INTRODUCTION

Although there have been great strides in medical technology and in the treatment of cardiovascular disease in general, the prevalence of congestive heart failure (CHF) continues to rise (Gheorghiade et al., 1998). Today many people who have suffered myocardial tissue damage from myocardial infarction (MI) survive only to progress to heart failure. Congestive heart failure is the number one hospital admitting diagnosis of persons over the age of 65 (Consensus Recommendations for Heart Failure, 1999). The six year mortality rate of patients diagnosed with CHF is 80% for men and 65% for women (American College of Cardiology/American Heart Association [ACC/AHA], 1995). According the American Heart Association, CHF is responsible either directly or indirectly for 260,000 deaths annually (Massie, & Shah, 1997).

Congestive heart failure affects approximately 4.8 million people in the United States with an estimated 400,000-700,000 new cases developing annually (Consensus Recommendations for Heart Failure, 1999). It is a major cause of morbidity and mortality in the U.S. and the monetary impact upon the public health system is enormous. It is estimated that CHF accounts for more than 2 million outpatient visits annually, costing over $10 billion, 75% of which is spent on patients hospitalized with this chronic condition (Polanczyk, Rohde, Dec, & DiSalvo, 2000).

The increased incidence of CHF, combined with the high morbidity and mortality, have prompted many research studies aimed toward improving care and management of these patients in the primary care setting. One recent study of an advanced practice nurse (APN) directed heart failure program illustrated that an intensive multidisciplinary approach produced positive patient outcomes as evidenced by decreased mortality, length of hospital stay, and readmission rates (Dahl, & Penque, 2000). A multidisciplinary model used by APNs for the management of patients with CHF can improve patient understanding about the disease and influence the patient to take an active role in staying healthy. The purpose of this article is to provide the APN with a greater understanding of the pathophysiology and management of CHF.

PATHOPHYSIOLOGY

Heart failure is defined as a condition that results from some abnormality in myocardial function. The abnormality, whatever the cause, results in the inability of the heart to deliver enough oxygenated blood to meet the metabolic needs of the body. When the right and left ventricles fail as pumps, pulmonary and systemic venous hypertension ensue, resulting in the syndrome of CHF. This syndrome is associated with decreased exercise tolerance, exertional and resting dyspnea, and lower extremity edema (Francis, 1998).

A normally functioning heart is capable of making precise adjustments in stroke volume to meet the body’s changing metabolic needs ranging from sleep
to aerobic exercise. These physiologic variations in stroke volume are possible because of the intrinsic elasticity of the myocardium that results in optimal ventricular emptying (cardiac output) without an increase in myocardial oxygen requirement or variation in mean arterial pressure. The intrinsic elasticity or contractility of the myocardium and cardiac output is affected by four interacting variables: inotropy, or the state of myocardial contractility; preload or the end diastolic filling volume; afterload, or that amount of pressure required by the ventricle to open the aortic valve (also known as systemic vascular resistance); and chronotropy, or heart rate.

The pathophysiologic outcome of decreased cardiac output is the constellation of symptoms known as CHF. Although there are many causes or conditions that can lead to CHF, the most common causes are uncontrolled hypertension, coronary artery disease, and mitral or aortic valve dysfunction. Despite the various etiologies, CHF represents a failure of cardiac output (CO) to meet the metabolic needs of the body. Once the diagnosis of CHF is made, the diagnostic distinction between systolic versus diastolic dysfunction should be made as early as possible because the long-term treatment of each differ.

Determining the ejection fraction (EF) is easiest way to differentiate between systolic and diastolic dysfunction. The two-dimensional transthoracic Doppler echocardiography yields the most diagnostic data and is the most cost effective way to evaluate the structure and function of the heart. Systolic dysfunction, characterized by an EF of less than 40%, is defined as a depression in the contractile force of the myocardium and often results in a thin and dilated heart muscle that is incapable of maintaining an adequate CO (Consensus Recommendations for Heart Failure, 1999). Systolic dysfunction is the most common cause of CHF and most often results from damage caused by MI (Abraham, 2000).

