SECOND EDITION

PRIMARY CARE

An Interprofessional Perspective

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SPRINGER PUBLISHING COMPANY

CHAPTER 10

Heart Failure

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Heart failure is a public health problem of enormous and growing significance. It affects more than 5 million people in the United States. As the population ages and the incidence of patients surviving other symptomatic cardiac diseases such as myocardial infarction (MI) continues to rise, the incidence of heart failure and its mortality rate will continue to increase. Heart failure is the only cardiovascular disease that is increasing in prevalence.

The management of heart failure has become one of the most challenging problems confronting our health care system today. Each year, the United States spends nearly $32 billion in managing patients with heart failure, with projected increases of 120% to $70 billion per year by 2030 (Go et al., 2013).

Treatment of heart failure is no longer confined to symptom relief. Because the underlying etiologies that contribute to ventricular dysfunction may progress independently from the development of symptoms, treatment to prevent and delay the progression of the disease is equally important. This chapter discusses the pathophysiology and treatment of heart failure, with emphasis on long-term management and patient education to prevent exacerbation and delay the disease progression to improve a patient's quality of life.

ANATOMY, PHYSIOLOGY, AND PATHOLOGY

Functional Anatomy of the Heart

The heart is a four-chambered structure consisting of two atria and two ventricles. The atria are relatively thin-walled, low-pressure chambers that lie superior to the ventricles. Their primary function is to act as a reservoir, filling their respective ventricles. The ventricles are thick-walled chambers that function at higher pressures and pump blood from the heart into the respective great vessels. The atria and ventricles are more specifically identified as either right or left, according to their orientation in the chest. The right atrium and ventricle receive deoxygenated blood as it returns from the body. The right ventricle then pumps this blood to the lungs through the pulmonary artery. After the blood is oxygenated in the lungs, it returns via the pulmonary veins to the left atrium and is then pumped out to the rest of the body by the left ventricle via the aorta. The circulatory system thus consists of two circuits in series, the pulmonary and systemic, through which the blood sequentially flows.

The unidirectional blood flow is maintained by the four valves located in the heart. These valves allow the forward flow of blood and when closed prevent retrograde flow. The two atrioventricular valves, mitral and tricuspid, in the right and the left sides of the heart respectively, separate the ventricles from their respective atria. The semilunar valves, aortic and pulmonic, divide the ventricles from their respective great vessel. The left ventricle is separated from the aorta by the aortic valve; the right ventricle is separated from the pulmonary artery by the pulmonic valve.

Cardiac Physiology and Hemodynamics

The primary physiological function of the heart is to pump blood to supply oxygen and nutrients to the different body organ systems. *Stroke volume* is the volume of blood pumped per beat by each ventricle. *Cardiac output* is the volume of blood pumped per minute. Cardiac output and stroke volume are therefore related by the following equation:

$$\text{Cardiac output (mL/min)} = \text{Stroke volume (mL/beat)} \times \text{Heart rate (beats/min)}$$

Stroke volume is regulated by three variables: the end-diastolic volume (EDV), the mean aortic or arterial blood pressure, and the contractility of the ventricles. The EDV is the amount of blood in the ventricles just before contraction. Because this is a workload imposed on the ventricles before contracting, it is clinically referred to as the preload. Arterial pressure represents an impedance to the ejection of blood from the ventricles, or an afterload imposed on the ventricles after contraction has begun. The stroke volume is directly proportional to the preload and contractility but inversely proportional to the afterload.

The portion of the EDV that is ejected (the ejection fraction or EF) against a given afterload depends on the strength of ventricular contraction. Normally, contraction strength is

sufficient to eject two thirds of the EDV with each heart beat. In a healthy heart, this fraction remains relatively constant, even with an increase in EDV. This implies that the strength of ventricular contraction must increase as the EDV increases in a normal heart. Such intrinsic control of contractile strength was first described by two physiologists, Frank and Starling, and thus is named the Frank–Starling law of the heart.

The other factor that controls cardiac output is the heart rate. Heart rate is regulated by the autonomic nervous system. Stimulation of the sympathetic nervous endings in the musculature of the atria and ventricles increases the strength of contraction (positive inotropic effect) and decreases slightly the time spent in systole. In contrast, enhancing the effect of the parasympathetic nervous system decreases cardiac rate and contractility.

Pathophysiology of Heart Failure

The pathophysiology of heart failure begins with myocardial cell damage caused by etiologies such as ischemic heart disease and hypertension. Table 10.1 details the causes of heart failure (Yancy et al., 2013). As myocardial damage becomes significant, either myocardial contractility decreases or the left ventricle stiffens and is unable to fill to full capacity. Both of these conditions lead to a decrease in cardiac output and intraventricular pressure increases to the point where body organ perfusion and functions are compromised. In the left ventricle, an increase in diastolic pressure leads to a rise in pressure in the pulmonary circulation. This causes pulmonary edema and prevents proper oxygenation of the blood, leading to dyspnea. In the right ventricle, an increase in diastolic pressure leads to an elevation of venous pressure, resulting in peripheral edema.

TABLE 10.1 Etiologies of Heart Failure

- Ischemic cardiomyopathy
  - Coronary artery disease
- Nonischemic, dilated cardiomyopathy
  - Hypertension
  - Valvular heart disease
  - Infectious etiologies
  - Toxic cardiomyopathy
    - Alcohol
    - Cocaine
    - Cardiotoxicity related to chemotherapy (e.g., doxorubicin)
    - Other cardiotoxins (e.g., ephedra, anabolic steroids)
  - Tachycardia induced
  - Cardiomyopathy from inflammation
    - Rheumatologic disorders (e.g., systemic lupus erythematosus, scleroderma)
    - Peripartum cardiomyopathy
    - Iron overload
    - Amyloidosis
    - Cardiac sarcoidosis
  - Genetic defects/familial cardiomyopathy
  - Obesity
  - Diabetes mellitus

In the presence of a primary abnormality in myocardial contractility or excessive hemodynamic stresses, the heart relies on three major adaptive mechanisms in attempts to maintain cardiac output:

- The Frank–Starling mechanism, in which an increase in preload brought about in part by salt and water retention helps sustain cardiac performance (stroke volume is directly proportional to preload)
- Increased release of catecholamines by adrenergic cardiac nerves and the adrenal medulla activation of the renin–angiotensin–aldosterone system, and other neurohormonal adjustments that act to maintain arterial pressure and vital organ perfusion
- Myocyte hypertrophy with or without chamber dilatation, in which left ventricular mass is increased in an attempt to enhance contractility

Figure 10.1 illustrates the interactions of the different compensatory mechanisms in heart failure.

Initially, these mechanisms can maintain cardiac output, arterial blood pressure, and organ perfusion, but they may also exert more stress on the already injured myocardium. Therefore, in the later phase of the disease, such compensatory mechanisms actually contribute to the worsening of symptoms. Table 10.2 illustrates the short-term and the long-term responses of the impaired myocardium to these compensatory mechanisms.

Hemodynamic Compensatory Mechanisms in Heart Failure

In the early phase of heart failure, activation of the sympathetic nervous system increases both the heart rate and the contractile force, thus increasing cardiac output. However, an important cardiac compensatory mechanism called myocardial remodeling, which occurs during myocardial injury and in a state of enhanced sympathetic nervous activities, may be both adaptive and maladaptive (Francis, 2001; Zipes, Libby, Bonow, & Braunwald, 2005). Unlike skeletal muscle cells, myocardial cells cannot divide to increase their numbers, but they undergo remodeling by which they increase in length or volume (dilatation and hypertrophy). Early in heart failure, dilatation and hypertrophy may enhance contraction, but chronically they often worsen cardiac damage. In addition, dilatation of heart chambers leads to an increase in myocardial wall stress, which is one of the determinants of myocardial oxygen demand and supply imbalance. Hypertrophy, if severe and long-standing, leads to loss of contractile force.

