Heart failure is a clinical syndrome, not a single disease entity, that results from any of a number of structural or functional conditions that cause diminished blood flow and decreased tissue oxygenation. Patients who have heart failure may present with signs and symptoms of volume overload, inadequate tissue perfusion, both, or neither. Because many patients who have the syndrome present without volume overload, the term “heart failure” is preferred to the more limited term “congestive heart failure.”

Estimating the prevalence and incidence of heart failure is difficult because of a lack of consensus in defining the syndrome. Additionally, the diagnosis may be missed in asymptomatic patients and those who have dyspnea only. Nonetheless, hospital discharges for heart failure, which underestimate prevalence, have increased over time from 377,000 in 1979 to 970,000 in 2002 [1]. The prevalence of heart failure in the US population is estimated at about 5 million, with men and women about equally represented. The incidence of heart failure increases in the elderly, blacks, and patients who have risk factors, especially coronary artery disease. It has often been said that mortality caused by heart failure has not improved, despite better understanding of the pathophysiology and treatment; however, outcome studies published since the 1990s indicate that long-term mortality due to heart failure has improved, although the 1-year mortality remains high at 20% to 30% [2,3].

Heart failure is progressive, and after recognition of the syndrome, patients experience greater morbidity and have more frequent encounters with the health care system. Heart failure results in an enormous cost to society in terms of morbidity, mortality, and health care expenditure. In 2005, the direct and indirect costs of heart failure are expected to approach $30
billion [1]. The prevalence of heart failure is likely to increase with aging of the population and improved survival after myocardial infarction (MI).

This article focuses on the outpatient diagnosis and management of heart failure. Treatment of the hospitalized patient who has heart failure is not discussed. The underlying causes of heart failure, such as ischemic cardiomyopathy, must be treated to optimize the patient’s condition; however, such a discussion is beyond the scope of this article. The authors endeavor to provide up-to-date information on syndromes of systolic and diastolic dysfunction, with the understanding that little evidence is available to guide treatment of the latter.

Pathophysiology and natural history

During the past 2 decades, a shift in the understanding of heart failure has taken place. Traditionally, the pathophysiology of heart failure was described in terms of the structural and functional alterations observed. Endogenous neurohormones (eg, angiotensin II, norepinephrine, etc.) were known to be elevated in patients who have heart failure, but were assumed to be compensatory [4]. In fact, these neurohormonal adaptations are initially important in maintaining perfusion, but ultimately result in maladaptive consequences that perpetuate heart failure. The neurohormonal hypothesis has led to modern pharmacologic therapies that exert their beneficial effects on heart failure by blocking many of these same neurohormonal pathways.

For most cardiac diseases, heart failure represents the final common pathway. The pathophysiology of heart failure can be illustrated by considering a patient who has ischemic cardiomyopathy, the most common cause of heart failure. The patient initially has an ischemic event, and if the event is severe enough, infarction occurs and myocardium is destroyed. Heart rhythm is disturbed and pump function is depressed, leading to poor tissue perfusion. To maintain perfusion of vital organs, cardiac output must increase, which occurs by activation of the neurohormonal axis. Norepinephrine, arginine-vasopressin (AVP), angiotensin II, endothelin, and other factors are released. Norepinephrine is the prototype compensatory hormone, causing increased cardiac contractility and rate, systemic vasoconstriction, and sodium retention. AVP causes retention of water to expand plasma volume. Angiotensin II promotes systemic vasoconstriction, induces sodium retention, and promotes pathologic remodeling of the myocardium. Endothelin is a potent vasoconstrictor that has positive inotropic effects and stimulates further secretion of AVP and aldosterone.

When these neurohormones are expressed chronically, a maladaptive pattern emerges, perpetuating heart failure. Analogous to flogging a tired horse, norepinephrine-induced alterations in heart rate and contractility eventually lead to myocardial hypertrophy and ischemia. When any neurohormone acts to increase peripheral sympathetic tone, afterload increases
and greater demand is placed on the heart, resulting in depressed cardiac function and myocardial damage [5]. Chronic activation of the renin-angiotensin-aldosterone system leads to left-ventricular hypertrophy and remodeling. The hypothesis is that activation of these once compensatory systems then causes progression of heart failure, arrhythmia, and death. The two leading causes of death in these patients are progressive inotropic failure and arrhythmia.