Diastolic dysfunction is an impairment in diastolic filling of the left ventricle and is secondary to the loss of muscle fiber elasticity. It is most often associated with long standing hypertension. Up to 40% of patients with symptomatic CHF have diastolic heart failure (Willerson, & Cohn, 1995). The mechanisms responsible for diastolic dysfunction, regardless of normal systolic function, are decreased ventricular compliance (or increased stiffness) and impaired ventricular relaxation. Impairment in diastolic filling of the left ventricle can result from ischemia, hypertrophy, reduction in beta-adrenergic tone, or increased myocardial connective tissue (Ruzumna, Gheorghiade, & Bonow, 1996). Patients with preserved systolic function (EF>50%) who have symptoms of heart failure are classified as having diastolic dysfunction (Ruzumna et al.). Over time, the increased work of the left ventricle causes hypertrophy of the muscle, further increases the contractility of the muscle fibers and results in impaired relaxation. The resulting non-compliant, hypertrophied left ventricle is unable to fill adequately leading to decreased stroke volume, decreased CO, and symptoms of CHF (Ruzumna, et al.). Subsequently, the low cardiac output stimulates the compensatory neurohormonal systems that increase circulating blood volume and filling pressures and result in further pulmonary congestion.

Compensatory Mechanisms

When there is a decreased CO and secondary decrease in mean arterial pressure, there is stimulation of several neurohormonal systems that act to maintain hemodynamic stability. Initially, these compensatory mechanisms are beneficial; however, over time they stress the myocardium further and exacerbate the CHF (McCance, & Huether, 1998).

In response to a decreased CO, baroreceptors located within the aortic arch and the carotid bodies stimulate the sympathetic nervous system (SNS) to release epinephrine and norepinephrine, which causes an increase in peripheral vascular resistance (PVR), heart rate, and contractility.

In turn, the redistribution of blood flow resulting from SNS stimulation decreases renal perfusion, causing activation of the renin-angiotensin-aldosterone system (RAAS). Renin secretion is stimulated by sodium and water depletion, decreased blood volume, and decreased renal perfusion. Subsequently, renin promotes the conversion of angiotensinogen to angiotensin I. Angiotensin I is converted to angiotensin II via the angiotensin converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor and also stimulates aldosterone secretion leading to sodium and water retention (McCance, & Huether, 1998). Figure 1 depicts this cascade of events.

Aldosterone plays a significant role in the pathophysiology of heart failure in many ways. It promotes the retention of sodium, stimulates the loss of magnesium and potassium, stimulates the activation of the SNS, inhibits parasympathetic effects, promotes myocardial and vascular fibrosis, causes baroreceptor dysfunction, and impairs arterial compliance (Pitt et al., 1999). The subsequent renal retention of sodium and water, coupled with an increase in PVR, ultimately leads to an increase in preload and afterload, which further contributes to pulmonary and vascular congestion and creates the cycle of symptoms characteristic of CHF (McCance, & Huether, 1998).

Chronic sympathetic stimulation, which happens in hypertension, can have adverse effects on the myocardium, including down-regulation of the beta receptors and ventricular remodeling. Down-regulation is a reduction of the beta-1 receptors within the myocardium, which over time results in a diminished response to catecholamine stimulation and leads to a diminished response to inotropic therapy (Connolly, 2000). This can present a problem when inotropic responsiveness is needed to treat an exacerbation of CHF.

Ventricular Remodeling

Ventricular remodeling is a complex pathophysiologic process that manifests as a change in size, shape, and function of the myocardium. Simply put, as a result of injury to the myocardium, myocytes die and do not regenerate. In an effort to maintain the stroke volume after the loss of contractile tissue, the surviving myocytes elongate and hypertrophy. As the ventricle enlarges and the myocytes slip, the ventricular wall thins out and begins to dilate, resulting in dilated cardiomyopathy (Cohn, Ferrari, & Sharpe, 2000). Remodeling can occur locally after an acute MI or globally as a result of cardiomyopathy. Left untreated, the change in left ventricular geometry can result in an alteration in papillary...
muscle function and lead to the development of secondary mitral insufficiency, which in turn leads to a further deterioration in cardiac output (Gheorghiade, et al., 1998).