Neurohormonal Compensatory Mechanisms in Heart Failure

One of the most significant compensatory mechanisms of heart failure is the activation of certain endogenous neurohormones. Reduction of cardiac output stimulates other body systems to try to maintain normal blood pressure and organ perfusion. The major neurohormonal systems that

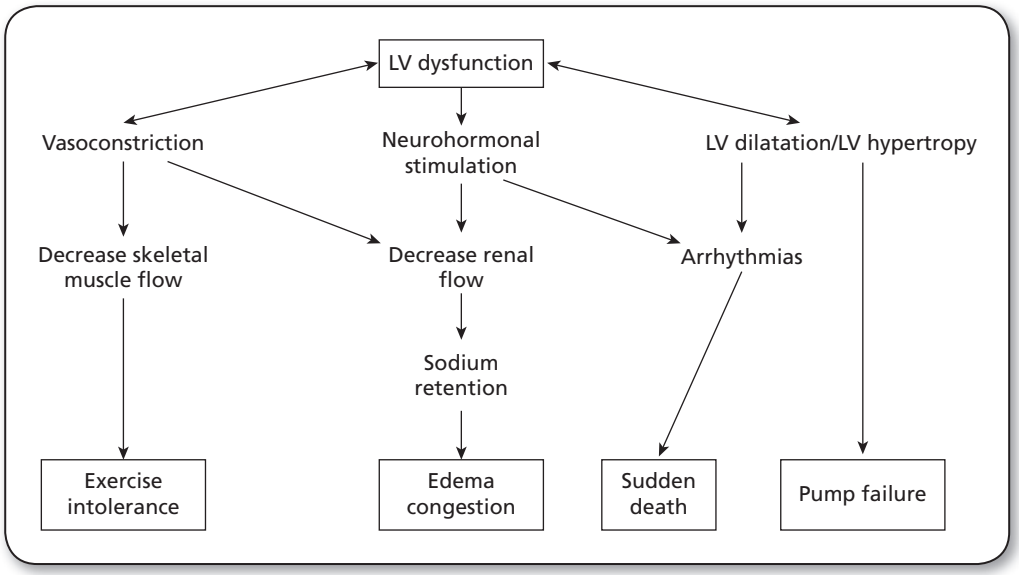

FIGURE 10.1
Pathophysiology of heart failure.

LV, left ventricular.

TABLE 10.2 Short-Term and Long-Term Responses of Impaired Myocardium to Different Compensatory Mechanisms

	SHORT-TERM EFFECTS	LONG-TERM EFFECTS
Frank–Starling mechanisms: salt and water retention (renin–angiotensin activation)	Increases preload	Causes peripheral and pulmonary congestion
Sympathetic nervous stimulation	Maintains blood pressure and organ perfusion by vasoconstriction	Increases energy expenditure; long-term stimulation also leads to desensitization of adrenergic receptors
Myocardial hypertrophy	May enhance contraction	Deterioration and death of cardiac cells

regulate these compensatory mechanisms are the sympathetic nervous system, the renin–angiotensin–aldosterone system, and the natriuretic peptides.

SYMPATHETIC NERVOUS SYSTEM

Plasma norepinephrine levels are increased in heart failure (Latini et al., 2004). With such activation of the sympathetic nervous system, there is an initial increase in cardiac contractility. Vasoconstriction also occurs and afterload increases. As mentioned, this is intended to enhance cardiac output and maintain vital organ perfusion. Chronically, however, this leads to an increase in systemic vascular resistance and adds to the strain of a failing heart. Increasing afterload leads to a decrease in forward blood flow to perfuse the body's organ systems. Decreased forward flow in turn results in a backup of venous blood returning to the heart, or increased preload. Vasoconstriction also leads to a reduction of blood flow to the kidneys, which stimulates the retention of sodium and water to compensate for the perceived lack of blood volume. Such retention of fluid and water does not improve stoke volume; rather, it contributes to the congestive symptoms of heart failure.

RENIN–ANGIOTENSIN SYSTEM

In states of low cardiac output, the renin–angiotensin–aldosterone system is activated (Ma, Kam, Yan, & Lam, 2010). This acts in concert with the activated adrenergic nervous–adrenal medullary system to maintain arterial pressure. Renin is released by the kidneys in response to reduced blood flow. Renin converts angiotensinogen to angiotensin I in the circulation. Angiotensin I circulates to the lungs and other tissues, where angiotensin-converting enzyme (ACE) converts it to angiotensin II. Angiotensin II is a potent vasoconstrictor and therefore significantly increases afterload. Aldosterone production is also increased by angiotensin II. Aldosterone has potent sodium-retaining properties and contributes to the general volume overload state of heart failure. Finally, angiotensin II plays an important role in the stimulation of the cell growth and development that leads to myocyte hypertrophy of the heart (Figure 10.2).

NATRIURETIC PEPTIDES

Atrial natriuretic peptide (ANP) is produced by the atrial tissue of the heart. Brain natriuretic peptide (BNP) is produced

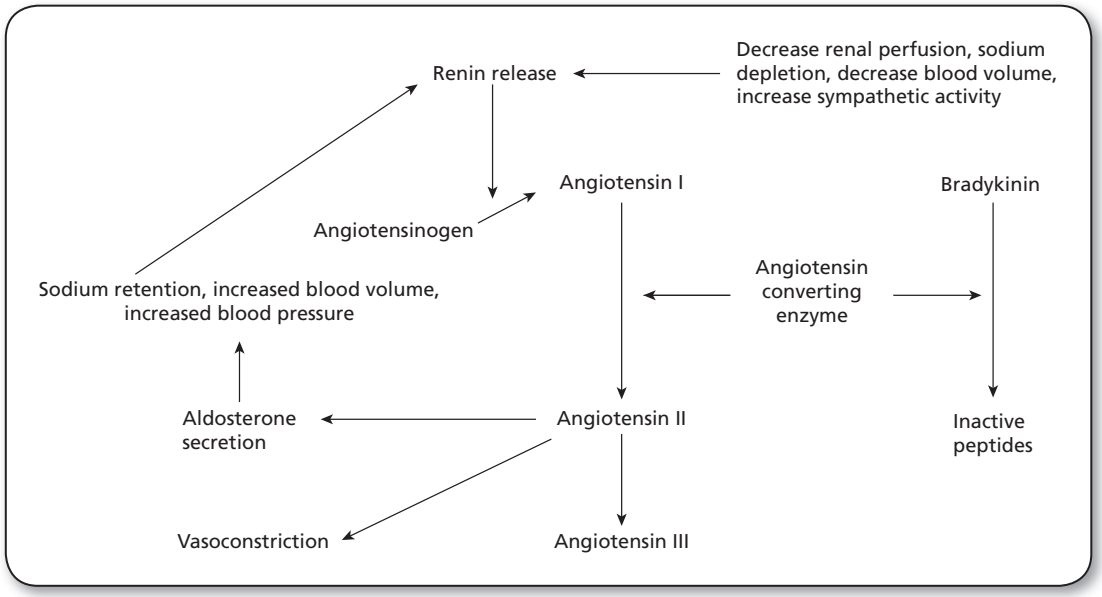

FIGURE 10.2
The renin–angiotensin–aldosterone system.

by the ventricular tissue. Secretion of ANP and BNP is regulated by wall tension; therefore, their levels are increased in chronic heart failure (Zipes et al., 2005). ANP and BNP are counterregulatory hormones that oppose the action of many of the vasoconstricting and salt- and water-retaining effects of the renin–angiotensin–aldosterone system. ANP and BNP act as vasodilating agents, suppress the formation of renin, and enhance the excretion of salt and water. However, because of their relatively weak action and short duration of effect, they cannot totally reverse the detrimental effects of the renin–angiotensin–aldosterone system and the sympathetic systems.

EPIDEMIOLOGY

More than 5 million Americans suffer from heart failure and more than 650,000 new cases are diagnosed each year (Go et al., 2013). Despite recent advances in the management of heart failure, both surgical and pharmacological, the mortality rate of this disease remains high. The 5-year mortality rate is approximately 50%. The increasing average age of the American population and the longer survival of people with other cardiac and comorbid diseases who subsequently develop heart failure at an older age add to the rapid increase in heart failure prevalence. Race-specific differences reflected a higher incidence of heart failure for African Americans, followed by Hispanic, White, and Chinese Americans. The risk of developing heart failure before age 50 years is higher in African Americans than Whites. This increased risk in African Americans is reflective of difference in risk factors such as hypertension, obesity, and diabetes mellitus, as well as differences in socioeconomic status (Go et al., 2013).

