

## Cardiovascular System

### Learning Unit 2: Coronary Artery Disease and Acute Coronary Syndrome, Heart Failure, and Cardiogenic Shock

#### Coronary Artery Disease and Acute Coronary Syndrome

Coronary Artery Disease and Acute Coronary Syndrome develop as a result of atherosclerosis. Atherosclerosis begins as a soft deposit of fat that hardens as it ages. With coronary artery disease and acute coronary syndrome, these deposits are found in the coronary arteries. They lead to a decreased oxygen supply to the heart tissue which can cause permanent damage. Cardiovascular disease is the major cause of death in the United States.

Atherosclerosis is characterized by a focal deposit of cholesterol and lipid within the intimal wall of the artery. The endothelial lining is altered from inflammation and injury. There are three developmental stages of development of the deposits. The first stage is the fatty streak stage. This is the earliest stage of lesion development. The fatty streaks are lipid filled smooth muscle cells. These streaks have been identified in persons as young as 15. They are potentially reversible with lowering the LDL cholesterol levels. The second stage is the fibrous plaque stage. There are now progressive changes in the arterial wall. The fatty streak is covered by collagen that forms a fibrous plaque that looks gray or white in color. The vessel lumen is now starting to narrow. This stage of lesion has been seen in persons as young as 30. The final developmental stage is the complicated lesion. Continued inflammation in the area results in plaque instability. It can ulcerate and rupture. The surface of the plaque is rough and platelets accumulate in the area. The narrowing of the artery increases and the lesion may completely block the lumen.

The presence of chronic ischemia contributes to the development of collateral circulation. Collateral circulation is the term used to describe the growth and development of branches off the main coronary arteries. This is a protective measure that occurs over time to try to maintain oxygenation to the heart tissue.

To understand how we are going to treat coronary artery disease, we must first look at the risk factors that are present that contribute to the development of the condition. Risk factors can be classified as nonmodifiable and modifiable. Nonmodifiable risk factors are those we cannot change. They include age, gender, ethnicity, family history, and genetic predisposition. Some facts related to these risk factors include that white, middle age men have the highest risk. After the age 65, the risk for white men and women equalize. African American women have a higher risk than white women.

Modifiable risk factors include elevated serum lipids, hypertension, tobacco use, physical inactivity, obesity, diabetes, metabolic syndrome, psychological states, and elevated homocystine levels. It is recommended that total cholesterol levels be <200 and triglyceride levels be <150. Blood pressure should be kept below 140/90. An elevated blood pressure actually increases the development of plaque formation. Tobacco use increases ones risk two to six times.

We need to know about risk factors so we can identify those at risk and initiate interventions to help reduce the risk of coronary artery disease. Since we cannot change the nonmodifiable risk factors, our interventions will be aimed at the modifiable ones. Health-promoting behaviors include becoming physically fit and modifying the diet. The FITT formula should be utilized to ensure physical fitness. FITT stands for frequency, intensity, type, and time. It is recommended that the individual exercise for 30 minutes for  $\geq 5$  days/week. The exercise should be aerobic, it should cause the individual to sweat, and it should increase the heart rate by 30 to 50 beats per minute. Exercise contributes to weight reduction, a decrease of  $\geq 10\%$  in systolic BP, and also can increase the HDL cholesterol level. Two web sites that may be helpful in designing an exercise program are [www.justmove.org](http://www.justmove.org) and [www.s2mw.com/choosetomove](http://www.s2mw.com/choosetomove).

Dietary changes are also important. The diet plan should decrease the intake of saturated fats and cholesterol and increase the intake of complex carbohydrates. Fat intake should be 30% or less of the total calories consumed and should come from monounsaturated fats from nuts and nut oils. Omega three fatty acids should be consumed on a regular basis (at least 2 times a week). These acids come from fatty fish such as salmon and tuna. The cholesterol level should be evaluated prior to diet changes. If there are no changes in the cholesterol level after 6 weeks, cholesterol lowering medications should be added to the regimen.

