Review Of Current Guidelines For The Treatment Of Chronic Heart Failure

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Abstract

Heart failure is a progressive clinical syndrome that impacts patients' quality of life. New research findings and recommendations have made it necessary for the American College of Cardiology and the American Heart Association to revise the guidelines for the treatment of chronic heart failure. This article is a review of the current heart failure guidelines. By implementing these guidelines, health care providers can deliver quality care and improve outcomes for those patients with chronic heart failure.

INTRODUCTION

Heart Failure (HF) continues to be a major health problem in the United States. Approximately 5 million patients are currently diagnosed with HF with an additional 500,000 new cases diagnosed each year . HF is a chronic disease and the leading diagnosis for hospitalization of older adults [2]. The number of people living with and disabled by HF continues to increase; this increase will have an enormous impact on health care in the near future[3]. The cost of treating HF in the United States is approximately $20 billion per year[4]. Due to the economic impact of HF and the effect it has on quality of life, it is imperative that health care providers deliver optimal therapy to their HF patients.

In order to provide consistent guidelines for the treatment of HF, the American College of Cardiology (ACC), American Heart Association (AHA), participants from the American College of Chest Physicians, the Heart Failure Society of America, the International Society for Heart and Lung Transplantation, the American Academy of Family Physicians, and the American College of Physicians-American Society of Internal Medicine formed a writing committee to develop a set of HF guidelines. This committee recognizes that optimal therapy for HF is “a work in progress” and future guidelines will follow[1].

CLINICAL SYNDROME: DEFINITION, SYMPTOMS, PROGRESSION OF DISEASE

The term “heart failure” is preferred over “congestive heart failure” because not all patients are volume overloaded at the time of evaluation1. HF is a complex clinical syndrome in which symptoms develop as a result of any structural or functional cardiac disorder.1 The diagnosis of heart failure is made by a careful history and physical examination. The primary signs and symptoms of HF are dyspnea, fatigue, and fluid retention. Dyspnea and fatigue may lead to decreased physical activity and limit exercise tolerance. Fluid retention may lead to pulmonary congestion and peripheral edema. The most common classification of functional limitation with regards to heart failure was developed by the New York Heart Association (NYHA). This classification system has 4 classes. Class I HF patients would have symptoms only at levels that normal individuals would experience symptoms. Class II HF patients have symptoms with ordinary exertion. Finally, Class III HF patients will have symptoms with less than ordinary exertion and Class IV HF patients may have symptoms at rest.

With the formulation of these new HF guidelines the writing committee decided to develop a new classification for HF that combined the evolution and progression of HF1. There are 4 stages of HF A through D1. Stage A HF is the least severe, whereas Stage D HF patients may require specialized treatment such as inotropic support, mechanical support, heart transplantation, or hospice care1.

Figure 1

Table 1: Stages of Heart Failure

Stage	Description
A	Those patients that are at high risk for developing heart failure, but have no structural disorders.
B	Those patients that have structural disorders, but no symptoms of heart failure.
C	Those patients that have current or previous symptoms of heart failure along with structural disorders.
D	Those patients that have end stage heart failure and require specialized treatments.

The functional limitation of HF patients is generally quantified by the NYHA functional class system. Patients tend to deteriorate with regards to functional class over time and tend to have fluctuation of symptoms even without changes in medications. Although HF has been regarded as a hemodynamic disorder, studies have not shown a correlation between cardiac performance and the symptoms the patient reports. Patients with low ejection fractions (EF) may not be symptomatic when compared to patients with preserved left ventricular function. An explanation of this may be due to ventricular distensibility, valvular regurgitation, pericardial restraint, and right ventricular dysfunction1. Other factors that may contribute include peripheral vascular dysfunction, skeletal muscle physiology, pulmonary dynamics, and neurohormonal and reflex autonomic activity1. Improvement in hemodynamics may improve rapidly with medications although symptoms may take weeks or longer to improve.