The management of heart failure patients exerts a heavy economic burden on society. Annually, more than 1 million patients are admitted to the hospital for heart failure management, accounting for a total Medicare expenditure exceeding $17 billion annually. Mortality and readmission rates following a heart failure hospitalization remain high, with nearly 1 in 4 patients readmitted to hospital within 30 days of discharge. As prevention of hospitalization and readmission has become a national priority, there is a growing fiscal imperative to develop strategies to improve the transition from hospital to home and provide more effective ambulatory heart failure management.

DIAGNOSTIC CRITERIA

Heart failure is characterized by a pathophysiological state in which the heart cannot provide adequate forward cardiac output to meet the perfusion and oxygenation requirements of the body organs and tissues. Although the etiologies of heart failure are numerous, the majority of cases can be classified as either heart failure with reduced ejection fraction (HFrEF), also referred to as systolic heart failure, in which there is impaired cardiac contractility; or heart failure with preserved ejection fraction (HFpEF), also referred to as diastolic heart failure, in which decreased compliance of the heart (manifesting as the heart's inability to relax) impairs ventricular filling. Distinguishing between these disorders is important clinically because they are managed differently. Nevertheless, there is common symptomatology, including fatigue, shortness of breath at rest, dyspnea on exertion, peripheral/pulmonary edema, and weight gain caused by fluid retention and congestion.

HFrEF is characterized by a dilated left ventricular chamber with a poorly contracting left ventricle and

TABLE 10.3 Diagnostic Criteria of Heart Failure

	HEART FAILURE WITH REDUCED EJECTION FRACTION (HFrEF)	HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFpEF)
Symptoms	Fatigue, shortness of breath, dyspnea on exertion, pulmonary or peripheral congestion, fluid retention	Similar to HFrEF
Left ventricular ejection fraction	≤40%	≥50%
Heart chamber size	Increased	Normal or increased
Wall thickness	Thinned	Typically enhanced

TABLE 10.4 Medications That May Induce or Exacerbate Heart Failure

MEDICATIONS	EFFECTS
Antiarrhythmic agents (e.g., quinidine, procainamide, flecainide, propafenone, sotalol)	Most antiarrhythmic agents have negative inotropic effects and may induce heart failure when used in patients with other underlying heart diseases
Non-dihydropyridine calcium channel blockers (verapamil, diltiazem)	Most first-generation, non-dihydropyridine calcium channel blockers have negative inotropic effects and may induce heart failure when used in patients with other underlying heart diseases. Second-generation, dihydropyridine-derivative calcium channel blockers (amlodipine and felodipine) may be considered for hypertension management, but they have not demonstrated any benefit in heart failure symptoms or survival
Heroin, cocaine, alcohol, amphetamines, doxorubicin, cyclophosphamide, sulfonamides, lead, arsenic, cobalt, phosphorus, ethylene glycol	Direct cardiac toxins
Corticosteroid and nonsteroidal anti-inflammatory agents (NSAIDs)	Cause salt and water retention
Thiazolidinedione (e.g., pioglitazone)	Causes plasma volume expansion

usually a thinned ventricular wall, ultimately producing a reduced left ventricular EF (≤40%; Yancy et al., 2013). In patients with HFpEF, the left ventricular chamber size may not increase and the wall thickness is usually enhanced. The most important diagnostic criterion for distinguishing HFrEF and HFpEF is the left ventricular EF. Patients with HFpEF may have a normal or increased left ventricular EF. Table 10.3 summarizes the major characteristics and diagnostic criteria of HFrEF and HFpEF.

HISTORY AND PHYSICAL EXAMINATION

Obtaining a complete medical history is extremely important in diagnosing heart failure. Identifying other health conditions or behaviors that may accelerate the progression of heart failure is necessary for effective management. Patients should be questioned about a previous history of angina or the equivalent (such as flash pulmonary edema); MI; hypertension; other heart diseases; diabetes; and renal, pulmonary, thyroid, or gastrointestinal diseases. A complete medication history is also important to obtain. Table 10.4 lists some common medications that may cause or exacerbate heart failure symptoms.

Symptoms suggestive of heart failure include:

- Decreased exercise tolerance
- Dyspnea on exertion
- Peripheral edema or ascites
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Unexplained confusion or altered mental status in an elderly patient (as a result of decreased cerebral perfusion)
- Abdominal symptoms associated with ascites or hepatic engorgement (e.g., nausea, abdominal pain, or early satiety)

If symptoms of progressive dyspnea, orthopnea, or paroxysmal nocturnal dyspnea are present in conjunction with a past cardiac history, the likelihood that the patient has heart failure dramatically increases. Patients with heart failure should be evaluated for activity tolerance, dyspnea,

orthopnea, paroxysmal nocturnal dyspnea, rapid weight gain, palpitations, presyncope or syncope, and defibrillator shocks, as well as adherence to the medication regimen and dietary restrictions, at each encounter.

The physical examination can provide important information about the etiology of the patient's symptoms and volume status to aid in the selection of appropriate therapies. Physical signs suggesting heart failure include:

- Elevated jugular venous pressure or positive hepatojugular reflux
- Third heart sound (positive S3)
- Laterally displaced apical impulse
- Bibasilar pulmonary rales that do not clear up with cough (this finding is rare in patients with chronic heart failure)
- Peripheral edema not caused by venous insufficiency
- Hepatomegaly and/or ascites

In addition, weight, supine and upright blood pressure, heart rate, and temperature of extremities should be assessed at each encounter.

DIAGNOSTIC STUDIES

Diagnostic studies for evaluating patients with suspected heart failure and for ongoing monitoring of the clinical status of patients with heart failure are listed in Table 10.5 (Yancy et al., 2013). Many of these tests are recommended to rule out other diseases that have clinical symptoms similar to those of heart failure and to delineate the underlying causes of heart failure so that they can be managed properly to reverse or prevent further progression of symptoms.

Central or obstructive sleep apnea is common in patients with heart failure. Patients with heart failure rarely report daytime somnolence. When a clinical suspicion exists, a patient should be referred for a sleep study. Treatment of obstructive sleep apnea with continuous positive airway pressure (CPAP) has demonstrated improvement in cardiac function and quality of life in patients with heart failure (Yancy et al., 2013).

Biomarkers

Biomarkers are useful in heart failure management. BNP and its amino acid N-terminal cleavage equivalent, NT-proBNP, are produced by cardiomyocytes in response to myocardial stretch. Assays for BNP and NT-proBNP are commonly used to aid in clinical decision making to diagnose or exclude heart failure. These biomarkers have a high negative predictive value. In the setting of dyspnea of unclear etiology, a BNP of <100 pg/mL or NT-proBNP of <300 pg/mL is associated with a decreased likelihood for heart failure as the cause of dyspnea. Patients with heart failure and dyspnea will likely have a BNP >400 pg/mL. NT-proBNP cutoff levels for diagnosing heart failure increase with age. For patients <50, 50 to 75, and >75 years of age, the optimal NT-proBNP cutoffs for diagnosing heart failure are 450 pg/mL, 900 pg/mL, and 1,800 pg/mL, respectively (Colucci & Chen, 2012). Although higher levels have a reasonable association with heart failure, other cardiac and noncardiac causes may result in high levels of these biomarkers. Heart failure treatment generally results in lower BNP or NT-proBNP levels over time. Other biomarkers, such as soluble ST2 and galectin-3, are being examined for their prognostic value in heart failure management; their use in routine clinical practice is still being evaluated.

