenings
   4. Dizziness and fainting when rising too quickly

18. A client is admitted with mitral valve disease and left ventricular dysfunction. What is the most reliable test to determine cardiac status?
   1. Electrocardiogram
   2. Stress test
   3. Cardiac angiogram
   4. Echocardiogram

19. What would be an important home care goal for a client who has bacterial endocarditis?
   1. To begin an exercise regimen as soon as possible
   2. To monitor urinary output
   3. To continue antibiotic therapy
   4. To decrease activity until pulse stabilizes

Answers and rationales to these questions are in the section at the end of the book titled Chapter Study Questions: Answers and Rationales.