HF may result from disorders of the pericardium, myocardium, endocardium, or great vessels with a majority of these patients resulting in symptoms due to left ventricular dysfunction. Left ventricular dysfunction may be either systolic or diastolic in nature. Systolic dysfunction is characterized by a dilated left ventricle with a decreased EF that is usually less than 40%. Diastolic dysfunction involves a normal sized ventricle with normal ejection of blood from the left ventricle, but impaired filling. Treatments for systolic and diastolic dysfunction are different. Coronary artery disease is the cause of left ventricular dysfunction in the majority of patients. Other causes are related to hypertension, thyroid disease, valvular disease, alcohol, myocarditis, or idiopathic causes.

HF is considered a progressive disorder in which ventricular remodeling occurs. Left ventricular dysfunction begins with an injury or insult to the myocardium. The actual geometrical shape of the left ventricle changes by dilating and hypertrophying, resulting in a more spherical shape. Due to the remodeling of the left ventricle, more stress is exerted on the ventricle wall, which decreases the mechanical performance and increases the flow through the mitral valve. This continues to perpetuate the remodeling of the ventricle, which precedes the development of symptoms, usually by months, but sometimes by years. Some of the factors that can accelerate the remodeling process are the neurohormonal systems, such as norepinephrine, angiotensin II, aldosterone, endothelin, vasopressin, and cytokines, which can adversely effect the heart's function. These hormones work to increase sodium retention and vasoconstriction. They may also act as cardiac toxins that can stimulate fibrosis1.

CLINICAL ASSESSMENT

Generally, there are 3 ways that HF patients present: decreased exercise tolerance, fluid retention, or with no symptoms or symptoms of another cardiac or non-cardiac disorder. Patients with HF that present with decreased exercise tolerance either describe more dyspnea with exercise or increased fatigue. These patients may be symptomatic even at rest. These symptoms in the elderly should not be inappropriately attributed to the aging process, deconditioning, or pulmonary disorders. HF should be considered as a differential diagnosis for patients with dyspnea and fatigue. Sometimes it is difficult to determine the cause due to comorbidities in patients. In some patients the only symptom may be fluid retention, while others may not have symptoms but may be found to have cardiac enlargement or dysfunction1.

The first step in evaluating patients is to obtain a complete history and physical examination. The history and physical exam may reveal evidence of a myocardial infarction, valvular disease, or congenital heart disease along with physical evidence of cardiac hypertrophy, murmurs, S3, and edema. Health care providers need to determine if there is a history of hypertension, diabetes, hypercholesterolemia, coronary or peripheral vascular disease, rheumatic fever, chest irradiation or chemotherapeutic drugs known to be cardiotoxic. Patients should be questioned about alcohol consumption, illegal drug use, and exposure to sexually transmitted diseases. They should also be asked about collagen diseases, infections, thyroid disease, and pheochromocytoma. A detailed family history should also be obtained to discover familial disposition to atherosclerotic disease, cardiomyopathies, and unexplained sudden death.

The most useful diagnostic test is a two-dimensional echocardiogram along with Doppler flow studies1. The echocardiogram will reveal the EF. Patients with an EF less than 40% are considered to have systolic dysfunction. An

echocardiogram can reveal important information about the cardiac dimensions, thickness, and motion of the ventricles. Other tests that may be helpful include radionuclide venticulography to measure global and regional functioning. MRI and CT scans are helpful in evaluating cardiac mass or differentiating pericardial disease. Chest x-rays can determine cardiac enlargement and pulmonary congestion. These tests help with the evaluation of HF but do not reveal the cause. The 12-lead electrocardiogram (ECG) may help diagnose the presence of a myocardial infarction (MI), left ventricular hypertrophy or cardiac dysrhythmias.

Laboratory testing is helpful to evaluate conditions that may exacerbate HF. The initial evaluation of HF patients should include a complete blood count, electrolytes, lipids, renal function, hepatic function, and urinalysis. Other tests that should be included are thyroid functions, ferritin levels, and tranferrin saturation. HIV screening, assays for connective tissue disorders, and pheochromocytoma should be performed if they are suspected. The measurement of brain natriuretic peptide (BNP) is a new marker, still undergoing research, which is used to identify patients with elevated left ventricular filling pressures who may present with signs and symptoms of HF. BNP cannot distinguish whether patients have systolic or diastolic dysfunction but it aids in distinguishing patients with dyspnea from HF or other disease processes1.