TREATMENT OPTIONS, EXPECTED OUTCOMES, AND COMPREHENSIVE MANAGEMENT

Heart failure management is driven by current clinical practice guidelines. In the United States, these guidelines are developed by two main task forces: the American College of Cardiology Foundation/American Heart Association (ACCF/AHA; circ.ahajournals.org/content/early/2013/06/03/CIR.0b013e31829e8807.citation) and the Heart Failure Society of America (www.heartfailureguideline.org/). A summary of these guidelines is presented in this chapter.

Heart Failure Classification

The ACCF/AHA and the New York Heart Association (NYHA) have developed classification systems for heart failure (Tables 10.6 and 10.7). Both systems provide useful and complementary information about the presence and severity of heart failure. The ACCF/AHA stages of heart failure emphasize the development and progression of disease and can be used to describe individuals and populations, whereas the NYHA classes focus on exercise capacity and symptomatic status of the disease (Yancy et al., 2013).

ACCF/AHA Stage A HFrEF

Primary prevention of heart failure should focus on prevention and treatment of underlying conditions associated with an increased risk of development of heart failure. Table 10.1 lists the etiologies for heart failure development. Ischemic heart disease/MI, hypertension, diabetes mellitus, and obesity encompass modifiable risk factors that may reduce the incidence or severity of heart failure. To understand how to prevent ischemic heart disease/MI and hypertension, one must first evaluate for risk factors for developing these conditions. To prevent heart failure, it is important to emphasize reduction in dietary fat and sodium consumption, weight maintenance, regular physical activity, and smoking cessation. In patients who already have a history of ischemic heart disease/MI, prevention of disease progression is also crucial (refer to Chapter 9, "Dyslipidemias"; Chapter 8, "Coronary Artery Disease"; Chapter 11, "Hypertension";

TABLE 10.5 Diagnostic Studies for Evaluating Patients With Heart Failure—Exercise Stress Testing

TEST	PURPOSE
Serum electrolytes, urea nitrogen, creatinine, glucose, phosphorus, magnesium, calcium, and albumin	To aid in determining fluid status; monitor renal function; evaluate side effects of medications; monitor hypoalbuminemia (a predictor of poor prognosis)
Complete blood count	To rule out anemia, which decreases the blood's oxygen-carrying capacity, thus leading to or aggravating heart failure
Urinalysis	To rule out nephrotic syndrome or glomerulonephritis, which may cause a fluid overload state similar to heart failure
Thyroid-stimulating hormones	To evaluate thyroid abnormalities, especially in patients with atrial fibrillation and unexplained heart failure
Fasting lipid profile	To evaluate hyperlipidemia
Liver function test	To evaluate liver dysfunction, congestion
Brain natriuretic peptide (BNP) or NT-proBNP	To aid in clinical decision making
Chest x-ray	To detect cardiomegaly and pulmonary congestion and to rule out other pulmonary causes of dyspnea such as pneumonia
Electrocardiogram	Abnormal changes such as ST-segment elevation/depression, T-wave inversion, and presence of Q waves may indicate ischemia or myocardial infarction, which if not managed promptly and appropriately may lead to heart failure Evaluate atrial and ventricular arrhythmias Low voltage may suggest amyloidosis Patients with a wide QRS may benefit from resynchronization therapy
Transthoracic two-dimensional echocardiography with Doppler	To assess ventricular function (ejection fraction), size, thickness, wall motion, and valvular function
Radionuclide ventriculography (also known as a multiple-gated acquisition or MUGA scan)	A nuclear imaging test to evaluate cardiac ejection fraction
Exercise stress test, coronary angiography (in patients with coronary artery disease)	To optimize management of coronary artery disease, which ultimately prevents or retards the progression of heart failure
Cardiopulmonary stress test	Measures gas exchange to determine aerobic capacity. Helps in determining causes of exertional dyspnea and aids in prognostication of patients with heart failure
Cardiac MRI	To evaluate myocardial infiltrative processes; also used to assess ventricular function if echocardiogram is inadequate
Right heart catheterization	Hemodynamic monitoring can be useful to guide therapy in patients with persistent symptoms despite adequate evidence-based therapies

and Chapter 15, "Diabetes Mellitus," for evidence-based guidelines related to the prevention, diagnosis, and management of these conditions).

ACCF/AHA Stage B HFrEF

In patients with evidence of reduced left ventricular EF who are yet to develop heart failure symptoms, ACE inhibitor and beta-blocker therapy should be initiated to prevent symptoms and reduce mortality. Medication dosages should be up-titrated to target doses listed in current clinical practice guidelines (see pharmacological recommendations for these agents in the ACCF/AHA Stage C HFrEF section). All recommendations for patients with Stage A heart failure should also be applied to reduce risk factors for heart failure progression.

TABLE 10.6	ACCF/AHA Stages of Heart Failure
Stage A	High risk for heart failure but no symptoms or structural heart disease
Stage B	Structural heart disease but without symptoms of heart failure (NYHA Class I)
Stage C	Structural heart disease with prior or current symptoms of heart failure (NYHA Class I–IV)
Stage D	Refractory heart failure requiring specialized interventions (NYHA Class IV)

ACCF, American College of Cardiology Foundation; AHA, American Heart Association; NYHA, New York Heart Association.

TABLE 10.7	New York Heart Association Functional Classification
Class I	No limitation of physical activities
Class II	Slight limitation of physical activity
Class III	Marked limitation of physical activity
Class IV	Unable to carry out any physical activity; symptoms at rest

ACCF/AHA Stage C HFrEF

The management of Stage C HFrEF includes all the general measures (e.g., exercise and dietary changes) listed earlier, in addition to pharmacotherapy and device therapies.

Pharmacotherapy

A pharmacotherapeutic plan includes:

- Neurohormonal blockade medications are the foundation of therapy for patient with HFrEF.
  - Patients should be started on a beta-blocker and an ACE inhibitor or angiotensin receptor blocker (ARB) and dosages up-titrated to those listed in current clinical practice guidelines.
  - An aldosterone antagonist should be added for patients with NYHA Class II–IV symptoms provided the estimated creatinine clearance is >30 mL/min and potassium is <5.0 mEq/L.
  - For African Americans with NYHA Class III or IV symptoms, hydralazine and nitrates should be added and up-titrated to dosages listed in current clinical practice guidelines.
- Relief of symptoms. For patients with signs of volume overload, loop diuretics should be added to reduce symptoms.
- Treatment of the underlying etiology whenever possible.
- Prevention and management of complications such as arrhythmia, cardiogenic shock, or thromboembolic events (stroke).
- Improvement of the mortality rate and prolongation of survival.

DIURETICS

Diuretics are used in heart failure to relieve circulatory congestion and peripheral edema. The role of diuretics in heart failure management is for symptomatic relief and should not be used if the patient has no signs of congestion. Table 10.8 lists common diuretics used in the management of heart failure. Thiazides are used to maintain a normal intravascular volume in mild states of congestion; more severe congestion usually requires the use of loop diuretics. If patients have persistent congestion, metolazone therapy may be added to loop diuretic therapy for a synergistic effect caused by their different sites of action at the renal tubule. Doses of diuretics are individualized based on the patient's condition. Patients receiving diuretic therapy should be monitored for electrolyte abnormalities, especially hypokalemia. Hypokalemia may result in cardiac arrhythmias or precipitate digoxin toxicity. Potassium supplements should be administered if necessary. Patients may also be advised to consume foods that are high in potassium, such as oranges and bananas. Potassium-sparing diuretics may be substituted to moderate the loss of potassium. However, potassium-sparing diuretics are not as potent a natriuretic agent as loop diuretics. Symptomatic hypotension and progressive elevation of the blood urea nitrogen concentration are signs of over-diuresis. Patients should be advised to weigh themselves daily to ensure efficacy and prevent over-diuresis. Some patients may be taught to self-adjust diuretic dosages based on changes in daily weights.

ACE INHIBITORS

ACE inhibitors can reduce preload, afterload, or both by relaxing the arterial and venous smooth muscle, reducing resistance to left ventricular ejection. This increases cardiac output, thus relieving congestion symptoms and improving exercise tolerance.

