It is more important to know what sort of person has a disease than to know what sort of disease a person has.

Hippocrates

Nursing Management

Heart Failure

Mary Ann House-Fancher and Hatice Y. Foell

LEARNING OBJECTIVES

1. Compare the pathophysiology of systolic and diastolic ventricular failure.
2. Discuss the compensatory mechanisms involved in heart failure.
3. Describe the nursing and collaborative management of the patient with acute decompensated heart failure and pulmonary edema.
4. Describe the collaborative care and nursing management, including drug and nutritional therapy, of the patient with chronic heart failure.
5. Describe the indications for cardiac transplantation and the nursing management of cardiac transplant recipients.

KEY TERMS

cardiac transplantation, p. ...
diastolic failure (dysfunction), p. ...
heart failure, p. ...
paroxysmal nocturnal dyspnea, p. ...
pulmonary edema, p. ...
systolic failure (dysfunction), p. ...

Electronic Resources

Supplemental content related to Chapter 35 can be found …

Companion CD

- Stress-Busting Kit for Nursing Students
- Interactive Case Study: Heart Failure
- NCLEX Examination Review Questions
- Animation: Anatomic Location of Sinuses
- Patient and Family Instruction Guide in English and Spanish: Congestive Heart Failure

Evolve Website

http://evolve.elsevier.com/Lewis/medsurg

- Content Updates
- Key Points
- Concept Map Creator
- Expanded Audio Glossary
- Key Term Flash Cards

- Customizable Nursing Care Plan: Heart Failure
- Electronic Calculators
- WebLinks

HEART FAILURE

Heart failure (HF) is an abnormal clinical condition involving impaired cardiac pumping. It results in the characteristic pathophysiologic changes of vasoconstriction and fluid retention. Heart failure, formerly called congestive HF, is the terminology preferred today since not all patients with HF have pulmonary congestion. HF is not a disease. It is associated with numerous types of cardiovascular diseases, particularly long-standing hypertension, coronary artery disease (CAD), and myocardial infarction (Table 35-1). HF is characterized by ventricular dysfunction, reduced exercise tolerance, diminished quality of life, and shortened life expectancy.

In most industrial nations, HF has become a major health problem. In contrast to other cardiovascular diseases, HF is increasing in incidence. This increase is due, in part, to improved survival after cardiovascular events, and in part to the increased aging population. Currently about 5 million people in the United States have HF. The American Heart Association (AHA) estimates that 470,000 new cases are diagnosed each year. HF dramatically increases with advancing age. As the elderly population increases, HF incidence and prevalence will continue to rise. Approximately 1 in every 100 older adults has HF. The incidence of HF is similar in men and women. HF is the most common reason for hospital admission in adults older than 65 years.1

Heart failure is associated with high rates of morbidity, mortality, and economic costs.1 The in-hospital mortality for these patients is 4.1%, with a mean length of hospital stay of 6.5 days. Hospital readmissions rates for patients with HF are 20% at 30 days and 50% at 6 to 12 months. With hospital readmission, mortality increases to 10% at 30 days and up to 20% to 40% at 12 months.2 These statistics reflect the increasing challenge of treating patients with HF.

Reviewed by Kathleen K. Salati, MSN, CCRN, NP-C, Adult Nurse Practitioner/Heart Failure, Christiana Care Health System, Newark, Del., and Erlinda C. Wheeler, RN, DNS, Associate Professor, University of Delaware, Newark, Del.

844
Acute Causes of Heart Failure

Precipitating Causes of Heart Failure

Mechanism

8/6/06 10:40:12 AM

CO, Cardiovascular System

Chapter 35 Nursing Management: Heart Failure 845

TABLE 35-1 Causes of Heart Failure

<table>
<thead>
<tr>
<th>Chronic</th>
<th>Acute</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>Acute myocardial infarction</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Dysrhythmias</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>Pulmonary emboli</td>
</tr>
<tr>
<td>Congenital heart disease</td>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>Hypertensive crisis</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>Rupture of papillary muscle (e.g., mitral valve)</td>
</tr>
<tr>
<td>Anemia</td>
<td>Ventricular septal defect</td>
</tr>
<tr>
<td>Bacterial endocarditis</td>
<td>Myocarditis</td>
</tr>
<tr>
<td>Valvular disorders</td>
<td></td>
</tr>
</tbody>
</table>

CULTURAL AND ETHNIC HEALTH DISPARITIES

Heart Failure

- African Americans have a higher incidence of HF, develop HF at an earlier age, and experience higher rates of mortality related to HF than whites.
- African Americans experience more ACE inhibitor-related angioedema than whites.
- Isosorbide dinitrate and hydralazine (BiDil) is used for the treatment of HF in African Americans; drug is only approved for use in this ethnic group.
- Asians have an extremely high risk (50%) for ACE inhibitor-related cough.

ACE, Angiotensin-converting enzyme; HF, heart failure.

Etiology and Pathophysiology

CAD and advancing age are the primary risk factors for HF. Other factors, such as hypertension, diabetes, cigarette smoking, obesity, and high serum cholesterol, can also contribute to the development of HF. Hypertension is a major contributing factor, increasing the risk of HF approximately threefold. The risk of HF increases progressively with the severity of hypertension, and systolic and diastolic hypertension equally predict risk. Diabetes predisposes an individual to HF regardless of the presence of concomitant CAD or hypertension. Diabetes is more likely to predispose women than men to HF.

HF may be caused by any interference with the normal mechanisms regulating cardiac output (CO). CO depends on (1) preload, (2) afterload, (3) myocardial contractility, (4) heart rate (HR), and (5) metabolic state of the individual. (Preload and afterload are discussed in Chapter 32.) Any alteration in these factors can lead to decreased ventricular function and the resultant manifestations of HF.

The major causes of HF may be divided into two subgroups: (1) primary causes (see Table 35-1) and (2) precipitating causes (Table 35-2). Precipitating causes often increase the workload of the ventricles, resulting in a decompensated condition that leads to decreased myocardial function.

Pathology of Ventricular Failure. Heart failure is classified as systolic or diastolic failure (or dysfunction).

Systolic Failure. Systolic failure, the most common cause of HF, results from an inability of the heart to pump blood. It is a defect in the ability of the ventricles to contract (pump). The left ventricle (LV) loses its ability to generate enough pressure to eject blood forward through the aorta. Over time, the LV becomes thick-walled, dilated, and hypertrophied. The hallmark of systolic dysfunction is a decrease in the left ventricular ejection fraction (the percentage of total ventricular filling volume that is ejected during each ventricular contraction). Normal ejection fraction (EF) is greater than 55% of the ventricular volume. Systolic failure is caused by impaired contractile function (e.g., myocardial infarction), increased afterload (e.g., hypertension), cardiomyopathy, and mechanical abnormalities (e.g., valvular heart disease).

Diastolic Failure. Diastolic failure is an impaired ability of the ventricles to relax and fill during diastole. Approximately 20% to 40% of patients with HF have diastolic failure with a normal EF and systolic function in the presence of HF symptoms. Decreased filling of the ventricles will result in decreased stroke volume and CO. Diastolic failure is characterized by high filling pressures due to stiff or noncompliant ventricles and results in venous engorgement in both the pulmonary and systemic vascular systems. The diagnosis of diastolic failure is made on the basis of the presence of pulmonary congestion, pulmonary hypertension, ventricular hypertrophy, and a normal EF.

Diastolic failure is usually the result of left ventricular hypertrophy from chronic systemic hypertension, aortic stenosis, or hypertrophic cardiomyopathy. Diastolic failure is commonly seen in older adults, and predominantly women, as a result of myocardial fibrosis and hypertension (see Gender Differences Box). However, the majority of patients who present with HF and normal systolic function do not have an identifiable heart disease.

Less common is isolated right ventricular diastolic failure. This results from pulmonary hypertension (chronic or acute) and causes reduced right ventricular emptying, resulting in a low left ventricular filling pressure and reduced CO. Acute right ventricular failure can cause rapid cardiac demise despite a normal LV.

TABLE 35-2 Precipitating Causes of Heart Failure

<table>
<thead>
<tr>
<th>Cause</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>↓ O2-carrying capacity of the blood stimulating ↑ in CO to meet tissue demands</td>
</tr>
<tr>
<td>Infection</td>
<td>↑ O2 demand of tissues, stimulating ↑ CO</td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
<td>Changes the tissue metabolic rate, ↑ HR and workload of the heart</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>Indirectly predisposes to ↑ atherosclerosis; severe hypothyroidism decreases myocardial contractility</td>
</tr>
<tr>
<td>Dysrhythmias</td>
<td>May ↓ CO and ↑ workload and O2 requirements of myocardial tissue</td>
</tr>
<tr>
<td>Bacterial endocarditis</td>
<td>Infarction: ↑ metabolic demands and O2 requirements</td>
</tr>
<tr>
<td>Pulmonary disease (e.g., pulmonary embolism)</td>
<td>Valvular dysfunction: causes stenosis and regurgitation</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>↑ Pulmonary pressure and exerts pressure on the RV, leading to RV hypertrophy and failure</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>↑ Workload of the heart by ↑ vascular bed in the skeletal muscle</td>
</tr>
<tr>
<td>Nutritional deficiencies</td>
<td>May ↓ cardiac function by ↑ myocardial muscle mass and myocardial contractility</td>
</tr>
<tr>
<td>Hypervolemia</td>
<td>↑ Preload causing volume overload on the RV</td>
</tr>
</tbody>
</table>

CO, Cardiac output; HR, heart rate; RV, right ventricle.
Cardiovascular System

Section 7 Problems of Oxygenation: Perfusion

GENDER DIFFERENCES

Heart Failure

Men
- Men experience systolic dysfunction more frequently than women.
- Men with asymptomatic systolic dysfunction experience greater mortality benefit from ACE inhibitor therapy than women.

Women
- Women experience diastolic dysfunction more frequently than men.
- Women have a higher risk of ACE inhibitor-related cough than men.
- Benefits of long-term use of digoxin in women may not justify the risks (e.g., drug-related death) when compared to men.
- Women with heart failure experience major depression more frequently than men.

ACE, Angiotensin-converting enzyme.

Mixed Systolic and Diastolic Failure. Systolic and diastolic failure of mixed origin is seen in disease states such as dilated cardiomyopathy (DCM). DCM is a condition in which poor systolic function (weakened muscle function) is further compromised by dilated left ventricular walls that are unable to relax (see Chapter 37). These patients often have extremely poor EFs (less than 35%), high pulmonary pressures, and biventricular failure (both ventricles may be dilated and have poor filling and emptying capacity).

The patient with ventricular failure of any type has low systemic arterial blood pressure (BP), low CO, and poor renal perfusion. Poor exercise tolerance and ventricular dysrhythmias are also common. Whether a patient arrives at this point acutely from a myocardial infarction (MI) or chronically from worsening cardiomyopathy or hypertension, the body’s response to this low CO is to mobilize its compensatory mechanisms to maintain CO and BP.

Compensatory Mechanisms. HF can have an abrupt onset as with acute MI, or it can be an insidious process resulting from slow, progressive changes. The overloaded heart resorts to compensatory mechanisms to try to maintain adequate CO. The main compensatory mechanisms include (1) sympathetic nervous system activation, (2) neurohormonal responses, (3) ventricular dilation, and (4) ventricular hypertrophy.

Sympathetic Nervous System Activation. Sympathetic nervous system (SNS) activation is often the first mechanism triggered in low-CO states. It is the least effective compensatory mechanism. In response to an inadequate stroke volume and CO, there is increased SNS activation, resulting in the increased release of catecholamines (epinephrine and norepinephrine). This results in an increased HR and myocardial contractility, and peripheral vasoconstriction. Initially this increase in HR and contractility improves CO. However, over time these factors result in an increase in cardiac workload, myocardial dysfunction, and peripheral vasoconstriction. However, this increase in HR and contractility may initially increase CO. However, an increase in venous return to the heart, which is already volume overloaded, actually worsens ventricular performance.