Ventricular remodeling has emerged as an important factor in the progression of cardiovascular disease and has become a focus in the treatment of CHF (Cohen et al., 2000). Recent research has focused on the interruption and regulation of chronic adrenergic activation because of the part it plays in the progressive deterioration of left ventricular (LV) dysfunction.

It is important to note that the remodeling process begins long before the symptoms of CHF appear (Abraham, 2000); therefore, treatment should focus on symptom control and regression of the remodeling process. Finding the delicate balance between the necessary compensatory actions to maintain cardiac output without promoting the harmful effects of remodeling is problematic.

**Figure 1. Compensatory Events in Congestive Heart Failure**

Cardiac Output

\[ \downarrow \]

Baroreceptors Activated

Sympathetic Nervous System

\[ \downarrow \]

Release of
Epinephrine and Norepinephrine

\[ \downarrow \]

\( \uparrow \)HR, \( \uparrow \)SVR, \( \uparrow \)CO

---Tachycardia

---\( \uparrow \)Afterload (arterial constriction)

\( \uparrow \)Preload (venous constrictions)

---Renal vasoconstriction; activation of RAAS

---Toxicity to myocytes via unopposed catecholamines

Neurohormonal Systems

\[ \downarrow \]

Activation of RAAS Systems

\[ \downarrow \]

Release of Renin from Kidney

---Angiotensin Converting Enzyme: powerful vasoconstrictor

---Stimulates Aldosterone secretion

\[ \downarrow \]

\( \uparrow \)Total Plasma Volume

\( \uparrow \)SVR, \( \uparrow \)CO

---Volume overload (pulmonary and vascular congestion)

---\( \downarrow \)vasoconstriction

\( \uparrow \)Cardiac workload

\( \uparrow \)Left ventricular dysfunction

Cardiac Remodeling

\[ \downarrow \]

Vicious Cycle of CHF
The signs and symptoms of CHF may appear insidiously in patients with a chronic underlying cardiovascular problem, such as poorly controlled hypertension, or may appear precipitously in patients who have an acute MI. The clinical presentation of CHF varies depending upon the degree of cardiac decompensation, the cause of the precipitating event, and the overall health of the patient (Consensus Recommendations for Heart Failure, 1999).

Some patients may be dyspneic and able to communicate only in short sentences, while others with chronic CHF may appear comfortable and asymptomatic. Any patient who presents with a new onset of dyspnea on exertion (DOE), paroxysmal nocturnal dyspnea (PND), or orthopnea should be evaluated for CHF. The dyspnea results from pulmonary vascular congestion and is often worse after lying down (orthopnea) because of the increased venous return to the failing left ventricle of the heart. As an assessment of the degree of orthopnea, many clinicians ask about the number of pillows the patient requires when supine (i.e., one pillow, two pillows). Percussion can reveal dullness in the dependent aspects of the lungs secondary to fluid accumulation. Patients with pulmonary congestion also describe a non-productive "hacking" cough that develops a few hours after lying down and is usually relieved by sitting. Other physical exam findings can include moist crackles (rales) in the lung bases or throughout all lung fields, which may be accompanied by wheezing (Consensus Recommendations for Heart Failure, 1999; Shamsham, & Mitchell, 2000). In the case of acute pulmonary edema, the patient may report a cough with production of frothy sputum.

The cardiac examination may reveal a displaced point of maximal impulse (PMI), an indication of cardiomegaly. Heart sounds may be irregular depending on the underlying cardiac rhythm. Auscultation of the heart may reveal systolic and/or diastolic murmurs. Evidence of a third heart sound (S3) or ventricular gallop is considered the hallmark of ventricular failure and is a result of ventricular volume overload (McCance, & Huether, 1998; Shamsham, & Mitchell, 2000). The S3, or atrial gallop, is a presystolic murmur associated with filling against a non-compliant left ventricle; it is most often associated with diastolic dysfunction. Pulsus alternans is a rhythmic alternation of weaker and stronger pulsations in the peripheral arteries and is indicative of a failing or damaged left ventricle (Shamsham, & Mitchell).