ACE inhibitors are the first choice of vasodilators used for HFrEF management because they not only relieve symptoms but also reduce hospitalizations and improve survival (Garg & Yusuf, 1995). ACE inhibitors reduce preload and afterload by inhibiting ACE, thus reducing the production of angiotensin II, which is a potent vasoconstrictor. Table 10.9 lists ACE inhibitors that have been tested in clinical trials for use in the management of HFrEF. Therapy with ACE inhibitors should be initiated at a low dose to avoid hypotension. Attempts should be made to achieve the target dosages identified in clinical trials because survival benefits have been demonstrated at these dosages. Renal function and potassium should be assessed 1 to 2 weeks after the initiation of therapy and with each dose increase. Moderate asymptomatic hypotension and azotemia (serum creatinine <2.5 mg/dL) are acceptable side effects of ACE inhibitors, and reduction or discontinuation of therapy is not warranted. Reduction of diuretic doses may be adequate to correct these problems. However, symptomatic hypotension, progressive azotemia, or intolerable cough occasionally requires discontinuation of ACE inhibitor therapy. Other side effects of ACE inhibitors include rash and angioedema.

TABLE 10.8 Use of Diuretics in the Management of Heart Failure

DRUGS	INITIAL DAILY DOSE	MAXIMUM TOTAL DAILY DOSE (mg)	COMMON ADVERSE EFFECTS
Thiazide Diuretics			
Hydrochlorothiazide	25 mg once or twice daily	200 mg	Hypokalemia, hyperglycemia, hyperuricemia, hypercholesterolemia
Chlorthalidone	12.5–25 mg once daily	100 mg	
Metolazone	2.5 mg once daily	20 mg	
Loop Diuretics			
Furosemide	20–40 mg once or twice daily	600 mg	Same as thiazide diuretics. High dose may also cause ototoxicity
Bumetanide	0.5–1 mg once or twice daily	10 mg	
Torsemide	10–20 mg once daily	200 mg	
Potassium-Sparing Diuretics/Aldosterone Receptor Antagonists			
Spironolactone	12.5–25 mg once daily	50 mg	Hyperkalemia, gynecomastia
Eplerenone	25 mg once daily	50 mg	

TABLE 10.9 ACE Inhibitors Used in Heart Failure With Reduced Ejection Fraction

DRUGS	INITIAL DOSE	TARGET DOSE
Captopril	6.25 mg three times daily	50 mg three times daily
Enalapril	2.5 mg twice daily	10 mg twice daily
Lisinopril	2.5–5 mg once daily	20–40 mg once daily
Quinapril	5 mg twice daily	20 mg twice daily
Ramipril	1.25–2.5 mg once daily	10 mg once daily

ANGIOTENSIN RECEPTOR BLOCKERS

For patients with HFrEF who cannot tolerate ACE inhibitors, ARBs can be substituted. Intolerable cough is the most common cause of ACE inhibitor intolerance. Caution should be used when substituting an ARB in a patient with a history of angioedema from an ACE inhibitor, as some patients have experienced angioedema on the ARB as well. Table 10.10 lists ARBs that have been tested in clinical trials for use in the management of HFrEF.

CLINICAL WARNING:

ACE inhibitors should be discontinued immediately in patients who present with signs of angioedema, including swelling of the tongue, lips, or face. A patient with a compromised airway requires immediate emergency department referral.

BETA-BLOCKERS

Beta-adrenergic antagonists or beta-blockers, in addition to ACE inhibitors, are the cornerstone of heart failure management. They can reduce heart failure symptoms in addition to reducing hospitalizations and improving survival. Increases in sympathetic nervous activities and downregulation of beta-receptors are believed to play an important role in the advanced stages of heart failure. Beta-blockers may theoretically be able to resensitize the endogenous beta-receptors, thus changing the natural history of the disease. Clinical studies support this theory. Trials with sustained-release metoprolol succinate and bisoprolol have both provided evidence that beta-adrenergic antagonists may have a favorable effect on the course and prognosis of HFrEF (CIBIS Investigators and Committees, 1994; Waagstein et al., 1993). Carvedilol, a second-generation beta-blocker with alpha-blockage activity and antioxidant properties, has shown a 48% reduction in mortality in patients with HFrEF (Packer et al., 1996).

When initiating beta-blocker therapy, the dosage should be started low and up-titrated slowly to the target dosage. Table 10.11 lists beta-blockers that have been tested in clinical trials for use in the management of HFrEF.

TABLE 10.10 Angiotensin Receptor Blockers Used in Heart Failure With Reduced Ejection Fraction

DRUGS	INITIAL DOSE	TARGET DOSE
Candesartan	4–8 mg once daily	32 mg once daily
Losartan	25–50 mg once daily	50–150 mg once daily
Valsartan	20–40 mg twice daily	160 mg twice daily

TABLE 10.11 Beta-Blockers Used in Heart Failure With Reduced Ejection Fraction

DRUGS	INITIAL DOSE	TARGET DOSE
Metoprolol succinate (sustained-release)	12.5–25 mg once daily	200 mg once daily
Carvedilol	3.125 mg twice daily	50 mg twice daily
Bisoprolol	1.25 mg once daily	10 mg once daily

Beta-blockers can be considered in patients with a history of reactive airway disease and should be up-titrated cautiously with close monitoring of worsening symptoms. Bisoprolol or sustained-release metoprolol succinate should be used in these patients for their beta-1 selective properties. Adverse events associated with beta-blockers include worsening heart failure symptoms, fatigue, bradycardia, and hypotension. Close assessment for worsening heart failure during medication up-titration is essential. Symptoms generally respond to an increase in diuretics. Once symptoms resolve, up-titration of the beta-blocker should continue.

ALDOSTERONE ANTAGONISTS

Aldosterone antagonists are recommended in patients with NYHA Class II to IV symptoms and who have a left ventricular EF of 35% or less, unless contraindicated, to reduce morbidity and mortality (Pitt et al., 1999; Yancy et al., 2013). Aldosterone antagonists are also recommended to reduce morbidity and mortality following an acute MI in patients with a left ventricular EF of 40% or less, who develop symptoms of heart failure, or who have a history of diabetes mellitus, unless contraindicated (Pitt et al., 2003). Table 10.8 lists starting and maximum dosages used in heart failure management. Patients with NYHA Class II should have a history of prior cardiovascular hospitalization or elevated plasma BNP or NT-proBNP levels to be considered for aldosterone receptor antagonists (Zannad et al., 2011).

Careful monitoring of potassium, renal function, and diuretic dosing should be performed at initiation and closely followed thereafter to minimize the risk of hyperkalemia. Aldosterone antagonists should not be initiated in patients with a creatinine clearance <30 mL/min or a serum potassium >5.0 mEq/L. A serum potassium >5.5 mEq/L should result in a dosage reduction or discontinuation of the aldosterone antagonist.

Spironolactone is associated with an increased incidence of gynocomastia and breast pain. If a patient experiences these symptoms the drug should be discontinued; eplerenone can be substituted with less risk of these adverse events.

DIGOXIN

Digoxin is a positive inotropic agent that is used in HFrEF to improve myocardial contractility. The therapeutic efficacy of digoxin in patients with HFrEF and normal sinus rhythm has always been controversial. Digoxin can prevent clinical deterioration in HFrEF and improve patients' symptoms. However, there is no significant reduction of the mortality rate (Digitalis Investigation Group, 1997; Packer et al., 1993). Digoxin should be used in patients with left ventricular dysfunction who remain symptomatic after optimization of ACE inhibitor, beta-blockers, aldosterone antagonists, and diuretic therapy.

The usual dose of digoxin is 0.125 to 0.25 mg given orally once a day. A loading dose is not required for the management of heart failure. Because digoxin has a vaguely defined but narrow therapeutic range, it is important to use doses that do not carry a risk of toxic effects. Digoxin dosing should be based on lean body mass, renal function, and concomitant medications (Lindenfeld et al., 2010). Lower doses (0.125 mg every other day) should be used in the older adult or patients with impaired renal function (Yancy et al., 2013). Doses greater than 0.25 mg daily are rarely used in heart failure management.

