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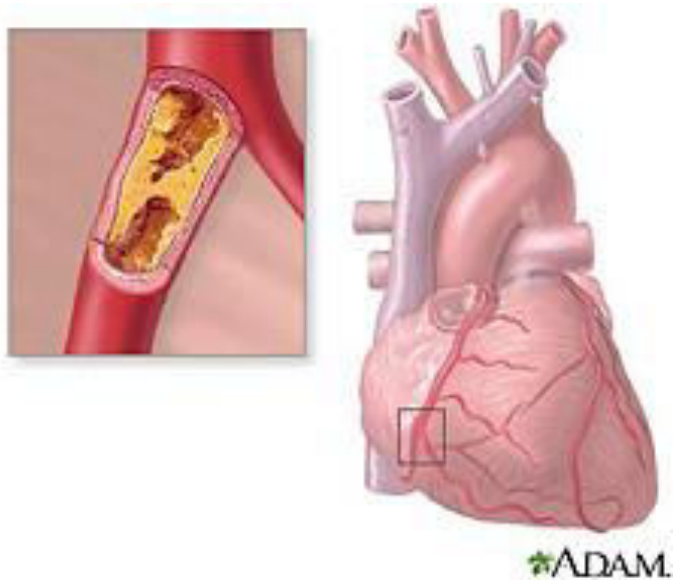
CORONARY ARTERY DISEASE

1. DEFINITION:

Coronary artery disease (CAD) is the leading cause of death in the United States. CAD is characterized by the accumulation of plaque within the layers of the coronary arteries. The plaques progressively enlarge, thicken, and calcify, causing a critical narrowing (> 70% occlusion) of the coronary artery lumen, resulting in a decrease in coronary blood flow and an inadequate supply of oxygen to the heart muscle.

An acute coronary syndrome (ACS) is a term used to define potential complications of CAD. This syndrome includes unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI).



2. PATHOPHYSIOLOGY AND ETIOLOGY:

- The most widely accepted cause of CAD is atherosclerosis
- Angina pectoris, caused by inadequate blood flow to the myocardium, is the most common manifestation of CAD.
    - Angina is usually precipitated by physical exertion or emotional stress, which puts an increased demand on the heart to circulate more blood and oxygen.

    - The ability of the coronary artery to deliver blood to the myocardium is impaired because of obstruction by a significant coronary lesion (> 70% narrowing of the vessel).
    - Angina can also occur in other cardiac problems, such as arterial spasm, aortic stenosis, cardiomyopathy, or uncontrolled hypertension.
    - Noncardiac causes include anemia, fever, thyrotoxicosis, and anxiety/panic attacks.
- ACS is caused by a decrease in the oxygen available to the myocardium due to:
    - unstable or ruptured atherosclerotic plaque.
    - coronary vasospasm.
    - atherosclerotic obstruction without clot or vasospasm.
    - inflammation or infection.
    - unstable angina due to a noncardiac cause (see angina).
    - thrombus formation with subsequent coronary artery occlusion (the most common cause).
- Risk factors for the development of CAD include:
    - Nonmodifiable: age (risk increases with age), male sex (women typically suffer from heart disease 10 years later than men due to the postmenopausal decrease in cardiac-protective estrogen), race (nonwhite populations have increased risk), and family history.
    - Modifiable: elevated lipid levels, hypertension, obesity, tobacco use, metabolic syndrome (obesity, hypertension, and diabetes mellitus), sedentary lifestyle, stress.
    - Recent studies have shown that there are new risk factors associated with the development of CAD. These include increased levels of homocysteine, fibrin, lipoprotein(a), and infection or inflammation (measured by C-reactive protein [CRP]).
    - The American Heart Association (AHA) also lists left ventricular hypertrophy (LVH) as a risk factor.

3. CORONARY ATHEROSCLEROSIS:

Atherosclerosis, which is an abnormal accumulation of lipid, or fatty and fibrous tissue in the vessel wall. These substances create blockages or narrow the vessel in a way that reduces blood flow to the myocardium.