Neurohormonal Response. As the CO falls, blood flow to the kidneys decreases. This is sensed by the juxtaglomerular apparatus in the kidney as decreased volume. In response, the kidneys release renin, which converts angiotensinogen to angiotensin I (see Chapter 45 and Fig. 45-4). Angiotensin I is subsequently converted to angiotensin II by a converting enzyme made in the lungs. Angiotensin II causes the adrenal cortex to release aldosterone, which results in sodium and water retention, and (2) increased peripheral vasoconstriction, which increases BP. This response is known as the renin-angiotensin-aldosterone system (RAAS).

Low CO causes a decrease in cerebral perfusion pressure. The posterior pituitary then secretes antidiuretic hormone (ADH). ADH increases water reabsorption in the renal tubules, causing water retention and therefore increased blood volume. As a result, blood volume is increased in a person who is already volume overloaded.

Other factors also contribute to the development of HF. The production of endothelin, produced by the vascular endothelial cells, is stimulated by ADH, catecholamines, and angiotensin II. Endothelin results in further arterial vasoconstriction and an increase in cardiac contractility and hypertrophy.

Locally, proinflammatory cytokines are released by cardiac myocytes in response to various forms of cardiac injury (e.g., MI). Two cytokines, tumor necrosis factor (TNF) and interleukin-1 (IL-1), further depress cardiac function by causing cardiac hypertrophy, contractile dysfunction, and myocyte cell death. Over time, a systemic inflammatory response is also mounted and accounts for the cardiac wasting, muscle myopathy, and fatigue that accompany advanced HF.

Activation of the SNS and the neurohormonal response lead to elevated levels of norepinephrine, angiotensin II, aldosterone, ADH, endothelin, and proinflammatory cytokines. Together, these factors result in an increase in cardiac workload, myocardial dysfunction, and ventricular remodeling. Remodeling involves hypertrophy of the cardiac myocytes, resulting in large, abnormally shaped contractile cells. This eventually leads to increased ventricular mass, changes in ventricular shape, and impaired contractility. Although the ventricles become larger, they become less effective pumps. All of these factors are overexpressed in HF and eventually perpetuate the downward spiral of progressive HF syndrome.

Dilation. Dilation is an enlargement of the chambers of the heart. It occurs when pressure in the heart chambers (usually the left ventricle) is elevated over time. The muscle fibers of the heart stretch in response to the volume of blood in the heart at the end of diastole. The degree of stretch is directly related to the force of the contraction (systole) (Frank-Starling law). Initially this increased contraction leads to increased CO and maintenance of arterial BP and perfusion. Initially dilation is an adaptive mechanism to cope with increasing blood volume. Eventually this mechanism becomes inadequate because the elastic elements of the muscle fibers are overstretched and can no longer contract effectively, thereby decreasing the CO.

Hypertrophy. In chronic HF, hypertrophy is an increase in the muscle mass and cardiac wall thickness in response to overwork and strain. It occurs slowly because it takes time for this increased muscle tissue to develop. Hypertrophy generally follows persistent or chronic dilation and thus further increases the contractile power of the muscle fibers. This will lead to an increase in CO and maintenance of tissue perfusion. However, hypertrophic heart muscle has poor contractility, requires more oxygen to perform work, has poor coronary artery circulation (tissue becomes more easily ischemic), and is prone to ventricular dysrhythmias.
Counterregulatory Mechanisms. The body’s ability to try to maintain balance is demonstrated by several counterregulatory processes. Natriuretic peptides (atrial natriuretic peptide [ANP] and b-type natriuretic peptide [BNP]) are hormones produced by the heart muscle that promote venous and arterial vasodilation (thus reducing afterload and preload). Natriuretic peptides are endothelin and aldosterone antagonists and enhance diuresis by increasing glomerular filtration rates (thus reducing preload and volume stress) and blocking the effects of the RAAS. In addition, they inhibit the development of cardiac hypertrophy and may have antiinflammatory effects. ANP is produced by the atrium, and BNP is produced by the ventricles. ANP is primarily triggered by increases in volume. BNP is primarily triggered by increased pressure. Prolonged atrial and ventricular distention (during HF) leads to a depletion of these factors. Nitric oxide (NO) is another substance released from the vascular endothelium in response to the compensatory mechanisms activated in HF. Like the natriuretic peptides, NO works to relax the arterial smooth muscle, resulting in vasodilation and decreased afterload.

Cardiac compensation occurs when compensatory mechanisms succeed in maintaining an adequate CO that is needed for tissue perfusion. Cardiac decompensation occurs when these mechanisms can no longer maintain adequate CO and inadequate tissue perfusion results.

Types of Heart Failure

HF is usually manifested by biventricular failure, although one ventricle may precede the other in dysfunction. Normally the pumping actions of the left and right sides of the heart are synchronized, producing a continuous flow of blood. However, as a result of pathologic conditions, one side may fail while the other side continues to function normally for a period of time. Because of the prolonged strain, both sides of the heart will eventually fail, resulting in biventricular failure.

Left-Sided Failure. The most common form of HF is left-sided failure (Fig. 35-1). Left-sided failure results from left ventricular dysfunction, which prevents normal blood flow and causes blood to back up into the left atrium and into the pulmonary veins. The increased pulmonary pressure causes fluid extravasation from the pulmonary capillary bed into the interstitium and then the alveoli, which is manifested as pulmonary congestion and edema.

![Fig. 35-1](https://example.com/picture.png)

Left-sided heart failure from elevated systemic vascular resistance. Left-sided heart failure leads to right-sided heart failure. Systemic vascular resistance and preload are exacerbated by the renin-angiotensin-aldosterone system. ADH, Antidiuretic hormone; LA, left atrial; LV, left ventricle; LVEDP, left ventricular end-diastolic pressure; RV, right ventricle.
Right-Sided Failure. Right-sided failure causes a backup of blood into the right atrium and venous circulation. Venous congestion in the systemic circulation results in jugular venous distention, hepatomegaly, splenomegaly, vascular congestion of the gastrointestinal (GI) tract, and peripheral edema. The primary cause of right-sided failure is left-sided failure. In this situation, left-sided failure results in pulmonary congestion and increased pressure in the blood vessels of the lung (pulmonary hypertension). Eventually, chronic pulmonary hypertension (increased right ventricular afterload) results in right-sided hypertrophy and failure. Cor pulmonale (right ventricular dilation and hypertrophy caused by pulmonary disease) can also cause right-sided failure (see Chapter 28). Right ventricular infarction may also cause isolated right ventricle (RV) failure.

Clinical Manifestations of Acute Decompensated Heart Failure

Regardless of etiology, acute decompensated heart failure (ADHF) typically manifests as pulmonary edema, an acute, life-threatening situation in which the lung alveoli become filled with serosanguineous fluid (Fig. 35-2). The most common cause of pulmonary edema is acute left ventricular failure secondary to CAD. (Other etiologic factors for pulmonary edema are listed in Chapter 28, Table 28-26.)

In most cases of ADHF, there is an increase in the pulmonary venous pressure caused by decreased efficiency of the LV. This results in engorgement of the pulmonary vascular system. As a result, the lungs become less compliant, and there is increased resistance in the small airways. In addition, the lymphatic system increases its flow to help maintain a constant volume of the pulmonary extravascular fluid. This early stage is clinically associated with a mild increase in the respiratory rate and a decrease in partial pressure of oxygen in arterial blood (PaO₂).

If pulmonary venous pressure continues to increase, the increase in intravascular pressure causes more fluid to move into the interstitial space than the lymphatics can drain. Interstitial edema occurs at this point. Tachypnea develops and the patient becomes symptomatic (short of breath out of proportion to activity level). If the pulmonary venous pressure increases further, the tight alveoli lining cells are disrupted and a fluid containing red blood cells (RBCs) moves into the alveoli (alveolar edema). As the disruption becomes worse from further increases in the pulmonary venous pressure, the alveoli and airways are flooded with fluid (see Fig. 35-2). This is accompanied by a worsening of the arterial blood gas values (i.e., lower PaO₂ and possible increased partial pressure of carbon dioxide in arterial blood [PaCO₂] and progressive respiratory acidemia).

Clinical manifestations of pulmonary edema are unmistakable. The patient is usually anxious, pale, and possibly cyanotic. The skin is clammy and cold from vasoconstriction caused by stimulation of the SNS. The patient has severe dyspnea, as evidenced by the use of accessory muscles of respiration, a respiratory rate greater than 30 breaths/minute, and orthopnea. There may be wheezing and coughing with the production of frothy, blood-tinted sputum. Auscultation of the lungs may reveal crackles, wheezes, and rhonchi throughout the lungs. The patient’s HR is rapid, and BP may be elevated or decreased depending on the severity of the HF.

Clinical Manifestations of Chronic Heart Failure

The clinical manifestations of chronic HF depend on the patient’s age, the underlying type and extent of heart disease, and which ventricle is failing to pump effectively. Table 35-3 lists the manifestations of right-sided HF and left-sided HF. The patient with chronic HF will probably have manifestations of biventricular failure.

Fatigue. Fatigue is one of the earliest symptoms of chronic HF. The patient notices fatigue after activities that normally are not tiring. The fatigue is caused by decreased CO, impaired perfusion to vital organs, decreased oxygenation of the tissues, and anemia. Anemia can result from poor nutrition, renal disease, or drug therapy (e.g., angiotensin-converting enzyme inhibitors).

Dyspnea. Dyspnea (shortness of breath) is a common manifestation of chronic HF. It is caused by increased pulmonary pressures secondary to interstitial and alveolar edema. Dyspnea can occur with mild exertion or at rest. Orthopnea is shortness of breath that occurs when the patient is in a recumbent position. Paroxysmal nocturnal dyspnea (PND) occurs when the patient is asleep. It is caused by the reabsorption of fluid from dependent body areas when the patient is recumbent. The patient awakens in a panic, has feelings of suffocation, and has a strong desire to seek relief by sitting up. Careful questioning of patients often reveals adaptive behavior such as sleeping with two or more pillows to aid breathing. Because there are increased pulmonary pressures and fluid accumulation in the lung tissues, the patient may have a persistent, dry cough, unrelied with position change or over-the-counter cough suppressants. A dry, hacking cough may be the first clinical symptom of HF.

Tachycardia. Tachycardia is an early clinical sign of HF. One of the body’s first mechanisms to compensate for a failing ventricle...
Angina-type pain may accompany either ADHF or chronic HF. A person with chronic HF who has decreased CO will also have impaired renal perfusion and decreased urinary output during the day. However, when the person lies down at night, fluid movement from interstitial spaces back into the circulatory system is enhanced. This causes increased renal blood flow and diuresis. The patient may complain of having to void 6 or 7 times during the night. A person with chronic HF who has decreased CO may take β-blocker medications and may not show an increase in response to SNS stimulation.

**Edema.** Edema is a common sign of HF. It may occur in dependent body areas (peripheral edema), liver (hepatomegaly), abdominal cavity (ascites), and lungs (pulmonary edema and pleural effusion). If the patient is in bed, sacral and scrotal edema may develop. Pressing the edematous skin with the finger may leave a transient indentation (pitting edema). The development of dependent edema or a sudden weight gain of more than 3 lb (1.4 kg) in 2 days is often indicative of exacerbated HF.

**Nocturia.** A person with chronic HF who has decreased CO will also have impaired renal perfusion and decreased urinary output during the day. However, when the person lies down at night, fluid movement from interstitial spaces back into the circulatory system is enhanced. This causes increased renal blood flow and diuresis. The patient may complain of having to void 6 or 7 times during the night.

**Skin Changes.** Because tissue capillary oxygen extraction is increased in a person with chronic HF, the skin may appear dusky. It may also be cool and damp to the touch from diaphoresis. Often the lower extremities are shiny and swollen, with diminished or absent hair growth. Chronic swelling may result in pigment changes, causing the skin to appear brown or brawny in areas covering the ankles and lower legs.

