Heart Failure

Purpose: This course provides an overview of heart failure. The recognition of signs and symptoms associated with heart failure will be discussed. The course will talk about treatment options of heart failure and the nurse's roles in the management of the disease.

Objectives:

- List three causes of heart failure
- Discuss five signs and symptoms suggestive of acute/decompensated heart failure
- Differentiate between diastolic and systolic heart failure
- List five medications used to treat heart failure
- Discuss the role of the nurse in the treatment of the patient with heart failure

Introduction

When the heart is unable to pump enough blood throughout the body; heart failure (HF) is present. Fluid and blood back up into the lungs, venous system, abdomen and legs leading to the hallmark signs and symptoms of heart failure.

HF is a devastating disease, not only because of its impact on morbidity and mortality but because of the sheer number of people it affects. HF is the cause of death in more than 287,000 people annually. It is the most common reason for hospitalization in those over the age of 65 and affects about 5 million people[1]. Heart failure directly costs the United States' health care system 29.6 billion dollars annually[1].

Race affects the prevalence of HF. It is more common in African Americans, Hispanics and Native Americans than in white Americans; this may be related to the increased prevalence of hypertension and diabetes in this population[2]. Men and women have an equal incidence of congestive heart failure, but women tend to have a later onset of symptoms. The incidence of HF increases with aging. Among older people the number newly diagnosed with HF has declined, but the number living with the condition has increased[3].

Classification of heart failure

Heart failure is labeled as: a) systolic versus diastolic and b) left versus right sided heart failure. Systolic heart failure occurs when the heart is unable to contract robustly. Systolic heart failure presents when the ejection fraction is less than 40%. Percentagewise, systolic heart failure is more common in the younger population, but as the age approaches 70 the prevalence of HF is almost equally divided between systolic and diastolic[4].

Diastolic heart failure – which is more common in women and older adults - is characterized by the left ventricle being unable to relax or fill properly. Diastolic heart failure is often called heart failure with normal ejection fraction. Death rates from diastolic heart failure are not as dire as systolic heart failure, but significantly worse than healthy controls[4].

It is not possible to differentiate between systolic and diastolic heart failure clinically. What differentiates the two groups are structure and function of the heart. Key criteria for diastolic heart failure are [2, 4, 5]:

- Signs and symptoms of heart failure
- Normal ejection fraction
- Ruling out other causes of the symptoms
- Structural changes such as increased end-diastolic pressure and stiffness

Doppler echocardiography is a method to identify abnormal diastolic function. The echocardiogram can determine if there is diminished early diastolic filling and reduced ventricular compliance associated with diastolic dysfunction [2, 4, 5]. In addition, the echocardiogram can determine the ejection fraction.

Heart failure is sometimes classified as left or right sided. Left sided heart failure is more common and leads to blood and fluid backing up into the lungs. Right sided heart failure typically arises with left-sided heart failure and presents with fluid backing up into the legs and abdomen often resulting in edema. Left and right sided HF often co-exists.

Signs and symptoms

Heart failure signs and symptoms present either gradually or acutely. Acute heart failure (decompensated) is an emergent condition that crops ups when the heart loses its ability to function properly. Its onset is rapid and may present with a variety of symptoms including:

- Shortness of breath
- Heart palpitations
- Productive cough
- Wheeze
- Chest pain
- Cardiac arrest

Most of the signs and symptoms are attributed to fluid overload as the heart's inability to effectively pump out blood causes blood to back up and overload the lungs and venous system. As fluid enters the lungs, the lungs ability to exchange gas effectively diminishes - leading to breathing difficulty. This is also the reason that rales are heard in the lungs of the patient with heart failure. Fluid that backs up into the venous system leads to increased pressure and fluid to leak out into the extracellular space leading to edema. Edema contributes to weight gain. An ineffective heart and fluid overload contributes to fatigue.