3.1. PATHOPHYSIOLOGY:

Unknown cause or due to genetic or environmental factors

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Fatty streaks, lipids that are deposited in the intima of the arterial wall

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Inflammatory response

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T lymphocytes and monocytes infiltrate the area to ingest the lipids and then die

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Smooth muscle cells within the cells to proliferate and form a fibrous cap over the dead fatty core, called as atheromas or plaques, protrude into the lumen of the vessel narrowing it and obstructing blood flow

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If the plaque is thick, the lipid pool remains relatively stable; it can resist the stress from blood flow and vessel movement. If the cap is thin, the lipid core may grow, causing it to rupture and hemorrhage into the plaque, allowing a thrombus to develop.

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The thrombus may obstruct the blood flow, leading to myocardial infarction.

4. Chronic Stable Angina Pectoris:

Chest pain or discomfort that is provoked by exertion or emotional stress and relieved by rest and nitroglycerin.

- Character—substernal chest pain, pressure, heaviness, or discomfort. Other sensations include a squeezing, aching, burning, choking, strangling, and/or cramping pain.
    - Pain may be mild or severe and typically presents with a gradual buildup of discomfort and subsequent gradual fading.
    - May produce numbness or weakness in arms, wrists, or hands.
    - Associated symptoms include diaphoresis, nausea, indigestion, dyspnea, tachycardia, and increase in blood pressure.
    - Women may experience atypical symptoms of chest pain, such as jaw pain, shortness of breath, or indigestion.
- Location—behind middle or upper third of sternum; the patient will generally make a fist over the site of the pain (positive Levine sign; indicates diffuse deep visceral pain) rather than point to it with his finger.
- Radiation—usually radiates to neck, jaw, shoulders, arms, hands, and posterior intrascapular area. Pain occurs more commonly on the left side than the right.
- Duration—usually lasts 2 to 15 minutes after stopping activity; nitroglycerin relieves pain within 1 minute.
- Other precipitating factors—exposure to weather extremes, eating a heavy meal, and sexual intercourse all increase the workload of the heart, thus increasing oxygen demand.

5. Unstable (Preinfarction) Angina Pectoris:

Chest pain occurring at rest; no increase in oxygen demand is placed on the heart, but an acute lack of blood flow to the heart occurs because of coronary artery spasm or the presence of an enlarged plaque or hemorrhage/ulceration of a complicated lesion. Critical narrowing of the vessel lumen occurs abruptly in either instance.

- A change in frequency, duration, and intensity of stable angina symptoms is indicative of progression to unstable angina.
- Unstable angina pain lasts longer than 10 minutes, is unrelieved by rest or sublingual nitroglycerin, and mimics signs and symptoms of impending MI.

6. SYMPTOMS:

Symptoms may be very noticeable, but sometimes you can have the disease and not have any symptoms.

Chest pain or discomfort (angina) is the most common symptom. You feel this pain when the heart is not getting enough blood or oxygen. How bad the pain is varies from person to person.

- It may feel heavy or like someone is squeezing your heart. You feel it under your breast bone (sternum), but also in your neck, arms, stomach, or upper back.
- The pain usually occurs with activity or emotion, and goes away with rest or a medicine called nitroglycerin.
- Other symptoms include shortness of breath and fatigue with activity (exertion).

Women, elderly people, and people with diabetes are more likely to have symptoms other than chest pain, such as:

- Fatigue
- Shortness of breath
- Weakness

7. DIAGNOSTIC EVALUATION:

- Characteristic chest pain and clinical history.
- Nitroglycerin test—relief of pain with nitroglycerin.
- Blood tests.
    - Hemoglobin to rule out anemia, which may reduce myocardial oxygen supply.
    - HbA_{1C} and fasting lipid panel to rule out modifiable risk factors for CAD.
    - Coagulation studies, CRP (determines inflammation), homocysteine (elevated levels can cause damage to the artery lining), and lipoprotein(a) (increased levels are associated with a twofold risk in developing CAD).