**Behavioral Changes.** Cerebral circulation may be impaired with chronic HF secondary to decreased CO. The patient or family may report unusual behavior, including restlessness, confusion, and decreased attention span or memory. This may also be secondary to poor gas exchange and worsening HF.

**Chest Pain.** HF can precipitate chest pain due to decreased coronary perfusion from decreased CO and increased myocardial work. Angina-type pain may accompany either ADHF or chronic HF.

**TABLE 35-3 Clinical Manifestations of Heart Failure**

<table>
<thead>
<tr>
<th>Right-Sided Heart Failure</th>
<th>Left-Sided Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Signs</strong></td>
<td></td>
</tr>
<tr>
<td>RV heaves</td>
<td>LV heaves</td>
</tr>
<tr>
<td>Murmurs</td>
<td>Pulsus alternans</td>
</tr>
<tr>
<td>Jugular venous distention</td>
<td>(alternating pulses: strong, weak)</td>
</tr>
<tr>
<td>Edema (e.g., anterior tibias, medial malleoli, scrotum, sacrum)</td>
<td>↑ HR</td>
</tr>
<tr>
<td>Weight gain</td>
<td>PMI displaced inferiorly and posteriorly (LV hypertrophy)</td>
</tr>
<tr>
<td>↑ HR</td>
<td>↓ PaO₂, slight ↑ PaCO₂ (poor O₂ exchange)</td>
</tr>
<tr>
<td>Ascites</td>
<td>Crackles (pulmonary edema)</td>
</tr>
<tr>
<td>Anasarca (massive generalized body edema)</td>
<td>S₃ and S₄ heart sounds</td>
</tr>
<tr>
<td>Hepatomegaly (liver enlargement)</td>
<td>Pleural effusion</td>
</tr>
<tr>
<td></td>
<td>Changes in mental status</td>
</tr>
<tr>
<td></td>
<td>Restlessness, confusion</td>
</tr>
</tbody>
</table>

**Symptoms**

Fatigue

Anxiety, depression

Dependent, bilateral edema

Right upper quadrant pain

Anorexia and GI bloating

Nausea

Weakness, fatigue

Anxiety, depression

Dyspnea

Shallow respirations up to 32-40/min

Paroxysmal nocturnal dyspnea

Orthopnea (shortness of breath in recumbent position)

Dry, hacking cough

Nocturia

Frothy, pink-tinged sputum (advanced pulmonary edema)

GI, Gastrointestinal; HR, heart rate; LV, left ventricle; PaO₂, partial pressure of oxygen in arterial blood; PaCO₂, partial pressure of carbon dioxide in arterial blood; PMI, point of maximal impulse; RV, right ventricle.

**Weight Changes.** Many factors contribute to weight changes. Initially there may be a progressive weight gain from fluid retention. However, over time the patient is often too sick to eat. Abdominal fullness from ascerts and hepatomegaly frequently causes anorexia and nausea. Renal failure may also contribute to fluid retention. In many cases the muscle and fat loss is masked by the patient’s edematous condition. The actual weight loss may not be apparent until after the edema subsides.

**Complications of Heart Failure**

**Pleural Effusion.** Pleural effusion results from increasing pressure in the pleural capillaries. A transudation of fluid occurs from these capillaries into the pleural space. (Pleural effusion is discussed in Chapter 28.)

**Dysrhythmias.** Chronic HF causes enlargement of the chambers of the heart. This enlargement (stretching of the atrial and ventricular tissues) may cause an alteration in the normal electrical pathway, especially in the atria. When numerous sites in the atria fire spontaneously and rapidly (atrial fibrillation), the organized spread of atrial depolarization (contraction or systole) no longer occurs. This loss of the atrial conduction can reduce CO by 10% to 20%. Atrial fibrillation also promotes thrombus formation within the atria, which may break loose and form emboli. Patients with atrial fibrillation are at risk for stroke and require treatment with cardioversion, antidysrhythmics, and/or anticoagulants. (Dysrhythmias are discussed in Chapter 36.)

**Left Ventricular Thrombus.** With ADHF or chronic HF, the enlarged LV and decreased CO combine to increase the chance of thrombus formation in the LV. Current guidelines of the American College of Cardiology (ACC) and American Heart Association...
Cardiovascular System

**TABLE 35-4** Comparison of NYHA Functional Classification of Persons With Heart Disease and ACC/AHA Stages of Heart Failure

<table>
<thead>
<tr>
<th>NYHA Functional Classification of Heart Disease</th>
<th>ACC/AHA Stages of Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I: No limitation of physical activity. Ordinary physical activity does not cause fatigue, dyspnea, palpitations, or anginal pain.</td>
<td>Stage A: Patients at high risk of developing left ventricular dysfunction because of the presence of conditions that are strongly associated with the development of HF.</td>
</tr>
<tr>
<td>Class II: Slight limitation of physical activity. No symptoms at rest. Ordinary physical activity results in fatigue, dyspnea, palpitations, or anginal pain.</td>
<td>Stage B: Patients who developed structural heart disease that is strongly associated with the development of HF but who have never shown signs of HF.</td>
</tr>
<tr>
<td>Class III: Marked limitation of physical activity. Usually comfortable at rest. Ordinary physical activity causes fatigue, dyspnea, palpitations, or anginal pain.</td>
<td>Stage C: Patients who have current or prior symptoms of HF associated with underlying structural heart disease.</td>
</tr>
<tr>
<td>Class IV: Inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of angina may be present even at rest. If any physical activity is undertaken, discomfort is increased.</td>
<td>Stage D: Patients with advanced structural heart disease and marked symptoms of HF at rest despite maximal medical therapy and who require specialized interventions.</td>
</tr>
</tbody>
</table>

ACC/AHA, American College of Cardiology/American Heart Association; HF, heart failure; NYHA, New York Heart Association

**TABLE 35-5** B-Type Natriuretic Peptide Levels*

<table>
<thead>
<tr>
<th>BNP</th>
<th>HF very improbable</th>
<th>BNP 100-500 pg/ml</th>
<th>BNP &gt;500 pg/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>HF</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Levels will be temporarily elevated in patients receiving nesiritide (Natrecor) and may be elevated in patients with chronic, stable HF.

BNP, B-type natriuretic peptide; HF, heart failure.

(ACC/AHA) recommend anticoagulation in patients with HF and atrial fibrillation or very poor left ventricular function (e.g., EF <20%). Once a thrombus has formed, it may also decrease left ventricular contractility, decrease CO, and further worsen the patient’s perfusion. The development of emboli from the thrombus also places the patient at risk for stroke.

**Hepatomegaly.** HF can lead to severe hepatomegaly, especially with RV failure. The liver lobules become congested with venous blood. The hepatic congestion leads to impaired liver function. Eventually liver cells die, fibrosis occurs, and cirrhosis can develop (see Chapter 44).

**Renal Failure.** The decreased CO that accompanies chronic HF results in a decrease perfusion to the kidneys and can lead to renal insufficiency or failure (see Chapter 47).

**Classification of Heart Failure**

The New York Heart Association (NYHA) has developed functional guidelines for classifying people with heart disease. The classification is based on the person’s tolerance to physical activity. The ACC/AHA have defined stages for people with HF beginning with people at risk for HF and progressing to people with advanced HF (Table 35-4).

**Diagnostic Studies**

Diagnosing HF is often difficult since neither patient signs nor symptoms are highly specific, and both may mimic many other medical conditions, such as anemia or lung disease. The primary goal in diagnosis is to determine the underlying etiology of HF.

Measures to assess the cause and degree of HF include a thorough history, physical examination, chest x-ray, electrocardiogram (ECG), laboratory data (cardiac enzymes, BNP, serum chemistries, liver function studies, thyroid function studies, and complete blood count), hemodynamic assessment, echocardiogram, stress testing, and cardiac catheterization. EF can be measured using echocardiography and/or nuclear imaging studies (see Table 32-7). EF can be used to differentiate between systolic and diastolic HF, an important distinction to make in the early treatment of HF. BNP levels are used to assist in the diagnosis of HF. In general, levels correlate positively with the degree of left ventricular dysfunction and can help to differentiate dyspnea caused by HF from other causes of dyspnea (e.g., exacerbation of chronic obstructive pulmonary disease) (Table 35-5). Diagnostic studies used for the patient with ADHF are presented in Table 35-6, and those for the patient with chronic HF are presented in Table 35-7.

A complete history, physical, diagnostic, and laboratory assessment is necessary to identify the cause of HF, aggravating factors, potential risk factors that may influence outcomes, and current clinical status. The patient’s comorbid conditions, especially active chronic conditions, may act as exacerbating factors affecting the plan of care and influencing the timing and intensity of therapies.

**NURSING and COLLABORATIVE MANAGEMENT**

**ACUTE DECOMPENSATED HEART FAILURE AND PULMONARY EDEMA**

With the addition of new pharmaceutical agents and device therapies, the management of ADHF and chronic HF has dramatically changed in the last 5 years. The goals of therapy for both ADHF and chronic HF are to decrease patient symptoms, reverse ventricular remodeling, improve quality of life, and decrease mortality and morbidity. Management therapies are similar for both clinical conditions.

The challenge of planning and providing nursing care that promotes the best possible clinical outcomes to the patient with...
ADHF remains a complex task. Because of the large number of patients and the high cost of care associated with hospital readmissions, acute and critical care nurses must develop and implement strategies that are associated with improved outcomes. The Joint Commission on Accreditation of Healthcare Organizations recently established four core measures in the acute management of patients with HF to promote adherence to basic standards of evidence-based care (Table 35-8).

The optimal treatment of ADHF remains a challenge and an important area of research today. The use of various diuretic regimens, vasoactive drugs, newer pharmacologic agents, ultrafiltration, and novel device therapy is being explored. Data from the Acute Decompensated Heart Failure National Registry (ADHERE) indicate that patients receiving early treatment with intravenous (IV) diuretics and vasoactive drugs have better outcomes and shorter hospital and intensive care unit (ICU) stays than patients who have delays in treatment. This information has led to new protocols from the ACC/AHA regarding the treatment of ADHF.7 This would include the use of oxygen therapy, diuretics, vasodilators, and possibly inotropic agents. Circulatory assist devices may also be used for the acutely ill patient to preserve heart and target organ function. Table 35-6 lists the major components of the therapeutic approach.

### Decreasing Intravascular Volume

Decreasing intravascular volume with the use of diuretics reduces venous return. A loop diuretic (e.g., furosemide [Lasix], metanide [Bumex]) may be used to decrease volume because it may be administered by IV bolus and its action within the kidney occurs rapidly. By decreasing venous return to the LV and thereby reducing preload, the overfilled LV may contract more efficiently and improve CO. This increases left ventricular function, decreases pulmonary vascular pressures, and improves gas exchange.