All of what has been stated so far applies to systolic heart failure, in which decreased pump function can be measured by functional cardiac studies (eg, echocardiogram, radionuclide ventriculography). The pathophysiologic mechanisms underlying heart failure with preserved systolic function, termed diastolic dysfunction or diastolic heart failure, are less well-understood. The symptoms of dyspnea and exercise intolerance in these patients are thought to be due to a “stiff ventricle.” Diastolic dysfunction is identifiable by findings on echocardiogram [6]. Abnormalities in active relaxation and passive stiffness cause increased pressures during ventricular filling (diastole). In turn, pulmonary venous pressures increase, causing reduced lung compliance. Cardiac output during exercise is not able to increase sufficiently, and patients experience dyspnea on exertion and exercise intolerance [7]. Although it appears clear that patients who have diastolic heart failure have greater mortality than age-matched, disease-free controls, two recent studies indicate that they seem to fare better than those who have systolic heart failure [8,9].

**Diagnosis**

Although at times the diagnosis of heart failure is straightforward, it often challenges physicians, because particular aspects of the syndrome lead to confusion. For instance, a patient presenting with dyspnea, which is the most common symptom of heart failure, will often have a comorbid condition that may also cause the symptom (eg, chronic obstructive pulmonary disease [COPD]). Additionally, a patient may present anywhere along a spectrum from asymptomatic to florid failure. Although the syndrome of heart failure is progressive, there are peaks and valleys along the way, and the point in time when a patient presents is likely to have an impact on the time to diagnosis. Simple clinical tests, such as chest radiography and electrocardiogram, are generally unhelpful in confirming heart failure. Finally, because heart failure is a clinical diagnosis, physicians sometimes disagree about it, resulting in delayed interventions. In elderly patients, making the diagnosis is more treacherous, because of a relative absence of typical signs and symptoms and the possibility of attributing heart failure symptoms to other conditions [10].

Although no single historical item can predict heart failure, patients generally present with dyspnea, fatigue, or decreased exercise tolerance. Less
commonly, patients present with fluid retention as the primary complaint. Symptoms are similar in systolic and diastolic heart failure. Dyspnea with exertion is present in most patients who have heart failure, and its complete absence should cause the clinician to reconsider the diagnosis [11]; however, dyspnea and other symptoms of heart failure are unreliable in the elderly [12]. Also, dyspnea is common, and therefore has poor specificity for heart failure. Likewise, a history of edema alone is unhelpful. Paroxysmal nocturnal dyspnea and orthopnea are generally thought to be more specific for heart failure [13]. A previous history of MI may be the most useful element, because it appears to be more strongly associated with the diagnosis of heart failure than are other historical items, and it is a known risk for developing heart failure [11]. Less commonly, heart failure may present with confusion (as in the elderly patient who has poor cerebral perfusion) or with abdominal symptoms (eg, nausea, pain, anorexia, ascites) caused by hepatic congestion. Finally, the history should include risk factors for heart failure, such as MI, hypertension, and diabetes, because these are associated with the ultimate development of the syndrome and have implications for its management.

When heart failure is suspected, certain elements of the physical examination aid in the diagnosis. Unfortunately, the examination often does not yield enough information for confirmation. Although several diagnostic criteria schemes (eg, Framingham, Boston, and others) are available, their clinical utility is questionable, and their concordance is poor [14]. The findings most strongly associated with increased left ventricular filling pressure in systolic heart failure are jugular venous distention (JVD), edema, and abnormal vital signs [15]. Likewise, JVD, edema, and a sustained or displaced apical impulse are associated with decreased ejection fraction [15]. Other clinical signs that support the diagnosis of heart failure include a third heart sound (S3), tachycardia, systolic hypotension, and pulmonary rales. The S3 has poor sensitivity (about 20%), but good specificity (99%), whereas pulmonary rales lack sensitivity and specificity [11,16]. These signs are helpful when present, but the lack of signs does not rule out heart failure, and they cannot be used to distinguish systolic from diastolic failure. In a patient who has a high likelihood of having heart failure, one or two clinical signs carry greater weight in the diagnosis than the same signs would in a patient who has low likelihood of heart failure.