- Cardiac markers, creatine kinase (CK) and its isoenzyme CK-MB, and troponin-I to determine the presence and severity of cardiac insult.

- Resting ECG—may show LVH, ST-T wave changes, arrhythmias, and Q waves.
- ECG stress testing—progressive increases of speed and elevation walking on a treadmill increase the workload of the heart. ST-T wave changes occur if myocardial ischemia is induced.
- Radionuclide imaging—a radioisotope, thallium 201, injected during exercise is imaged by camera. Low uptake of the isotope by heart muscle indicates regions of ischemia induced by exercise. Images taken during rest show a reversal of ischemia in those regions affected.
- Radionuclide ventriculography (gated blood pool scanning)—red blood cells tagged with a radioisotope are imaged by camera during exercise and at rest. Wall motion abnormalities of the heart can be detected and ejection fraction estimated.
- Cardiac catheterization—coronary angiography performed during the procedure determines the presence, location, and extent of coronary lesions.
- Positron-emission tomography (PET)—cardiac perfusion imaging with high resolution to detect very small perfusion differences caused by stenotic arteries. Not available in all settings.
- Electron-beam computed tomography (CT)—detects coronary calcium, which is found in most, but not all, atherosclerotic plaque. It is not routinely used due to its low specificity for identifying significant CAD.

8. MANAGEMENT:

8.1. DRUG THERAPY

Antianginal medications (nitrates, beta-adrenergic blockers, calcium channel blockers, and angiotensin-converting enzyme [ACE] inhibitors) are used to maintain a balance between oxygen supply and demand. Coronary vessel relaxation promotes blood flow to the heart, thereby increasing oxygen supply. Reduction of the workload of the heart decreases oxygen demand and consumption. The goal of drug therapy is to maintain a balance between oxygen supply and demand.

- Nitrates—cause generalized vasodilation throughout the body. Nitrates can be administered orally, sublingually, transdermally, I.V., or intracoronary (I.C.) and may provide short- or long-acting effects.
    - Short-acting nitrates (sublingual) provide immediate relief of acute anginal attacks or prophylaxis if taken before activity.
    - Long-acting nitrates prevent anginal episodes and/or reduce severity and frequency of attacks.
- Beta-adrenergic blockers—inhibit sympathetic stimulation of receptors that are located in the conduction system of the heart and in heart muscle.
    - Some beta-adrenergic blockers inhibit sympathetic stimulation of receptors in the lungs as well as the heart ("nonselective" beta-adrenergic blockers); vasoconstriction of the large airways in the lung occurs; generally contraindicated for patients with chronic obstructive lung disease or asthma.
    - "Cardioselective" beta-adrenergic blockers (in recommended dose ranges) affect only the heart and can be used safely in patients with lung disease.
- Calcium channel blockers—inhibit movement of calcium within the heart muscle and coronary vessels; promote vasodilation and prevent/control coronary artery spasm.
- ACE inhibitors—have therapeutic effects by remodeling the vascular endothelium and have been shown to reduce the risk of worsening angina.
- Antilipid agents—reduce total cholesterol and triglyceride levels and have been shown to assist in the stabilization of plaque.
- Antiplatelet agents—decrease platelet aggregation to inhibit thrombus formation.
- Folic acid and B complex vitamins—treat increased homocysteine levels.

8.2. PERCUTANEOUS CORONARY INTERVENTIONS:

- Percutaneous transluminal angioplasty
    - A balloon-tipped catheter is placed in a coronary vessel narrowed by plaque.

    - The balloon is inflated and deflated to stretch the vessel wall and flatten the plaque
    - Blood flows freely through the unclogged vessel to the heart.
- Intracoronary atherectomy
    - A blade-tipped catheter is guided into a coronary vessel to the site of the plaque.
    - Depending on the type of blade, the plaque is either cut, shaved, or pulverized, and then removed.
    - Requires a larger catheter introduction sheath so its use is limited to larger vessels.
- Intracoronary stent
    - A diamond mesh tubular device is placed in the coronary vessel.
    - Prevents restenosis by providing a "skeletal" support.
    - Drug-eluting stents contain an anti-inflammatory drug, which decrease the inflammatory response within the artery.

