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CRITCAL CARE NURSING CARE PLANS

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CARDIOVASCULAR SYSTEM

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Congestive Heart Failure

Heart failure is the inability of the heart to supply blood flow to meet physiologic demands, without utilizing compensatory changes. There may be failure involving one or both sides of the heart, and over time, causes the development of pulmonary and systemic congestion and complications. Congestive heart failure, or CHF, is a common complication after myocardial infarction and can be attributed to one-third of the deaths of patients with MIs. Usually following MI, the heart failure is left-sided since most infarctions involve damage to the left ventricle.

Heart failure can also be classified as acute or chronic. In chronic heart failure, the body experiences a gradual development as the heart becomes unable to pump a sufficient amount of blood to meet the body's demands. Chronic heart failure can become acute without any overt cause.

Often, the patient will have no early symptoms of left-sided heart failure. Symptoms of decreased cardiac output will develop once the heart fails to pump enough blood into the systemic circulation. The pressure in the left ventricle increases, which in turn causes retrograde increases of pressure in the left atrium because of the increased difficulty for blood to enter the atrium from the pulmonary veins. Blood backs up in the lung vasculature, and when the pulmonary capillary pressure is exceeded by the oncotic pressure of the proteins in the plasma fluid (usually > 30 mmHg), the fluid leaks into the interstitial spaces. When this fluid moves into the alveoli, shortness of breath, coughing, and crackles (rales) occur, and the patient progresses into overt pulmonary edema, with the classic sign of coughing up copious amounts of pink frothy sputum.

Right-sided heart failure is usually caused by left-sided heart failure, but can also be caused by pulmonary emboli, pulmonary hypertension, COPD, and the presence of right ventricular infarctions.

The lungs can accept a certain amount of fluid build-up, but eventually, if no intervention is taken, the pressure in the lungs increases to the point whereby the right ventricle cannot eject its blood into the lungs. The right ventricle fails and then the blood in the right atrium cannot drain completely, and thus cannot accept the total amount of blood from the vena cavae. Venous pooling occurs with the impairment of venous blood flow, and eventually the organs become congested with venous blood.

Treatment of heart failure involves attempts to improve contractility of the ventricle by use of positive inotropic drugs, decrease of afterload by the use of nitrates and vasodilators, and in some instances, by use of the IABP, and decrease of preload by the use of diuretics, IV nitroglycerin, and fluid/sodium restrictions.

MEDICAL CARE

Oxygen: to increase available oxygen supply

Morphine: used to induce vasodilation, decrease venous return to the heart, reduce pain and anxiety, and decrease myocardial oxygen consumption

Cardiac glycosides: digitalis (Digoxin, Lanoxin) PO or IV to increase the force and strength of ventricular contractions and to decrease rate of contractions in order to increase cardiac output

Diuretics: furosemide (Lasix) PO or IV, chlorothiazide (Diuril) PO, bumetanide (Bumex) PO or IV to promote excess fluid removal, to decrease edema and pulmonary venous pressure by preventing sodium and water reabsorption

Vasodilators: hydralazine (Apresoline) PO or IV, isosorbide dinitrate (Isordil) SL or PO, prazosin (Minipress) PO, minoxidil (Loniten) PO, diazoxide (Hyperstat) IV, sodium nitroprusside (Nipride) IV, nitroglycerine (Nitrostat, Tridil) PO, SL, IV to relax vascular smooth muscle, decrease preload and afterload, decrease oxygen demand, decrease systemic vascular resistance, and increase venous capacitance

Renin-angiotensin system inhibitors: captopril (Capoten) PO used to inhibit angiotensin converting enzyme to reduce the production of angiotensin II to enable the decrease in vasoconstriction and to reduce afterload

Inotropic agents: dopamine, dobutamine (Dobutrex) IV, amrinone (Inocor) IV used to increase myocardial contractility, without increasing the heart rate, to produce peripheral vasodilation and decrease preload and afterload

Electrolytes: mainly potassium to replace that which is lost during diuretic therapy

Laboratory: electrolyte levels to monitor for imbalances; renal profiles to monitor for kidney function problems; digoxin levels to monitor for toxicity; platelet count to monitor for thrombocytopenia from amrinone

Chest x-ray: shows any enlargement of the heart and pulmonary vein, presence of pulmonary edema or pleural effusion

Electrocardiography: used to monitor for dysrhythmias which may occur as a result of the heart failure or as a result of digitalis toxicity

Echocardiography: used to study structural abnormalities and blood flow through the heart

Intra-aortic balloon pump: decreases the workload on the heart, decreases myocardial oxygen demand, increases coronary perfusion, decreases afterload, decreases preload, improves cardiac output and tissue perfusion

NURSING CARE PLANS

Fluid volume excess

Related to: increased sodium and water retention, decreased organ perfusion, compromised regulatory mechanisms, decreased cardiac output, increased ADH production

Defining characteristics: edema, weight gain, intake greater than output, increased blood pressure, increased heart rate, shortness of breath, dyspnea, orthopnea, crackles (rales), S_3 gallop, oliguria, jugular vein distention, pleural effusion, specific gravity changes, altered electrolyte levels

Outcome Criteria

Blood pressure will be maintained within normal limits and edema will be absent or minimal in all body parts.