A majority of patients with HF have coronary artery disease. Revascularization of these patients may actually improve their symptoms of HF. Some patients with nonischemic cardiomyopathy present with chest pain that may resemble angina, in these cases coronary angiography is recommended. About one-third of patients with HF have normal coronary arteries; therefore, other diseases of the myocardium are responsible for their HF1. Many patients do not have a defined cause for their cardiomyopathy, while in other patients the cause is related to a systemic disorder such as hyperthyroidism, hemochromatosis, or exposure to cardiotoxic drugs or the presence of myocardial inflammation. Endocardial biopsy may be used to detect inflammatory cells, confirm the diagnosis of sarcoidosis, amyloidosis, and hemochromatosis. Endocardial biopsy is not routinely recommended but may be a useful tool to confirm the diagnosis responsible for producing HF symptoms.

Once a diagnosis of HF has been confirmed, the ongoing clinical evaluation of the patient is critical to the management of HF. The patient must be evaluated on the type, severity, and duration of symptoms that the patient experiences that may decrease their functional capacity. Health care providers must question patients about their activities and the activities that they would like to do, but cannot, due to exacerbation of HF symptoms. The 6-minute walk test may be used to measure functional capacity along with the subjective NYHA functional classification1.

Evaluation of volume status is another important factor in effectively treating HF patients. Volume status must be determined in order to prescribe diuretic therapy. The physical exam is the critical element in evaluating volume status by determining the presence and degree of jugular venous distention (JVD), the presence of organ congestion (hepatomegaly, crackles in lung fields), and peripheral edema. Many patients with end-stage HF or chronic HF do not have pulmonary crackles. Short-term fluid balance is best measured by weighing patients. For patients that have long follow-up periods, weight measures are not reliable indicators of volume status due to the loss of muscle and fat with the progression of HF.

HF patients should have routine labs to test electrolytes and renal function. Hypokalemia is a side effect of diuretic therapy whereas hyperkalemia may complicate the use of ACE inhibitors and spirolactone. Serial chest radiographs, routine echocardiograms, and the use of invasive hemodynamic monitoring are not recommended1. Prognosis of HF patients is difficult to determine. The majority of patients' prognoses may be determined by the clinical assessment and their functional capacity. With disease progression, the measurement of renal function and sodium concentration can be prognostic indicators.

HEART FAILURE THERAPY

STAGE A HEART FAILURE

Stage A HF patients are those at risk for developing left ventricular dysfunction. By modifying the risk factors, the health care provider may prevent the development of HF. Hypertension is a major risk factor for developing HF. Both systolic and diastolic blood pressure must be adequately controlled in order to reduce the risk of HF. It is recommended that the same medications used for the treatment of HF such as diuretics and angiotensin converting enzyme (ACE) inhibitors, be used in the treatment of hypertension1.

Diabetes can negatively effect patients with HF, although control of diabetes has not been shown to reduce the

development of HF. ACE inhibitors have been shown to prevent end organ disease in diabetic patients. Patients with known atherosclerotic disease should be aggressively treated due to the risk of development of HF. Hypercholesterolemia must be treated adequately to decrease the progression of atherosclerotic disease.

Other measures to prevent HF include not smoking or the use of illicit drugs such as cocaine. Alcohol is toxic to the myocardium; therefore, intake should be minimal. There is no evidence that sodium restriction prevents the development of HF. Health care providers should treat thyroid diseases and tachyarrhythmias along with frequent assessments to evaluate patients for HF symptoms.

STAGE B HEART FAILURE

Stage B HF patients are those who have not yet developed symptoms. These patients have had a MI or insult to the myocardium and evidence of left ventricular dysfunction. The recommendations are the same for those with Stage A HF. There is no evidence that sodium restriction, regular exercise, or nutritional supplements can prevent the development of HF for patients in Stage B.

The prevention of cardiac events in Stage B HF patients is priority. Patients with an acute MI should be appropriately treated with thrombolytics or percutaneous coronary interventions (PCI)1. ACE inhibitors and beta blockers should be given to decrease the risk of death and reinfarction[5]. ACE inhibitors have also been shown to decrease death and hospitalization of HF in asymptomatic patients[6]. No evidence exists concerning the benefit of giving digoxin to symptomatic patients1. Those patients with mitral regurgitation or stenosis should be considered for valve replacements. Again, patients should be routinely assessed for signs and symptoms of HF.