Cholesterol lowering agents include drugs that restrict lipoprotein production like the statins and niacin, drugs that increase lipoprotein removal like the bile acid sequestrants, and drugs that decrease the absorption of cholesterol like Zetia.

Other medications will also be prescribed for the individual at risk. They include low dose aspirin therapy and Plavix.

Coronary artery disease manifests itself as chronic angina and acute coronary syndrome. Let's look at both of these conditions. Angina occurs when there is reversible (temporary) myocardial ischemia. This occurs any time  $O_2$  demand  $>$   $O_2$  supply. The primary reason for insufficient blood flow is narrowing of coronary arteries by atherosclerosis. For ischemia to occur, the artery is usually 75% or more stenosed. Chronic stable angina is intermittent chest pain that occurs over a long period with the same pattern of onset, duration, and intensity of symptoms.

Some other types of angina include silent angina (they have no symptoms) associated with diabetes and hypertension, nocturnal angina which occurs only at night, angina decubitus which occurs only when lying down, and Prinzmetal's angina which is caused from spasms of the coronary arteries in the absence of coronary artery disease.

The classic manifestation of chronic stable angina is chest pain. The pain lasts 3 to 5 minutes and subsides when the precipitating factor is relieved or with the administration of sublingual nitroglycerin. Precipitating factors can include physical exertion, temperature extremes, strong emotions, heavy (large) meals, smoking, sex, or stimulants. Other manifestations that may occur are shortness of breath, diaphoresis, and anxiety.

Drug therapy for chronic stable angina is aimed at decreasing oxygen demand and/or increasing oxygen supply. Drugs utilized can include nitrates, beta adrenergic blockers, calcium channel blockers, and angiotensin converting enzyme inhibitors. Nitrates are usually one of the first drugs implemented. Nitrates work by dilating peripheral blood vessels and decreasing the blood systemic vascular resistance which decreases the workload of the heart, reducing the myocardial oxygen demand. They also dilate the coronary arteries and collateral vessels, increasing the blood flow to the heart tissue.

Nitrates are available in both short acting and long acting forms. Short acting nitrates are prescribed for acute angina attacks and the long acting nitrates are utilized to prevent angina attacks. The most common type of short acting nitrate is the sublingual nitroglycerin tablet. It works in 3 minutes and has a duration of 30 to 60 minutes. Long acting nitrates come in pill form, ointment form, and transdermal controlled release forms.

Beta adrenergic blockers decrease the heart rate, decrease the blood pressure, and decrease the systemic vascular resistance. This decreases the myocardial oxygen demand. The most common beta blocker that is prescribed is metoprolol. ACE inhibitors are indicated if there are problems with congestive heart failure associated with the angina.

Calcium channel blockers are utilized if beta blockers are poorly tolerated, contraindicated, or do not control symptoms. They are also the drug of choice for Prinzmetal's angina. They work by causing coronary and systemic vasodilation which decreases the systemic vascular resistance. They also decrease myocardial contractility. Common calcium channel blockers include Norvasc, Plendil, Cardizem, and Cardene.

Diagnostic studies for chronic stable angina include a health history and physical exam, various lab studies, a 12 lead ECG, a chest x-ray, an echocardiogram, and an exercise stress test. A diagnostic and therapeutic test is the cardiac catheterization, also referred to as a coronary arteriogram or angiogram. This involves inserting a catheter through either the femoral artery in the groin or the brachial artery in the arm and threading it up to the coronary arteries. A radio opaque dye is injected and the coronary arteries are evaluated for narrowed or blocked areas from atherosclerosis. If narrowed areas are identified, a percutaneous coronary intervention can be done. This is normally a balloon angioplasty with stent placement.

Nursing interventions for chronic stable angina involves patient teaching. At the health promotion level this is teaching on risk factor modification. At the ambulatory and home care level this includes precipitating factors and how to avoid them, risk factor modification, and medications. Acute interventions are varied. During an acute anginal attack, oxygen should be applied, nitrates given, patient assessment done, and continued monitoring for the effectiveness of treatment.