The use of a loop diuretic may be delayed until the patient is assessed for hemodynamic and renal function and started on other vasodilator agents. The use of IV loop diuretic therapy alone has

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**Table 35-6: Collaborative Care for Acute Decompensated Heart Failure and Pulmonary Edema**

<table>
<thead>
<tr>
<th>Diagnostic</th>
<th>Collaborative Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>History and physical examination</td>
<td>Treatment of underlying cause</td>
</tr>
<tr>
<td>ABGs, serum chemistries, cardiac enzymes, BNP level, liver function tests</td>
<td>High Fowler’s position</td>
</tr>
<tr>
<td>Chest x-ray</td>
<td>O2 by mask or nasal catheter; BiPAP</td>
</tr>
<tr>
<td>Hemodynamic monitoring</td>
<td>BP, HR, RR, urinary output at least q1hr</td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>Continuous ECG and pulse oximetry monitoring</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>Hemodynamic monitoring (e.g., intraarterial BP, PAWP, CO)</td>
</tr>
<tr>
<td>Nuclear imaging studies</td>
<td>Drug therapy: diuretics IV (furosemide [Lasix]); nitroglycerin IV; morphine IV; nesiritide (Natrecor); inotropic therapy (see Table 35-9)</td>
</tr>
<tr>
<td>(see Table 32-7)</td>
<td>Daily weights</td>
</tr>
<tr>
<td>Cardiac catheterization</td>
<td>Possible cardioversion (e.g., atrial fibrillation)</td>
</tr>
<tr>
<td></td>
<td>Endotracheal intubation and mechanical ventilation</td>
</tr>
<tr>
<td></td>
<td>Circulatory assist devices (e.g., intraaortic balloon pump, ventricular assist device)</td>
</tr>
</tbody>
</table>

**Table 35-7: Collaborative Care for Chronic Heart Failure**

<table>
<thead>
<tr>
<th>Diagnostic</th>
<th>Collaborative Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>History and physical examination</td>
<td>Treatment of underlying cause</td>
</tr>
<tr>
<td>Serum chemistries, cardiac enzymes, BNP level, liver function tests</td>
<td>Oxygen therapy at 2-6 L/min by nasal catheter</td>
</tr>
<tr>
<td>Chest x-ray</td>
<td>Rest-activity periods</td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>Drug therapy (see Table 35-9)</td>
</tr>
<tr>
<td>Echocardiography</td>
<td>Daily weights</td>
</tr>
<tr>
<td></td>
<td>Sodium-restricted diet</td>
</tr>
<tr>
<td>Nuclear imaging studies</td>
<td>Circulatory assist devices (e.g., ventricular assist device)</td>
</tr>
<tr>
<td>(see Table 32-7)</td>
<td>Cardiac resynchronization therapy with internal cardioverter-defibrillator</td>
</tr>
<tr>
<td>Cardiac catheterization</td>
<td>Cardiac transplantation</td>
</tr>
</tbody>
</table>

**Table 35-8: Core Measures for Heart Failure**

- Written discharge instructions or educational material must be given to the patient or caregiver and include all of the following: activity level, diet, discharge medications, follow-up appointment, weight monitoring, and symptom management.
- Patients with known systolic dysfunction of moderate to severe impairment (ejection fraction less than 40%) and without contraindication to angiotensin-converting enzyme inhibitor will be prescribed an angiotensin-converting enzyme inhibitor at hospital discharge. An angiotensin receptor blocker is an acceptable alternative for patients with contraindication to angiotensin-converting enzyme inhibitors.
- Patients who are current smokers or former smokers who quit in the past 12 months will be given smoking cessation advice or counseling during the hospital stay.

**BNP, B-type natriuretic peptide; ECG, electrocardiogram**
Cardiovascular System

been shown to be associated with an increased risk of fatal dysrhythmias, aggravated renal dysfunction, and enhanced activation of the RAAS and SNS. Overall, the effects of loop diuretics actually increase systemic vascular resistance (afterload), decrease preload, and cause electrolyte imbalances.10

Ultrafiltration, or aquapheresis, is an option for the patient with volume overload. Ultrafiltration has generally been achieved through hemodialysis or central venous access. Newer technology allows removal of up to 500 ml/hr of fluid through either a peripheral or central venous line without significantly changing mean arterial pressure. When ultrafiltration is performed, volume is removed similar to hemodialysis but without hemodynamic instability. This therapeutic modality may be an appropriate adjuvant therapy for patients with HF and renal failure. Current research trials are ongoing to evaluate this modality for the patient with ADHF.9 (Ultrafiltration is discussed in Chapter 47.)

■ Decreasing Venous Return
Decreasing venous return (preload) reduces the amount of volume returned to the LV during diastole. This can be accomplished by placing the patient in a high Fowler’s position with the feet horizontal in the bed or dangling at the bedside. This position helps decrease venous return because of the pooling of blood in the extremities. This position also increases the thoracic capacity, allowing for improved ventilation. IV nitroglycerin is a vaso dilator used in the treatment of ADHF. It reduces circulating volume by decreasing preload and also increases coronary artery circulation by dilating the coronary arteries. Therefore nitroglycerin reduces preload, slightly reduces afterload (in high doses), and increases myocardial oxygen supply. When titrating IV nitroglycerin, BP is monitored frequently (every 5 to 10 minutes) to avoid symptomatic hypotension.

■ Decreasing Afterload
Afterload is the resistance against which the LV must pump; that is, it is the amount of work the LV has to produce to eject blood into the systemic circulation. Systemic vascular resistance (SVR) is a determinant of afterload, as is left ventricular filling. If afterload is reduced, the CO of the LV improves and thereby decreases pulmonary congestion.

IV sodium nitroprusside (Nipride) is a potent vasodilator that reduces preload and afterload. Because of its rapid onset of action and potent effects on the vascular system, it is the drug of choice for the patient with ADHF and pulmonary edema. By reducing both preload and afterload (by arterial and venous dilation), myocardial contracture improves, increasing CO and reducing pulmonary congestion. Complications of IV sodium nitroprusside include (1) hypotension and (2) thiocyanate toxicity, which can develop after 48 hours of use. Patients receiving sodium nitroprusside must have their BP monitored frequently (every 5 to 10 minutes) during the titration of the drug because profound hypotension is the principal adverse effect.

Morphine sulfate also reduces preload and afterload and is frequently used in the treatment of ADHF and pulmonary edema. It dilates both the pulmonary and systemic blood vessels, a goal in decreasing pulmonary pressures and improving the gas exchange. Morphine sulfate also reduces anxiety and may assist in reducing dyspnea.

Nesiritide (Natrecor) is an IV vasoactive therapy for ADHF. It is a recombinant form of BNP and causes both arterial and venous dilation. The main hemodynamic effects of nesiritide include (1) a reduction in pulmonary artery wedge pressure (PAWP) and (2) an increase in CO without increasing myocardial oxygen consumption or the occurrence of dysrhythmias.11 In addition, renal perfusion is enhanced, thus protecting the kidney. Nesiritide also has direct renal effects, resulting in the inhibition of the RAAS and promotion of natriuresis and diuresis.9 Although classified as a vasodilator, nesiritide is also referred to as a neurohormonal blocking agent and is indicated for short-term treatment of ADHF. The main adverse effect of nesiritide is asymptomatic hypotension, so BP should be closely monitored.

■ Improving Gas Exchange and Oxygenation
Gas exchange may be improved by several measures. IV morphine sulfate decreases oxygen demands, which may be raised as a result of anxiety and subsequent increased musculoskeletal and respiratory activity. Administration of oxygen helps increase the percentage of oxygen in inspired air. (Oxygen therapy is discussed in Chapter 29.) In severe pulmonary edema the patient may need noninvasive ventilatory support (e.g., bilevel positive airway pressure [BiPAP]) or intubation and mechanical ventilation. (Ventilatory support is discussed in Chapter 66.)

■ Improving Cardiac Function
In a patient who is or becomes hemodynamically unstable—that is, becomes progressively hypotensive, has an HR that is abnormally fast or slow, develops dysrhythmias, or becomes hypoxic with cool and clammy skin—nursing care becomes more urgent and treatment protocols may require aggressive, complex therapies. The use of diuretics, morphine sulfate, and vasodilators may not be sufficient to control symptoms. The addition of inotropic therapy may be warranted, as well as the initiation of hemodynamic monitoring to evaluate the effectiveness of interventions. Once a pulmonary artery catheter is in position, accurate measurement of CO, pulmonary artery pressure (PAP), and PAWP may be made and therapy instituted and titrated to maximize CO. A PAWP of 14 to 18 mm Hg will generally achieve the goal of increasing CO. (Hemodynamic monitoring is discussed in Chapter 66.)

Digitalis is a positive inotrope that improves left ventricular function. Digitalis increases contractility but also increases myocardial oxygen consumption. Because digitalis requires a loading dose and time to accomplish hemodynamic improvement, it is not recommended for the initial treatment of ADHF.

Other positive inotropes that can be considered include the β-adrenergic agonists (e.g., dopamine [Intropin], dobutamine [Dobutrex], epinephrine, norepinephrine [Levophed]). Stimulation of β-adrenergic receptors results in an increase in cyclic adenosine monophosphate (cAMP) within the myocardial cells and an increase in contractility (inotropic effect) and HR. The β-adrenergic agonists are typically used as a short-term treatment of ADHF in the ICU and, most recently, on step-down or intermediate care units with ECG monitoring capability. Dopamine is a β-adrenergic

Drug Alert - Nitroprusside (Nipride)

- Too rapid rate of IV administration can reduce BP too quickly.
- Headache, nausea, dizziness, dyspnea, blurred vision, sweating, and restlessness can occur.
- Assess BP prior to administration and continuously during administration.

IV, Intravenous.

8/6/06 10:40:20 AM
agonist used for treatment of severe ADHF and cardiogenic shock. In addition to increasing myocardial contractility and SVR, activation of dopamine receptors in the kidneys dilates the renal blood vessels and enhances urine output. Unlike dopamine, dobutamine is a selective β-adrenergic agonist and works selectively on the receptors in the heart. Dobutamine does not increase SVR and may be preferred for short-term treatment of ADHF.12

**Drug Alert • Dopamine (Intropin)**
- Extravasation with tissue sloughing may occur with IV administration.
- Monitor IV site for extravasation to prevent necrosis.
- High dosages may produce ventricular dysrhythmias.

Potential problems related to long-term treatment with β-adrenergic agonists include tolerance, increased ventricular irritability, and increased need for oxygen by the myocardium. In addition, these drugs are only available for IV use. Inamrinone (Inocor) and milrinone (Primacor) are two phosphodiesterase inhibitors that have been called inodilators because they increase myocardial contractility (inotropic effect) and promote peripheral vasodilation. Inhibition of phosphodiesterase increases cAMP, which enhances calcium entry into the cell and improves myocardial contractility. They increase CO and reduce arterial pressure (decrease afterload). Studies have shown inamrinone to be superior to dobutamine or dopamine in improving cardiac function.12 Like dopamine and dobutamine, these drugs are only available for IV use. Adverse effects include dysrhythmias, thrombocytopenia, and hepatotoxicity.

Although these inotropic agents can effectively increase CO, reduce filling pressures, and lead to short-term clinical improvement, some data suggest that their use may be associated with increased overall mortality.11 Currently, inotropic therapy is only recommended for use in the short-term management of patients with ADHF who have not responded to conventional pharmacotherapy (e.g., diuretics, vasodilators, morphine sulfate).7

**Reducing Anxiety**

Reduction of anxiety is an important nursing function, since anxiety may increase the SNS response and further increase myocardial workload. Reducing anxiety may be facilitated by a variety of nursing interventions (see NCP 35-1) and the use of sedative medications (e.g., benzodiazepines, morphine sulfate). When morphine sulfate is used, the patient often experiences relief from dyspnea and, consequently, the anxiety that is often associated with dyspnea. Though morphine-induced respiratory depression is rare, the patient’s respiratory rate should be monitored.

Once the patient is more stable, determination of the cause of pulmonary edema is important. Diagnosis of systolic or diastolic failure will then determine further management protocols. Aggressive drug therapy may continue with IV forms of diuretics, vasodilators, and inotropes. Nursing care focuses on continual physical assessment, hemodynamic monitoring, and evaluating the patient’s response to treatment.

**Collaborative Care: Chronic Heart Failure**

The main goal in the treatment of chronic HF is to treat the underlying cause and contributing factors, maximize CO, provide treatment to alleviate symptoms, improve ventricular function, improve quality of life, preserve target organ function, and improve mortality and morbidity (see Table 35-7). The management of dysrhythmias is discussed in Chapter 36, hypertension in Chapter 33, valvular disorders in Chapter 37, and coronary artery disease in Chapter 34.

In a person with HF, oxygen saturation of the blood is reduced because the blood is not adequately oxygenated in the lungs. Administration of oxygen improves saturation and assists greatly in meeting tissue oxygen needs. Thus oxygen therapy helps relieve dyspnea and fatigue. Optimally either pulse oximetry or arterial blood gases (ABGs) are used to monitor the effectiveness of oxygen therapy.