Other Interventional Strategies

- Coronary artery bypass graft (CABG) surgery
    - A graft is surgically attached to the aorta, and the other end of the graft is attached to a distal portion of a coronary vessel.
    - Bypasses obstructive lesions in the vessel and returns adequate blood flow to the heart muscle supplied by the artery
- Transmyocardial revascularization—by means of a laser beam, small channels are formed in the myocardium to encourage new blood flow.

9. SECONDARY PREVENTION:

According to the AHA/American College of Cardiology (ACC) Guidelines for Secondary Prevention for Patients with Coronary and Other Atherosclerotic Vascular Disease (2006):

- Cessation of smoking

- Control of high blood pressure (below 130/85 mm Hg in those with renal insufficiency or heart failure; below 130/80 mm Hg in those with diabetes; below 140/90 mm Hg in all others)
- Diet low in saturated fat (< 10% of calories), cholesterol (<300 mg/day), trans-fatty acids, sodium (>6 g/day), alcohol (2 or fewer drinks/day in men, 1 or fewer in women)
- Low-dose aspirin daily for those at high risk
- Physical exercise (at least 30 minutes of moderate intensity exercise most days)
- Weight control (ideal body mass index 18.5 to 24.9 kg/m^2); waist circumference less than 40 inches for men, less than 35 inches for women
- Control of diabetes mellitus (fasting glucose <110 mg/dL and HbA_{1C} < 7%)
- Control of blood lipids with low-density lipoprotein (LDL) goal less than 100.

10. COMPLICATIONS:

- Sudden death due to lethal dysrhythmias
- Heart failure
- MI

11. MYOCARDIAL INFARCTION

MI refers to a dynamic process by which one or more regions of the heart experience a severe and prolonged decrease in oxygen supply because of insufficient coronary blood flow; subsequently, necrosis or "death" to the myocardial tissue occurs. The onset of the MI process may be sudden or gradual, and the progression of the event to completion takes approximately 3 to 6 hours. MI is one manifestation of ACS.

11.1. PATHOPHYSIOLOGY AND ETIOLOGY:

- Acute coronary thrombosis (partial or total)—associated with 90% of MIs.
  - Severe CAD (> 70% narrowing of the artery) precipitates thrombus formation.

- The first step in thrombus formation involves plaque rupture. Platelets adhere to the damaged area.
- Activation of the exposed platelets causes expression of glycoprotein IIb/IIIa receptors that bind fibrinogen.
- Further platelet aggregation and adhesion occurs, enlarging the thrombus and occluding the artery.

- Other etiologic factors include coronary artery spasm, coronary artery embolism, infectious diseases causing arterial inflammation, hypoxia, anemia, and severe exertion or stress on the heart in the presence of significant CAD (ie, surgical procedures or shoveling snow).
- Different degrees of damage occur to the heart muscle
- Zone of necrosis—death to the heart muscle caused by extensive and complete oxygen deprivation; irreversible damage
- Zone of injury—region of the heart muscle surrounding the area of necrosis; inflamed and injured, but still viable if adequate oxygenation can be restored
- Zone of ischemia—region of the heart muscle surrounding the area of injury, which is ischemic and viable; not endangered unless extension of the infarction occurs

11.2. CLASSIFICATION OF MI:

- STEMI—whereby ST-segment elevations are seen on ECG. The area of necrosis may or may not occur through the entire wall of heart muscle.
- NSTEMI—no ST-segment elevations can be seen on ECG. ST depressions may be noted as well as positive cardiac markers, T-wave inversions, and clinical equivalents (chest pain). Area of necrosis may or may not occur through the entire myocardium.