Chronic heart failure – whose symptoms come on more gradually - is the more common presentation of heart failure and presents with:

- Shortness of breath on exertion
- Orthopnea or shortness of breath when lying down
- Cough
- Wheeze
- Edema in the legs or abdomen
- Weight gain

- Anorexia
- Nausea
- Confusion
- Fatigue
- Nocturnal cough
- Intolerance to cold
- Anorexia

Signs of heart failure can also be variable depending on whether the presentation is acute or chronic. Those with acute heart failure may appear in acute distress and look well nourished. Those in chronic compensated heart failure do not appear in acute distress but may suffer from cardiac cachexia. Acute heart failure may present with an elevated heart rate, pallor, diaphoresis, cyanosis, and pale, cool extremities. Rales in the lung are common and may be accompanied by wheezing if HF is severe. Increased jugular vein distention is noted with fluid overload. A positive hepatojugular reflex is elicited by pushing over the liver which results in a budging of the jugular vein is also present when there is fluid overload. Edema is not usually present until at least 5 liters of extra fluid are on the patient[2]. Edema is not a specific finding to heart failure as it may be noted in other conditions such as liver/renal failure, chronic venous insufficiency and hypoproteinemia. An enlarged liver – hepatomegaly - is common in chronic right-sided heart failure, or in acute heart failure. Acute heart failure associated with hepatomegaly is typically associated with a tender liver. An S_3 gallop is common in decompensated heart failure. Cardiomegaly is present in some patients with chronic heart failure as evidence by a displaced point of maximal impulse (PMI) or an enlarged heart on x-ray [2, 4, 5, 7].

Clinicians have developed strategies for staging HF. The American College of Cardiology and the American Heart Association[5] developed a strategy based on symptoms that place patients in one of 4 categories (A, B, C or D). A class A patient has risk factors for HF but no symptoms. Class B patients have diagnosed HF but are asymptomatic. Class C patients are symptomatic and Class D patients have refractory symptoms. Another classification is the New York Heart Association Classification, which places patients in categories I through IV (See Table 1). Both scales have limitations but can be helpful to give a broad classification of patients.

While it is difficult to clinically distinguish between diastolic and systolic heart failure, some key features distinguish between the two types of failure. Diastolic heart failure typically presents with a greater elevation in blood pressure. Increased PMI is more common in systolic HF. Pulsus alterans – the arterial pulse shows an altering of strong and weak beats - is more common in diastolic HF.

Table 1: New York Heart Association Classification of Heart Failure

Class	Characteristics
I	Patients with cardiac disease, but without limitation of physical

	activity. Ordinary activity does not cause symptoms.
II	Patients with cardiac disease and slight limitation in their physical activity. Ordinary physical activity such as showering causes symptoms.
III	Patients with cardiac disease and with marked limitation in their physical activity. Less than ordinary activity, such as combing their hair or tying their shoes causes symptoms.
IV	Patients have cardiac disease and are unable to carry on physical activity. Symptoms are present at rest.
	Symptoms: dyspnea, fatigue, chest pain

Risk Factors

Heart failure is more common in those who are afflicted with certain conditions. It only takes one of these conditions for heart failure to develop but the presence of more than one increases the risk of developing heart failure. Hypertension is present in about 70% of patients who are diagnosed with heart failure[1].

The following is a list of factors that increase the risk:

- Hypertension
- Previous heart attack
- Coronary heart disease
- Diabetes
- Alcohol use
- Cardiac arrhythmias
- Certain medications: doxorubicin, cocaine, rosiglitazone (Avandia) and pioglitazone (Actos)
- Kidney disease
- Congenital heart defects
- Viruses
- Sleep apnea

Physiology

Heart failure is the inability of the heart to circulate the blood at a rate satisfactory to preserve the metabolic requirements of peripheral tissues and organs[2]. Whatever the cause of heart failure, the response is a fall in cardiac

output. Multiple compensation mechanisms take place to help the heart maintain its function.

Ventricle remodeling is a system of adaptation that the heart goes through in response to injury such as prolonged hypertension or myocardial infarction. Cells hypertrophy, abnormal contractile proteins are produced and collagen is deposited between myocytes. Angiotensin II and catecholamines are partly responsible for these changes. The result is a progressive loss of myocardial function.

As cardiac output falls, the left ventricle hypertrophies in an endeavor to compensate. The heart is attempting to contract with more strength so it can circulate blood more efficiently. While initially this helps, it leads to a situation where the heart muscle thickens and requires more blood from the coronary arteries.