Upon completion of the examination, patients can be categorized as having low, intermediate, or high likelihood of heart failure. If a patient presents with typical symptoms of heart failure and has three or more signs of systolic dysfunction (tachycardia, hypotension, S3, pulmonary rales, abnormal apical impulse), he has a high likelihood of systolic heart failure [15]. If the clinical likelihood of heart failure is high, an empiric trial of diuretic therapy may be appropriate [17]. In such a patient, weight loss and clinical improvement support the diagnosis of heart failure. If a patient has a low likelihood of heart failure based on atypical symptoms and lack of any
examination findings to support the diagnosis, other causes for the symptoms should be considered; however, most patients do not seem to fit neatly into one of these two categories and are then classified as intermediate likelihood. Table 1 provides an approach to the patient in each category.

A useful test in the office evaluation of the patient who has an intermediate likelihood of heart failure is the B-type natriuretic peptide (BNP). BNP is a neurohormone secreted from the ventricles in response to stress from volume expansion and pressure overload [18]. As such, BNP has good sensitivity for heart failure, and can be used to differentiate dyspnea caused by heart failure from pulmonary causes. For primary care physicians, BNP has the added value of being studied and validated in the outpatient setting [19]. The cutoff level for BNP above which heart failure is diagnosed is generally set at 80 to 100 pg/ml. Using this cutoff is most sensitive and specific for patients who have no history of heart failure and new onset symptoms. Because patients who have a history of heart failure may have elevated BNP at baseline, the diagnostic utility of BNP declines in these patients (who may have dyspnea due to other causes but still have a high BNP). Therefore, establishing a baseline BNP level for heart failure patients allows this test to be used to identify exacerbations of heart failure. Also, most studies have shown that the sensitivity of BNP is superior to its specificity, because elevated BNP levels are caused by any stress on the ventricles, including MI, cardiomyopathy, pulmonary embolism, etc. Finally, BNP is known to have prognostic implications and correlates well with functional limitations [20]. Higher levels of BNP are generally associated with more severe symptoms and a poorer prognosis.

Most patients who have a new diagnosis of heart failure should undergo echocardiography. The American College of Cardiology and American Heart Association (ACC/AHA) guidelines recommend a two-dimensional echocardiogram with Doppler flow studies in these patients [21]. Other authors have suggested that patients who have a high likelihood of heart failure need not undergo this test initially [13,17]; however, echocardiogram is invaluable in distinguishing systolic from diastolic heart failure, a nearly impossible feat to accomplish on clinical grounds alone. Patients who have systolic heart failure will have an ejection fraction less than 40%, and those

<table>
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<th>Probability of HF based on H &amp; P</th>
<th>First steps</th>
<th>Alternative steps</th>
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<tr>
<td>Low</td>
<td>Consider alternative diagnoses</td>
<td>CXR, BNP</td>
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<tr>
<td>Intermediate</td>
<td>ECG, CXR, BNP</td>
<td>Echocardiogram</td>
</tr>
<tr>
<td>High</td>
<td>Trial of diuretic</td>
<td>ECG, CXR, BNP, Echocardiogram</td>
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Abbreviations: BNP, B-type natriuretic peptide; CXR, chest radiograph; ECG, electrocardiogram; HF, heart failure; H & P, history and physical examination.
who have diastolic failure will have preserved ejection fractions, but problems with ventricular filling [6]. Some patients may have both systolic and diastolic abnormalities on echocardiogram. Another reason to perform echocardiography is to identify structural defects (e.g., valvular disease, wall motion abnormalities) that will affect future interventions.

A number of laboratory tests, which are listed in Table 2, are recommended in the patient who have newly diagnosed heart failure. Laboratory testing is aimed at detecting conditions which cause or exacerbate heart failure (e.g., thyroid disease) as well as the function of organs affected by heart failure (e.g., liver function). Coronary angiography deserves special mention, because it is not necessarily recommended in all patients who have heart failure. There is good evidence for the ACA/AHA recommendation for coronary angiography in patients who have new-onset systolic heart failure and angina, MI, or other evidence of cardiac ischemia [21]. In systolic heart failure without evidence of cardiac ischemia, the role of coronary angiography is less well-defined. In patients who have diastolic heart failure, no recommendations currently exist to assist in the decision regarding coronary angiography.