Jugular venous distention (JVD), although not specific to heart failure, is commonly found on physical examination of patients with CHF. Patients with CHF may present with JVD because of fluid overload, right-sided heart failure, or a combination of both. Peripheral edema in ambulatory patients with CHF is almost always dependent and bilateral (McCance, & Huether, 1998). If the edema is unilateral, the clinician should consider other possible causes such as deep vein thrombosis.

Radiographic signs of CHF are manifested on the CXR when interstitial edema in the lungs increases. As the pulmonary congestion increases, there is a classic bilateral hilar haziness or opacity often referred to as a "butterfly" or "bat wing" pattern on the CXR. The degree of fluid overload will determine the height of the interstitial pattern. Evidence of Kerley B lines, horizontal lines representing dilated and thickened interlobular septa, are typically found in the bases of the lungs (Diettenmeier, 1995). Depending on whether the heart failure is acute or chronic, the CXR may also show an enlarged cardiac silhouette suggestive of cardiomegaly.

The ECG is often abnormal in patients with heart failure, but there are no specific changes that are diagnostic of CHF. Abnormalities on the ECG can include increased voltage through the precordial leads and left axis deviation (indicative of LV hypertrophy), atrial arrhythmias (such as atrial fibrillation or flutter) and sinus tachycardias. The ECG is always included as a part of the workup in CHF as it can be a quick diagnostic tool for detecting the presence of acute cardiac ischemia or MI. Any patient who presents with ischemic changes associated with an acute episode of heart failure should be hospitalized and have an individualized cardiac work up by a cardiologist.

Two-dimensional transthoracic Doppler echocardiography is the gold standard for evaluating systolic, diastolic, and valvular dysfunction, providing valuable information regarding ventricular chamber size, shape, wall motion, as well as an estimate of the EF. It is used for initial evaluation and for ongoing assessment of the progression or regression of ventricular remodeling (Consensus Recommendations for Heart Failure, 1999).

TREATMENT

Successful treatment of CHF requires a multidisciplinary approach that encompasses both pharmacologic and non-pharmacologic interventions. Advanced practice nurses are in an ideal position to provide such a multidisciplinary approach. Because patients with heart failure are known to be frequent users of the health care system, they require close monitoring of their disease (Connolly, 2000). Multiple studies have shown that patients who are educated and knowledgeable about their disease process and understand how their medications work and the importance of compliance have fewer hospital readmissions and experience an increase in functional quality of life (Rich, Bechham, & Wittenburg, 1995; Fonarow, Stevenson, & Walden, 1997; Dahl, & Penque, 2000). Patients should be taught the signs and symptoms of worsening CHF: increased dyspnea, development or worsening of orthopnea or PND, weight gain, and exercise intolerance or inability to perform the normal activities of daily living without increased fatigue. Up to 65% of hospital admissions are due to noncompliance with both pharmacologic and nonpharmacologic treatment regimes (Dahl, & Penque).

Nonpharmacologic therapies include a no added salt diet, which constitutes about 2-3 grams of salt per day (Weinberger, & Kenny, 2000). Patients should be instructed to avoid foods containing large amounts of sodium, such as highly processed foods, canned foods, and luncheon meats. A nutrition consult is helpful especially if the patient is overweight. Patients should be taught to look for words other than “salt” on food labels, for example sodium or potassium hydrochloride. Some patients may need to have their daily fluid restricted to 1.5-2.0 liters per day. This is a clinical judgment based on signs of congestion, fluid
overload, and weight gain. Patients should be instructed to weigh themselves daily or every other day and record the data in a log, which should be taken to every visit with the clinician. A weight gain of 2-3 pounds should trigger a visit to the clinician.