Fluid volume will be stabilized with balanced intake and output.

INTERVENTIONS	RATIONALES
Monitor vital signs and hemodynamic readings if available.	Fluid volume excess will cause increases in blood pressure, and CVP and pulmonary artery pressures, and these changes will be reflected from the development of pulmonary congestion and heart failure.
Auscultate lungs for presence of crackles (rales), or other adventitious breath sounds. Observe for presence of cough, increased dyspnea, tachypnea, orthopnea or paroxysmal nocturnal dyspnea.	May indicate pulmonary edema from cardiac decompensation and pulmonary congestion. Pulmonary edema symptoms reflect left-sided heart failure. Right-sided heart failure may have slower onset, but symptoms of dyspnea, orthopnea, and cough are more difficult to reverse.

INTERVENTIONS	RATIONALES
Observe for jugular vein distention and dependent edema. Note presence of generalized body edema (anasarca).	May indicate impending congestive failure and fluid excess. Peripheral edema begins in feet and ankles, or other dependent areas and ascends as failure progresses. Pitting will usually occur only after 10 or more pounds of excess fluid is retained. Anasarca will be seen only with right heart failure or bi-ventricular failure.
Investigate abrupt complaints of dyspnea, air hunger, feeling of impending doom or suffocation.	Excessive fluid build-up can promote other complications such as pulmonary edema or pulmonary embolus and intervention must be immediate.
Determine fluid balance by measuring intake and output, and observing for decreases in output and concentrated urine.	Renal perfusion is impaired with excessive fluid volume, which causes decreased cardiac output leading to sodium and water retention and oliguria.
Weigh daily and notify MD of greater than 2 lb/day increase.	Abrupt changes in weight usually indicate excess fluid.
Provide patient with fluid intake of 2 L/day, unless fluid restriction is warranted.	Fluids may need to be restricted due to cardiac decompensation. Fluids maintain hydration of tissues.
Administer diuretics as ordered (furosemide, hydralazine, spirolactone with hydrochlorothiazide).	Drugs may be necessary to correct fluid overload depending on emergent nature of problem. Diuretics increase urine flow rate and may inhibit reabsorption of sodium and chloride in the renal tubules.
Monitor electrolyte for imbalances. Note increasing lethargy, hypotension, or muscle cramping.	Hypokalemia can occur with the administration of diuretics. Signs of potassium and sodium deficits may occur due to fluid shifts with diuretic therapy.
Place and maintain patient in semi-Fowler's position.	Diuresis may be enhanced by recumbent position due to increased glomerular filtration and decreased production of ADH.
Auscultate bowel sounds and observe for abdominal distention, anorexia, nausea, or constipation. Provide small, easily-digestible meals.	CHF progression can impair gastric motility and intestinal function. Small, frequent meals may enhance digestion and prevent abdominal discomfort.
Measure abdominal girth if warranted.	Progressive right-sided heart failure can cause fluid to shift into the peritoneal space and cause ascites.
Palpate abdomen for liver enlargement; note any right upper quadrant tenderness or pain.	Progressive heart failure can lead to venous congestion, abdominal distention, liver engorgement, and pain. Liver function may be impaired and can impede drug metabolism.
Assist with dialysis or hemofiltration as warranted.	Mechanically removing excess fluid may be performed to rapidly reduce circulating volume in cases refractory to other medical therapeutics.

Instruction, Information, Demonstration

INTERVENTIONS	RATIONALES
Instruct patient regarding dietary restrictions of sodium.	Fluid retention is increased with intake of sodium.
Instruct patient to observe for weight changes and report these to MD.	Weight gain may be first overt sign of fluid excess and should be monitored to prevent complications.
Consult with dietitian.	May be required to ensure adequacy of caloric intake with fluid and sodium restriction requirements.

INTERVENTIONS	RATIONALES
Instruct patient in medications prescribed after discharge, with dose, effect, side effects, contraindications.	Promotes knowledge and compliance with treatment regimen.
Monitor chest x-rays.	Reveal changes in pulmonary status regarding improvement or deterioration.

Discharge or Maintenance Evaluation

- Patient will have no edema or fluid excess.
- Fluid balance will be maintained and blood pressure will be within normal limits of baseline.
- Lung fields will be clear, without adventitious breath sounds, and weight will be stable.
- Patient will be able to accurately verbalize understanding of dietary restrictions and medications.

Decreased cardiac output

Related to: damaged myocardium, decreased contractility, dysrhythmias, conduction defects, alteration in preload, alteration in afterload, vasoconstriction, myocardial ischemia, ventricular hypertrophy, accumulation of blood in lungs or in systemic venous system

Defining characteristics: dependent edema, elevated blood pressure, elevated mean arterial pressure greater than 120 mmHg, elevated systemic vascular resistance greater than 1400 dyne-seconds/cm^5, cardiac output less than 4 L/min or cardiac index less than 2.5 L/min/m^2, tachycardia, cold, pale extremities, absent or decreased peripheral pulses, EKG changes, hypotension, S_3 or S_4 gallops, decreased urinary output, diaphoresis, orthopnea, dyspnea, crackles (rales), frothy blood-tinged sputum, jugular vein distention, edema, chest pain, confusion, restlessness

Outcome Criteria

Vital signs and hemodynamic parameters will be within normal limits for patient, with no dysrhythmias noted.