- The region(s) of the heart muscle that becomes affected depends on which coronary artery(s) becomes obstructed
  - Left ventricle is a common and dangerous location for an MI because it is the main pumping chamber of the heart.
  - Right ventricular infarctions commonly occur with damage to the inferior and/or posterior wall of the left ventricle.

- The severity and location of the MI determines prognosis.

11.3. CLINICAL MANIFESTATIONS:

- Chest pain
    - Severe, diffuse, steady substernal pain; may be described as crushing, squeezing, or dull
    - Not relieved by rest or sublingual vasodilator therapy, but requires opioids
    - May radiate to the arms (usually the left), shoulders, neck, back, and/or jaw
    - Continues for more than 15 minutes
    - May produce anxiety and fear, resulting in an increase in heart rate, BP, and respiratory rate
    - Some patients exhibit no complaints of pain.
- Diaphoresis, cool clammy skin, facial pallor
- Hypertension or hypotension
- Bradycardia or tachycardia
- Premature ventricular and/or atrial beats
- Palpitations, severe anxiety, dyspnea
- Disorientation, confusion, restlessness
- Fainting, marked weakness
- Nausea, vomiting, hiccups

Atypical symptoms: epigastric or abdominal distress, dull aching or tingling sensations, shortness of breath, extreme fatigue

11.4. DIAGNOSTIC EVALUATION:

ECG Changes

- Generally occur within 2 to 12 hours, but may take 72 to 96 hours.
- Necrotic, injured, and ischemic tissue alters ventricular depolarization and repolarization.

- ST-segment depression and T-wave inversion indicate a pattern of ischemia.
- ST elevation indicates an injury pattern.
- Q waves indicate tissue necrosis and are permanent. A pathologic Q wave is one that is greater than 3 mm in depth or greater than one-third the height of the R wave.

Location of the infarction (anterior wall, anteroseptal) is determined by the leads in which the ischemic changes are seen.

Other Findings

- Elevated CRP and lipoprotein(a) due to inflammation in the coronary arteries.
- Abnormal coagulation studies (prothrombin time [PT], partial thromboplastin time [PTT]).
- Elevated white blood cell (WBC) count and sedimentation rate due to the inflammatory process involved in heart muscle cell damage.
- Radionuclide imaging allows recognition of areas of decreased perfusion.
- PET determines the presence of reversible heart muscle injury and irreversible or necrotic tissue; extent to which the injured heart muscle has responded to treatment can also be determined.
- Cardiac muscle dysfunction noted on echocardiography or cardiac magnetic resonance imaging (MRI).

11.5. MANAGEMENT:

Therapy is aimed at reversing ischemia to preserve cardiac muscle function, reduce the infarct size, and prevent death. Innovative modalities provide early restoration of coronary blood flow. The use of pharmacologic agents improves oxygen supply and demand, reduces and prevents dysrhythmias, and inhibits the progression of CAD.

The pharmacologic therapy for treatment of MI is standard

MONA—acronym that outlines the immediate pharmacologic interventions used to treat MI.

- M (Morphine)—given I.V. Used to treat chest pain. Endogenous catecholamine release during pain imposes an increase in the workload on the heart, thus causing an increase in oxygen demand. Morphine's analgesic effects decrease the pain, relieve anxiety, and improve cardiac output by reducing preload and afterload.
- O (Oxygen)—given via nasal cannula or face mask. Increases oxygenation to ischemic heart muscle.
- N (Nitrates)—given sublingually, spray, or I.V. Vasodilator therapy reduces preload by decreasing blood return to the heart and decreasing oxygen demand.
- A (Aspirin)—immediate dosing by mouth is recommended to halt platelet aggregation.

Other Medications

- Thrombolytic agents, such as tissue plasma activator (Activase), streptokinase (Streptase), and reteplase (Retavase), reestablish blood flow in coronary vessels by dissolving thrombus.
    - No effect on the underlying stenosis that precipitated the thrombus to form.
    - Administered I.V. or I.C.
- Anti-arrhythmics, such as amiodarone, decrease the ventricular irritability that occurs after MI.
    - Given I.V. via bolus, then infusion over 24 hours.