All patients with CHF should be encouraged to exercise to improve overall physical conditioning. The amount and type of exercise will depend on patient preference and functional class of the disease according to the New York Heart Association classification (NYHA). The NYHA classifies patients into one of four classes according to their dyspnea and fatigue. The established standard for assessment of physical capacity is an exercise test, which provides objective data regarding exercise time, distance, peak workload, and oxygen consumption (ACC/AHA, 1995).

This type of treadmill testing is expensive and is often bypassed for subjective data regarding the patient’s ability to tolerate common daily activities such as bathing, dressing, walking or climbing stairs. The basic differentiation between class is as follows: Class IV-patients with symptoms of CHF (dyspnea, fatigue, palpitations) while at rest; Class III-comfortable at rest but symptoms with less than ordinary exertion; Class II-symptoms on ordinary exertion; Class I-symptoms only occur as they would in a normal individual (Dahl, & Penque, 2000). This classification is useful only for assessment of functional capacity and is not meant to be used as a measure of the severity of left ventricular dysfunction. Casual walking or low intensity pool exercises are usually acceptable starting points from which patients can increase their intensity and endurance.

Patients with other comorbid conditions such as hypertension, obesity, hyperlipidemia, or diabetes mellitus should maintain optimum control of their disease processes. Patients should avoid non-steroidal anti-inflammatory medication because of the potential interaction with ACE inhibitors (Pinkowski, 1997). Patients who smoke should be instructed to quit and offered a referral to a smoking cessation group. Alcohol is a known cardiotoxin and patients with CHF should limit consumption to one drink a day. Patients with dilated cardiomyopathy secondary to long term alcohol consumption need to abstain completely and should be encouraged to attend Alcoholics Anonymous™ to maintain long-term sobriety.

Traditionally, the treatment of CHF has been based solely on symptom relief. It is now clear that deleterious changes in the myocardium occur before symptoms appear; therefore, treatment must include medications known to forestall these changes. Pharmacologic management includes the treatment of symptoms, as well as long-term management focused on modulating the neurohormonal systems and preventing further ventricular remodeling (Abraham, 2000; Connolly, 2000).

**Pharmacologic Treatment**

The pharmacologic treatment of CHF can include diuretics, beta-blockers, ACE inhibitors, digoxin, aspirin, and other agents (Dahl, & Penque, 2000). The following discussion centers around how these different classes of medications are useful in the management of CHF. The most commonly prescribed medications used in the treatment of CHF are listed in Table 1.

**ACE Inhibitors.** The use of angiotensin-converting enzyme (ACE) inhibitors in the treatment of heart failure is now considered a gold standard. The American College of Cardiology regards ACE inhibitors as the most important advancement in the management of chronic heart failure in the last decade (Nash, 1999). The unfortunate reality is that only 30%-40% of patients with CHF are receiving treatment with ACE inhibitors. Providing information regarding the function and therapeutic dosing of ACE inhibitors may improve the rate of utilization and treatment with this class of drugs.

The ACE inhibitors act by inhibiting the enzymatic conversion of angiotensin I to angiotensin II, a potent vasoconstrictor. This decreases afterload by blocking the production of angiotensin II and decreases preload and water retention by inhibiting the stimulation of the adrenal cortex to release aldosterone. Additionally, ACE inhibitors also inhibit the breakdown of bradykinin, a potent vasoactive peptide that stimulates the endothelium to produce nitric oxide, another potent vasodilator. The use of ACE inhibitors not only provides symptomatic improvement for patients; the cardioprotective effect helps prevent further ventricular remodeling (Greisinger, Espadas, & Ashton, 1998; Pinkowski, 1997). The benefits of ACE inhibition are usually seen during the first 90 days of therapy and extend to all patients, regardless of age, sex, etiology of CHF or NYHA class (Pinkowski). The ACE inhibitors most commonly used in the treatment of CHF are found in Table 1 with suggested dosing regimens.

