**Home Care**

Many clients with stable angina manage their pain effectively, continuing to live active and productive lives. To promote effective management of this disorder, include the following topics in teaching for home care.

- CHD and the processes that cause chest pain, including the relationship between the pain and reduced blood flow to the heart muscle
- Use and effects (desired and adverse) of prescribed medications; importance of not discontinuing medications abruptly
- Nitroglycerine use for acute angina: Always carry several tablets (not the entire supply); prophylactic use before activities that often cause chest pain; take tablet at first indication of pain rather than waiting to see if the pain develops; seek immediate medical assistance if three nitroglycerin tablets over 15 to 20 minutes do not relieve the pain
- The importance of calling 911 or going to the emergency department immediately for unrelieved chest pain
- Appropriate storage of nitroglycerin: This unstable compound needs to be stored in a cool, dry, dark place; no more than a 6-month supply should be kept on hand

For the client who has undergone cardiac surgery, also include the following:

- Respiratory care, activity, and pain management
- The importance of actively participating in rehabilitation
- Manifestations of infection or other potential complications and their management

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**Nursing Care Plan**

**A Client with Coronary Artery Bypass Surgery**

Six weeks ago, John Clements, age 50, was discharged from the hospital after emergency triple bypass surgery. Despite having emergency surgery, his postoperative recovery was uneventful, and he was discharged 6 days after admission. He returns to the clinic for a postoperative stress test and to discuss his cardiac rehabilitation program. Anne Wagner, RN, CNS, a cardiac clinical nurse specialist and the program coordinator, meets Mr. Clements to obtain specific information regarding his medical status.

**ASSESSMENT**

Mr. Clements’s medical history reveals significant CHD, an anterior wall myocardial infarction that led to his emergency triple bypass, and hyperlipidemia. Current medications include Cardizem, Isordil, Ecrothrin, and Transderm-Nitro 5. The ECG reveals sinus rhythm with some ST segment and T wave flattening. Resting heart rate 68, and blood pressure 136/84.

Mr. Clements has a strong family history of CHD. He does not smoke and uses alcohol occasionally in social situations. He enjoys “good Southern-style cooking” and watching television. Mr. Clements states his only regular exercise used to be an evening of dancing with his wife and friends about once a month, “But I get short of breath walking around the block now, so I guess I can’t go dancing anymore!”

Mr. Clements owns his own contracting business and states that he typically works about 50 to 60 hours per week. He tells Ms. Wagner, “I don’t know what this program is supposed to do for me. I have got to get back to work! You just can’t sit around in my business—you have to make sure that the work is getting done on time, and you have to check on supplies and equipment and the like. But I feel like a weakling—I need to get my energy back!”

**DIAGNOSES**

Ms. Wagner formulates the following nursing diagnoses with Mr. Clements.

- Activity intolerance related to general weakness and fatigue
- Ineffective role performance related to health crisis

**EXPECTED OUTCOMES**

Mr. Clements and Ms. Wagner mutually agree that he will:

- Verbalize an understanding of the definition and components of his structured cardiac rehabilitation program.
- Verbalize a desire to make lifestyle changes.
- Identify resources available in the community to assist with lifestyle changes.
- Participate in his activity program without suffering any complications.
- Verbalize an increase in energy after 6 weeks on the program.
- Accept the reality of the temporary change in his usual work responsibilities.

**PLANNING AND IMPLEMENTATION**

Ms. Wagner plans and implements the following nursing interventions for Mr. Clements.

- Define the purpose and components of a cardiac rehabilitation program.
- Enroll in “heart health” classes, including cardiac anatomy, physiology, and coronary heart disease; exercise and activity prescriptions; lifestyle modifications, including diet counseling and stress management; emotional reactions to CAD; sexual activity; use of cardiac medications; and self-responsibility for health.
- Plan an exercise program based on stress test results, physical examination, and interview.
- Encourage to schedule rest periods before and after activity/exercise.
- Review signs and symptoms of overexertion.
- Provide information about community resources for emotional and educational support.
- Assist to identify strategies for dealing with concerns about his business role.