STAGE C HEART FAILURE

Stage C HF patients have established left ventricular dysfunction with current or prior symptoms. Recommendations for Stage C HF include those considered appropriate for Stage A and Stage B HF patients. Patients with Stage C HF should be educated on sodium restriction and the need for daily weights. They should also be given influenza and pneumoccocal immunizations to prevent respiratory infections. In addition, physical activity should be encouraged to prevent deconditioning.

The three classes of drugs that should be avoided in HF patients are: antiarrhythmic medications, calcium channel blockers, and non-steroidal anti-inflammatory drugs (NSAIDS). Many of the antiarrhythmic agents can depress cardiac dysfunction, except for amiodarone. Calcium channel blockers can actually worsen HF and NSAIDS can cause sodium retention and peripheral vasoconstriction1. Patients should be closely monitored for signs and symptoms of HF.

The medications recommended for Stage C HF are diuretics, ACE inhibitors, beta blockers, and sometimes digoxin1. Diuretics interfere with sodium reabsorption; loop diuretics are the preferred class of diuretics in HF. Diuretics decrease fluid retention, which will be demonstrated by decreasing signs of JVD, pulmonary congestion, peripheral edema, and body weight. The effects of morbidity and mortality from diuretic therapy are unknown. Diuretics will produce symptomatic relief quicker than any other drug for HF. Diuretics help control fluid retention, but should never be used alone in the treatment of HF. Diuretics should be initiated in all patients with a history of fluid retention. Also, they should be started at low doses and increased until urine output increases and body weight decreases at a rate of 0.5-1kg per day1. Electrolytes should be treated and if hypotension or increased BUN results, diuresis should be slowed but not stopped. Once fluid retention has been resolved, a maintenance dose of diuretics should be prescribed. Diuretics should be adjusted as needed to treat fluid retention. Patients should be monitored for hypokalemia and hyperkalemia; hypokalemia increases the risk for developing dysrhythmias. Be aware that patients may be less responsive to diuretic therapy if they have a high intake of sodium or use NSAIDS1.

ACE inhibitors prevent the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor. ACE inhibitors have been found to alleviate symptoms of HF, reduce death, and hospitalizations5. These benefits have been reported in patients with mild, moderate, and severe symptoms along with patients with or without coronary artery disease. ACE inhibitors should be given to all patients with left ventricular dysfunction unless contraindicated due to allergies or renal failure. ACE inhibitors should be used in combination with diuretics, beta blockers and usually digoxin1. Selection of ACE inhibitors should be based on those used in clinical trials to treat HF, such as captopril, enalapril, lisinopril, or ramipril. Treatment with ACE inhibitors should begin at low doses and titrated up. The patient's renal function and potassium levels should be performed within 1-2 weeks of initiation of ACE inhibitor therapy. Again, fluid retention

can inhibit the therapeutic benefits of ACE inhibitors; therefore, appropriate diuretic therapy is imperative1.

Adverse effects of ACE inhibitors fall into 2 categories related to angiotensin suppression and kinnin potentiation. The most common side effect of angiotensin suppression is hypotension. Most patients treated with ACE inhibitors will have a reduction in blood pressure. It is only a problem if patients are symptomatic with postural hypotension, worsening renal function, blurred vision, or syncope. These side effects may improve by increasing sodium intake and/or decreasing diuretic doses. Worsening renal function is another common adverse effect of ACE inhibitors. Renal function usually improves by reducing the dose of ACE inhibitors. Hyperkalemia may be another side effect, but this is more common in patients with diabetes and in patients that have been taking potassium supplements. The use of ACE inhibitors potentiates kinin, which is most commonly associated with a cough. This is the most common reason for withdrawal of ACE inhibitors1. The other severe adverse effect is angioedema. Angioedema rarely occurs, but is a life threatening event. If a patient reports angioedema from an ACE inhibitor this class of drug should never be used again in this patient.