Once the diagnosis is established, heart failure can be further staged and classified based on various scoring systems. In 2001, the ACC/AHA

<table>
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<th>Table 2</th>
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<td><strong>Initial laboratory evaluation in the patient diagnosed with heart failure</strong></td>
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<tr>
<td><strong>Test</strong></td>
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<tr>
<td>ECG</td>
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<tr>
<td>CXR</td>
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<tr>
<td>Echocardiogram (2D with Doppler)</td>
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<tr>
<td>Thyroid-stimulating hormone</td>
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<tr>
<td>CBC</td>
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<tr>
<td>Renal function (creatinine and urinalysis)</td>
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<tr>
<td>Liver function</td>
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<tr>
<td>Electrolytes</td>
</tr>
<tr>
<td>Lipids</td>
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<tr>
<td>Ferritin, transferrin saturation, HIV, ANA</td>
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<tr>
<td>Coronary angiography</td>
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<tr>
<td>Endomyocardial biopsy</td>
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</tbody>
</table>

These tests are generally recommended but do not apply to all HF patients.

*Abbreviations:* ANA, antinuclear antibody test; CAD, coronary artery disease; CBC, complete blood count; 2D, two-dimensional.
published practice guidelines for evaluation and management of heart failure [21], which proposed new staging (Table 3) analogous to staging for cancer. One impetus for this method of staging was to promote recognition of presymptomatic stages of heart failure, so that intervention could occur earlier. This staging system compliments the New York Heart Association (NYHA) functional classification scheme, and the two can be employed together. For example, a patient who has a previous MI and depressed systolic function would be categorized as stage C in the ACC/AHA system, but could also have NYHA Class II heart failure (symptoms with some physical activity). Although the ACC/AHA system follows a stepwise progression of the disease, the NYHA classification is fluid with patients moving from one class to another, depending on the degree of symptomatology at a given time.

Management

Heart failure is a complex syndrome, and patients who have this syndrome may present at different ages, having comorbid conditions, having various etiologies of heart failure, and possessing different expectations from the health care team. All management decisions should begin by establishing goals of care, negotiated between the patient and physician. Patient education is essential in this process. Without a firm understanding of the prognosis and natural history of the syndrome, patients will not be able to participate fully in their own care.

Nonpharmacologic therapy

Optimal management of heart failure relies on risk factor control, lifestyle modification, and patient self-assessment and self-management. These

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<tr>
<th>ACC/AHA staging system</th>
<th>NYHA functional classification system</th>
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<tr>
<td>A At high risk for HF without structural heart disease or symptoms of HF.</td>
<td>I Cardiac disease but no symptoms of HF with ordinary activity</td>
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<tr>
<td>B Structural heart disease without symptoms of HF</td>
<td>II Cardiac disease that limits function slightly, with HF symptoms occurring during ordinary activity but not at rest</td>
</tr>
<tr>
<td>C Structural heart disease with prior or current symptoms of HF</td>
<td>III Cardiac disease that limits function significantly, with HF symptoms occurring during less-than-ordinary activity but not at rest</td>
</tr>
<tr>
<td>D Refractory HF requiring specialized interventions</td>
<td>IV Any physical activity causes HF symptoms; symptoms may occur at rest and get worse with activity</td>
</tr>
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Abbreviations: ACC, American College of Cardiology; AHA, American Heart Association; HF, heart failure; NYHA, New York Heart Association.
interventions should be implemented when heart failure is initially diagnosed, or when a patient is found to be at risk for developing heart failure (ACC/AHA stage A or B). The health care team should assist patients in identifying modifiable risk factors and effecting positive change. The health care team should screen for hypertension, diabetes, and dyslipidemia, and treat these diseases if present. Lifestyle modification should include tobacco cessation, moderation or elimination of alcohol consumption, avoidance of illicit drugs, and weight loss in obese patients. Dietary sodium restriction is often recommended, but has little evidence to support its use in patients who do not have edema or hypertension [21]. In patients who have symptomatic chronic heart failure, exercise has been shown to reduce mortality and hospital admission when patients exercise to a mean peak heart rate of 60% to 80% predicted, and certain patients may benefit from a supervised exercise program [22]. One study of heart failure patients in NYHA Class II or III showed a 32% reduction in all-cause mortality for patients who had inserted an automatic implantable defibrillator compared with those started on amiodarone or placebo [23].