11.6. PERCUTANEOUS CORONARY INTERVENTIONS:

- Mechanical opening of the coronary vessel can be performed during an evolving infarction.
- Percutaneous coronary interventions (PCIs), including percutaneous transluminal coronary angioplasty, coronary stenting, and atherectomy, can be used instead of, or as an adjunct to, thrombolytic therapy
- Should be performed within 30 minutes of initial diagnosis of MI.

Surgical Revascularization

- Emergency CABG surgery can be performed within 6 hours of evolving infarction.
- Benefits of this therapy include definitive treatment of the stenosis and less scar formation on the heart.

11.7. COMPLICATIONS:

- Dysrhythmias
- Sudden cardiac death due to ventricular arrhythmias
- Infarct expansion (thinning and dilation of the necrotic zone)
- Infarct extension (additional heart muscle necrosis occurring after 24 hours of acute infarction)
- Heart failure (with 20% to 35% left ventricle damage)
- Cardiogenic shock
- Reinfarction
- Ischemic cardiomyopathy
- Cardiac rupture
- Papillary muscle rupture
- Ventricular mural thrombus
- Thromboemboli
- Ventricular aneurysm
- Cardiac tamponade
- Pericarditis (2 to 3 days after MI)
- Dissection of coronary arteries during angioplasty
- Psychiatric problems—depression, personality changes

12. NURSING ASSESSMENT:

- Ask patient to describe anginal attacks.
- Obtain a baseline 12-lead ECG.
- Assess patient's and family's knowledge of disease.

- Identify patient's and family's level of anxiety and use of appropriate coping mechanisms.
- Gather information about the patient's cardiac risk factors. Use the patient's age, total cholesterol level, LDL and HDL levels, systolic BP, and smoking status to determine the patient's 10-year risk for development of CHD according to the Framingham Risk Scoring
- Evaluate patient's medical history for such conditions as diabetes, heart failure, previous myocardial infarction (MI), or obstructive lung disease that may influence choice of drug therapy.
- Identify factors that may contribute to noncompliance with prescribed drug therapy.
- Review renal and hepatic studies and complete blood count (CBC).
- Discuss with patient current activity levels. (Effectiveness of antianginal drug therapy is evaluated by patient's ability to attain higher activity levels.)
- Discuss patient's beliefs about modification of risk factors and willingness to change.

Nursing Diagnoses:

- Acute Pain related to an imbalance in oxygen supply and demand
- Decreased Cardiac Output related to reduced preload, afterload, contractility, and heart rate secondary to hemodynamic effects of drug therapy
- Anxiety related to chest pain, uncertain prognosis, and threatening environment

Nursing Interventions:

Relieving Pain

- Determine intensity of patient's angina.
    - Ask patient to compare the pain with other pain experienced in the past and, on a scale of 0 (no pain) to 10 (worst pain), rate current pain.

    - Observe for other signs and symptoms, including diaphoresis, shortness of breath, protective body posture, dusky facial color, and/or changes in level of consciousness (LOC).
- Position patient for comfort; Fowler's position promotes ventilation.
- Administer oxygen if prescribed.
- Obtain BP, apical heart rate, and respiratory rate.
- Obtain a 12-lead ECG as directed.
- Administer antianginal drug(s) as prescribed.
- Report findings to health care providers.
- Monitor for relief of pain, and note duration of anginal episode.
- Take vital signs every 5 to 10 minutes until angina pain subsides.
- Monitor for progression of stable angina to unstable angina: increase in frequency and intensity of pain, pain occurring at rest or at low levels of exertion, pain lasting longer than 5 minutes.
- Determine level of activity that precipitated anginal episode.
- Identify specific activities patient may engage in that are below the level at which anginal pain occurs.
- Reinforce the importance of notifying nursing staff when angina pain is experienced.