Beta blockers are another important drug in the treatment of HF. These drugs work by inhibiting the sympathetic nervous system in HF patients. Activation of the sympathetic nervous system over time can cause ventricular hypertrophy, increase heart chamber volumes, and cause vasoconstriction. Beta blockers stop these effects of the sympathetic nervous system and decrease the effects of ventricular remodeling. Beta blockers have shown to reduce mortality and hospitalization[7]. A combination of ACE inhibitors and beta blockers suggests the suppression of 2 neurohormonal systems is beneficial to patients with HF. Beta blockers should also be given to all patients with stable HF due to left ventricular dysfunction unless contraindicated. Patients who should not be started on beta blockers are those who are in an intensive care unit, have evidence of fluid retention or volume depletion, or recent use of inotropic therapy. These patients should be reevaluated for beta blockers once they have stabilized. Beta blockers should be started at low doses and slowly increased. Patients should be monitored for HF symptoms and be instructed to weigh themselves daily due to the potential for fluid retention with beta blockers. Patients may not have improvement of symptoms for 2-3 months1. Beta blockers should not be abruptly withdrawn due to the potential for decompensation. If patients develop fluid retention diuretics should be increased while beta blockers are continued. Beta blockers should only be discontinued if there is evidence of hypoperfusion.

There are 4 adverse effects related to beta blocker use: fluid retention and worsening HF symptoms; fatigue; bradycardia and heart block; and hypotension. Fluid retention is usually asymptomatic and is usually not volume overload. Fatigue generally spontaneously disappears within a few weeks. If fatigue persists reduction of the beta blocker may be warranted. Bradycardia is usually asymptomatic; if it is associated with dizziness then a reduction in the dose is needed. Hypotension my only be present in the first 24-48 hours of starting the drug. If hypotension persists the dose may need to be lowered. Also, patients need to be advised to take ACE inhibitors and beta blockers at different times to reduce the potential for hypotension1.

Digoxin has long been considered a positive inotropic drug. Recent studies imply that digoxin may actually prevent the activation of neurohormonal systems1. The use of digoxin has shown to decrease the combination of death and hospitalization6. Digoxin should not be used as primary treatment of HF and should be used cautiously in patients with sinus or atrioventricular blocks. Little evidence exists to support the use of digoxin levels to determine therapeutic levels1. Recommended doses are 0.125-0.25mg per day. Caution should be used in treating patients that are elderly, have impaired renal function, or low lean body mass with digoxin in order to prevent the risk of toxicity1.

STAGE D HEART FAILURE

Stage D HF patients are those that are considered to have refractory, end-stage HF. These patients continue to experience symptoms at rest or with minimal exertion despite appropriate medical therapy. These patients are unable to perform most activities of daily living. They are frequently cachexic and require frequent and prolonged hospitalizations for treatment of their symptoms. These patients need to be considered for mechanical circulatory support, continuous intravenous inotropic support, cardiac transplantation, or hospice referral. Appropriate treatments include those for patients with Stage A, B, and C HF1.

Volume overload can be treated with loop diuretics along with sodium restriction. As HF progresses there is usually a decline in the renal function[8]. With this decline diuretics may not be as effective and the addition of intravenous drugs such as dopamine and dobutamine may be needed to improve renal function [9]. The addition of these drugs can

cause increased diuresis, but may worsen renal function. The use of hemofiltration or ultrafiltration may be appropriate to remove fluid1. The goal should be to discharge HF patients home in euvolemic volume status. Once the dry weight has been established it may be possible for patients to adjust diuretics according to their weight.

Neurohormonal blocking agents have been found to cause worsening HF symptoms in patients with end stage HF. Therefore, these patients may tolerate low doses or may not tolerate ACE inhibitors and beta blockers at all. Health care providers should not initiate these drugs if the systolic blood pressure is less than 80 mm Hg or the patient exhibits signs of hypoperfusion. Patients should not be started on beta blockers if they have significant fluid retention or have recently been treated with intravenous inotropic drugs. ACE inhibitors and beta blockers should be initiated at low doses, if these doses are tolerated then they may be titrated up1.