Patient will be eupneic with no adventitious breath sounds or abnormal heart tones.

INTERVENTIONS	RATIONALES
Determine level of cardiac function and existing cardiac and other conditions.	Additional disease states and complications may place an additional workload on an already compromised heart.
Auscultate apical pulses and monitor heart rate and rhythm. Monitor BP in both arms.	Decreased contractility will be compensated by tachycardia, especially concurrently with heart failure. Blood volume will be lowered if blood pressure is increased resulting in increased afterload. Pulse decreases may be noted in association with toxic levels of digoxin, and peripheral pulses may be hard to accurately determine if perfusion is decreased. Hypotension may occur as a result of ventricular dysfunction and poor perfusion of the myocardium.
Measure cardiac output and cardiac index, and calculate hemodynamic pressures every 4 hours and prn.	Provides measurement of cardiac function and calculated measurements of preload and afterload to facilitate titration of vasoactive drugs and manipulation of hemodynamic pressures.
Monitor EKG for dysrhythmias and treat as indicated.	Conduction abnormalities may occur due to ischemic myocardium affecting the pumping efficiency of the heart.
Observe for development of new S_3 or S_4 gallops.	S_3 gallops are usually associated with congestive heart failure but can be found with mitral regurgitation and left

INTERVENTIONS	RATIONALES
	ventricular overload after MI. S_4 gallops can be associated with myocardial ischemia, ventricular rigidity, pulmonary hypertension, or systemic hypertension, which can decrease cardiac output.
Auscultate for presence of murmurs and/or rubs.	Indicates disturbances of normal blood flow within the heart related to incompetent valves, septal defects, or papillary muscle/chordae tendonae complications post-MI. Presence of a rub with an MI is associated with pericarditis and/or pericardial effusion.
Observe lower extremities for edema, distended neck veins, cold hands and feet, mottling, oliguria.	Reduced venous return to the heart can result in low cardiac output; oliguria results from decreased venous return due to fluid retention.
Position in semi-Fowler's position.	Promotes easier breathing and prevents pooling of blood in the pulmonary vasculature.
Administer cardiac glycosides, nitrates, vasodilators, diuretics, and antihypertensives as ordered.	Used in the treatment of vasoconstriction and to reduce heart rate and contractility, reduces blood pressure by relaxation of venous and arterial smooth muscle which then in turn increases cardiac output and decreases the workload on the heart.
Titrate vasoactive drugs as ordered per MD parameters.	Maintains blood pressure and heart rate at levels to optimize cardiac output function.
Weigh every day.	Weight gain may indicate fluid retention and possible impending congestive failure.
Arrange activities so as to not over-tax patient.	Avoids over-fatiguing patient and decreasing cardiac output further. Balancing rest with activity minimizes energy expenditure and myocardial oxygen demands by maintaining cardiac output.
Avoid Valsalva-type maneuvers with straining, coughing or moving.	Increasing intra-abdominal pressure results in an abrupt decrease in cardiac output by preventing blood from being pumped into the thoracic cavity and thus, less blood being pumped into the heart which then decreases the heart rate. When the pressure is released, there is a sudden overload of blood which then increases preload.
Provide small, easy to digest, meals and restrict caffeine.	Large meals increase the workload on the heart. Caffeine directly stimulates the heart and increases heart rate.
Have emergency equipment and medications available at all times.	Coronary occlusion, lethal dysrhythmias, infarct extensions or intractable pain may precipitate cardiac arrest that requires life support and resuscitation.

Information, Instruction, Demonstration

INTERVENTIONS	RATIONALES
Instruct on medications, dose, effects, side effects, contraindications, and avoidance of over-the-counter drugs without MD approval.	Promotes knowledge and compliance with regimen. Prevents any adverse drug interactions.
Instruct in activity limitations. Demonstrate exercises to be done.	Promotes compliance. Reduces decrease in cardiac output by lessening the workload placed on the heart.
Instruct to report chest pain.	May indicate complications of decreased cardiac output.
Instruct patient/family regarding placement of pulmonary artery catheter, and post-procedure care.	Alleviates fear and promotes knowledge. Pulmonary artery catheter necessary for direct measurement of cardiac output and for obtaining values for other hemodynamic measurements.

INTERVENTIONS	RATIONALES
Assist with insertion and maintainance of pacemaker when needed.	Cardiac pacing may be necessary during the acute phase of MI or may be necessary as a permanent measure if the MI severely damages the conduction system.

Discharge or Maintenance Evaluation

- Patient will have no chest pain or shortness of breath.
- Vital signs and hemodynamic parameters will be within normal limits for age and disease condition.
- Minimal activity will be tolerated without fatigue or dyspnea.
- Urinary output will be adequate.
- Cardiac output will be adequate to ensure adequate perfusion of all body systems.