Serum digoxin levels should be measured to ensure safety; many laboratories make this test available. The therapeutic range is generally defined as 0.8 to 2 ng/mL. However, the correlation between therapeutic levels and efficacy is not strong. For HFrEF, it is recommended that the digoxin level should be 0.5 to 0.9 ng/mL (Yancy et al., 2013). Serum concentrations >1.1 mg/dL are believed to increase mortality (Digitalis Investigation Group, 1997). Routine monitoring of digoxin levels is not necessary to ensure efficacy or safety. Patients should be educated about the possible side effects of digoxin (see Table 10.12) and if necessary a serum level can be obtained to confirm toxicity.

HYDRALAZINE AND NITRATES

Hydralazine (a direct arterial dilator that reduces afterload) and nitrate (a venous dilator that reduces preload) combinations have also been shown to improve hemodynamics as well as prolong survival, especially in African American patients when used in addition to diuretics, ACE inhibitors, and beta-blockers (Taylor et al., 2004). In terms of survival benefit, this combination therapy did not compare as favorably as ACE inhibitors (Cohn et al., 1986, 1991); however,

TABLE 10.12	Adverse Effects of Digoxin
Nausea, vomiting (first sign presented)	
Diarrhea	
Abdominal pain	
Headache	
Fatigue	
Depression	
Yellow-green halo	
Cardiac arrhythmia	

it did provide a more favorable effect on left ventricular function and exercise capacity. This combination should be reserved for patients who cannot tolerate ACE inhibitors or ARBs, or in African American patients in addition to other disease-modifying therapies.

The target dosage of hydralazine is 75 mg three times a day. The target dosage of nitrates, given as isosorbide dinitrate, is 40 mg three times a day. Many patients cannot tolerate the target dose due to the development of headaches from the nitrate therapy. It is important that both drugs be used at the same time to ensure mortality and hemodynamic benefits. The need to take medications three times a day makes participation in this regimen difficult. Substituting long-acting nitrate formulations such as isosorbide mononitrate (administered once daily) may help. A nitrate-free interval of 10 to 14 hours is desirable to minimize the development of nitrate tolerance. Other side effects of this combination regimen include lupus syndrome from hydralazine, which is common at a daily dose of 200 mg or more.

Other Drug Therapies

ANTIARRHYTHMICS

Ventricular arrhythmia is one of the two leading causes of death in patients with heart failure (the other being cardiogenic shock). It is not currently recommended that antiarrhythmic agents be routinely administered to suppress ventricular arrhythmias, except in the event of sustained ventricular tachycardia or ventricular fibrillation. Patients with heart failure are also susceptible to developing atrial fibrillation because of the fluid stretch of the atrium during a fluid overload state. Conversion to normal sinus rhythm should be attempted because patients with heart failure do not tolerate symptoms of atrial fibrillation. Amiodarone is the most often used antiarrhythmic agent in patients with heart failure because of the low incidence of proarrhythmia and the lack of negative inotropic effect (Doval et al., 1994; Singh et al., 1995). Antiarrhythmic therapies have not been proven to prolong survival in patients with heart failure.

ANTICOAGULANTS

Thromboembolism is a potential complication in patients with heart failure. A poorly pumping heart promotes formation of blood clots (thrombi) in the ventricles. These thrombi may dislodge and eventually form emboli in the brain (stroke), the lungs (pulmonary emboli), or the coronary arteries (myocardial ischemia). Routine use of prophylactic anticoagulation is not recommended because of a lack of any prospective trial supporting its efficacy. However, patients who are at particularly high risk of developing thromboembolic events, such as those who have a history of atrial fibrillation or prior thromboembolic events, should receive maintenance anticoagulation therapy. The choice of anticoagulant should be based on a patient's risk factors, clinical characteristics, and preferences. Warfarin is commonly used but requires monitoring of the International Normalized Ratio (INR) to achieve a therapeutic level between 2 and 3. Newer agents, such as dabigatran or rivaroxaban, which do not require blood test monitoring, could also be considered. Anticoagulation is not recommended in patients with heart failure who do not have atrial fibrillation, a prior history of thromboembolic event, or evidence of a cardioembolic source.

OMEGA-3 FATTY ACIDS

The use of omega-3 polyunsaturated fatty acid (PUFA) has been found to modestly reduce mortality and hospitalizations related to cardiovascular events in patients with NYHA Class II–IV heart failure (Lavie, Milani, Mehra, & Ventura, 2009). A dose of 800 to 1,000 mg of combined eicosapentaenoic acid (EPA)/docosahexaenoic acid (DHA) omega-3 PUFA is reasonable to use as adjunctive therapy. Further research is needed, however, to determine optimal dosing.

Device Therapies

Patients with a left ventricular EF of 35% or less are at an increased risk of ventricular tachyarrhythmias that may result in sudden cardiac death. For this reason, implantable cardioverters/defibrillators (ICDs) are recommended for patients with HFrEF who are at least 40 days post-MI, on optimal medical therapy for a minimum of 3 to 6 months, and with NYHA Class II–III symptoms, as a primary prevention measure against sudden cardiac death. Patients with NYHA Class I symptoms and a left ventricular EF <30% should also be considered for ICD implantation. The consideration for ICD implantation should be individualized taking into account a patient's life expectancy, age, comorbidities, risk for complications, and individual preferences. As heart failure progresses and end of life nears, discussions should be had with patients and families concerning deactivation of the ICD.

Patients with a left bundle branch block pattern; a QRS interval >150 ms on electrocardiogram; and NYHA Class II, III, or ambulatory Class IV symptoms should be considered for cardiac resynchronization (biventricular pacing) therapy in addition to ICD implantation. Prolongation of the QRS is associated with worse outcomes. Biventricular pacing, along with optimal medical management, has been shown

to improve functional status, exercise capacity, and quality of life by improving ventricular contractility (Santangeli et al., 2011; Young et al., 2003).

ACCF/AHA Stage D HFrEF

In patients refractory to chronic heart failure medical therapy, continuous intravenous inotropic support (intravenous dopamine, dobutamine, or milrinone) is reasonable as a bridge therapy to those eligible for mechanical circulatory support (ventricular assisted device or total artificial heart) or cardiac transplantation. Long-term inotropic support may also be considered as palliative care for symptom control in Stage D patients who are not candidates for mechanical circulatory support or cardiac transplantation.

Patients with advanced heart failure or those with frequent hospitalizations related to decompensation should be referred to a heart failure management program for evaluation of advanced therapies. The discussion of these therapies is beyond the scope of this chapter. For those patients with contraindications to mechanical circulatory support or cardiac transplantation, palliative care or hospice should be considered with goals of minimizing symptoms, reducing hospitalizations, improving quality of life, and planning for end of life.

Heart Failure With Preserved Ejection Fraction

The treatment of HFpEF has both similarities to and differences from the treatment of HFrEF. Similar to HFrEF, any underlying causal or aggravating conditions must be corrected, if possible. Unlike HFrEF, there is limited clinical evidence supporting the positive benefits of a specific pharmacological regimen in patients with HFpEF. Management is typically focused on reducing symptoms and controlling comorbidities.

Hypertension is a major cause of HFpEF; therefore, tight control of blood pressure is a major goal of management for patients with HFpEF. ACE inhibitors, ARBs, and beta-blockers are reasonable agents for blood pressure control, although no direct mortality benefits have been seen with these agents in HFpEF. The use of ARBs might be considered to decrease hospitalization (Yusuf et al., 2003). Diuretics should be used for the relief of symptoms due to volume overload. The use of aldosterone antagonists in patients with HFpEF is currently being studied.

Right Heart Failure

While the majority of attention in heart failure diagnosis and treatment revolves around the left ventricle, failure of the right ventricle may occur as well. This is usually the result of increase in pressure from left ventricular failure which in turn damages the right ventricle. Other causes of right ventricular failure include pulmonary hypertension, MI involving the right ventricle, or pulmonary embolus. Patient with evidence of right ventricular failure or pulmonary hypertension should be referred to a cardiologist for ongoing management. The primary care provider's involvement is essential for control of comorbid conditions and comanagement of symptoms.