B-type natriuretic peptide (BNP) is a cardiac neurohormone secreted from the heart's ventricles in response to increased pressure in the ventricle. The natriuretic peptide system decreases blood pressure and increases salt excretion. It is also used as a lab test to evaluate HF.

As cardiac out falls, the blood flow to the kidneys decrease which initiates the hormonal cascade of the renin-angiotensin-aldosterone system (RAAS). The RAAS axis starts off a series of chemical events that result in vasoconstriction and increased afterload. Renin is a hormone given off by the juxtaglomerular apparatus of the kidney which stimulates the conversion of angiotensin to angiotensin I. Then, angiotensin converting enzyme converts angiotensin I into angiotensin II. Angiotensin II is a constrictor and helps the body retain water and salt. Aldosterone leads to sodium and water retention and helps maintain cardiac output. These systems help the body compensate to meet metabolic demands during the acute event, but over the long-term contributes to the progressive nature of heart failure[2].

Norepinephrine and epinephrine – as part of the sympathetic nervous system – increase the heart rate, cardiac contractility and arterial vasoconstriction. These compensatory mechanisms temporarily increase cardiac performance. Unfortunately, they increase preload and afterload, which increases the oxygen requirement and workload of the heart. Eventually, the amplified left ventricular work affects the failing heart, which cannot endure the increased workload, and the cardiac output decreases setting off the cycle again.

The result of remodeling is the left ventricle chamber becomes rounder and larger. This can have a profound effect on the condition and pumping force of the heart. This leads to weaker heart cells and over the long-term the heart cells do not function well and eventually lead to cell death. Because of the remodeling many HF patients die from sudden cardiac death (SCD). The electrical system of the heart changes as the cells gets stretched out; this leads to an increased risk of cardiac conduction disturbances.

Treatment options are partially focused on stopping the negative impact of the activated nervous and endocrine systems to modulate the long-term course of the disease.

Complications

Left ventricle hypertrophy is an adaptation that the heart makes in response to heart failure. While this initially helps the body compensate, the heart will dilate and become less efficient. The bigger heart needs more oxygen that the diseased heart cannot supply. The heart will beat faster in attempt to compensate, but this typically leads to progressive heart failure. In addition, the hormonal system attempts to help the body adapt by increasing levels of certain hormones. This leads to increased vasoconstriction and makes the heart work harder.

Surgery is more complicated in those with heart failure. Those with heart failure have an increased risk for re-hospitalization and death after non-cardiac surgery[6].

HF has a direct affect on the heart. HF patients have more cardiac arrhythmias than patients without heart failure and the risk is higher in those who are decompensated (acute symptoms of HF) or have more advanced heart failure. Those with heart failure have more hypoxemia which predisposes the patient to a heart attack or ischemia [2, 4, 5, 7].

The liver and kidney are affected by the failing heart. Those with heart failure are at greater risk for renal insufficiency. The failing heart does not get blood to the kidney which increases the risk of renal failure. HF increases the risk of liver dysfunction. Fluid backs up into the liver especially in those with right sided HF leading to jaundice, elevated liver enzymes and at times fulminate liver failure. In addition, certain medications are not cleared as quickly in those with liver congestion leading to increased levels of medications. Decompensated heart failure may progress to respiratory failure and intubation [2, 4, 5, 7].

Differential diagnosis

When the patient presents with signs and symptoms suggestive of heart failure other diagnoses must be considered. Common conditions that may mimic heart failure include:

- Anemia
- Depression/anxiety
- Myocardial ischemia
- Chronic obstructive pulmonary disease/asthma
- Pulmonary embolism
- Sleep apnea
- Deconditioning
- Renal or hepatic failure
- Venous stasis
- Hypoalbuminemia

Diagnostic Tests

Diagnostic tests are typically employed after signs and symptoms suggest the diagnosis of heart failure. Chest x-ray is a common test used for patients with heart failure. It can differentiate between HF and other disease processes. In

acute heart failure chest x-ray changes after onset of symptoms may be delayed by 12 hours [2].