Impaired gas exchange

Related to: ventilation/perfusion imbalance caused from excess fluid in alveoli and reduction of air exchange area in lung fields, fluid collection shifts into the interstitial space

Defining characteristics: confusion, restlessness, irritability, hypoxia, hypercapnea, dyspnea, orthopnea, abnormal ABGs, abnormal oxygen saturation

Outcome Criteria

Patient will have adequate oxygenation with respiratory status within limits of normal based on age and other conditions, and ABGs will be within normal limits.

INTERVENTIONS	RATIONALES
Monitor respiratory status for rate, regularity, depth, ease of effort at rest or with exertion, inspiratory/expiratory ratio.	Changes in respiratory pattern or patency of airway may result in gas exchange imbalances.
Observe for presence of cyanosis and mottling; monitor oximetry for oxygen saturation; monitor ABGs for ventilation/perfusion problems.	Cyanosis results from decreases in oxygenated hemoglobin in the blood and this reduction leads to hypoxia. Reading of 90% on pulse oximeter correlates with pO_2 of 60.
Monitor for mental status changes, deterioration in level of consciousness, restlessness, irritability, easy fatigueability.	Hypoxia affects all body systems and mental status changes can result from decreased oxygen to brain tissues.
Position in semi- or high-Fowler's position.	Promotes breathing and lung expansion to enhance gas distribution.
Administer oxygen via nasal cannula at 2-3 L/min, or other delivery systems.	Maintains adequate oxygenation without depression of respiratory drive. CO_2 may be retained with higher flow rates when used in patients with COPD.
Assist with placement of ETT and placement on mechanical ventilation.	Mechanical ventilation may be required if respiratory failure is progressive and adequate oxygen levels cannot be maintained by other delivery systems.

Instruction, Information, Demonstration

INTERVENTIONS	RATIONALES
Instruct in breathing exercises as warranted.	Assists to restore function to diaphragm, decreases work of breathing, and improves gas exchange.
Assess for nausea and vomiting.	May indicate effects of hypoxia on gastrointestinal system.
Avoid activities that promotes dyspnea or fatigue. Allow for periods of rest between activities.	Activity increases oxygen consumption and demand, and can impair breathing pattern.

INTERVENTIONS	RATIONALES
Instruct in safety concerns with oxygen use.	Promotes safety with oxygen and provides knowledge.
Instruct patient/family in need for placement on mechanical ventilation, what to expect, what benefits are to be received, what potential problems may be encountered.	Promotes knowledge and decreases anxiety and fear of the unknown.

Discharge or Maintenance Evaluation

- Patient will exhibit no ventilation/perfusion imbalances.
- Patient will be eupneic with no adventitious breath sounds.
- ABGs will be within acceptible ranges for patient with adequate oxygenation of all tissues.
- Patient will be able to verbalize/demonstrate the correct use of oxygen.

Risk for impaired skin integrity

Related to: bed rest, decreased tissue perfusion, edema, immobility, decreased peripheral perfusion, shearing forces or pressure, secretions, excretions, altered sensation, skeletal prominence, poor skin turgor, altered metabolic rate

Defining characteristics: disruption of skin surface, pressure areas, reddened areas, blanched areas, mottling, warmth, firmness to area of skin, irritated tissues, excoriation of skin, maceration of skin, lacerations of skin, pruritis, dermatitis

Outcome Criteria

Patient will have and maintain skin integrity.

INTERVENTIONS	RATIONALES
Monitor mobility status and patient's ability to move self.	Immobility is the primary cause of skin breakdown.
Inspect all skin surfaces, especially bony prominences, for skin breakdown, altered circulation to areas, or presence of edema.	Skin is at risk because of decreased tissue perfusion, immobility, decreased peripheral perfusion, and possible nutritional alterations.
Provide skin care to blanched or reddened areas.	Stimulates blood flow and decreases tissue hypoxia. Excess dryness or moistness of skin can promote breakdown.
Provide eggcrate mattress, alternating pressure mattress, sheepskin, elbow protectors, heel protectors, etc.	These items can reduce pressure on skin and may improve circulation.
Reposition frequently, at least every 2 hours. Assist with ROM exercises. Maintain body alignment. Raise head of bed no higher than 30 degrees.	Improves circulation by reduction of time pressure is on any one area. Proper body alignment prevents contractures. Elevations higher than this may promote pressure and friction from sliding down, and shearing force may result in breakdown of skin.
Avoid subcutaneous or IM injections when possible.	Edema and tissue hypoxia impede circulation which can cause decreased absorption of medication and can predispose patient to tissue breakdown and development of abscess/infection.

Instruction, Information, Demonstration

INTERVENTIONS	RATIONALES
Instruct on safety precautions in bed—avoiding bumping against rails, falls, etc.	May cause breaks in skin integrity.
Instruct on hazards of immobility; avoid lying or sitting in one position for prolonged time.	Bedrest promotes pressure to skin and tissues.
Instruct on the use of lotions and oil to apply to skin.	Prevents skin dryness and chance of tissue breakdown.