During the frequent hospital admissions of end stage HF patients, they are commonly started on positive inotropic drugs such as dopamine, dobutamine, and milrinone. These drugs are used to improve symptoms and promote diuresis. Once the patient has been stabilized on intravenous inotropes then an oral regimen should be initiated. Some physicians use hemodynamic measurements from pulmonary artery catheters to guide their use of inotropic drugs. For those patients who cannot be successfully weaned off the inotropic drips, they may require a long term catheter for long term or outpatient therapy. Cardiac transplantation is a surgical option to treat end stage HF patients. There are established indications for heart transplant, including severe functional impairment, oxygen consumption of less than 15 mL/kg/min, or the need for continuous inotropic therapy1. Left ventricular assist devices are also available as a bridge to transplant and are currently under investigation for destination therapy for patients with end stage HF.

DIASTOLIC DYSFUNCTION

Diastolic dysfunction is diagnosed in those patients with HF symptoms that are found to have preserved systolic dysfunction and an EF greater than 40%. Some of the disorders associated with diastolic dysfunction include: restrictive cardiomyopathy, obstructive and non-obstructive cardiomyopathy, and infiltrative cardiomyopathies1. With diastolic dysfunction, filling of the ventricles is impeded due to fibrosis and the lack of relaxation. HF is predominately found in elderly women with hypertension[10].

Making the diagnosis of diastolic dysfunction is often difficult. The diagnosis is generally made by findings on an echocardiogram. These patients that are found to have signs or symptoms of HF with a normal ejection fraction are diagnosed with diastolic dysfunction1. There have been few clinical trials to evaluate the treatments for diastolic dysfunction. Treatment is generally based on physiologic factors such as blood pressure, heart rate, uncontrolled blood volume and ischemia. Hypertension can cause both functional and structural changes in the heart. Both systolic and diastolic blood pressure should be controlled using published guidelines to prevent the development of diastolic dysfunction1. Tachycardia shortens both the ventricular filling time and perfusion of the coronary arteries. Those medications that slow the heart rate can improve symptoms in diastolic dysfunction. Diuretics may also decrease blood volume, thus improving shortness of breath in these patients. If patients are ischemic they should be considered for revascularization to alleviate ischemia and ultimately improve their symptoms1.

THERAPY FOR SELECTED HEART FAILURE PATIENTS

Some therapies that have been used in the treatment of HF have not been proven to be beneficial for all HF patients or are only effective in certain groups of patients. Some of these therapies are the use of aldosterone antagonists, angiotensin receptor blockers (ARB), combination of hydralazine and isosorbide dinitrate, and exercise.

Aldosterone antagonists, such as spirolactone, should be considered for those patients with symptoms at rest who are receiving diuretics, ACE inhibitors, and beta blockers. Patients need to have a serum potassium level less than 5.0 mmol/L and a serum creatinine level less than 2.5 mg/dL before therapy is begun. Potassium supplements should be stopped due to the potential for hyperkalemia. The medication should be stopped if hyperkalemia and/or painful gynecomastia develops. The benefit of spirolactone in the treatment of patient with mild to moderate HF is not known and therefore should not be used1. ARBs should not be substituted for ACE inhibitors; but ARBs may be considered for those patients who had adverse reaction to ACE inhibitors such as angioedema or cough. ARBs can produce hypotension, hyperkalemia, and decrease renal function. Data is inconclusive on the benefits of using ACE inhibitors and ARBs simultaneously in HF patients. The combination of hydralazine and isosorbide dinitrate should not be substituted for ACE inhibitors in HF patients. The combination of these drugs may be considered for those

patients intolerant to ACE inhibitors. Beta blockers should be the first choice rather than hydralazine and isosorbide. Exercise training should be prescribed to all stable outpatients to prevent deconditioning in addition to drug therapy for optimal outcomes1.