Electrocardiogram (ECG) is another test commonly used in heart failure. This will help detect any underlying arrhythmias contributing to or causing HF. This may include: atrial fibrillation/flutter, another tachyarrhythmia or bradyarrhythmia. It will also help identify any underlying cardiac ischemia or previous heart attacks. The ECG can discover enlargement of the atrium or ventricles. Holter monitor testing is sometimes carried out to look for cardiac arrhythmias.

Echocardiography with Doppler is used to determine size and function of the heart, non-invasively. The test determines the size of the cardiac chambers, any wall motion abnormalities, the thickness of the wall, the ejection fraction and any abnormalities in the heart valves.

Laboratory evaluation can be variable. B-type natriuretic peptide (BNP) is often used as a diagnostic test in heart failure. It is not recommended as a screening test[7], but is recommended when HF is suspected but the diagnosis is uncertain. The BNP levels are higher in those with pressure overload and ventricular volume expansion and are correlated with severity of HF. Levels greater than 100 pg/ml suggest heart failure; while normal BNP may indicate normality, but not necessarily. BNP is elevated in diastolic HF but not quite as much as in systolic HF. Many factors alter BNP levels. Older age is correlated to higher levels as is sex. Older women have higher levels of BNP when compared to men[2]. BNP goes up with age, atrial fibrillation and renal insufficiency. BNP goes down with obesity and treated heart failure.

Other lab tests should be done to assess for contributing factors and complications of heart failure. A complete blood count may suggest anemia or leukocytosis. Anemia is a factor that may complicate or cause heart failure. Infection may be indicated by an elevated white blood cell count which may precipitate heart failure. A thyroid panel can help determine if hyper or hypothyroidism is contributing or causing the HF.

Electrolyte levels have value in the monitoring of HF as well as the medications used to treat the disease. Sodium levels may be decreased by severe HF and is one indication that a sodium restricted diet may be appropriate. Potassium levels may be depressed due to diuretic use or elevated due to many of the medications used to treat HF.

Kidney and liver function should be monitored. Kidney function can deteriorate in heart failure for many reasons. Decreased blood flow to the kidneys due to a reduced cardiac output or from large doses of diuretics may increase creatinine and blood urea nitrogen (BUN) levels. Liver enzymes may increase due to liver congestion secondary to fluid overload. Heart failure has the potential to increase the bilirubin level[2].

Cardiac blood pool scans look at the heart chambers using radioactive dye to determine how well the heart is pumping. An exercise stress test may be done in some to look for ischemia, non-invasively.

Cardiac catheterization can be done to help diagnosis heart failure or determine the cause of heart failure. The use of a pulmonary arterial catheter to measure pulmonary capillary wedge pressure can assist in determining if the

heart failure is caused by cardiac or non-cardiac factors. Coronary angiography determines if any ischemia or an infarction is contributing to HF.

Treatment

Heart failure is treated with a combination of lifestyle interventions, medications, devices, and surgical options. Patients with heart failure and those at risk for heart failure should have risk factors aggressively treated. Those with risk factors for heart failure may have early changes in the heart's function and structure and treatment of these risk factors may help reduce the risk of the patient progressing to clinically evident heart failure.

Multiple non-pharmacological interventions are helpful in the treatment of HF. Light exercise should be encouraged unless there is a specific contraindication to exercise. Exercise has the potential to improve the overall function of the patient with HF. It also is helpful in the treatment of the risk factors associated with HF. Exercise reduces weight, improves blood pressure and lipid levels, improves glucose metabolism and reduces cardiovascular risk. Alcohol consumption should be minimized (equal to or less than one drink per day in women and two drinks in men) or eliminated in those with HF. Smoking cessation should be encouraged.

Dietary interventions may help treat the signs and symptoms of heart failure. Sodium intake should be reduced to 2-3 grams per day and those with severe heart failure benefit from less than 2 grams of sodium per day[7]. Selected patients – those with serum sodium less than 130mEq/L and those with fluid overload while on diuretics and sodium restriction – should have their fluid restricted to less than 2 liters per day[7]. Those who are overweight or obese should be counseled about weight loss. Multivitamins improve nutritional intake.