Nonsteroidal anti-inflammatory drugs (NSAIDS) and aspirin can blunt the effects of ACE inhibitors by interfering with bradykinin and other vasoactive prostaglandins and should not be prescribed concurrently. The potential adverse effects of using ACE inhibitors include: hypotension, a decline or worsening of renal function, hyperkalemia, and cough. Hypotension is the most common side effect and is of primary concern if it is associated with a decrease in renal function (Pinkowski, 1997; Nash, 1999). Patients who have a propensity toward hypotension can usually tolerate ACE inhibitors if they are introduced at low doses and titrated upward. Diuretic-mediated intravascular volume contraction also increases the risk of hypotension, especially when prescribed prior to the introduction of ACE inhibitor therapy. When using diuretics with ACE inhibitors it is prudent to adjust the diuretic to the lowest effective dose. Acute renal failure can result from the decreased renal perfusion pressures associated with the use of ACE inhibitors. In patients with renal artery stenosis, ACE inhibitors must be used with caution because they can potentiate decreased renal perfusion (Pinkowski). Potassium levels should be monitored along with renal function as ACE inhibitor therapy is initiated. Nonproductive cough is the most common side effect associated with the use of ACE inhibitors (Connolly, 2000). Patients who do not tolerate ACE inhibitors should be started on an angiotensin receptor blocker (ARB).

Research has shown that ARBs are often better tolerated by patients who have developed a cough with ACE inhibitors. The ARBs prevent and reverse all the effects of angiotensin II without the associated production and accumulation of bradykinin, which is linked to the persistent nonproductive cough and
Angioedema found with ACE inhibitors (Greisinger et al., 2000). Angiotensin receptor blockers are not recommended for first line therapy; they should only be used by those patients who cannot tolerate ACE inhibitors. Clinical trials are underway to determine whether the use of ARBs as monotherapy will have the same mortality benefit as ACE inhibitors (Gheorghiade et al., 1998).

**Beta-blockers.** Beta-blockers are another group of drugs that may be used judiciously in the treatment of CHF. Beta-blockers not only slow conduction time through the atrioventricular node, decreasing the chance of tachyarrhythmias, but they also help prevent the complications associated with acute ischemia. Beta-blockers act through inhibition of the sympathetic nervous.

<table>
<thead>
<tr>
<th><strong>DRUG (generic)</strong></th>
<th><strong>INITIAL DOSE</strong></th>
<th><strong>TARGET DOSE (maximum dose)</strong></th>
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</thead>
<tbody>
<tr>
<td><strong>ACE INHIBITORS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>captopril</td>
<td>6.25-25.0mg TID</td>
<td>50-100mg TID</td>
</tr>
<tr>
<td>enalapril maleate</td>
<td>2.5mg QD / BID</td>
<td>2.5-10mg BID</td>
</tr>
<tr>
<td>fosinopril sodium</td>
<td>5-10mg QD</td>
<td>20-40mg QD</td>
</tr>
<tr>
<td>lisinopril</td>
<td>2.5-5mg QD</td>
<td>5-20mg QD</td>
</tr>
<tr>
<td>quinapril HCl</td>
<td>5mg BID</td>
<td>10-20mg BID</td>
</tr>
<tr>
<td>ramipril</td>
<td>1.25-2.5mg BID</td>
<td>5mg BID</td>
</tr>
<tr>
<td>trandolapril</td>
<td>1.0mg QD</td>
<td>4.0mg QD</td>
</tr>
<tr>
<td><strong>BETA-BLOCKERS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>carvedilol</td>
<td>3.125-6.25mg BID</td>
<td>25-50mg BID</td>
</tr>
<tr>
<td>metoprolol</td>
<td>6.25-25mg BID</td>
<td>50-150mg QD</td>
</tr>
<tr>
<td><strong>DIURETICS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>orothiside</td>
<td>12.5-25mg QD</td>
<td>as needed +</td>
</tr>
<tr>
<td>metolazone</td>
<td>2.5mg QD</td>
<td>as needed +</td>
</tr>
<tr>
<td>furosemide</td>
<td>2.5-40mg QD</td>
<td>as needed +</td>
</tr>
<tr>
<td>spironolactone</td>
<td>12.5-25mg QD</td>
<td>12.5-50mg QD</td>
</tr>
<tr>
<td><strong>OTHERS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.125-0.25mg QD</td>
<td>0.125-0.5mg QD</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>10-25mg TID/QID</td>
<td>75mg TID/QID</td>
</tr>
<tr>
<td>isosorbide dinitrate</td>
<td>2.5-20mg QID</td>
<td>10-40mg QID</td>
</tr>
<tr>
<td>aspirin</td>
<td>81-162mg QD</td>
<td>81-162mg QD</td>
</tr>
</tbody>
</table>