Nursing interventions for a patient undergoing a cardiac catheterization includes assessing the patient for allergies prior to the procedure. If allergies to iodine or shellfish are present the physician should be notified and a special contrast material will be needed. Teaching on the procedure needs to be completed. The patient may feel a hot feeling when the dye is injected. They also may feel palpitations. Following the procedure the patient is on bedrest for 4-6 hours. The access site needs to be monitored

frequently for bleeding. Peripheral pulses need to be frequently monitored. The patient must also be frequently assessed for pain. If pain occurs it usually indicates restenosis of the vessel and they may need to have the catheterization repeated.

Acute Coronary Syndrome is when the heart ischemia is prolonged and not immediately reversible. ACS encompasses both unstable angina (UA), non-ST-segment-elevation myocardial infarction (NSTEMI), and ST-segment-elevation myocardial infarction (STEMI). The stable plaque that was seen with chronic angina has now deteriorated, allowing platelets to adhere to the plaque and for thrombus formation. A partial occlusion of the coronary artery results in UA or NSTEMI, while a total occlusion of the coronary artery results in STEMI. Unstable angina is angina that is new in onset, occurs at rest, or has a worsening pattern. It is a medical emergency as there is an increased risk of myocardial infarction with unstable angina.

A myocardial infarction is the result of sustained ischemia (> 20 minutes) that causes irreversible myocardial cell death and necrosis. Necrosis of the entire thickness of the myocardium takes 4 to 6 hours. As the cells die, enzymes are released into the blood stream that can be measured with lab tests. Within 24 hours, leukocytes infiltrate the area. Proteolytic enzymes from the neutrophils and macrophages remove all of the necrotic tissue by the second or third day, and scar tissue starts to replace the necrotic tissue. By six weeks after the myocardial infarction, the area is said to be healed, but it is less compliant and does not function as well. Ventricular remodeling occurs to compensate for the area of damage. The degree of altered function depends on the area of the heart involved and the size of the infarct.

Clinical manifestations of an acute myocardial infarction include chest pain. The total occlusion of the artery causes the heart cells to change to an anaerobic metabolism that produces lactic acid. The lactic acid production leads to severe, immobilizing chest pain that is not relieved by rest, position change, or nitrate administration. It is often described as a heaviness, constriction, tightness, burning, pressure, or crushing type pain. It may be substernal, retrosternal, or epigastric in location. It also may radiate.

The sympathetic nervous system is stimulated with a myocardial infarction. This results in the release of glycogen, diaphoresis, and vasoconstriction of the peripheral blood vessels. The skin becomes ashen, clammy, and cool to the touch. Initially the heart rate and blood pressure will increase, and then the blood pressure will fall secondary to decreased cardiac output. Crackles may develop in the lungs. JVD and abnormal heart sounds may develop. Nausea and vomiting may occur from the severe pain or from stimulation of the vomiting center. A fever will also develop. It is a normal response from the inflammatory process caused by cell death. It may rise as high as 102 degrees.

The most common complication from a MI is cardiac dysrhythmia. Dysrhythmia occurs in 80% of all MI patients and is the most common cause of prehospital death. Other complications include heart failure, cardiogenic shock, papillary muscle dysfunction, ventricular aneurysm, acute pericarditis, and Dressler syndrome.

Diagnostic studies for the patient with a MI include a 12 lead ECG, serum cardiac markers and coronary angiography. An echocardiogram and exercise stress testing may be done during hospitalization to

determine cardiac function following the MI. One of the first diagnostic tests that will be done is the 12 lead ECG.

The 12 lead ECG will show changes associated with a ST segment elevation MI before other diagnostic test will. It will also allow for identification of where the damage is occurring in the heart. The change we look for is ST segment elevation. This is a key sign of prolonged ischemia and heart damage. We look for ST elevation of > 1 mm in more than one lead. The specific leads that the changes are occurring in can identify the area of the heart affected. The ECG is also utilized to monitor the progression of the MI. The ECG will show changes as the MI progresses through the healing process.