Those with advanced heart failure should be watched for weight loss. Cardiac cachexia needs to be monitored for and may present when there is severe muscle wasting and anorexia in association with chronic cardiac failure. It results in weight loss and confers a poor prognosis.

Currently there are several medications that have proven effective at reducing morbidity and mortality in the heart failure patient.

Medications to treat heart failure work by ridding the body of extra fluid, adjusting the hormonal balance of the body and helping the heart function better. It typically requires more than one medication to treat HF.

Angiotensin converting enzyme inhibitors

Angiotensin-converting enzyme inhibitors (ACE-I) are a well studied drug that reduces the level of angiotensin II in the body by blocking angiotensin converting enzyme. Angiotensin II is one of the hormones that are elevated in the heart failure patient as a compensatory mechanism. This hormone leads to vasoconstriction and retention of water and salt. While this is helpful for the short-term, overtime angiotensin II stresses the heart and is one mechanism that leads to progressive heart failure.

ACE-I should be given to all patients with systolic HF (and many with diastolic dysfunction) unless there is a contraindication[9]. In addition to improving morbidity

and mortality in HF, these medications are also used in the treatment of hypertension. Enalapril (Vasotec), lisinopril (Prinivil, Zestril) and captopril (Capoten) are examples of commonly used ACE-I. No evidence recommends the use of one ACE-I over another[9].

Hypotension, renal failure, angioedema and hyperkalemia are four special conditions that should be watched for in those on or being considered for ACE-I treatment. ACE-I lower the blood pressure and for those with a systolic reading of less than 100 mmHg should use caution when on ACE-I. Creatinine levels of greater than 2.5 mg/dl should also use caution when on ACE-I as these medications have the potential to worsen renal dysfunction. In addition, those with renal insufficiency and/or baseline hyperkalemia are at risk for worsening hyperkalemia. Those afflicted with angioedema from ACE-I may be tried on other medications.

There are times when medications should be substituted for ACE-I. For those individuals who are bothered with a cough from the ACE-I may be switched to an angiotensin II receptor blocker (ARB). Those troubled with either worsening renal failure or hyperkalemia while on ACE-I should be switched to the combination of an oral nitrate and hydralazine. Those who are afflicted by angioedema may be switched to the hydralazine/nitrate combination or possibly an ARB.

Angiotensin II receptor blockers (ARB)

These drugs, which include losartan (Cozaar) and valsartan (Diovan), have many of the beneficial effects of ACE inhibitors, but they do not cause a persistent cough. They may be an alternative for people who cannot tolerate ACE inhibitors due to cough or possibly due to angioedma[7]. Generally ACE-I are recommended as first line agents and when they are not tolerated, an ARB is substituted. In some individuals they may be used as first line agents such as those with heart failure post myocardial infarction and those with chronic HF with systolic dysfunction[10].

Some attention has been given to the combination use of ACE-I and ARB as this may more fully block the negative effects of angiotensin II. The combination of ACE-I and ARB may be indicated for some patients. While the combination of the two medications does not improve mortality rates, they do reduce hospitalizations[7].

Beta blockers

This class of drugs is one of the cornerstones for patients with systolic HF. The combination of beta blockers (BB) with ACE-I should be routine therapy for all patients with a left ventricular ejection fraction of less than 40%[7]. Beta-blockers improve left ventricular ejection fraction[12]**.** The three BB that have proven effective in heart failure include: carvedilol (Coreg), metoprolol (Lopressor) and bisoprolol (Zebeta). It cannot be assumed that other BB have beneficial effects on HF.

This class of medication should be used with caution in those with severe/recurrent hypoglycemia, peripheral vascular disease, hypotension or

asthma. Absolute contraindications include: bradycardia, heart block and severe reversible airway disease. BB are recommended for patients who are clinically stable and euvolemic after a bout of HF. They should not be used in those who are short of breath at rest with fluid overload or are hemodynamically unstable. The dose is started low and gradually titrated upwards toward the target in table 2. Medications should be titrated up every 2-4 weeks.