Physical and emotional rest allows the patient to conserve energy and decreases the need for additional oxygen. The degree of
rest recommended depends on the severity of HF. A patient with severe HF may be on bed rest with limited activity. A patient with mild to moderate HF can be ambulatory with a restriction of strenuous activity. The patient should be instructed to participate in limited activities with adequate recovery periods.

Nonpharmacologic therapies are now being used in the management of HF patients who are receiving maximum medical therapy, continue to have NYHA Functional Class III or IV symptoms, and have a widened QRS interval. One therapy is biventricular pacing. Traditional pacemakers pace one or two chambers (e.g., atrium and/or ventricle). Cardiac resynchronization therapy (CRT) coordinates right and left ventricle contractility through biventricular pacing. The ability to have normal electrical conduction within the right and left ventricles increases left ventricular performance and CO. This additional therapy allows patients to increase their exercise capacity and decrease their overall symptoms. CRT does not prolong life, but does improve quality of life in patients with NYHA Functional Class III and IV HF. CRT therapy can be combined with traditional pacing capability as well as defibrillator technology. If the patient has ischemia-induced HF and an EF of <35%, the implementation and use of an implantable cardioverter-defibrillator (ICD) with CRT may be warranted. Life-threatening ventricular dysrhythmias (e.g., ventricular tachycardia) are a complication of the ischemic myocardium and can cause sudden cardiac death. The addition of the ICD in these patients has reduced the overall mortality. (Pacemakers and defibrillators are discussed in Chapter 36.)

Cardiac transplantation is one form of treatment for ADHF and chronic HF. However, the lack of donor hearts and the challenges of care make it an option for only a small number of patients with HF. Stringent criteria are necessary to select the few patients with advanced HF who can even hope to receive a transplanted heart. (Heart transplantation is discussed later in this chapter.)

Several mechanical options are available to sustain HF patients with deteriorating conditions, especially those awaiting cardiac transplantation. The intraaortic balloon pump (IABP) is frequently employed in the setting of MI or perioperatively during cardiac surgery. The IABP can be useful in the hemodynamically unstable HF patient because it decreases SVR, PAWP, and PAP as much as 25%, leading to improved CO. However, the limitations of bed rest, infection, and vascular complications preclude long-term use. (IABPs are discussed in Chapter 66.) Ventricular assist devices (VADs) provide highly effective long-term support for up to 2 years and have become standard care in many heart transplant centers. VADs are used as a bridge to transplantation, effectively increasing cardiac function until a donor heart becomes available for the patient. The use of a permanent, implantable VAD, known as destination therapy, is an option for patients with advanced NYHA Functional Class IV HF who are not candidates for heart transplantation. (VADs are discussed in Chapter 66.)

Drug Therapy: Chronic Heart Failure

General therapeutic objectives for drug management of chronic HF include the following: (1) identification of the type of HF and underlying causes, (2) correction of sodium and water retention and volume overload, (3) reduction of cardiac workload, (4) improvement of myocardial contractility, and (5) control of precipitating and complicating factors. The aims of treating HF are to improve symptoms, minimize side effects of treatment, decrease
morbidity, improve quality of life, and prolong survival. Current therapeutic approaches stress the role of diuretics, vasodilators, β-blockers, and inotropic agents (Table 35-9).

**Diuretics.** Diuretics are used in HF to mobilize edematous fluid, reduce pulmonary venous congestion, and reduce preload (see Table 33-8). If excess extracellular fluid is excreted, blood volume returning to the heart can be reduced and cardiac function improved.

Diuretics act on the kidney by promoting excretion of sodium and water. Many varieties of diuretics are available, and some have specific indications for use. Thiazide diuretics may be the first choice in chronic HF because of their convenience, safety, low cost, and effectiveness. They are particularly useful in treating edema secondary to HF and in controlling hypertension. The thiazides inhibit sodium reabsorption in the distal tubule, thus promoting excretion of sodium and water.

Loop diuretics (e.g., furosemide [Lasix], bumetanide [Bumex], torsemide [Demadex]) are potent diuretics. These drugs act on the ascending loop of Henle to promote sodium, chloride, and water excretion. Furosemide is commonly used in ADHF and chronic HF because it is slightly more predictable in its response. Problems in using loop diuretics include reduction in serum potassium levels, ototoxicity, and possible allergic reaction in the patient who is sensitive to sulfa-type drugs.

Spironolactone (Aldactone) is an inexpensive, potassium-sparing diuretic that promotes sodium and water excretion but blocks potassium excretion by blocking receptors for aldosterone in the distal renal tubules. More important, aldosterone receptor antagonism has been shown to be effective because it blocks the harmful neurohormonal effects of aldosterone on the heart blood vessels. Spironolactone appears to add to the benefits of angiotensin-converting enzyme (ACE) inhibitors, and is appropriate to use while renal function is adequate. Spironolactone may also be used in conjunction with other diuretics, such as furosemide.

**Drug Alert - Spironolactone (Aldactone)**
- Assess for hyperkalemia during treatment.
- Use with caution in patients taking digoxin as hyperkalemia may reduce the effects of digoxin.
- Instruct patient to avoid foods high in potassium (e.g., bananas, oranges, dried apricots).

**Vasodilators.** Vasodilator drugs are a class of drugs clearly shown to improve survival in HF. The goals of vasodilator therapy in the treatment of HF include (1) increasing venous capacity, (2) improving EF through improved ventricular contraction, (3) slowing the process of ventricular dysfunction, (4) decreasing heart size, (5) avoiding stimulation of the neurohormonal responses initiated by the compensatory mechanisms of HF, and (6) enhancing neurohormonal blockade.

Angiotensin-Converting Enzyme Inhibitors. The benefits of ACE inhibitors in the treatment of all stages of HF have been well documented. ACE inhibitors are useful in both systolic and diastolic HF, and they are the first-line therapy in the treatment of chronic HF. Examples of ACE inhibitors include captopril (Capoten), benazepril (Lotensin), and enalapril (Vasotec). Other examples of ACE inhibitors are discussed in Chapter 33 and listed in Table 33-8.

**Drug Alert - Captopril (Capoten)**
- Excessive hypotension may occur.
- Monitor patient for first-dose hypotension (first-dose syncope).
- Skipping doses or discontinuing the drug can result in rebound hypertension.

The conversion of angiotensin I to the potent vasoconstrictor angiotensin II requires the presence of ACE (see Chapter 45, Fig. 45-4). ACE inhibitors exert their effects by blocking this enzyme, resulting in decreased levels of angiotensin II. As a result, plasma aldosterone levels are also reduced. Thus ACE inhibitors are now regarded as neurohormonal blocking agents in the treatment of HF.

Because CO is dependent on afterload in chronic HF, the reduction in SVR seen with the use of ACE inhibitors produces a significant increase in CO. Furthermore, with the use of ACE inhibitors, although BP may be decreased, tissue perfusion is maintained or increased as a result of improved CO and diuresis is enhanced by the suppression of aldosterone. Other hemodynamic changes include a reduction in (1) PAP, (2) right arterial pressure, and (3) left ventricular filling pressure. As a neurohormonal blocker, ACE inhibitors also decrease the development of ventricular remodeling by inhibiting ventricular hypertrophy.

ACE inhibitor therapy used in combination with diuretic therapy has shown to be beneficial in decreasing mortality in patients with chronic HF. ACE inhibitors counterbalance the alteration in renal mechanisms responsible for sodium and water retention in patients with chronic HF. The use of ACE inhibitor and diuretic therapy has the effect of increasing exercise tolerance, improving EF, and decreasing hospital readmissions in patients with chronic HF.

Major side effects of ACE inhibitors include symptomatic hypotension, intractable cough, hyperkalemia, angioedema (allergic reaction involving edema of the face and airways), and renal insufficiency (when ACE inhibitors are used in high doses). Aging and baseline renal insufficiency slow the metabolism of ACE inhibitors and may therefore lead to increased serum drug levels. It is recommended that these drugs be started at the lowest dose and that BP and renal function (e.g., serum creatinine) be monitored at regular intervals. Adequate titration of ACE inhibitors to target doses that are associated with increased survival is critical. Overall, ACE inhibitors are well tolerated by patients.

**Nitrites.** Nitrites cause vasodilation by acting directly on the smooth muscle of the vessel wall. Their effects primarily involve increasing venous capacitance, dilating the pulmonary vasculature, and improving arterial compliance. Therefore the major hemody-
namic effect of nitrates is to decrease preload. Nitrates are of particular benefit in the management of myocardial ischemia related to HF because they promote vasodilation of the coronary arteries. One specific deterrent to the use of nitrates in HF is nitrate tolerance. In addition, men with HF may experience erectile dysfunction and as a result take an erectile agent (e.g., sildenafil [Viagra]). Erectile agents are contraindicated in patients taking nitrates as together they could precipitate profound hypotension.

**Human n-Type Natriuretic Peptide.** Nesiritide is a synthetic form of human BNP. Nesiritide has gained importance in the treatment of ADHF and is being studied for its use in the ongoing treatment of patients with chronic HF (see earlier discussion). Outpatient management of ADHF and chronic HF using parenteral natriuretic peptides is an emerging strategy that appears to be clinically effective, may lead to fewer hospitalizations, and may improve quality of life. When combined with the control of cardiovascular risk factors and correction of precipitating mechanisms for decompensation, it is hopeful that treatment of HF with natriuretic peptides in the outpatient setting may reduce morbidity and mortality.2

**β-Adrenergic Blockers.** The use of β-adrenergic blockers in the management of HF continues to evolve. Marked improvement in patient survival has been shown with the use of β-adrenergic blockers, specifically carvedilol (Coreg) and metoprolol (Toprol XL).12 β-Adrenergic blockers directly block the negative effects of the SNS on the failing heart, such as increased HR. It is recommended that they be used in combination with other therapies (e.g., ACE inhibitors, diuretics, digitalis). Because β-adrenergic blockade can reduce myocardial contractility, care must be taken to start gradually, increasing the dosage slowly every 2 weeks as tolerated by the patient. Major adverse effects include edema, worsening of HF, hypotension, fatigue, and bradycardia.12

**Drug Alert - Carvedilol (Coreg)**
- Overdose can produce profound bradycardia, hypotension, bronchospasm, and cardiogenic shock.
- Assess BP and pulse at beginning of treatment and q4hr.
- Abrupt withdrawal may result in sweating, palpitations, and headaches.

**Positive Inotropes.** The use of positive inotropic agents in the patient with HF is directed at improving cardiac contractility to increase CO, decrease left ventricular diastolic pressure, and decrease SVR. Types of inotropic agents used in treating HF are listed in Table 35-9.

**Digitalis Glycosides.** Digitalis glycosides (e.g., digoxin [Lanoxin]) have been used for more than 200 years and remain the mainstay in the treatment of HF. However, the use of digitalis preparations has recently been questioned. While they have shown to reduce symptoms, they have not been shown to prolong life.12 Digitalis preparations are particularly useful in the treatment of HF accompanied by atrial flutter and/or fibrillation with a rapid ventricular rate. They increase the force of cardiac contraction (inotropic action). They also decrease the conduction speed within the myocardium and slow the HR (chronotropic action). These actions allow for more complete emptying of the ventricles, thus diminishing the volume remaining in the ventricles during diastole. CO increases because of an increased stroke volume from improved contractility.

An individual receiving a digitalis preparation is prone to develop digitalis toxicity (Table 35-10). Early symptoms of toxicity include anorexia, nausea, and vomiting. Visual disturbances, such as “yellow” vision, can occur with digitalis toxicity. Dysrhythmias are a common but often a late indication of digitalis toxicity. Although almost any dysrhythmia can occur, the types most frequently found are premature ventricular beats, atrial fibrillation, and first-degree heart block.13

Hypokalemia, secondary to the use of potassium-depleting diuretics (e.g., thiazides, loop diuretics), is one of the most common causes of digitalis toxicity. Low serum potassium enhances the action of digitalis, causing a therapeutic dose to achieve toxic levels. Similarly, hyperkalemia inhibits the action of digitalis, causing a therapeutic dose to become subtherapeutic. Both hypo- and hyperkalemia also precipitate the development of dysrhythmias. Monitoring the serum potassium levels of patients receiving digitalis preparations and potassium-depleting and potassium-sparing diuretics is essential. Other electrolyte imbalances, such as hypercalcaemia and hypomagnesaemia, can also precipitate digitalis toxicity. (Manifestations of electrolyte imbalances are discussed in Chapter 17.)