Cardiac Markers are also an important test to determine if there has been a MI. Cardiac markers include the CK-mb units and the troponin level. The CK-mb level will rise in 3-6 hours after the MI, peak in 24 hours, and return to normal in 2 – 3 days. The troponin level will rise within 1 hour, peak in 24-48 hours, and return to normal in 5-14 days. Cardiac markers are drawn on admission, and then at intervals to monitor the rise, peak, and fall.

Emergency management of the patient with acute coronary syndrome involves the initial interventions of putting the patient on the cardiac monitor, starting oxygen and IV therapy, getting the 12 lead ECG and labs drawn, and giving nitroglycerin, aspirin, and Morphine. Emergent PCI (percutaneous coronary intervention) is the treatment of choice for a confirmed MI. It needs to be completed within 90 minutes of arrival to the emergency room. They will open the occluded artery and place a drug eluding stent. If cardiac catheterization is not available fibrinolytic therapy will be considered.

Fibrinolytic therapy will dissolve the thrombus in the coronary artery to restore blood flow to the heart tissue. It needs to be started within 6 hours of the onset of symptoms to be effective. ECG changes that indicate a MI must be present as well. The patient must also have no contraindications for therapy, such as internal bleeding, a stroke within 3 months, or surgery within 3 months. Facility protocol must be followed for delivery. The fibrinolytic therapy is effective if the ST segment returns to baseline, the patient's pain is relieved, and there is reperfusion ectopi. The biggest complication from the treatment is bleeding. Bleeding will occur wherever the patient has been stuck for lab work. Pressure dressings will need to be applied to these sites. Major bleeding can be indicated by a decreased blood pressure and increased heart rate or a decreased level of consciousness. These would be indicators to stop the therapy.

Other therapies for acute coronary syndrome include medications, nutritional therapy and coronary revascularization surgery. Nutritional therapy will include initially making the patient NPO, then progressing to a low salt, low saturated fat, low cholesterol diet. Drug therapy will include:

- IV nitroglycerin
  - To decrease preload, decrease afterload, decrease oxygen demand, and increase collateral blood flow
- Morphine sulfate
  - Reduces anxiety, fear, and pain; vasodilates and decreases cardiac workload

- $\beta$ -adrenergic blockers
  - decrease HR, preload, myocardial contractility; can reduce the size of MI if given in the first hours
- Angiotensin-converting enzyme inhibitors
  - To prevent or slow heart failure
- Antidysrhythmia drugs
  - To control dysrhythmias
- Cholesterol-lowering drugs
  - To lower cholesterol levels
- Stool softeners
  - To prevent straining and increasing the workload of the heart when having a bowel movement.

Coronary surgical revascularization is what is commonly referred to as coronary artery bypass grafting. It is indicated if the patient fails medical management, if they have left main coronary artery disease or three vessel disease, if they are not a candidate for PCI, or the PCI has failed.

Coronary artery bypass graft (CABG) surgery requires the use of cardiopulmonary bypass. It uses the internal mammary artery or the saphenous vein as the graft. With the use of the saphenous vein the patient has a leg incision as well as the chest incision. Minimally invasive direct coronary artery bypass (MIDCAB) is an alternative to traditional CABG. It is completed using a laparoscopic approach. Our patient must be taught that surgery will relieve their symptoms, but it will not correct the underlying cause. They will still need to make dietary modifications, take medications, and exercise.

Nursing management of acute coronary syndrome includes pain management with nitroglycerin, morphine and oxygen. We also need to continuously monitor the ECG, VS, pulse oximetry, and heart and lung sounds. Activity will be restricted for the first 12 – 24 hours with the patient being on bed rest. Then we will gradually increase the activity level.

Anxiety and emotional reactions may also occur. Denial should be expected and teaching delayed until they are asking questions. Cardiac rehab teaching should also be done when the patient is ready for it. We may also see depression and low self- esteem.

Following bypass surgery the patient is typically in ICU for the first 24 to 36 hours. Intensive monitoring of all body systems will be needed, focusing on assessing for bleeding, monitoring fluid status, replacing electrolytes, restoring temperature, monitoring respiratory and cardiac status.