* = titrate dose to patient need; QD= every day; BID= twice a day; TID= three times a day; QID= four times a day
(Data from Young, 2000; Branon, 1999; Frantz, 2000).
by inhibiting sarcolemmal sodium-potassium adenosine triphosphatase (ATP-ase) activity. Inhibition of ATP-ase within the kidney decreases renal tubular reabsorption of sodium and suppresses the renin-angiotensin system. The inhibitory effects on the sympathetic nervous system in combination with reduced neurohormonal activity make digoxin an important pharmacologic weapon for controlling CHF.

Digoxin is not recommended for use in patients with pure diastolic dysfunction unless it is used as adjunct therapy in the modulation of the neurohormonal system or for control of atrial arrhythmias. Dosing of digoxin is affected by the patient's age, weight, and renal function. There is a narrow therapeutic index (0.8-2.0ng/mL) which must be monitored to help guide maintenance dosing of digoxin. Digoxin toxicity is potentiated by hypoxemia, electrolyte imbalances (hypokalemia and hypomagnesemia), renal insufficiency, dehydration, and hypothyroidism. The side effects associated with digoxin toxicity include cardiac arrhythmias, particularly ventricular arrhythmias and heart block, gastrointestinal disturbances (anorexia, nausea, and vomiting) and visual disturbances.

**Diuretics.** Patients who present with fluid overload associated with CHF will usually require diuretic therapy. Caution must be used when initiating diuretics in combination with ACE inhibitors and beta-blocker therapy because of resultant hypotension. Dietary restriction of sodium and water should be initiated in conjunction with diuretic therapy. Diuretics inhibit the reabsorption of sodium and chloride at different sites along the renal tubules.

As diuretic therapy is introduced, patients must be monitored for overdosing of diuretics, which can cause azotemia and hypotension, as well as for under-dosing, which can result in fluid overload and a diminished response to ACE inhibitors and beta-blockers. Within two weeks of initiating diuretic therapy, patients should have a routine assessment of blood chemistry and electrolytes to determine renal function and electrolyte status. Diuretics are not recommended as monotherapy for CHF and are more easily adjusted for symptomatic relief in acute CHF when used in concert with other medications.

Thiazide diuretics are used for the initial treatment of patients with normal renal function and mild fluid overload. Those patients who present with more serious pulmonary congestion will benefit most from a loop diuretic such as furosemide. Dosing of diuretics is highly individualized with the goal of treatment being symptomatic relief of congestion. Electrolyte imbalances are a common side effect of diuretics. Hypokalemia in particular can potentiate digoxin toxicity. Patients who seem to have a propensity for developing hypokalemia may benefit from the addition of spironolactone, a potassium-sparing diuretic. Patients should be instructed to log their daily weight and any increase of 2 to 3 pounds or more should be reported to the clinician, who can then provide instructions for a supplemental dose of diuretics until the weight has returned to baseline.