Patients should be instructed to monitor their weight on a daily basis and to determine their baseline “dry weight.” This is often accomplished by having patients weigh themselves on the same scale in the morning after voiding but before eating [17]. A significant change in weight (five pounds or more) indicates a significant change in volume status. Patients who are well-educated about their heart failure may adjust their diuretic based on experience and education, or may contact the health care team for instructions, thereby avoiding a visit to the office or emergency department. In fact, patient education, self-monitoring, and contact with the health care team has been associated with reduced hospitalization [24].

**Pharmacologic therapy**

An evolution in the understanding of heart failure has occurred in the past 2 decades. As described previously, the compensatory neurohormonal mechanisms in heart failure have been well-characterized, and are now targeted for disease-modifying interventions. There are now two avenues of pharmacologic therapy to pursue simultaneously when prescribing medications for heart failure: disease treatment and symptom treatment. Pharmacotherapy for disease treatment includes medications that have been shown to reduce mortality and affect the natural history of heart failure. Symptom treatment relies on diuretics and positive inotropes to reduce symptom severity, but it has a less certain effect on outcomes. Unless otherwise stated, all that now follows applies to systolic heart failure rather than diastolic heart failure. A suggested approach for initiating pharmacotherapy in systolic heart failure patients is provided in Table 4.

Angiotensin-converting enzyme (ACE) inhibitors decrease the production of angiotensin II and the destruction of bradykinins. Numerous clinical
trials have demonstrated the beneficial effects across all functional classes of heart failure for a variety of ACE inhibitors [25]. In fact, ACE inhibitors reduce the risk of developing symptomatic heart failure in patients at high risk, including those who have asymptomatic left ventricular systolic dysfunction, previous MI, and older persons (over age 55) who have diabetes or other vascular disease [26,27]. The ACA/AHA guidelines recommend starting an ACE inhibitor in all patients who have structural heart disease, even without symptoms of heart failure (stage B heart failure). ACE inhibitors should be started at low doses (eg, captopril 6.25 mg three times a day or enalapril 2.5 mg twice a day) and increased as tolerated to reach the recommended target dose for the particular drug. Generally, ACE inhibitors should be the earliest pharmacologic therapy started in heart failure. Serum potassium and creatinine should be measured before and after starting an ACE inhibitor, because hyperkalemia and azotemia are potential adverse effects that may limit the use of the drug.

According to the ACC/AHA guidelines, angiotensin II receptor blockers (ARBs) are recommended as alternative agents for patients who cannot tolerate ACE inhibitors [21]. Compared with placebo, ARBs significantly reduce mortality in heart failure, and the benefit of ARBs appears to be similar to that seen with ACE inhibitors [28]. Combining an ARB and an ACE inhibitor has been evaluated, but so far the results are mixed, and for most patients this approach cannot be recommended with the current evidence.

Based on a structural understanding of heart failure and the observation that beta-blockers worsened acute exacerbations of heart failure, beta-blockers were historically contraindicated; however, strong evidence exists to refute this observation, and beta-blockers are now considered standard therapy in heart failure. Although carvedilol and metoprolol have more recent prospective randomized, controlled trials to support reduced mortality in heart failure, the benefit seems to be a class effect [29]. After ACE