Some medications and therapies are not approved and are under clinical investigation. Vasopeptidase inhibitors are being evaluated as a possible better drug than ACE inhibitors in the treatment of HF. Cytokine antagonists were believed to block cytokines that have a cardiac depressant effect; but a large clinical trial had to be stopped due to the likeliness it would be beneficial in the treatment of HF. Endothelin is a potent vasoconstrictor; endothelin antagonists are currently under investigation in the treatment of HF. These drugs are those that block the endothelin receptor and those that block the enzyme to convert endothelin. Synchronized biventricular pacing is used to synchronize ventricular conduction in order to improve the pump in HF patients. This has shown to improve HF symptoms and long term clinical trials are currently underway. External counterpulsation is intended to mimic the therapeutic effects of an intra-aortic balloon pump by an inflatable suit worn by the patient. Studies are also being conducted on respiratory support, such as nocturnal oxygen and continuous positive airway pressure. Some therapies that are unproven and not recommended include: nutritional supplements and hormonal therapies, intermittent intravenous positive inotropic therapy, and dynamic cardiomyoplasty1.

HEART FAILURE TREATMENT IN SPECIAL POPULATIONS

Many regard HF as a disease of men when women are the majority of HF patients, especially elderly women with diastolic dysfunction1. Many HF clinical trials have not included many women; currently, women are being included in more clinical trials as mandated by governmental agencies. Another group that has a high incidence of HF is African-Americans. African-Americans tend to exhibit HF symptoms at an earlier age and disease progression is quicker than whites possibly due to hypertension, diabetes, and the sodium retention found in African-Americans. Again, there is an under representation of racial minorities in clinical trials to determine appropriate treatments for each race. Elderly patients are not well represented in clinical trials for HF either, although HF is a common reason for hospital admissions in the elderly[11]. Also, elderly patients are not often aggressively treated for risk factors associated with HF and they often take medications that may interfere with treatment, such as NSAIDS.

HF patients frequently develop dysrhythmias. Atrial fibrillation is the most common atrial dysrhythmia in HF patients. Atrial fibrillation is associated with decreased exercise and worsening HF symptoms[12]. There are 3 ways that atrial dysrhythmias may have adverse effects: emboli may develop due to stasis blood in the atria; ventricular filling may be compromised; and ventricular response may be diminished[13]. Therefore, ventricular rate and prevention of emboli is important in HF patients. Digoxin is mostly used in the treatment of atrial fibrillation in HF. Beta blockers have been found to be more effective at rate control with exercise than digoxin[14]. According to the recommendations, heart rates should be controlled to 80-90 beats per minute at rest and 110-130 beats per minute during exercise1. Nodal ablation or the addition of amiodarone may need to be implemented if the heart rate is uncontrolled. Until further research is complete current recommendations are to convert HF patients from atrial fibrillation to normal sinus rhythm1. The use of anticoagulants in HF remains inconclusive. HF patients are prone to thromboembolic events due to venous stasis and the frequency of atrial fibrillation in HF. Warfarin is needed for those patients who have had a prior emboli event or those patients with chronic atrial fibrillation[15].

HF patients are also at high risk for sudden death. Most HF patients experience ventricular dysrhythmias. The current research suggests that sudden death in HF is a result of an ischemic event or a bradyarrhythmia rather than a sustained ventricular arrhythmia1. Health care providers should not use ambulatory electrocardiographic monitoring to detect asymptomatic ventricular dysrhythmias. Three interventions that need to be implemented in order to prevent sudden death in HF are: beta blockers, amiodarone, and implantable cardiac defibrillator (ICD)1.

END OF LIFE ISSUES

With regards to end of life, patients and families should receive education concerning expected course of illness, final treatment options, and plans if the patient becomes too ill to make their own health care decisions. Patients and families should be given information about initiating advance directives. A recommendation is that the same health care team be involved in both outpatient and inpatient care in order to facilitate continuity of care. Hospice should be an option for patients for support and symptom relief at

the end of life1.

IMPLEMENTATION OF HEART FAILURE GUIDELINES

Despite previous HF guidelines there is still suboptimal treatment of HF patients. In the past, simple distribution of guidelines to physicians did not improve the quality of care. Therefore, dissemination of guidelines along with behavioral interventions to improve practice must occur. Disease management programs have been shown to be effective in the treatment of chronic diseases. These programs are effective for patients at high risk for hospitalization and clinical deterioration. Therefore, the use of these guidelines in a disease management model may improve the treatment and quality of care for HF patients1.

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