Maintaining Cardiac Output

- Carefully monitor the patient's response to drug therapy.
    - Take BP and heart rate in a sitting and a lying position on initiation of long-term therapy (provides baseline data to evaluate for orthostatic hypotension that may occur with drug therapy).
    - Recheck vital signs as indicated by onset of action of drug and at time of drug's peak effect.
    - Note changes in BP of more than 10 mm Hg and changes in heart rate of more than 10 beats/minute.
    - Note patient complaints of headache (especially with use of nitrates) and dizziness (more common with ACE inhibitors).

        - Administer or teach self-administration of analgesics as directed for headache.
        - Encourage supine position to relieve dizziness (usually associated with a decrease in BP; preload is enhanced by this mechanism, thereby increasing BP).
    - Institute continuous ECG monitoring or obtain 12-lead ECG as directed. Interpret rhythm strip every 4 hours for patients on continuous monitoring (beta-adrenergic blockers and calcium channel blockers can cause significant bradycardia and varying degrees of heart block).
    - Evaluate for development of heart failure (beta-adrenergic blockers and some calcium channel blockers decrease contractility, thus increasing the likelihood of heart failure).
        - Obtain daily weight and intake and output.
        - Auscultate lung fields for crackles.
        - Monitor for the presence of edema.
        - Monitor central venous pressure (CVP) if applicable.
        - Assess jugular vein distention.
    - Monitor laboratory tests as indicated (cardiac markers).
- Be sure to remove previous nitrate patch or paste before applying new paste or patch (prevents hypotension) and to reapply on different body site. To decrease nitrate tolerance, transdermal nitroglycerin may be worn only in the daytime hours and taken off at night when physical exertion is decreased.
- Be alert to adverse reaction related to abrupt discontinuation of beta-adrenergic blocker and calcium channel blocker therapy. These drugs must be tapered to prevent a "rebound phenomenon": tachycardia, increase in chest pain, hypertension.
- Discuss use of chromotherapeutic therapy with health care provider (tailoring of anti-anginal drug therapy to the timing of circadian events).
- Report adverse drug effects to health care provider.

Decreasing Anxiety

- Assess patient for signs of hypoperfusion, auscultate heart and lung sounds, obtain a rhythm strip, and administer oxygen as prescribed. Notify the health care provider immediately.
- Document all assessment findings, health care provider notification and response, and interventions and response.
- Explain to patient and family reasons for hospitalization, diagnostic tests, and therapies administered.
- Encourage patient to verbalize fears and concerns about illness through frequent conversations—conveys to patient a willingness to listen.
- Administer medications to relieve patient's anxiety as directed. Sedatives and tranquilizers may be used to prevent attacks precipitated by aggravation, excitement, or tension.
- Explain to patient the importance of anxiety reduction to assist in control of angina. (Anxiety and fear put an increased stress on the heart, requiring the heart to use more oxygen.) Teach relaxation techniques.
- Discuss measures to be taken when an anginal episode occurs. (Preparing patient decreases anxiety and allows patient to accurately describe angina.)
    - Review the questions that will be asked during anginal episodes.
    - Review the interventions that will be employed to relieve anginal attacks.

Patient Education and Health Maintenance

Instruct Patient and Family about CAD

- Review the chambers of the heart and the coronary artery system, using a diagram of the heart.
- Show patient a diagram of a clogged artery; explain how the blockage occurs; point out on the diagram the location of patient's lesions.
- Explain what angina is (a warning sign from the heart that there is not enough blood and oxygen because of the blocked artery or spasm).
- Review specific risk factors that affect CAD development and progression; highlight those risk factors that can be modified and controlled to reduce risk.

- Discuss the signs and symptoms of angina, precipitating factors, and treatment for attacks. Stress to patient the importance of treating angina symptoms at once.
- Distinguish for patient the different signs and symptoms associated with stable angina versus preinfarction angina.
- Give patient and family handouts to review and encourage questions for a later teaching session.

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