Ambulatory and home care interventions include patient teaching. They need teaching on medications, diet changes, physical activity, and the resumption of sexual activity. Physical activity should not increase the heart rate more than 20 beats per minute. If it does, they need to stop and rest until the heart rate comes down. Sexual activity should be gradually resumed. The energy expended with sexual activity equals the energy needed to climb two flights of stairs. When they can climb two flights of stairs without chest pain or shortness of breath they are ready to resume sexual activity.

Sudden Cardiac Death (SCD) is when there is an unexpected death from cardiac causes. Most deaths occur outside of hospital and 80% of them are related to coronary artery disease. With SCD there is an abrupt disruption in cardiac function that results in the loss of CO and cerebral blood flow. Most are caused by ventricular dysrhythmias. Death usually within 1 hour of onset of acute symptoms (e.g., angina, palpitations). If the patient survives the episode a diagnostic workup needs to be completed to rule out or confirm a MI. It will include cardiac markers, ECG, cardiac catheterization, 24 hour holter monitoring, exercise stress testing, signal averaged ECG, and electrophysiologic studies. Treatment will include antidysrhythmic medications and/or an implantable cardioverter-defibrillator. Psychosocial adaptations will also need to be made by the patient. They have had a near death experience and may have fear that will need to be addressed. They also may have role changes related to the experience.

## Heart Failure

Heart failure is an abnormal condition involving impaired cardiac pumping. The heart is unable to produce an adequate cardiac output (CO) to meet the metabolic needs. It is characterized by ventricular dysfunction, reduced exercise tolerance, diminished quality of life, and a shortened life expectancy. It is a syndrome that is associated with long standing hypertension (hypertension overworks the heart muscle) and coronary artery disease. About 5 million people in the United States are affected by heart failure and it is the leading cause of hospitalization in adults over 65.

Heart failure is classified as systolic or diastolic failure or dysfunction. Systolic failure is the most common cause from a decrease in the left ventricular ejection fraction (EF). We see this type of heart failure develop from impaired contractile function (MI), increased afterload (hypertension), cardiomyopathy, or mechanical abnormalities (valve problems). Diastolic failure is the result of an impaired ability of the ventricles to relax and fill during diastole that results in a decreased stroke volume and CO. The EF remains normal in this type of failure. Diastolic heart failure can be caused by left ventricular hypertrophy, aortic stenosis, and hypertrophic cardiomyopathy. Some patients will have components of both types of failure.

Heart failure affects all body systems as the body's compensatory mechanisms are activated to try to maintain an adequate cardiac output. The sympathetic nervous system is activated first, and it is the least effective mechanism. Catecholamines (epinephrine and norepinephrine) are released that increase the heart rate, increase myocardial contractility, and cause peripheral vasoconstriction. Over time, these mechanisms are detrimental as they actually increase the myocardial oxygen demand and workload.

The neurohormonal response to the decreased blood flow to the kidneys causes them to release rennin. Renin converts angiotensinogen to angiotensin I. Angiotensin I is converted to angiotensin II by a converting enzyme made in the lungs. Angiotensin II causes the adrenal cortex to release aldosterone which causes sodium and water retention. The angiotensin II also causes peripheral vasoconstriction to increase the BP.

A low cardiac output also causes a decrease in cerebral perfusion pressure. This causes the release of antidiuretic hormone (ADH) which increases water reabsorption in the renal tubules leading to water retention and an increase in blood volume. Endothelin is stimulated by the ADH release, the

catecholamines, and angiotensin II to cause arterial vasoconstriction, an increase in cardiac contractility, and hypertrophy of the heart tissue. Proinflammatory cytokines are also released that depress cardiac function. Over time, a systemic inflammatory response is triggered that further weakens the heart muscle.

The compensatory mechanisms have significant consequences on the heart. Ventricular dilation occurs that eventually contributes to a further decrease in cardiac output. Hypertrophy of the heart muscle occurs that contributes to poor contractility, higher oxygen needs for the heart, poor coronary artery circulation, and an increase in ventricular dysrhythmias.