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**Table 4**

A stepwise approach to instituting pharmacotherapy in systolic heart failure

<table>
<thead>
<tr>
<th>Step</th>
<th>Drug class</th>
<th>Comments</th>
</tr>
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<tr>
<td>Step 1</td>
<td>Diuretic</td>
<td>All patients with past or present hypervolemia</td>
</tr>
<tr>
<td></td>
<td>ACE inhibitor</td>
<td>Start low and titrate to maximum tolerated dose</td>
</tr>
<tr>
<td></td>
<td>Beta-blocker</td>
<td>Start a low dose when stable on a low dose of ACE inhibitor; titrate to maximum tolerated dose</td>
</tr>
<tr>
<td>Step 2</td>
<td>ARB</td>
<td>Substitute if patient unable to tolerate ACE inhibitor</td>
</tr>
<tr>
<td></td>
<td>Hydralazine + nitrate</td>
<td>Substitute if patient unable to tolerate ACE inhibitor or ARB</td>
</tr>
<tr>
<td>Step 3</td>
<td>Aldosterone inhibitor</td>
<td>For persistent NYHA Class III or IV HF</td>
</tr>
<tr>
<td>Step 4</td>
<td>Digoxin</td>
<td>For persistent symptomatic HF or recurrent hospitalization</td>
</tr>
</tbody>
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*Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blockers; HF, heart failure; NYHA, New York Heart Association.*
inhibitor therapy, a beta-blocker should be given to all patients who have heart failure, regardless of severity. Like ACE inhibitors, beta-blockers are started at a low dose and titrated to the maximum dose tolerated. When starting a beta-blocker, patients should be euvolemic and relatively stable (eg, not in the intensive care unit).

Aldosterone inhibition has demonstrated benefit in patients who have more severe functional impairment from heart failure (NYHA Classes III and IV). The effect on heart failure with lesser symptoms is not well-known. Spironolactone, a nonselective aldosterone inhibitor, reduces mortality and hospitalization for patients on standard therapy for NYHA Class III or IV heart failure, but is associated with hyperkalemia and gynecomastia [30]. Additionally, there are concerns that spironolactone is being used in patients who are not necessarily appropriate for the drug [31]. Appropriate patients for spironolactone are those who have more severe heart failure on standard therapy and have normal potassium and creatinine less than 2.5 mg/dl [21]. Patients should be kept on a low dose (25 mg every other day to 50 mg daily) and have their potassium and creatinine monitored closely. Another member of this class is eplerenone, a selective aldosterone inhibitor less likely to cause gynecomastia. It has been shown to reduce mortality in a very specific set of patients—those who have heart failure due to left ventricular dysfunction after MI [32]. The usual dose of eplerenone is 25 to 50 mg per day. Hyperkalemia remains a concern, so potassium and creatinine should be monitored closely.

Although no clinical trials exist to determine their effects on mortality, diuretics persist as the mainstay of symptomatic treatment for heart failure. The reason is simple: heart failure presents as a hypervolemic condition, therefore achieving euvolemia should improve symptoms. Thiazide diuretics are appropriate for patients who have mild symptoms, but most patients who have heart failure require loop diuretics. Diuretics are indicated in all patients who have heart failure and who have signs of hypervolemia. For most outpatients who are diuretic-naïve, furosemide 20 to 40 mg (or its equivalent) once daily is a reasonable starting dose [21]. The dose of diuretic should be adjusted so that symptoms of hypervolemia are controlled while maintaining hemodynamic stability, renal function, and electrolyte balance. This tends to be an individualized trial-and-error (or trial-and-success) process.

Diuretic resistance may result from several causes: progression of heart failure, excessive dietary sodium consumption, nonsteroidal anti-inflammatory drugs (NSAIDs), or anything else that reduces renal perfusion [21,33]. If diuretic resistance is encountered, the first step is to increase the dose. The usual maximum single oral dose of furosemide is 200 to 250 mg, or an equivalent dose of torsemide or bumetanide could be employed. Because the half-lives of loop diuretics are on the order of hours, frequent dosing may increase total daily diuresis [33]. The concurrent use of an ACE inhibitor or aldosterone antagonist may facilitate diuresis [34]. Nonpharmacologic
measures, such as water and sodium restriction and avoidance of NSAIDs, should be used. Another option is the addition of a small dose of a thiazide diuretic, such as metolazone or hydrochlorothiazide, to the loop diuretic. If loop and thiazide diuretics are used in combination, potassium, renal function, and hemodynamics must be carefully monitored.

Digoxin, which has historically been part of routine management of heart failure, now plays a more limited role. There is good evidence to suggest that digoxin does not improve mortality in patients who have mild-to-moderate systolic heart failure [35]. It does reduce the risk of hospitalization for heart failure, and is therefore considered a symptomatic treatment, generally employed in patients who are not well-controlled with ACE inhibitors, beta-blockers, and diuretics. Because digoxin has a narrow therapeutic window, a dose range of 0.125 to 0.250 mg per day is appropriate in patients who have normal hepatic and renal function. Patients should be monitored for symptoms of digoxin toxicity. Elderly patients and those who have hypokalemia may present with digoxin toxicity despite having serum digoxin levels within the reference range.