Diseases of the kidney and liver increase the susceptibility to digitalis toxicity because most preparations are metabolized and eliminated by these organs. An older adult is especially prone to digitalis toxicity because digitalis accumulation occurs sooner with decreased liver and kidney function and slowed body metabolism, which occurs with aging.

The usual treatment of toxicity consists of withholding the drug until the symptoms subside. In the case of life-threatening toxicity, digoxin immune Fab (ovine) (Digibind) is an antidote that can be given IV. The treatment of life-threatening dysrhythmias is instituted as needed (see Chapter 36).

**β-Adrenergic Agonists.** β-Adrenergic agonists are typically used in the short-term treatment of ADHF refractory to conventional pharmacotherapy. Their role in long-term therapy of HF is controversial. Potential problems related to long-term treatment with β-adrenergic agonists include tolerance, increased ventricular irritability, and increased need for oxygen by the myocardium.12

**Calcium Sensitziers.** Calcium sensitizers are novel positive inotropic agents in the treatment of HF. They improve cardiac performance by interacting directly with contractile proteins without affecting intracellular calcium concentrations or increasing myocardial oxygen demand. They are used in patients who need inotropic support who are also at risk for myocardial ischemia. They produce cardioprotective effects while simultaneously enhancing ventricular contractile function. Levosimendan (Simdax) is a calcium sensitizer recently available in Europe and currently undergoing clinical trials in the United States.9

**Angiotensin II Receptor Blockers.** In patients who are unable to tolerate ACE inhibitors because of angioedema or intractable cough, angiotensin II receptor blockers such as losartan (Cozaar)
and valsartan (Diovan) may be used.\textsuperscript{16,17} These agents prevent the vasoconstrictor and aldosterone-secreting effects of angiotensin II by binding to the angiotensin II receptor sites. Efficacy of these drugs is similar to ACE inhibitors except that the incidence of adverse effects (e.g., intractable cough) is lower (see Table 33-8).

**Bidil.** A combination drug containing isosorbide dinitrate and hydralazine (BiDil) is used for the treatment of HF in African Americans who are already being treated with standard therapy. The drug is only approved for use with this ethnic group. As an antihypertensive agent, hydralazine relaxes the arteries and decreases the work of the heart. The antiangiotal agent, isosorbide dinitrate, relaxes the veins as well as the arteries. Isosorbide seems to work by releasing nitric oxide at the blood vessel wall, but its effect usually wears off after half a day. Hydralazine may prevent this loss of effect. How these two drugs work together is not fully known, but studies have shown a decrease in HF symptoms, hospitalizations, and mortality in African Americans. Common side effects of BiDil are headache and dizziness.

**Nutritional Therapy: Chronic Heart Failure**

Diet education and weight management are critical to the patient’s control of chronic HF. The nurse or diettitian should obtain a detailed diet history, determining not only what foods the patient eats and when, but also the sociocultural value of food to the patient. The nurse can use this information to assist the patient in making appropriate dietary choices when developing a diet plan. The National Heart, Lung, Blood Institute (NHLBI) provides useful dietary guidelines for heart healthy food preparation for people of various cultures (e.g., Hispanics, Native Americans, Asian Americans, African Americans). These are available online at www.nhlbi.nih.gov/health/index.htm#recipes. Diet and weight management recommendations must be individualized and culturally sensitive if the necessary changes are to be realized.

The edema of chronic HF is often treated by dietary restriction of sodium. The patient should be taught what foods are low and high in sodium and ways to enhance food flavors without the use of salt (e.g., substituting lemon juice and various spices). The degree of sodium restriction depends on the severity of the HF and the effectiveness of diuretic therapy. Diets that are severely restricted in sodium are rarely prescribed because they are unpalatable and patient compliance is poor. The Dietary Approaches to Stop Hypertension (DASH) diet is effective as a first-line therapy for many individuals with isolated systolic hypertension (see Chapter 33, Table 33-7). This diet is now also widely used for the patient with HF, with or without hypertension.

The average American adult’s daily dietary intake of sodium ranges from 7 to 15 g. A commonly prescribed diet for a patient with mild HF is a 2.5-g sodium diet (Table 35-11). All foods high in sodium should be eliminated (Tables 35-12 and 35-13). For more severe HF, sodium intake is restricted to 500 to 1000 mg (see Table 35-11). On this diet, milk, cheese, bread, cereals, canned soups, and some canned vegetables must be severely restricted. The patient and family must be instructed on how to read labels to look for sodium content.

Fluid restrictions are not commonly prescribed for the patient with mild to moderate HF. Diuretic therapy, ACE inhibitors, and digitalis preparations act as effective diuretics to promote fluid excretion. However, in moderate to severe HF and renal insufficiency, fluid restrictions are usually implemented.

Instructing patients to weigh themselves daily is important for monitoring fluid retention, as well as weight reduction. Patients should be instructed to weigh themselves at the same time each day, using the same scale and preferably before breakfast, while wearing the same type of clothing. This helps ensure valid comparisons from day to day and helps identify early signs of fluid retention. If a patient experiences a weight gain of 3 lb (1.4 kg) over 2 days or a 3- to 5-lb (2.3 kg) gain over a week, the primary care provider should be called.\textsuperscript{18}

**TABLE 35-11 NUTRITIONAL THERAPY Low-Sodium Diets**

<table>
<thead>
<tr>
<th>Sodium (mg)</th>
<th>Breakfast</th>
</tr>
</thead>
<tbody>
<tr>
<td>161</td>
<td>½ cup bran cereal</td>
</tr>
<tr>
<td>3</td>
<td>(½ cup Shredded Wheat cereal)†</td>
</tr>
<tr>
<td>149</td>
<td>1 slice whole wheat bread</td>
</tr>
<tr>
<td>1</td>
<td>1 medium banana</td>
</tr>
<tr>
<td>85</td>
<td>6 oz fruit yogurt, fat free</td>
</tr>
<tr>
<td>126</td>
<td>1 cup fat-free milk</td>
</tr>
<tr>
<td>5</td>
<td>2 tbs jelly</td>
</tr>
<tr>
<td>5</td>
<td>Coffee, 8 oz</td>
</tr>
</tbody>
</table>

| Lunch | 65 | 2 slices (3 oz) chicken breast, skinless |
| 299 | 2 slices whole wheat bread |
| 328 | 1 slice (% oz) American cheese |
| 54 | (1 slice [% oz Swiss cheese, natural]† |
| 90 | Large-leaf romaine lettuce |
| 90 | 2 slices tomato |
| 7 | 1 tbs mayonnaise, low fat |
| 7 | 1 medium peach |

| Dinner | 459 | ½ cup vegetarian spaghetti sauce |
| 260 | (6 oz no-salt-added tomato paste)† |
| 1 | 1 cup spaghetti |
| 349 | 3 tbs Parmesan cheese |
| 24 | Spinach salad |
| 10 | 1 cup fresh spinach leaves |
| 1 | ¼ cup fresh carrots (grated) |
| 1 | ½ cup fresh mushrooms (sliced) |
| 0 | 2 tbs vinaigrette dressing |
| 4 | ½ cup canned pears, juice pack |
| 4 | ½ cup corn, cooked from frozen |

| Snack | 4 | ½ cup almonds |
| 3 | ¼ cup dried apricots |
| 85 | 6 oz fruit yogurt, fat free |

**General Principles**

- Do not add salt or seasonings containing sodium when preparing foods.*
- Do not use salt at the table.*
- Avoid high-sodium foods (e.g., canned soups, processed meats, cheese, frozen meals).

**Sample Menu Plans for 2400-mg Sodium Diet**

- Limit milk products to 2 cups daily.

**Modifications for Other Low-Sodium Diets**

<table>
<thead>
<tr>
<th>Sodium (mg)</th>
<th>500-mg Sodium Diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>126</td>
<td>Restricted milk products to 1 cup daily. Limit meat to 4 oz daily. Use salt-free butter, bread, vegetables, and starches.</td>
</tr>
<tr>
<td>260</td>
<td>Use salt-free butter and vegetables.</td>
</tr>
</tbody>
</table>

*1 tbs of salt equals 2.3 g of sodium.
†Substitutes to reduce to 1500-mg sodium diet.
TABLE 35-13  NUTRITIONAL THERAPY
Sodium Content in Different Food Groups

<table>
<thead>
<tr>
<th>Food Groups</th>
<th>Sodium (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grains and Grain Products</strong></td>
<td></td>
</tr>
<tr>
<td>Cooked cereal, rice, pasta, unsalted, ½ cup</td>
<td>0-5</td>
</tr>
<tr>
<td>Ready-to-eat cereal, 1 cup</td>
<td>100-360</td>
</tr>
<tr>
<td>Bread, 1 slice</td>
<td>110-175</td>
</tr>
<tr>
<td><strong>Vegetables</strong></td>
<td></td>
</tr>
<tr>
<td>Fresh or frozen, cooked without salt, ½ cup</td>
<td>1-70</td>
</tr>
<tr>
<td>Canned or frozen with sauce, ½ cup</td>
<td>140-480</td>
</tr>
<tr>
<td>Tomato juice, canned, ½ cup</td>
<td>820</td>
</tr>
<tr>
<td><strong>Fruit</strong></td>
<td></td>
</tr>
<tr>
<td>Fresh, frozen, canned, ½ cup</td>
<td>0-5</td>
</tr>
<tr>
<td><strong>Low-Fat or Fat-Free Dairy Foods</strong></td>
<td></td>
</tr>
<tr>
<td>Milk, 1 cup</td>
<td>120</td>
</tr>
<tr>
<td>Yogurt, 8 oz</td>
<td>160</td>
</tr>
<tr>
<td>Natural cheeses, ½ oz</td>
<td>110-450</td>
</tr>
<tr>
<td>Processed cheeses, ½ oz</td>
<td>600</td>
</tr>
<tr>
<td><strong>Nuts, Seeds, and Dry Beans</strong></td>
<td></td>
</tr>
<tr>
<td>Peanuts, salted, ½ cup</td>
<td>120</td>
</tr>
<tr>
<td>Peanuts, unsalted, ½ cup</td>
<td>0-5</td>
</tr>
<tr>
<td>Beans, cooked from dried or frozen, without salt, ½ cup</td>
<td>400</td>
</tr>
<tr>
<td><strong>Meats, Fish, and Poultry</strong></td>
<td></td>
</tr>
<tr>
<td>Fresh meat, fish, poultry, 3 oz</td>
<td>30-90</td>
</tr>
<tr>
<td>Tuna, canned, water pack, no salt added, 3 oz</td>
<td>34-45</td>
</tr>
<tr>
<td>Tuna, canned, water pack, 3 oz</td>
<td>250-350</td>
</tr>
<tr>
<td>Ham (lean) roasted, 3 oz</td>
<td>1020</td>
</tr>
</tbody>
</table>

NURSING MANAGEMENT

CHRONIC HEART FAILURE

**Nursing Assessment**

Subjective and objective data that should be obtained from a patient with HF include those presented in Table 35-14.

**Nursing Diagnoses**

Nursing diagnoses for the patient with HF include, but are not limited to, those presented in NCP 35-1.