**EVALUATION**

Mr. Clements decides to “give the rehab program a try.” Ms. Wagner and an exercise physiologist work with him to plan an individualized exercise/activity program. A registered dietitian provides dietary counseling. Ms. Wagner emphasizes stress management strategies. Mr. Clements is able to list manifestations of overexertion and states that he realizes the need for gradual activity progression.
**THE CLIENT WITH ACUTE MYOCARDIAL INFARCTION**

An acute myocardial infarction (AMI), necrosis (death) of myocardial cells, is a life-threatening event. If circulation to the affected myocardium is not promptly restored, loss of functional myocardium affects the heart’s ability to maintain an effective cardiac output. This may ultimately lead to cardiogenic shock and death.

Heart disease remains the leading cause of death in the United States. Of the major heart diseases, myocardial infarction or heart attack, and other forms of ischemic heart disease cause the majority of deaths. Annually, approximately 650,000 people in the United States experience their first MI; another 450,000 suffer an MI subsequent to the initial one. Nearly 530,000 people died of coronary heart disease in 2000, with most of these deaths related to MI (NHLBI, 2002).

The majority of deaths from MI occur during the initial period after symptoms begin: approximately 60% within the first hour, and 40% prior to hospitalization. Heightening public awareness of the manifestations of MI, the importance of seeking immediate medical assistance, and training in cardiopulmonary resuscitation (CPR) techniques are vital to decrease deaths due to MI.

Myocardial infarction rarely occurs in clients without pre-existing coronary heart disease. While no specific cause has been identified, the risk factors for MI are those for coronary heart disease: age, gender, heredity, race; smoking, obesity, hyperlipidemia, hypertension, diabetes, sedentary lifestyle, diet, and others. See the previous section of this chapter on coronary heart disease for further discussion of these risk factors.

**PATHOPHYSIOLOGY**

Atherosclerotic plaque may form stable or unstable lesions. Stable lesions progress by gradually occluding the vessel lumen, whereas unstable (or complicated) lesions are prone to rupture and thrombus formation. Stable lesions often cause angina (discussed in the previous section); unstable lesions often lead to acute coronary syndromes, or acute ischemic heart diseases. Acute coronary syndromes include unstable angina, myocardial infarction, and sudden cardiac death (McCance & Huether, 2002).

Myocardial infarction occurs when blood flow to a portion of cardiac muscle is blocked, resulting in prolonged tissue ischemia and irreversible cell damage. Coronary occlusion is usually caused by ulceration or rupture of a complicated atherosclerotic lesion. When an atherosclerotic lesion ruptures or ulcerates, substances are released that stimulate platelet aggregation, thrombin generation, and local vasomotor tone. As a result, a thrombus (clot) forms, occluding the vessel and interrupting blood flow to the myocardium distal to the obstruction.

Cellular injury occurs when the cells are denied adequate oxygen and nutrients. When ischemia is prolonged, lasting more than 20 to 45 minutes, irreversible hypoxemic damage causes cellular death and tissue necrosis. Oxygen, glycogen, and ATP stores of ischemic cells are rapidly depleted. Cellular metabolism shifts to an anaerobic process, producing hydrogen ions and lactic acid. Cellular acidosis increases cells’ vulnerability to further damage. Intracellular enzymes are released through damaged cell membranes into interstitial spaces.

Cellular acidosis, electrolyte imbalances, and hormones released in response to cellular ischemia affect impulse conduction and myocardial contractility. The risk of dysrhythmias increases, and myocardial contractility decreases, reducing stroke volume, cardiac output, blood pressure, and tissue perfusion.

The subendocardium suffers the initial damage, within 20 minutes of injury, because this area is the most susceptible to changes in coronary blood flow. If blood flow is restored at this point, the infarction is limited to subendocardial tissue (a subendocardial or non-Q wave infarction). The damage progresses to the epicardium within 1 to 6 hours. When all layers of the myocardium are affected, it is known as a transmural infarction. A significant Q wave develops with a transmural infarction, so this also may be called a Q wave MI. Complications...