Discharge or Maintenance Evaluation

- Patient will have intact skin, free of redness, irritation, rashes, or bruising.
- Patient will be able to verbally relate measures to reduce chance of tissue injury.

Anxiety
[See MI]

Related to: change in health status, fear of death, threat to body image, threat to role functioning, pain

Defining characteristics: restlessness, insomnia, anorexia, increased respirations, increased heart rate, increased blood pressure, difficulty concentrating, dry mouth, poor eye contact, decreased energy, irritability, crying, feelings of helplessness

Knowledge deficit
[See MI]

Related to: lack of understanding, lack of understanding of medical condition, lack of recall

Defining characteristics: questions regarding problems, inadequate follow-up on instructions given, misconceptions, lack of improvement of previous regimen, development of preventable complications

LEFT-SIDED HEART FAILURE

Burden placed on cardiovascular system by any of the following: hypertension, myocardial infarction, valvular heart disease, dysrhythmias, tachy/bradycardia, cardiomyopathy, cardiac tamponade, constrictive pericarditis, aortic stenosis, mitral insufficiency, or anemia ↓

Decreased cardiac output → Sympathetic nervous system stimulated
Fatigue, weakness
- Heart rate increases
- Arteriolar vasoconstriction
- Myocardial contractility increases
- Venous tone increases
- Venous and ventricular filling pressures increase

↓

Decreased effective arterial blood volume

↓

Renal compensatory changes occur
- Renal blood flow decreases
- GFR decreases
- Cortical blood flow decreases
- Renin and angiotensin increase
- Aldosterone increases
- ADH increases

↓

Sodium reabsorption and free water clearance decreases
Effective blood volume increases

↓

Accumulation of blood in lungs

↓

LV cannot pump blood from lungs into systemic circulation

↓

LV pressures and LV volume increase

↓

LA pressures and volume increase

↓

Fluid backs up into pulmonary vasculature = pulmonary congestion
- Paroxysmal nocturnal dyspnea, orthopnea

↓

Pulmonary hypertension, PA diastolic and PCWP pressure increases

↓

Fluid leaks into interstitial and alveoli

↓

Pulmonary edema
- Cough
- Frothy, blood-tinged sputum
- Rales (crackles)

↓

Decreased oxygen in blood
Impaired gas exchange and hypoxia
- Tachypnea
- Cyanosis
- Confusion
- Restlessness
- Pulmonary effusion
- Pulsus alternans

RIGHT-SIDED HEART FAILURE

Burden placed on the cardiovascular system by any of the following: left-sided heart failure, pulmonary hypertension, COPD, cor pulmonale, pulmonary embolus, anemia, thyrotoxicosis, pulmonary stenosis, or mitral stenosis.

↓

Accumulation of blood in systemic venous system
Lung pressure increases
Pressure in pulmonary vasculature increases

↓

Increased right atrial and ventricular pressures
Increased peripheral venous pressure

↓

Right heart cannot pump blood into pulmonary system
Right-sided heart failure
Bounding pulses
Dysrhythmias
S_3 or S_4 gallop

↓

Venous return decreases
Organs become congested with blood
Peripheral dependent edema occurs

↓

Congestion of portal circulation
Hepatomegaly, hepatojugular reflux
JVD, weight gain
Anorexia
Ascites, abdominal pain, anorexia, nausea
Fatigue, cyanosis

↓

Advanced heart failure

↓

Air hunger, gasping
Tachycardia
Crackles, frothy blood-tinged sputum
Skin cool and moist
Cyanotic lips, nailbeds
Confusion, stupor
Enlarged RA and RV
Tricuspid murmur

Myocardial Infarction (MI)

Myocardial infarction (MI) is a critical emergency that requires timely management to save heart muscle and limit damage that may evolve over several hours. Blood flow is abruptly decreased or stopped through the coronary arteries and results in ischemia and necrosis to the myocardium if not treated. Many people die prior to receiving medical care due to the denial that anything may be wrong and postponement of seeking medical care. Cardiac dysrhythmias, mainly ventricular fibrillation, is usually the cause of death in these individuals. An MI is diagnosed based on type of chest pain, electrocardiographic changes, and increase of cardiac enzymes, such as CK, SGOT, and LDH. Precordial pain is similar to but usually more intense and prolonged than anginal pain, and in the instance of MI, the chest pain is usually constant and not relieved with nitroglycerin or rest.

Atherosclerosis of the arteries is usually the most common finding in patients. Atherosclerosis and arteriosclerosis are used interchangeably when discussing the fatty plaques that adhere to the inner layer of the arteries. The continuous build-up of these plaques, as well as the potential for hemorrhage at the intimal layer may result in alterations of the blood flow through the coronary arteries and abnormalities in platelet aggregation may contribute to changes in coronary perfusion. Infarction may occur without coronary artery disease or occlusion, and if the patient has developed an adequate collateral circulation, coronary occlusion may occur without infarction.