Decompensated Heart Failure

Heart failure continues to be the leading cause of hospitalizations for patients older than 65 years. Readmissions for heart failure are a major concern for patients, health care providers, hospitals, and insurers. An admission for decompensated heart failure is a sentinel event associated with a high risk for recurrent hospitalizations and one-year mortality.

Although many admissions for heart failure are necessary and unavoidable, some admissions can be prevented through open communication between the patient and provider and close outpatient follow-up. Patients requiring hospitalization for heart failure are typically older, equally male and female, and with a history of hypertension in addition to other comorbidities (Adams et al., 2005; Fonarow et al., 2007). There tends to be an equal distribution between HFpEF and HFrEF in hospitalized patients, although those with HFpEF tend to be older females with hypertension.

When evaluating patients with decompensated heart failure, their hemodynamic status and peripheral perfusion can be classified into one of four categories (Figure 10.3). This classification can be helpful to identify which patient should be admitted and which patient could be managed as an outpatient with close follow-up. A patient with normal perfusion (warm) and no evidence of congestion (dry) is the typical compensated patient with heart failure. These patients should undergo up-titration of evidence-based medications and continue outpatient follow-up for monitoring and education. The warm and wet patient is one who shows evidence of volume overload but maintains

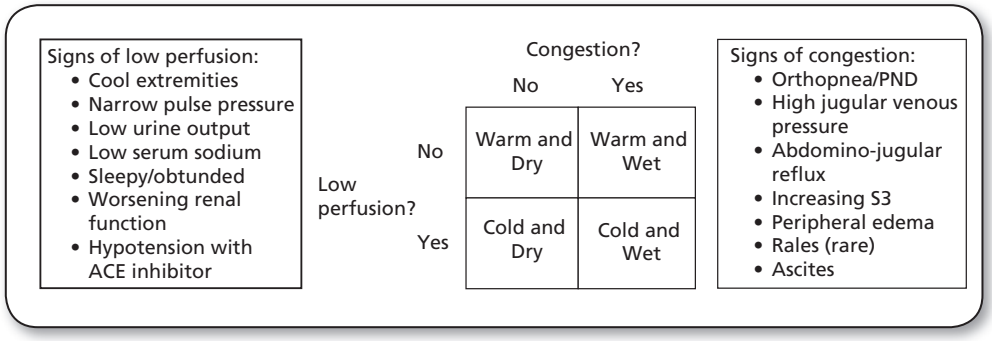

FIGURE 10.3
Heart failure hemodynamic profiles.

PND, paroxysmal nocturnal dyspnea.
Source: Adapted from Nohria, Lewis, and Stevenson (2002). Reprinted with permission from Dr. L. W. Stevenson.

adequate perfusion. The patients may be able to undergo a trial of outpatient diuresis by adjusting dosages of diuretics and up-titrating ACE inhibitor dosages. Patients should receive reinforcement of heart failure self-care strategies and be followed closely in the outpatient setting. Failure to achieve adequate and timely diuresis may result in hospitalization. The cold and dry patient is relatively uncommon. This patient has evidence of poor profusion but no signs of congestion. It is possible that congestion in this patient is unappreciated due to body habitus. This may also be a sign of being hypovolemic and steps should be taken to correct this state such as decreasing diuretic dosages. A patient who presents as wet and cold, that is, with signs of volume overload and poor perfusion, should be immediately referred for admission. This profile requires more intensive therapies, such as vasodilators and inotropic agents, which must be delivered in a monitored setting.

For patients hospitalized with a heart failure exacerbation, the opportunity should be seized to initiate and/or up-titrate evidence-based medications to target dosages while optimal volume status is achieved. The 2013 ACCF/AHA Heart Failure Management guidelines (Yancy et al., 2013) emphasized the importance of using performance improvement systems for transition of care from the inpatient to outpatient setting as a valuable prevention strategy for rehospitalization. It is recommended that throughout the hospitalization as appropriate, before hospital discharge, at their first post-discharge visits (scheduled ideally within 7–14 days after discharge or phone visit within 3 days after discharge), and at subsequent follow-up visits, the following should be addressed with the patient:

- Initiation of disease-modifying medical therapy if not previously established and not contraindicated
- Up-titration and optimization of oral heart failure therapies to target doses, as per current clinical practice guidelines
- Precipitant causes of heart failure, barriers to optimal care transitions, and limitations in post-discharge support
- Assessment of volume status and supine/upright hypotension with adjustment of heart failure therapies as appropriate
- Assessment of renal function and electrolytes where appropriate
- Assessment and management of comorbid conditions
- Reinforcement of heart failure education, self-care, emergency plans, and importance of participation in the care plan
- Consideration for palliative care in certain patients

The transition period from hospital to home is a particularly vulnerable time as a result of the progressive nature of the disease, the complexity of the medical regimen, the presence of comorbid conditions, and the number of care providers involved. Patient education is essential throughout the hospitalization and transition period. Patients must be provided with clear discharge instructions and directions for how to take their medications. Communication to the next care provider is essential, as is early patient follow-up. The first post-discharge follow-up should occur within 7 to 14 days, or sooner for higher-risk patients. At this visit, education should continue, medication reconciliation should occur, and heart failure therapies should continue to be optimized. Identifying errors in medications or knowledge deficits early after discharge may help reduce rehospitalizations. For patients deemed at high risk of decompensation and rehospitalization, referral to a heart failure disease management program should be considered.

TEACHING AND SELF-CARE

The provision of easily understood, culturally sensitive, and evidence-based education for patients with heart failure and their caregivers is an essential component of disease management. After the diagnosis of heart failure is made, patients and their families or caregivers should receive patient- and family-centered education regarding the following aspects (Lindenfeld et al., 2010; Yancy et al., 2013):

- Nature of the disease: explanation of heart failure, etiology, expected symptoms, and symptoms of worsening heart failure
- Prognosis: life expectancy, advance directives, advice for family members in the event of sudden death
- Activity recommendations: recreation, leisure and work activities, exercise, and sexual activity
- Dietary recommendations: sodium restriction, avoidance of excessive fluid (or fluid restriction if necessary), and alcohol restriction
- Medications: effects of medication, dosing, side effects, financial assistance with medications, strategies to follow the complex medication regimen
- Other treatment plans: self-monitoring of daily weights, smoking and recreational drug use cessation, the role of patients and family members in the treatment plan, availability of support groups, what to do when symptoms worsen, and the importance of obtaining vaccinations against influenza and pneumococcal disease
- Importance of active participation with the whole treatment/care plan

Patients must be educated on self-care strategies and reinforcement should occur at every encounter. In addition, an assessment for psychosocial, behavioral, and socioeconomic barriers (including access to care, cognitive decline, and inability to afford medications) should occur. A trusting relationship between the patient and caregiver and the primary care provider should be established, thus fostering an environment where a patient can share barriers to care and strategies to overcome barriers can be developed.

Self-Care

Self-care is an essential component to heart failure management. Self-care includes both self-maintenance and self-management strategies. *Self-maintenance* is the ability of a patient to follow the prescribed treatment while monitoring for and recognizing the symptoms of decompensation. *Self-management* occurs when patients make appropriate adjustments to self-care behaviors in response to self-assessments. Patients should understand how to monitor for symptoms, make recommended lifestyle modifications, and follow a complex medication regimen. Ongoing education and reinforcement are needed on self-care behaviors.

Dietary sodium restriction is an important self-maintenance strategy recommended in many guidelines. Data to support this recommendation are modest and there is variation among studies evaluating this activity. It is known that sodium intake is linked to hypertension and cardiovascular disease. Current ACCF/AHA guidelines, therefore, recommend that sodium be restricted to 1,500 mg daily in patients with Stage A and B heart failure to reduce risk of disease progression (Yancy et al., 2013). Sodium consumption in patients with Stage C and D heart failure should also be reduced, but the data to guide this recommendation are limited. Current ACCF/AHA guidelines recommend <3,000 mg daily in these patients to lessen symptoms (Yancy et al., 2013). A diet with 3,000 mg of sodium can be achieved fairly easily by not adding salt to foods and by avoiding salty foods. Many patients, especially the elderly, may find this unpalatable. Counseling and flexibility are required to promote participation and to ensure that patients do not become malnourished. Referral to a dietitian and involvement of the spouse, companion, or family members may be necessary to increase patient participation.