The debate continues and research is inconclusive about the most effective of the three approved BB, but current thinking is that there is similar efficacy between the three agents. Within one year the risk of re-hospitalization for HF was not significantly different in patients receiving atenolol, shorter-acting metoprolol tartrate, or carvedilol[13]. Carvedilol (Coreg) – if used at hospital discharge for those with HF and systolic dysfunction – has survival benefits, improves treatment rates and is well tolerated[14].

Table 2: Beta-Blocker to be used in HF

Medication	Target dose
Carvedilol (Coreg)	25 mg twice a day
Metoprolol succinate (Toprol XL)	200 mg every day
Bisoprolol (Zebeta)	10 mg every day

Aldosterone antagonists

Aldosterone antagonists (AA) are potassium sparing diuretics that help the heart function better and include the drugs spironolactone (Aldactone) and eplerenone (Inspra). In addition to their diuretic effect they also reduce the level of aldosterone, which is a hormone that leads to progression of heart failure. This class is recommended in those with class III or IV New York Heart Association systolic heart failure with an ejection fraction of less than 35% while being treated with an ACE-I or ARB, BB and diuretics. They are also indicated in those after a heart attack when the ejection fraction is reduced to less than 40% [2, 4, 5, 6, 9].

They are not recommended in those with renal failure or hyperkalemia. Those on AA should have their kidney function and potassium level monitored regularly. Spironolactone is also more likely to cause gynecomastia in men.

Diuretics

Diuretics are helpful in the patient with fluid overload. The use of diuretics requires a delicate balance of providing enough effect to offload fluid and not too much to cause side effects such as hypotension, dehydration or electrolyte imbalance. As heart function improves with other pharmacological agents such as ACE-I, ARB, BB, AA and with sodium restriction, patients may be able to tolerate a lower dose of diuretics. Patients should be weaned down or off diuretics, if possible, as there is some evidence to suggest that the use of diuretics in HF may increase death rates[14]. If patients are weaned down, it is important to monitor weight and signs and symptoms of HF closely.

For those in acute heart failure large doses often need to be given to provide adequate diuresis. Commonly prescribed loop diuretics for heart failure include

bumetanide (Bumex) and furosemide (Lasix). The addition of thiazide diuretics such as metolazone can be helpful in offloading fluid when combined with a loop diuretic.

Common side effects include electrolyte shifts, dehydration and hypotension. Potassium, magnesium and kidney function need to be monitored regularly.

Nitrates and Hydralazine

A medication called BiDil is a single pill that combines hydralazine and isosorbide dinitrate — both of which dilate and relax the blood vessels. This combination increases survival when added to standard therapy in black people with advanced heart failure[15]. This is the first drug studied and approved for a specific racial group. Further studies will be necessary to determine if this combination medicine will be helpful for others with heart failure. It is also recommended to use this medication combination in non-African Americans in addition to ACE-I and BB in those who remain symptomatic[7]. They are also used in some patients who cannot tolerate ACE-I and ARB.

A recent study showed improvement in morbidity and mortality, including a 39% reduction in hospitalization, improvements in quality of life and functional status, and a 43% reduction in mortality[15] with the combined use of hydralazine and nitroglycerine in African Americans on standard therapy.

Side effects include hypotension, so blood pressure needs to be watched and headache which tends to lessen with continued use.

Digoxin (Lanoxin).

Digoxin increases the strength of heart muscle contractions but its use is less frequent today than it was years ago. It is very helpful in those with systolic heart failure and co-morbid atrial fibrillation, but its use in those with normal sinus rhythm is not as well established. It is also helpful for those with systolic dysfunction and symptomatic while on standard therapy[7]. This drug has shown not to prolong life, but can be effective in reducing hospitalizations and helping the patient feel better, especially patients with systolic heart failure[22].

Therapeutic range for digoxin should be between 0.5 and 1.0 ng/ml and the level should be measured at least 6 hours after dosing. It also tends to slow the heartbeat. Digoxin reduces heart failure symptoms and improves the ability to live with the condition[7].

Other therapies

Cholesterol lowering drugs called statins (atorvastatin, simvistatin) have been shown to reduce death rates among those with HF[16].