There are counterregulatory mechanisms that the body institutes to try to prevent heart damage from the compensatory mechanisms. Natriuretic peptides are released in response to increases in atrial volume and ventricular pressure. They enhance diuresis by blocking the effects of the renin-angiotensin-aldosterone system. Nitric oxide is released from the vascular endothelium as well. It relaxes arterial smooth muscle that causes vasodilation and a decrease in afterload.

Heart failure is classified into left sided and right sided heart failure. Manifestations vary according to the type of failure that is present. With left sided heart failure the blood backs up into the left atrium and pulmonary veins. The manifestations that will be seen include pulmonary congestion and pulmonary edema. Right sided heart failure usually develops from left sided heart failure or changes in the lungs related to COPD. Blood backs up into the right atrium and the venous systemic circulation. Manifestations include an elevated right atrial pressure, jugular venous distention, hepatomegaly, splenomegaly, ascites, and peripheral edema.

Acute decompensated heart failure (ADHF) is the terminology that refers to pulmonary edema. Early signs of pulmonary edema include an increase in the respiratory rate and a decrease in the PaO<sub>2</sub>. As it progresses more manifestations develop. They include orthopnea, dyspnea, tachypnea, anxiety, use of accessory muscles, cyanosis, cool and clammy skin, a cough with frothy blood tinged sputum, tachycardia, and changes in the blood pressure. Lung sounds will reveal crackles, wheezes, or rhonchi.

Chronic heart failure manifestations include fatigue from a decreased cardiac output, dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. With paroxysmal nocturnal dyspnea the patient wakes at night with dyspnea from fluid being reabsorbed from the extremities pooling in the lungs with sleep. A persistent, dry cough may also be present. It is unrelieved by over the counter cough suppressants. It is from an increase in pulmonary pressures and fluid in the lung tissue. Edema is another manifestation. It is usually dependent in nature. Nocturia may also occur. The skin may be dusky in color and cool to the touch.

Complications from heart failure can include pleural effusions, atrial fibrillation, ventricular dysrhythmias, and renal failure.

A multitude of diagnostic tests can be utilized to help in the diagnosis of heart failure. These can include a chest x-ray, ECG, lab studies such as cardiac enzymes and a BNP (B type natriuretic peptide), an echocardiogram, hemodynamic assessment, stress testing, and cardiac catheterization. The BNP level is

one of the newer lab tests that is being done in the clinical area. Remember, B type natriuretic peptide is secreted when there is an increase in ventricular pressures, which occur with heart failure. An elevation in the BNP level is suggestive of heart failure.

The overall goals of treating both ADHF and chronic heart failure are to decrease patient symptoms, improve left ventricular function, reverse ventricular remodeling, improve the quality of life, and decrease mortality and morbidity. There are core measures that the Joint Commission on Accreditation of Healthcare Organization have identified for the treatment of ADHF. They are to decrease intravascular volume, decrease venous return (preload), decrease afterload, improve gas exchange and oxygenation, improve cardiac function, and reduce anxiety.

Decreasing intravascular volume in ADHF reduces the preload and venous return. Decreasing the volume and venous return allows the left ventricle to contract more effectively and increase cardiac output. The most common treatment for this is loop diuretics such as lasix.

Decreasing venous return (preload) in the patient with ADHF reduces the amount of volume returned to the left ventricle during diastole. This can be accomplished by placing the patient in a high fowler's position with the legs horizontal and utilizing IV nitroglycerin. IV nitroglycerin decreases preload and increases the blood flow to the coronary arteries.

Decreasing afterload in ADHF improves cardiac output and decreases pulmonary congestion. This can be accomplished with IV sodium nitroprusside, IV morphine sulfate, and IV nesiritide (Natrecor). Natrecor dilates both arterial and venous blood vessels and decreases both preload and afterload. With the use of any of these agents we need to maintain a SBP > 90.

Improving gas exchange and oxygenation for the patient in ADHF is done with the use of supplemental oxygen, noninvasive ventilatory support (BiPAP), and morphine sulfate. Morphine also helps to reduce anxiety associated with ADHF. This is important because anxiety increases the SNS response and makes the condition worse.