Spironolactone is not only a diuretic but also an aldosterone antagonist that, when used in conjunction with ACE inhibitors, is considered a more effective modulator of the RAAS system.
then either one alone. Treatment with spironolactone was once contraindicated because of the potential for developing hyperkalemia; however, the Randomized Aldactone Evaluation Study (RALES) found that this was uncommon (Pitt et al., 1999). The RALES study demonstrated that treatment with spironolactone, in combination with an ACE inhibitor, loop diuretic, and digitalis, reduced the risk of death from all causes, death from cardiac causes, and hospitalization for cardiac causes among patients with CHF resulting from left ventricular systolic dysfunction.

**Aspirin and Warfarin.** The use of aspirin (ASA), an antiplatelet agent, has been clinically proven to reduce ischemic events in those patients with angina and in those recovering from MI (Gheorghieade et al., 1998). Because coronary artery disease (CAD) is a major cause of heart failure, ASA has become an integral part of medical management. Aspirin should be prescribed to all patients with heart failure secondary to CAD. However, because ASA may attenuate the effects of ACE inhibitors, lower doses are recommended.

Anticoagulation therapy (warfarin) in patients with CHF is usually reserved for those with a history of prior embolic events, atrial fibrillation, or those with an EF less than or equal to 20%. According to ACC/AHA task force guidelines, the risk of arterial thromboembolism in this population is between 0.9% and 5.5%. The most common contributing factor is systolic dysfunction with an EF between 20% and 25%. Another indication for long-term warfarin use is the presence of an intracardiac thrombus, although the value of this has not been firmly established. The optimum dose of warfarin is determined by achieving a therapeutic international normalized ratio (INR) between 2.0-3.0 for patients with atrial fibrillation and up to 3.5 for patients with a history of prior embolic event (Gheorghieade et al., 1998).

**Calcium Channel Blockers.** Calcium channel blockers (CCB) work through dilation of the arterial vessels in the coronary and systemic circulation. They are often effective in treatment of myocardial ischemia and/or angina. Although first and second generation CCBs are not beneficial in the treatment of CHF, amiodipine and felodipine have been used to enhance ventricular relaxation and improve compliance of the ventricle (Gheorghieade et al., 1998; Abraham, 1998). Calcium channel blockers also counteract the vasoconstricting effects of angiotensin II, which facilitates vasoconstriction.

**Diastolic Dysfunction**

Therapy for CHF secondary to diastolic dysfunction is aimed at treating underlying high blood pressure, reducing left ventricular hypertrophy, and reducing left ventricular filling pressure without reducing cardiac output (Ruzumna et al., 1996). The pharmacologic management of CHF due to diastolic dysfunction differs only because treatment is aimed at relaxation of the ventricle and therefore avoids inotropic agents. Patients with diastolic dysfunction require adequate preload and ventricular filling in order to maintain cardiac output, making maintenance of a normal sinus rhythm extremely important. The treatment of choice is a combination of a diuretic and a nitrate, most commonly hydralazine and isosorbide dinitrate (ACC/AHA Task Force Report, 1995). Nitrates act by enhancing ventricular relaxation and, together with diuretics, decrease ventricular pressure and volume and thus enhance cardiac output. All patients receiving nitrates should have an 8-10 hour nitrate-free period to prevent the development of nitrate tolerance.

**SUMMARY**

The management of CHF is very complex. Advanced practice nurses, armed with a better understanding of CHF and current guidelines for treatment, can positively impact the quality of life of their patients while simultaneously decreasing hospital admissions and health care costs. Many patients are maintained at optimal levels of functioning only through strict adherence to pharmacological and dietary treatment regimens. Although there is limited evidence thus far, it has been suggested that comprehensive chronic CHF treatment programs may produce better outcomes than “usual care” (Greisinger et al., 2000).

Advanced practice nurses take a multidisciplinary approach to treatment, emphasizing patient education on the importance of taking an active role in the treatment program and being compliant with the medical regimen. As the number of older adults with CHF continues to rise, so will the need to care for them effectively. In-depth knowledge of the pathophysiology and management of CHF is the first step in creating change. Establishing APN-directed multidisciplinary care for this growing population will have a significant impact on the quality of life of patients with CHF as well as decreasing the cost to an ever-increasing health care budget.

**REFERENCES**