Cardiac dysrhythmias are a common cause of death in those with heart failure. The use of antiarrhythmic drugs have not shown to reduce death rates in heart failure but the use of implantable cardioverter defibrillators (ICD) are helpful. Patients with heart disease and an ejection fraction of less than 30-35% could benefit from an ICD in respect to reducing the risk of sudden death from cardiac dysrhythmias[18, 19]. This therapy is vastly underused as it is costly and patients often do not want this treatment.

Biventricular pacing should be considered in those with class III New York Association heart failure with persistent moderate to severe symptoms. Criteria include: normal sinus rhythm with severe systolic dysfunction and a wide QRS interval[7].

A variety of surgical options may be considered in those with heart failure. Heart transplant is recommended for those with severe HF, rhythm disturbances that are not controlled with other therapies or for those with refractory angina[7].

Treatment of Diastolic Heart Failure

Not a lot of studies report on how to treat diastolic HF. There are principles that should apply to the treatment of diastolic HF. Treatment should be tailored to the symptoms that they have, disease they have or the mechanism that underlies that disease. Controlling blood pressure reduces the risk of heart failure exacerbation. If atrial fibrillation is present than the atrial rate should be controlled. Those with volume overload should be treated with diuretics.

Acute heart failure is treated similarly in systolic and diastolic heart failure. Reduction of venous congestion through the use of nitrates, diuretics, morphine and oxygen is critical. Aggressive diuresis is not recommended as it may lead to severe hypotension in the patient with diastolic heart failure. Rate needs to be controlled as diastolic HF patients do not tolerate tachycardia well.

Recent evidence suggests that the use of ARB may be helpful in the treatment of diastolic HF. Fibrillary collagen is controlled by the RAAS system and treatment with ARB reduce it. ARB reduce the rate of hospitalization and may reduce death from cardiac causes in those with diastolic failure[17]. ARB are the only medication that increases exercise tolerance. Exercise duration is increased with losartan and there is a decrease in peak exercise blood pressure[20].

In addition to ARB, ACE-I may be beneficial in the treatment of diastolic HF[7]. Long-term mortality is reduced in those with diastolic heart failure who are treated with ACE-I[21].

The use of BB in diastolic heart failure is recommended in those with hypertension, atrial fibrillation in need of rate control and after a heart attack[7]. In addition to diuretics, sodium and water restriction may also reduce fluid overload in diastolic heart failure.

Nursing Interventions

The nurse's role in the management of heart failure is to help identify cases of heart failure, identify exacerbations, educate patients and monitor treatments. Identifying patients with heart failure is accomplished by thoroughly evaluating all patients. Searching for signs and symptoms suggestive of heart failure is critical. Identifying exacerbation is easier then identifying new cases as those with a previous diagnosis are already on the radar and should more readily be identified. Factors that may suggest worsening heart failure are included in Table 3.

Heart failure is a chronic disease and patients need to be taught health maintenance techniques. Annual influenza vaccinations and staying up to date

on the pneumonia vaccination are critical to prevent these two very deadly diseases in the heart failure patient. Teaching patients to avoid nonsteroidal anti-inflammatory medications such as ibuprofen or naproxen is important as these medications increase the risk of an exacerbation of HF.

Monitoring patients for stress, anxiety and depression is critical as these conditions are very common in the patient with HF.

Common nursing diagnosis used for those with HF include:

- Activity intolerance related to fatigue
- Decreased cardiac output related to impaired cardiac function
- Fear related to the disease process
- Excess fluid volume related to impaired secretion of water and sodium
- Impaired gas exchange related to excessive fluid in the lungs

Given these nursing diagnoses the nurse needs to monitor patients for shortness of breath, vital signs including oxygen saturation, lung sounds, edema and weight gain. Nurses should teach patients energy conserving techniques, how to use oxygen if indicated, how to take medications, encourage patients to weigh themselves every morning and report significant changes to the primary care provider.

Optimal nursing and medical care has the potential to significantly improve the quality and quantity of life in the patient afflicted with HF.

Table 3: Signs/Symptoms that Heart Failure is Worsening

- Weight gain
- Increased shortness of breath on exertion
- Needing to prop yourself up to sleep on more pillows
- Increased edema
- Increased fatigue

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