MI is usually a disease involving the left ventricle but the damage may extend to other areas, such as the atria or right ventricle. A right ventricular myocardial infarction usually has high right ventricular filling pressures and often has severe tricuspid regurgitation. Transmural infarcts involve the entire thickness of the myocardium and are characterized by Q waves on the electrocardiogram. Nontransmural infarcts are characterized by S-T segment and T wave changes. Subendocardial infarcts usually involve the inner portion of the myocardium where wall tension is highest and the blood flow is most vulnerable to circulatory problems. Occlusion of the right coronary artery will result in an inferior infarction that may also include posterior portions of the heart. Occlusion of the left main artery, known as "the widow maker," usually results in death due to the extensive damage. Occlusion of the left anterior descending artery results in an anterior infarction and may include some inferior parts of the heart, and occlusion of the circumflex artery results in a lateral infarction.

Precipitating factors that preclude MIs include heredity, age, gender, presence of hypertension, presence of diabetes mellitus, cigarette smoking, hyperlipidemia, obesity, sedentary lifestyles, and stress.

The main goals in treating myocardial infarction are to increase blood flow to the coronary arteries and thus decrease infarction size, increase oxygen supply and decrease oxygen demand to prevent myocardial death or injury, and control or correct dysrhythmias.

MEDICAL CARE

Oxygen: to increase available oxygen supply

Analgesics: morphine is the drug of choice, given in incremental doses IV every 5 minutes as needed; IM injections are avoided because they can raise the enzyme levels and do not act as quickly

Thrombolytic agents: Streptokinase, Urokinase, or Tissue Plasminogen Activator (tPa) given either intracoronary or intravenously to activate the body's own fibrinolytic system to dissolve the clot and resume coronary blood perfusion

Cardiac glycosides: digitalis to increase force and strength of ventricular contractions and to decrease the conduction and rate of contractions in order to increase cardiac output; usually not used in the acute phase

Diuretics: furosemide (Lasix) to promote excess fluid removal, to decrease edema and pulmonary venous pressure by preventing sodium and water reabsorption

Vasodilators: hydralazine (Apresoline), nifedipine (Procardia, Adalat), nitroglycerin (Nitropaste, Nitrodur, Nitrostat, Tridil, Nitroglycerine), prazosin (Minipres), captopril (Capoten)—used to relax venous and/or arterial smooth muscle to decrease preload, decrease afterload, and decrease oxygen demand

Beta-adrenergic blockers: used to decrease blood pressure, decrease elevated plasma renins, and with non-selective blockers, may do so without related reflex tachycardias; used to treat ventricular dysrhythmias and for the prophylaxis of angina

Aspirin: used to decrease platelet aggregation and helps with vasodilation of peripheral vessels

Thrombolytics: used in the treatment of acute MI; acts by activating mechanisms for conversion of plasminogen to plasmin which is able to dissolve the clot; commonly used are streptokinase, urokinase, alteplase, or anistreplase

Heparin: used with thrombolytic protocols, and in the treatment of MI; prevents conversion of fibrinogen to fibrin and prothrombin to thrombin by its action on antithrombin III

Laboratory: leukocyte count, sed rate and blood glucose may be elevated; creatinine phosphokinase (CK, CPK) will normally increase within 4-6 hours, peak between 12-24 hours, and last 2-3 days but should not be used as sole indicator due to possibility of elevation with other problems such as surgery or trauma; lactate dehydrogenase (LDH) will normally increase within 8-12 hours, peak between 2-4 days, and last 10-14 days but should not be used as sole indicator due to possibility of elevation with other problems such as liver failure; serum glutamic oxaloacetic transaminase (SGOT) is occasionally used as an infarct indicator; isoenzymes of CPK are very specific with CPK-MB most specific for MI, and levels will not rise with transient chest pain or in surgical procedures; a definitive level for CPK-MB is greater than or equal to 4% of the total CPK; LDH isoenzymes, specifically LDH1 is more specific for MI; if the total LDH is elevated and LDH1 is most predominant, MI is confirmed; both CPK-MB and LDH1 will return to normal 72-96 hours after elevation

Chest x-ray: shows any enlargement of the heart and pulmonary vein, presence of pulmonary edema or pleural effusion

Electrocardiography: shows indicative changes associated with sites of acute infarcts using Q waves, S-T segment elevation, and T wave inversion. Also reveals changes with atrial and ventricular enlargement, rhythm and conduction abnormalities, ischemia, electrolyte abnormalities, drug toxicity, and presence of dysrhythmias

Echocardiography: used to study structural abnormalities and blood flow through the heart; M-mode echocardiography measures structures with a single ultrasonic beam that provides a narrow view of the heart; two-dimensional (2D) echocardiography shows a two-dimensional and

wider look at the heart that is more useful in diagnosing right ventricular infarcts; documents increased right ventricular size, performance and segmental wall motion abnormalities, and blood flow through the heart

Nuclear cardiologic testing: MUGA (multiple gated acquisition study) provides information that approximates ejection fractions and the analysis of the ventricular wall motion; 99mTc (Technetium-99 pyrophosphate scan) shows infarcted areas as increased levels of radioactivity, or "hot spots" that appear 12-36 hours after infarct and remain for 4-7 days; PET (positron emission tomography) allows measurement of myocardial blood flow, fatty acid and glucose metabolism, and blood volume; thallium scans can determine size and location of damage as a "cold spot"