Patients with heart failure should also be advised to avoid excessive fluid intake. Strict restriction is not necessary unless the patient develops hyponatremia. Alcohol consumption should also be discouraged because acute ingestion of alcohol may depress myocardial contractility in patients with cardiac diseases.

As heart failure is a lifelong disease, aggressive rehabilitation plans should be implemented to help patients live the fullest, least disabling life possible. Patients should be encouraged to perform regular exercise, such as walking or cycling if possible, to improve functional status (Yancy et al., 2013). Heart failure, when properly managed, should not prohibit patients from performing regular daily activities. Referral to supervised rehabilitation programs may also benefit patients who are anxious; are dyspneic at a low work level; or have angina, a recent MI, or a post-coronary bypass surgery.

In addition to knowledge, patients must have the skills necessary to implement self-care behaviors. Skills to review with patients and families include reading food labels to choose low-sodium items, adapting recipes with low-sodium healthy alternatives, and developing a system for active participation with the complex medication regimen. Patients and caregivers should understand how to monitor for worsening signs and symptoms. A plan should be established for what to do when symptoms worsen, even if that is simply contacting the primary care provider.

Educating patients on the importance of daily weight monitoring is an important self-maintenance strategy. Patients should be instructed to weigh themselves each morning upon waking and before eating, so they are weighed wearing approximately the same amount of clothing at the same time each day. Weights should be recorded in a log that can be reviewed at each follow-up visit. Patients should be taught to call their provider if they gain 3 pounds or more in 3 days or less, as this is likely a sign of fluid retention. Early detection of fluid retention can be managed with adjustments in diuretic dosages and possibly prevent further decompensation and hospitalization. Some patients can be taught a self-directed diuretic regimen, where diuretic dosages are adjusted based on changes in weight.

Heart failure knowledge should be continually reassessed. Ongoing education should be directed toward perceived barriers and developing individualized strategies to motivate patients to overcome those barriers. Such education is most effectively provided by an interprofessional team involving different health care providers, including the primary care provider, cardiologist, nurses, pharmacists, physical therapists, dietitians, and social workers. Motivation and participation from patients themselves and their family members are crucial.

GENERAL MANAGEMENT SUMMARY

The following is a summary of recommendations for the management of HFrEF and HFpEF.

In General

- Mild to moderate exercise to tolerance, such as walking or biking, should be encouraged.
- Salt restriction (<3,000 mg/d) should be recommended.
- Underlying etiologies should be treated whenever possible.
- Heart transplantation or mechanical circulatory support should be considered in patients with heart failure refractory to medical therapy.

Heart Failure With Reduced Ejection Fraction

- ACE inhibitors and beta-blockers should be administered and up-titrated to target doses listed in current clinical practice guidelines for all patients with significantly reduced left ventricular EF (≤40%) unless contraindicated. ARBs can be used if patients develop cough on ACE inhibitors.

- A combination of hydralazine and isosorbide dinitrate can be substituted if patients have contraindications to ACE inhibitors and ARBs. Hydralazine and isosorbide dinitrate should also be considered in African American patients after optimizing other disease-modifying therapy (ACE inhibitors, beta-blockers).
- Aldosterone antagonists should be added after optimizing ACE inhibitor and beta-blocker therapy, provided there are no contraindications.
- Diuretic therapy should be administered to patients with fluid overload.
- Digoxin should be given to patients with HFrEF not adequately responsive to ACE inhibitor, beta-blocker, and diuretic therapy.
- Digoxin should also be given to patients with HFrEF and atrial fibrillation with rapid ventricular rates.

Heart Failure With Preserved Ejection Fraction

- Diuretics are the drugs of choice for patients with congestive symptoms.
- ARBs may reduce hospitalization.
- Optimal control of blood pressure is important. Beta-blockers, calcium channel blockers, ACE inhibitors, and ARBs are some therapeutic options.

COMMUNITY RESOURCES

There are numerous organizations that provide patient education and support groups for patients with heart failure. Support groups offer patients the chance to talk to others about their feelings and experiences dealing with the disease. Many support groups also offer educational programs about heart problems. For more information about support groups or patient education resources, patients can contact:

- The American Heart Association, 7272 Greenville Ave., Dallas, TX 75231-4596 (1-800-AHA-USA1); www.heart.org/HEARTORG/Conditions/HeartFailure/Heart-Failure_UCM_002019_SubHomePage.jsp
- Heart Failure Society of America, 5425 Wisconsin Avenue-Suite 600, Chevy Chase, MD 20815 (1-301-718-4800); www.hfsa.org/heart_failure_education_modules.asp
- American Association of Heart Failure Nurses, 15000 Commerce Parkway, Suite C, Mount Laurel, NJ 08054 (1-888-45-AAHFN); www.aahfnpatienteducation.com; Patient education heartline (1-856-539-9006)
- The Mended Hearts, Inc., 8150 N. Central Expressway, M2248, Dallas, TX 75206 (1-888-HEART99); www.mendedhearts.org

Referral Points and Clinical Warnings

Primary care providers should be aware of the appropriate indications for referral to cardiologists and hospitalization of patients with heart failure. In general, any patient with NYHA Class IV symptoms or those who demonstrate a failure to respond to evidence-based treatment should be referred for specialty care. Any of the findings listed in Table 10.13 usually signifies a need for referral to specialty heart failure care (Lindenfeld et al., 2010; Yancy et al., 2013). Table 10.14 outlines recommendations to patients who could benefit from a referral to a heart failure disease management program (Lindenfeld et al., 2010). These programs may be physician or nurse managed and specialize in evaluation and education for patients with heart failure. Patients with Stage D heart failure and a poor prognosis should be referred for evaluation of advanced therapies such as heart transplant and mechanical assist devices. Patients with Stage C heart failure and frequent episodes of decompensation despite optimal medial therapy should also be referred for early evaluation of advanced therapies. Patients exhibiting symptoms of acute myocardial ischemia, respiratory distress, or ongoing decompensation despite escalating diuretic doses, especially with signs of worsening end-organ dysfunction, should be referred for inpatient hospital evaluation and treatment.

TABLE 10.13 Clinical Findings Signifying the Need for Heart Failure Cardiology Specialty Referral

- Difficulty initiating, up-titrating, or maintaining beta-blocker or ACE inhibitor therapies
- Considerable functional limitations despite optimal medical therapy
- High-dose diuretic requirements
- Recurrent hospitalizations for decompensated heart failure
- Early signs of renal or hepatic dysfunction
- Hemodynamic instability (e.g., symptomatic hypotension or syncope)
- Recurrent malignant arrhythmias (recurrent ventricular tachycardia)
- Right ventricular failure or pulmonary hypertension

TABLE 10.14 Consider Referring to a Heart Failure Disease Management Plan

- Recent or repeated hospitalizations for heart failure
- Patients at high risk of decompensation, including patients with renal insufficiency, NYHA Class III or IV symptoms, diabetes, or chronic obstructive pulmonary disease
- History of depression, cognitive dysfunction, inadequate social supports, low health literacy, or persistent nonparticipation in evidence-based regimens

NYHA, New York Heart Association.

Clinical Pearls

- ACE inhibitors and beta-blockers are the cornerstone of heart failure pharmacotherapy. Dosages should be up-titrated to target doses used in clinical trials.
- Aldosterone antagonists are beneficial to patients with NYHA Class II, III, and IV heart failure.
- Self-care strategies (low-sodium diet, daily weights, self-monitoring for signs and symptoms) are essential to achieving positive patient outcomes.
- Patients with a left ventricular EF ≤35% despite optimal medical therapy should be referred for ICD placement as a primary prevention measure to reduce the risk of sudden cardiac death.
- For patients with HFpEF, management should be aimed at achieving target blood pressure levels and volume management.

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