**TABLE 35-14  NURSING ASSESSMENT**

**Heart Failure**

**Subjective Data**

**Important Health Information**

Past health history: CAD (including recent MI), hypertension, cardiomyopathy, valvular or congenital heart disease, diabetes mellitus, thyroid or lung disease, rapid or irregular heart rate

**Medications:** Use of and compliance with any cardiac medications; use of diuretics, estrogens, corticosteroids, nonsteroidal antiinflammatory drugs, over-the-counter drugs, herbal supplements

**Functional Health Patterns**

Health perception–health management: Fatigue, depression, anxiety

Nutritional–metabolic: Usual sodium intake; nausea, vomiting, anorexia, stomach bloating; weight gain, ankle swelling

Elimination: Nocturia, decreased daytime urinary output, constipation

Activity–exercise: Dyspnea, orthopnea, cough; palpitations; dizziness, fainting

Sleep–rest: Number of pillows used for sleeping; paroxysmal nocturnal dyspnea, insomnia

Cognitive–perceptual: Chest pain or heaviness; RUQ pain, abdominal discomfort; behavioral changes; visual changes

**Objective Data**

**Integumentary**

Cool, diaphoretic skin; cyanosis or pallor, peripheral edema (right-sided heart failure)

**Respiratory**

Tachypnea, crackles, rhonchi, wheezes; frothy, blood-tinged sputum

**Cardiovascular**

Tachycardia, S3, S4, murmurs; pulsus alternans, PMI displaced inferiorly and posteriorly, jugular vein distention

**Gastrointestinal**

Abdominal distention, hepatosplenomegaly, ascites

**Neurologic**

Restlessness, confusion, decreased attention or memory

**Possible Findings**

Altered serum electrolytes (especially Na⁺ and K⁺), ↑ BUN, creatinine, or liver function tests; chest x-ray demonstrating cardiomegaly, pulmonary congestion, and interstitial pulmonary edema; echocardiogram showing increased chamber size, decreased wall motion, decreased EF; atrial and ventricular enlargement on ECG; ↓ O₂ saturation

**Planning**

The overall goals for the patient with HF include (1) a decrease in symptoms (e.g., shortness of breath, fatigue), (2) a decrease in peripheral edema, (3) an increase in exercise tolerance, (4) compliance with the medical regimen, and (5) no complications related to HF.

**Nursing Implementation**

Health Promotion. An important measure used to prevent HF is the treatment or control of the underlying heart disease. For example, in valvular disease, valve replacement should be planned before lung congestion develops. Coronary revascularization procedures should be performed in patients with CAD. Another important preventive measure concerns early and continued treatment of hypertension. Hyperlipidemic states in persons with CAD should be managed with diet, exercise, and medication. The use of antisympathomimetic agents or pacemakers is indicated for people with serious dysrhythmias or conduction disturbances. In addition, patients with HF should be counseled to obtain vaccinations against the flu and pneumonia.

**TABLE 35-12  Sodium Label Language**

<table>
<thead>
<tr>
<th>Phrase</th>
<th>What It Means</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium free or salt free</td>
<td>&lt;5 mg per serving</td>
</tr>
<tr>
<td>Very low sodium</td>
<td>35 mg or less sodium per serving</td>
</tr>
<tr>
<td>Low sodium</td>
<td>140 mg or less of sodium per serving</td>
</tr>
<tr>
<td>Low-sodium meal</td>
<td>140 mg or less of sodium per 3.5 oz</td>
</tr>
<tr>
<td>Reduced or less sodium</td>
<td>At least 25% less sodium than regular</td>
</tr>
<tr>
<td>Light in sodium</td>
<td>50% less sodium than regular</td>
</tr>
<tr>
<td>Unsalted or no salt added</td>
<td>No salt added to the product during process</td>
</tr>
</tbody>
</table>

Use caution with products advertised as no-salt replacements—they may contain high potassium.
When a patient is diagnosed with HF, preventive care should focus on slowing the progression of the disease. Knowledge of the importance of following the medication, diet, and exercise regimens is essential. Research has shown that exercise training (e.g., cardiac rehabilitation) does improve symptoms of chronic HF but is often underprescribed. The acute care nurse may request home nursing care for the patient and family to provide for follow-up care and to monitor the patient’s response to treatment. Early detection of signs and symptoms of worsening failure may help modify care and prevent an acute episode requiring future hospitalization.

**Acute Intervention.** Successful HF management depends on several important principles: (1) HF is a progressive disease, and treatment plans are established with quality-of-life goals; (2) symptom management is controlled by the patient with self-management tools (e.g., daily weights, drug regimens, diet and exercise plans); (3) salt and water must be restricted; (4) energy must be conserved; and (5) support systems are essential to the success of the entire treatment plan.

Many persons with HF will experience one or more episodes of ADHF. When they do, they are usually admitted through the emergency department, initially stabilized, and then managed in an ICU. Once their condition has improved, they will be transferred to a step-down or general unit. The nursing care plan for the patient with HF applies to the patient with stabilized ADHF or chronic HF (see NCP 35-1).

**Ambulatory and Home Care.** HF is a chronic illness for most persons. Important nursing responsibilities are (1) teaching the patient about the physiologic changes that have occurred, (2) assisting the patient to adapt to both the physiologic and psychological changes, and (3) integrating the patient and the patient’s family or
support system in the overall care plan. Research has revealed that patients with NYHA Functional Class III or IV HF have a high risk for anxiety and depression.2 In one large study, major depression was found to be more prevalent in female patients and patients less than 60 years of age.23 It must be emphasized to the patient that it was found to be more prevalent in female patients and patients less for anxiety and depression.22 In one large study, major depression patients with NYHA Functional Class III or IV HF have a high risk support system in the overall care plan. Research has revealed that is possible to live productively with this chronic health problem. It may be asymptomatic when HF is under control. It must be stressed that the disease is chronic and that medication must be continued to keep the HF under control.

The patient should evaluate the action of the prescribed drugs and be taught to recognize the manifestations of drug toxicity. The patient should also be taught how to take her or his pulse rate and to know under what circumstances drugs, especially digitalis and β-adrenergic blockers, should be withheld and a health care provider consulted. The pulse rate should always be taken for 1 full minute. A pulse rate lower than 50 beats/minute may be a contra-indication to taking a digitalis preparation or β-adrenergic blocker unless specified otherwise by the health care provider. However, in the absence of symptoms (e.g., heart block, ventricular ectopy, syncope), a pulse rate of less than 50 beats/minute may be acceptable. It may also be appropriate to instruct patients in home BP monitoring, especially for those HF patients with hypertension.

### OUTCOMES (NOC)

#### Respiratory Status: Gas Exchange
- Ease of breathing _____
- Oxygen saturation _____
- PaO2 _____

**Measurement Scale**

1 = Severely compromised
2 = Substantially compromised
3 = Moderately compromised
4 = Mildly compromised
5 = Not compromised

- Dyspnea with exertion _____
- Dyspnea at rest _____

**Measurement Scale**

1 = Severe
2 = Substantial
3 = Moderate
4 = Mild
5 = None

### OUTCOMES (NOC)

#### Anxiety Self-Control
- Uses effective coping strategies _____
- Seeks information to reduce anxiety _____
- Controls anxiety response _____

**Measurement Scale**

1 = Never demonstrated
2 = Rarely demonstrated
3 = Sometimes demonstrated
4 = Often demonstrated
5 = Consistently demonstrated

### INTERVENTIONS (NIC) and RATIONALES

#### Respiratory Monitoring
- Monitor rate, rhythm, depth, and effort of respirations to evaluate changes in respiratory status.
- Auscultate breath sounds, noting areas of decreased/absent ventilation and presence of adventitious sounds, to assess congestion.
- Monitor for dyspnea and events that improve and worsen it to detect events that can influence ADLs.

#### Oxygen Therapy
- Administer supplemental O2 as ordered to maintain O2 levels.
- Change O2 delivery device from mask to nasal prongs during meals as tolerated to sustain O2 levels while doing ADLs.
- Monitor the effectiveness of O2 therapy to identify hypoxemia and establish range of O2 saturation.

#### Positioning
- Position to alleviate dyspnea (e.g., semi-Fowler’s position), as appropriate, to improve ventilation by decreasing venous return to the heart and increasing thoracic capacity.

#### Anxiety Reduction
- Use a calm, reassuring approach to increase confidence in caregiver and relieve anxiety.
- Explain all procedures, including sensations likely to be experienced during a procedure, to promote sense of security.
- Help patient identify situations that precipitate anxiety to plan appropriate use of anxiety-reducing techniques.
- Create an atmosphere to facilitate trust (e.g., answer call bell promptly and make frequent checks) to promote sense of security.
- Instruct patient in use of relaxation techniques (e.g., imagery) to help alleviate anxiety.
NURSING CARE PLAN 35-1—cont’d

Patient With Heart Failure—cont’d

NURSING DIAGNOSIS
Deficient knowledge related to disease process as evidenced by questions about the disease and patient’s statement, “I don’t know why I keep getting sick.”

PATIENT GOAL
Describes disease process and rationales for dietary and medication regimen

OUTCOMES (NOC)

Knowledge: Disease Process
- Description of specific disease process
- Description of complications
- Descriptions of precautions to prevent complications

Measurement Scale
1 = None
2 = Limited
3 = Moderate
4 = Substantial
5 = Extensive

INTERVENTIONS (NIC) and RATIONALES

Teaching: Disease Process
- Appraise the patient’s current level of knowledge related to specific disease process to identify needed areas of teaching.
- Describe common signs and symptoms of the disease so patient will know signs and symptoms to report to health care provider.
- Instruct the patient on measures to prevent/minimize side effects of treatment for the disease so patient may be able to decrease number of acute episodes of HF.

Teaching: Prescribed Diet
- Review patient’s knowledge of medications to determine where further teaching is needed.
- Appraise the patient’s current level of knowledge about prescribed diet to assess areas needing additional instruction.
- Include the family/significant others to provide support for the patient.

Teaching: Prescribed Medication
- Review patient’s knowledge of medications to determine where further teaching is needed.
- Appraise the patient’s current level of knowledge about prescribed diet to assess areas needing additional instruction.
- Include the family/significant others to provide support for the patient.

TABLE 35-15

PATIENT AND FAMILY TEACHING GUIDE
Heart Failure

Health Promotion
1. Obtain annual flu vaccination.
2. Obtain pneumococcal vaccine (e.g., Pneumovax) and revaccination after 5 years (for people at high risk of infection or serious disease).
3. Consider smoking cessation and weight reduction, if appropriate.

Rest
1. Plan a regular daily rest and activity program.
2. After exertion, such as exercise and ADLs, plan a rest period.
3. Shorten working hours or schedule rest period during working hours.
4. Avoid emotional upsets. Verbalize any concerns, fears, feelings of depression, etc. to health care provider.

Drug Therapy
1. Take each drug as prescribed daily.
2. Develop a check-off system (e.g., daily chart) to ensure medications have been taken.
3. Take pulse rate each day before taking medications (if appropriate). Know the parameters that your health care provider wants for your heart rate.
4. Learn to take own BP at determined intervals (if appropriate). Know your target BP limits.
5. Know signs and symptoms of orthostatic hypotension and how to prevent them (see Table 33-14).
6. Know signs and symptoms of internal bleeding (bleeding gums, increased bruises, blood in stool or urine) and what to do if on anticoagulants.
7. Know own INR if taking warfarin (Coumadin) and how often to have blood monitored.

Dietary Therapy
1. Consult the written diet plan and list of permitted and restricted foods.
2. Examine labels to determine sodium content. Also examine the labels of over-the-counter drugs such as laxatives, cough medicines, and antacids.
3. Avoid using salt when preparing foods, or adding salt to foods.

4. Weigh yourself in the early morning after arising and emptying your bladder. Use the same scale and wear the same or similar clothes every day.
5. Report weight gain of 3 lb (1.4 kg) in 2 days, or 3-5 lb (2.3 kg) in a week.
6. Eat smaller, more frequent meals.

Activity Program
1. Increase walking and other activities gradually, provided they do not cause fatigue and dyspnea. Consider a cardiac rehabilitation program.
2. Avoid extremes of heat and cold.