Magnetic resonance imaging (MRI): provides a three-dimensional view that can detect changes in tissues before structural damage is done and is safe for pregnant women and children

Cardiac catheterization: used to assess pathophysiology of the patient's cardiovascular disorder, to provide left ventricular function information, to allow for measurement of heart pressures and cardiac output, to evaluate stenotic lesions, and to measure blood gas content

Intra-aortic balloon pump (IABP): decreases the workload on the heart, decreases myocardial oxygen demand, increases coronary perfusion, decreases afterload, decreases preload, and helps to limit infarct size if quickly initiated, improves cardiac output and tissue perfusion; used in cardiogenic shock, for support post cardiac surgery, intractable chest pain, and in cardiac catheterizations or other cardiovascular procedures of high-risk patients

Ventricular assist device (VAD): used on either or both ventricles to provide total support to the heart and circulation in order to allow recovery to the heart; usually indicated in patients who are awaiting cardiac transplantation or in those patients with cardiogenic shock and ventricular failure; may be used in conjunction with IABP

Pacemakers: either temporary or permanent, used in anticipation of lethal dysrhythmias and/or conduction problems

Surgery: coronary artery bypass grafting to reroute the coronary blood flow around the diseased vessel to enable coronary perfusion

NURSING CARE PLANS

Alteration in comfort

Related to: chest pain due to decreased blood flow to myocardium, myocardial ischemia or infarct, post-procedure discomfort, chest wall pain post-surgery, pericarditis

Defining characteristics: chest pain with or without radiation, facial grimacing, clutching of hands or chest, restlessness, diaphoresis, changes in pulse and blood pressure, dyspnea, dizziness

Outcome Criteria

Chest pain will be relieved or controlled to patient's satisfaction.

INTERVENTIONS	RATIONALES
Evaluate chest pain as to type, location, severity, relief, change with activity or rest, other symptoms concurrently noted, such as pallor, diaphoresis, radiation of pain, nausea, vomiting, shortness of breath, and vital sign changes.	Variations may occur with patients regarding specific complaints and behavior. Most MI patients look acutely ill and can only focus on their pain. Respirations may be increased as a result of anxiety and pain. Heart rate

INTERVENTIONS	RATIONALES
	may increase due to increased catecholamines, stress, and pain, which can also increase blood pressure.
Obtain description of intensity using 0-10 scale, with 0 being no pain and 10 being the worst pain experienced.	Pain is a subjective experience and personal to that patient. Intensity scales are useful to gauge improvement or deterioration as perceived by the patient.
Obtain history (when possible) of previous cardiac pain and familial history of cardiac problems.	This provides information that may help to differentiate current pain from previous problems, as well as identify new problems and complications.
Administer oxygen by nasal cannula or mask as indicated.	Supplemental oxygen can increase the available oxygen and can relieve pain associated with myocardial ischemia.
Administer analgesic as ordered, such as morphine sulfate, meperidine (Demerol), or Dilaudid IV.	Morphine is the drug of choice to control MI pain, but other analgesics may be used to reduce pain and reduce the workload on the heart. IM injections should be avoided because they can alter cardiac enzymes and are not absorbed well in tissue that is non- or under-perfused.
Administer beta-blockers as ordered (such as atenolol, pindolol, and propranolol).	These drugs block sympathetic stimulation, reduce heart rate and systolic blood pressure, and thus lowers the myocardial oxygen demand. Beta-blockers should not be given in severely impaired contractility states due to the negative inotropic properties.
Administer calcium-channel blockers as ordered (such as verapamil, diltiazem, or nifedipine).	These drugs can increase coronary blood flow and collateral circulation, reduce preload and myocardial oxygen demands, which can decrease pain due to ischemia.
Maintain bedrest during pain, with position of comfort; nurse to stay with patient during pain.	Reduces oxygen consumption, and demand; alleviates fear and provides caring atmosphere.
Maintain relaxing environment to promote calmness.	Reduces competing stimuli and reduces anxiety.

Information, Instruction, Demonstration

INTERVENTIONS	RATIONALES
Instruct to notify nurse immediately of any chest pain.	Delay in notification can delay pain relief and may require increased amounts of medication in order to finally achieve relief. Pain can cause further damage to an already-injured myocardium, and may signal extension of MI, spasm, or other complication.
Instruct in relaxation techniques, deep breathing, guided imagery, visualization, etc.	Helps to decrease pain and anxiety and provides distraction from pain.
Instruct in nitroglycerin SL administration after hospitalization; 1 q5 minutes up to 3 times, and if pain is unrelieved, patient should seek emergency medical care.	Knowledge facilitates cooperation and compliance with medical regimen. Pain unrelieved with NTG may be indicative of MI.
Instruct in activity alterations and limitations.	Decreases myocardial oxygen demand and workload on the heart.
Instruct in medication effects, side effects, contraindications, and symptoms to report.	Promotes knowledge and compliance with therapeutic regimen. Alleviates fear of unknown.

Discharge or Maintenance Evaluation

- Patient will report pain being absent or controlled with medication administration.
- Medication will be administered prior to pain becoming severe.
- Patient will be able to recall effects, side effects, and contraindications of medications accurately.
- Activity will be modified in such a way as to prevent onset of chest pain.