For patients with ADHF who do not respond to the conventional pharmacologic agents (diuretics, vasodilators, morphine sulfate) inotropic agents can be used to improve cardiac function. Some commonly used inotropic agents are digitalis, dobutamine, and Inocor. Hemodynamic monitoring is often used when administering these agents.

Management of chronic heart failure includes oxygen therapy, physical and emotional rest, nonpharmacological therapies such as pacemakers, cardiac transplantation, or ventricular assist devices, pharmacological therapy, and nutritional therapy. Patient teaching is also a very important aspect of care. With proper education and management of heart failure, the patient can lead a productive life.

Drug therapy can include diuretics, vasodilators, and positive inotropic agents. Diuretic therapy is used for the same purposes that it was used in ADHF. Vasodilators are important as they increase the venous capacity and improve the ejection fraction. They also contribute to improved ventricular contraction and

slow the development of ventricular dysfunction. Vasodilators include ACE inhibitors, angiotensin II receptor blockers, nitrates, beta adrenergic blockers, and Nesiritide.

ACE inhibitors reduce the systemic vascular resistance. This contributes to an increase in cardiac output, a decrease in the pulmonary artery pressure, and a decrease in the right atrial pressure. ACE inhibitors are used in conjunction with diuretic therapy. Capoten is one of the commonly prescribed ACE inhibitors. Some nursing interventions related to capoten include having the patient call for assistance when getting up as it can cause orthostatic hypotension. It is an agent that is potassium sparing, so the potassium should not be increased in the diet. It also should be taken one hour before meals to promote better absorption. If the patient is having difficulty managing the medication regimen, they may need referral to a home care agency for assistance.

Nutritional therapy for chronic heart failure includes weight reduction if overweight, and the most important modification is reducing the sodium intake. Sodium intake is restricted to 2.5 g per day. The normal dietary intake is between 7-15 g. Our patient will need instruction on low sodium foods. Dairy products and processed food should be avoided. There are tables in the textbook on page 834 that outline the sodium content of foods.

Fluid intake is not normally restricted, but the patient needs to be taught the importance of monitoring their weight on a daily basis. They should weigh themselves at the same time each day, with the same amount of clothing, and using the same scale. A weight gain of 3 pounds (1.4 kg) over a two day period needs to be reported to the health care provider. This could be an indication of an exacerbation of their heart failure.

Cardiac transplantation is a treatment option for end stage chronic heart failure. Candidacy is based on many things, and many patients die while waiting for a transplant. The major complications from transplantation are infection and rejection. Endomyocardial biopsies are obtained from the right ventricle weekly for the first month, monthly for the next 6 months and then yearly to assess for rejection. Nursing care focuses on promoting adaptation to the transplant process, monitoring cardiac function, managing lifestyle changes, and providing teaching.

### **Cardiogenic Shock**

Cardiogenic shock occurs when the heart cannot pump effectively resulting in a decrease in cardiac output. There is no decrease in the circulating volume or vasodilation that contributes to the decrease in cardiac output. The most common cause of cardiogenic shock is myocardial infarction.

The manifestations of cardiogenic shock are signs and symptoms of left ventricular failure. They include tachycardia, hypotension, narrowed pulse pressure, tachypnea, pulmonary congestion, cyanosis, pallor, cool and clammy skin, decreased urine output, and anxiety or delirium.

Treatment will include oxygen therapy and IV fluids to maintain fluid balance. Hemodynamic monitoring will be utilized to monitor heart function and fluid balance. Monitoring will show an increase in the pulmonary artery wedge pressure (PAWP) which indicates increased preload. Medications will be utilized to increase myocardial contractility to increase the blood pressure and heart rate PAWP. One of

the common medications utilized for this is dopamine. IV nitroglycerin may also be used. The best way to tell if medications are effective is to monitor the PAWP. It should decrease if the medications are effective. The patient will also need intensive monitoring of all body systems. They will be prone to cardiac dysrhythmias due to ischemia of the heart tissue. Kidney function must also be monitored carefully. Urine output needs to be at least 30 ml/hr. Decreased urine output indicates decreased renal perfusion.

Recovery from cardiogenic shock is possible with the proper monitoring and treatment of the patient.

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