Ongoing Monitoring
1. Check the signs and symptoms of recurring or progressing heart failure.
2. Recall the symptoms experienced when illness began; reappearance of previous symptoms may indicate a recurrence.
3. Report immediately to health care provider any of the following:
   • Difficulty breathing, especially with exertion or when lying flat
   • Waking up breathless at night
   • Frequent dry, hacking cough, especially when lying down
   • Fatigue, weakness
   • Swelling of ankles, feet, or abdomen; swelling of face or difficulty breathing (if taking ACE inhibitors)
   • Nausea with abdominal swelling, pain, and tenderness
   • Dizziness or fainting
   • Weight gain of 3 lb (1.4 kg) in 2 days, or 3-5 lb (2.3 kg) in a week
4. Follow up with health care provider on regular basis.
5. Consider joining a local support group with your family members and/or support person(s).

ADLs, Activities of daily living; BP, blood pressure; CVP, central venous pressure; HF, heart failure; MAP, mean arterial pressure; PaO₂, partial pressure of oxygen in arterial blood; PAWP, pulmonary artery wedge pressure.

ACE, Angiotensin-converting enzyme; ADLs, activities of daily living; BP, blood pressure; INR, international normalized ratio.
The patient should also be taught the symptoms of hypo- and hyperkalemia if diuretics that deplete or spare potassium are being taken. Frequently the patient who is taking thiazide or loop diuretics is given supplemental potassium.

The nurse, physical therapist, or occupational therapist can instruct the patient in energy-conserving and energy-efficient behaviors after an evaluation of daily activities has been done. For example, once the nurse understands the patient’s daily routine, suggestions can be made for simplification of work or modification of an activity. Frequently the patient needs a prescription for rest after an activity. Many hard-driving persons need the “permission” to not feel “lazy.” Sometimes an activity that the patient enjoys may need to be eliminated. In such situations the patient should be helped to explore alternative activities that cause less physical and cardiac stress. The physical environment may require modification in situations in which there is an increased cardiac workload demand (e.g., frequent climbing of stairs). The nurse can help the patient identify areas where outside assistance can be obtained.

The home health nurse is essential in the care of the HF patient and family. Frequent physical assessments, including vital signs and weight, are extremely important. Home health nurses frequently work within protocols set up with the patient’s health care team. The protocols may enable the nurse and patient to identify problems, such as an increase in weight and HR as evidence of worsening HF, and institute interventions to prevent hospitalization. This may include altering medications and initiating fluid restrictions. Home health nursing care of HF patients is paramount in reducing the number of hospitalizations, increasing functional capacity, and increasing quality of life.

### Evaluation

The expected outcomes for the patient with HF are presented in NCP 35-1.

### CARDIAC TRANSPLANTATION

The first heart transplant was performed in 1967. Since that time, cardiac transplantation (transfer of a heart from one person to another) has become the treatment of choice for patients with refractory end-stage HF, cardiomyopathy, and inoperable CAD. Table 35-16 identifies the indications for transplantation in patients with refractory end-stage HF.

Once an individual meets the criteria for cardiac transplantation, the goal of the evaluation process is to identify patients who would most benefit from a new heart. After a complete physical examination and diagnostic workup, the patient and family then undergo a comprehensive psychologic profile that includes assessing coping skills, family support systems, and motivation to follow the rigorous regimen that is essential to a successful transplantation. The complexity of the transplant process may be overwhelming to a patient with inadequate support systems and a poor understanding of the lifestyle changes required after transplant.

Once an individual is accepted as a transplant candidate (this may happen rapidly during an acute illness or over a longer period), he or she is placed on a transplant list. Patients may wait at home and receive ongoing medical care if their medical condition is stable. If their condition is not stable, they may require hospitalization for more intensive therapy. Unfortunately, the overall waiting period for a transplant is long, and many patients die while waiting for a transplant (see Ethical Dilemmas box).

### Table 35-16 Indications for Cardiac Transplantation for Patients With End-Stage Heart Failure

<table>
<thead>
<tr>
<th>Absolute Indications</th>
<th>Relative Indications</th>
<th>Insufficient Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>For hemodynamic compromise due to HF:</td>
<td>Peak exercise oxygen consumption &lt; 55% of predicted normal</td>
<td>Low EF</td>
</tr>
<tr>
<td>Refractory cardiogenic shock</td>
<td>Persistent unstable ischemia that is not amenable to pharmacologic or revascularization interventions</td>
<td>History of NYHA Functional Class III or IV</td>
</tr>
<tr>
<td>Dependence on IV inotropes to maintain organ perfusion</td>
<td>Persistent fluid overload/renal dysfunction in spite of adherence to medical regimen</td>
<td></td>
</tr>
<tr>
<td>Peak exercise oxygen consumption &lt; 50% of predicted normal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**CARDIAC TRANSPLANTATION**

Donor and recipient matching is based on body and heart size and ABO type. Negative lymphocyte crossmatch (explained in Chapter 14) and avoidance of a transplantation from a cytomegalovirus (CMV)-positive donor to a CMV-negative recipient are important.

Most donor hearts are obtained at sites distant from the institution performing the transplant. The maximum acceptable time from harvesting the donor heart to transplantation is 4 to 6 hours.

The recipient is prepared for surgery, and cardiopulmonary bypass is used. The usual surgical procedure involves removing the recipient’s heart, except for the posterior right and left atrial walls and their venous connections. The recipient’s heart is then replaced with the donor heart, which has been trimmed to match. Care is taken to preserve the integrity of the donor sinoatrial (SA) node so that a sinus rhythm may be achieved postoperatively.

Immunosuppressive therapy usually begins in the operating room. Regimens vary, but they usually include azathioprine (Imuran), corticosteroids, and cyclosporine. Tacrolimus (Prograf) is also used as an immunosuppressant. (The mechanisms of action and side effects of these and other immunosuppressants are discussed in Chapter 14 and Table 14-18). Currently cyclosporine is used with corticosteroids for maintenance immunosuppression. Its use has resulted not only in reduced rejection, but also in slowing the rejection process so that early treatment can be instituted. Because of the use of immunosuppressants, infection is the primary complication following transplantation.

Endomyocardial biopsies are typically obtained from the right ventricle (via the right internal jugular vein) on a weekly basis for the first month, monthly for the following 6 months, and yearly thereafter to detect rejection. The HeartBreath test is used along with endomyocardial biopsy to assess organ rejection in heart transplant patients. The test works by measuring the amount of methylated alkanes (natural chemicals found in the breath and air) in a pa-
as a bridging device for transplantation and for recovery of the heart after cardiac surgery.

Artificial Heart

The lack of available transplant hearts and the increasing number of patients in need have triggered the movement to develop artificial hearts. Two implantable artificial hearts, the CardioWest Total Artificial Heart and the AbioCor Implantable Replacement Heart, have been developed. They are both designed with materials that minimize coagulation and contain motor-driven pumping systems (artificial ventricles) that operate on both internal and external batteries (Fig. 35-3). An electronic package in the abdomen monitors the system, including adjusting the heart rate based on the patient’s activity. An external battery pack allows for periods of independence from the console. The total artificial heart requires no immunosuppression and may hold promise for short-term survival in patients with end-stage HF.

Ongoing Research

The use of adult and embryonic stem cells to replace myocardial cells damaged from heart disease and to establish new blood vessels to supply the regenerated heart muscle is gaining attention. Research is focused on directing stem cells to become cardiomyocytes (the contractile cells of the heart), vascular endothelial cells (cells that form the inner lining of the blood vessels), and smooth muscle cells (cells that form the wall of the blood vessels). Scientists hope that providing replacement tissue for the damaged heart will have immense advantages over heart transplantation and the use of artificial hearts.

CRITICAL THINKING EXERCISE

CASE STUDY

Heart Failure

Patient Profile. Mrs. E., a 70-year-old Hispanic woman, was admitted to the medical unit with complaints of increasing shortness of breath.

Subjective Data

• Had a severe MI at 58 years of age
• Has experienced increasing shortness of breath during the last 2 weeks
• Recently had a respiratory tract infection; has persistent cough, and edema in legs 2 weeks ago
• Cannot climb a flight of stairs without getting short of breath
• Sleeps with head elevated on three pillows
• Does not always remember to take medication

Objective Data

Physical Examination

• In respiratory distress, use of accessory muscles, respiratory rate 36 breaths/minute
• Systolic heart murmur
• Moist crackles in both lungs
• Cyanotic lips and extremities
• Skin cool and diaphoretic

Diagnostic Studies

• Chest x-ray results: cardiomegaly with right and left ventricular hypertrophy; fluid in lower lung fields
• Echocardiogram results: EF 20%
**CRITICAL THINKING EXERCISE—cont’d**

**Collaborative Care**
- digoxin 0.25 mg PO daily
- furosemide (Lasix) 40 mg IV bid
- potassium 40 mEq PO bid
- enalapril (Vasotec) 5 mg PO daily
- 2-g sodium diet
- Oxygen 6 L/min by nasal catheter
- Daily weights
- Daily 12-lead ECG, serum electrolytes; cardiac enzymes

**Critical Thinking Questions**

1. Explain the pathophysiology of Mrs. E.’s heart disease and dyspnea.
2. What clinical manifestations of HF did Mrs. E. exhibit?
3. What is the significance of the findings of the diagnostic studies?
4. How would a serum BNP be beneficial in the diagnosis of HF?
5. Explain the rationale for each of the medical orders prescribed for Mrs. E.
6. What are the priority nursing interventions for Mrs. E.?
7. Based on the assessment data presented, write one or more priority nursing diagnoses. Identify any collaborative problems.
8. What teaching measures should be instituted to prevent recurrence of ADHF?

**NCLEX EXAMINATION REVIEW QUESTIONS**

The number of the question corresponds to the same-numbered objective at the beginning of the chapter.

1. The nurse recognizes that primary manifestations of systolic failure include
   - a. ↓ Afterload and ↓ left ventricular end-diastolic pressure
   - b. ↓ Ejection fraction and ↑ PAWP.
   - c. ↓ PAWP and ↓ left ventricular EF.
   - d. ↓ Pulmonary hypertension associated with normal EF.
2. A compensatory mechanism involved in HF that leads to inappropriate fluid retention and additional workload of the heart is
   - a. ventricular dilation.
   - b. ventricular hypertrophy.
   - c. neurohormonal response.
   - d. sympathetic nervous system activation.
3. A drug used in the management of a patient with ADHF and pulmonary edema that will decrease both preload and afterload and provide relief of anxiety is
   - a. amrinone.
   - b. furosemide.
   - c. dobutamine.
   - d. morphine sulfate.
4. A patient with chronic HF and atrial fibrillation is treated with a digitals glycoside and a loop diuretic. To prevent possible complications of this combination of drugs, the nurse needs to
   - a. monitor serum potassium levels.
   - b. keep an accurate measure of intake and output.
   - c. teach the patient about dietary restriction of potassium.
   - d. withhold the digitals and notify the health care provider if the heart rate is irregular.
5. The primary causes of death in patients with heart transplants in the first year include
   - a. infection and dysrhythmias.
   - b. rejection and dysrhythmias.
   - c. infection and acute rejection.
   - d. myocardial infarction and cancer.

**REFERENCES**


*Nursing research–based reference.*

**RESOURCES**

**American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR)**
312-321-5146
www.aacvpr.org

**American Association of Heart Failure Nurses**
888-452-2436
www.aahfn.org

**American College of Cardiology**
Heart House
800-253-4636, ext. 694 or 301-897-5400
www.acc.org

**American Heart Association**
800-AHA-USA-1 (242-8721)
www.americanheart.org

**Council on Cardiovascular Nursing**
American Heart Association
214-373-6300
http://216.185.112.5/presenter.html?identifier = 1148

**Heart Failure Society of America**
www.hfsa.org

**International Society for Heart and Lung Transplantation**
972-490-9495
www.ishlt.org

**National Heart, Lung, and Blood Institute**
National Institutes of Health
301-592-8573
www.nhlbi.nih.gov

**The Mended Hearts**
888-432-7899 or 214-706-1442
www.mendedhearts.org

For additional Internet resources, see the website for this book at http://evolve.elsevier.com/Lewis/medsurg.