Altered tissue perfusion: cardiopulmonary, cerebral, peripheral

Related to: tissue ischemia, reduction or interruption of blood flow, vasoconstriction, hypovolemia, shunting, depressed ventricular function, dysrhythmias, conduction defects

Defining characteristics: abnormal hemodynamic readings, dysrhythmias, decreased peripheral pulses, cyanosis, decreased blood pressure, shortness of breath, dyspnea, cold and clammy skin, decreased mental alertness, changes in mental status, oliguria, anuria, sluggish capillary refill, abnormal electrolyte and digoxin levels, hypoxia, ABG changes, chest pain, ventilation perfusion imbalances, changes in peripheral resistance, impaired oxygenation of myocardium, EKG changes (S-T segment, T wave, U wave), LV enlargement, palpitations

Outcome Criteria

Blood flow and perfusion to vital organs will be preserved and circulatory function will be maximized.

Patient will be free of dysrhythmias.

Hemodynamic parameters will be within normal limits.

INTERVENTIONS	RATIONALES
Monitor vital signs. Obtain hemodynamic values, noting deviations from baseline values.	Provides information about the hemodynamics of the patient and facilitates early intervention for problems.
Monitor EKG for disturbances in conduction and for dysrhythmias and treat as indicated.	Decreased cardiac perfusion may instigate conduction abnormalities. Ventricular fibrillation is the most common dysrhythmia following MI. Reperfusion dysrhythmias may occur after the administration of thrombolytic therapy.
Administer oxygen by nasal cannula as ordered, with rate dependent on disease process and condition.	Provides oxygen necessary for tissues and organ perfusion.
Auscultate lungs for crackles (rales), rhonchi, or wheezes.	May indicate fluid overload that will further decrease tissue perfusion.
Auscultate heart sounds for S_3 or S_4 gallop, new murmurs, presence of jugular vein distention, or hepatojugular reflex.	May indicate impending or present heart failure.
Monitor oxygen status with ABGs, S_vO_2 monitoring, or with pulse oximetry.	Provides information about the oxygenation status of the patient. Continuous monitoring of saturation levels provide an instant analysis of how activity affects oxygenation and perfusion for the patient.
Monitor for changes in respiratory status, increased work of breathing, dyspnea, etc.	Decreased cardiac perfusion may result in pump failure and precipitate respiratory distress and failure.
Determine the presence and character of peripheral pulses, capillary refill time, skin color and temperature.	May indicate decreased perfusion resulting from impaired coronary blood flow.

INTERVENTIONS	RATIONALES
Discourage any non-essential activity.	Ambulation, exercise, transfers, and Valsalva-type maneuvers can increase blood pressure and decrease tissue perfusion.
Assist patient with planned, graduated levels of activity.	Allows for balance between rest and activity to decrease myocardial workload and oxygen demand. Gradual increases help to increase patient tolerance to activity without pain.
Titrate vasoactive drugs as ordered.	Maintain blood pressure and heart rate at parameters set by MD for optimal perfusion with minimal workload on heart.
Administer thrombolytic drugs as ordered.	Drugs lyse the clot that may be occluding the coronary artery and promote restoration of oxygen and blood flow to increase perfusion.
Auscultate for bowel sounds and monitor for complaints of nausea, vomiting, anorexia, abdominal distention, abdominal pain, or constipation.	Decreased perfusion to mesentery may result in loss or change in peristalsis, resulting in GI use of analgesics, and change in surroundings may contribute to changes in GI status.
Monitor urine output for adequate amounts, character of urine, presence of sediment, and specific gravity.	Decreased perfusion to renal arteries may result in oliguria. Dehydration secondary to nausea and vomiting may affect renal perfusion.
Monitor labwork such as renal or liver profiles.	May indicate organ dysfunction and decreased perfusion.

Information, Instruction, Demonstration

INTERVENTIONS	RATIONALES
Instruct on medications, dosage, effects, side effects, and contraindications.	Promotes compliance with regimen and knowledge base.
Instruct to refrain from smoking.	Smoking causes vasoconstriction with can decrease perfusion.
Instruct in dietary requirements, menu planning, sodium restrictions, foods to avoid.	Reduction of high-cholesterol and sodium foods will help to control atherosclerosis, hyperlipidemia, fluid retention, and the effects on coronary blood flow.

Discharge or Maintenance Evaluation

- Lung fields will be clear and free of adventitious breath sounds.
- Extremities will be warm and pink, with easily palpable pulses.
- Vital signs and hemodynamic parameters will be within normal limits for patient.
- Oxygenation will be optimal as evidenced by pulse oximetry greater than 90%, S_vO_2 greater than 75%, or normal ABGs.
- Patient will be free of chest pain and shortness of breath.
- Patient will be able to verbalize information accurately regarding medications, diet and activity limitations.

Decreased cardiac output

Related to: damaged myocardium, decreased contractility, dysrhythmias, conduction defects, alteration in preload, alteration in afterload, vasoconstriction, myocardial ischemia, ventricular hypertrophy