Hydralazine and nitrates reduce afterload and may be prescribed in patients who cannot tolerate ACE inhibitors or ARBs [21]. In patients of African descent who have NYHA Class III or IV heart failure and who are already taking an ACE inhibitor and a beta-blocker, the addition of hydralazine and a nitrate reduces mortality [36]. Aside from these patients, there is no evidence to support routine use of this combination of drugs in heart failure.

Diastolic dysfunction

There is little evidence to guide the treatment of diastolic dysfunction. Management relies on identifying and treating the cause of diastolic dysfunction and relieving the symptoms [7]. Generally, blood pressure should be controlled, initially with an ACE inhibitor, ARB, thiazide diuretic, or a combination of antihypertensives. Tachycardia should be controlled, usually with a beta-blocker or calcium channel blocker. Hypervolemia is treated with diuretics and sodium restriction. None of these therapies has been shown to improve mortality in patients who have diastolic heart failure, however. To date, only one large randomized, controlled trial [37] has shown any benefit: compared with placebo, candesartan reduced the risk of hospitalization in patients who have heart failure and preserved systolic function. There was no effect on mortality, but this study suggests that ARBs should be considered in patients who have diastolic heart failure.

Collaborative care and referral considerations

Patients who have heart failure are most commonly cared for by their primary care physicians (in the United States that includes family physicians, general internists, or geriatricians). Heart failure is a chronic disease and
may be best cared for in the context of a team approach, applying the chronic care model to address the unique problems of these patients [38–40]. Disease management programs using a nurse case manager have been studied and found to reduce the hospital readmission rate of patients who have heart failure [41]. Studies of post-myocardial infarction patients have shown improved mortality of patients when there is shared care between generalists and cardiologists [42]. It is unclear if applying these data to heart failure patients is appropriate. Hospitalized severely ill patients who had heart failure were less likely to be referred to cardiologists based on sociodemographic factors such as race and lower income and education level [43]. Primary care physicians should insure that their patients are not limited to specialized care services except based on informed patient preferences.

In the ambulatory settings, reasons for referral to cardiologists depend on several factors. First, the cause of the heart failure must be identified, and importantly for systolic heart failure, identifying whether ischemic heart disease is the etiology is paramount. Because revascularization procedures are effective treatments for heart failure, improving morbidity and mortality, patients who are candidates for revascularization should be referred for diagnostic evaluation, including coronary angiogram. If revascularization procedures are not an option based on patient preference, maximizing medical management is required. Many well-trained primary care physicians can medically manage patients who have heart failure. Referral to a cardiologist is appropriate when patient response to a medication is atypical, the diagnosis remains in doubt, or more intensive treatment is required through hospitalization. All patients who desire maximal technological interventions with support by artificial pumps or who are candidates for cardiac transplant should be managed with the aid of cardiologists and cardiac surgeons.

Finally, referral to a cardiac electrophysiologist for dysrhythmias associated with heart failure may assist the primary care physician in managing patients who have high risk for mortality.

**Palliative care**

The palliative care of patients who have heart failure is important, because of the advanced age and poor health status of many patients. Heart failure carries a mortality rate very similar to some forms of cancer, and thus early discussion of advanced directives should be a routine part of patient management. This discussion will guide the physician in planning management issues, such as hospitalization and referral, and also in arranging supportive and end-of-life care.

End-stage heart failure is often classified as NYHA functional Class IV—patients who have dyspnea at rest despite maximal medical management and usually suffer from repeated hospitalizations. Treatment approaches
for these patients include all the management strategies outlined above; however, because of age, comorbid conditions, and personal preferences, these patients often request palliative measures to maintain comfort. When patients are identified as appropriate for a palliative approach, intensive home nursing and traditional end-of-life medical therapies, including morphine and anxiolytics for dyspnea, may be employed. Patients who have end-stage heart failure are excellent candidates for hospice services, and can be managed by the primary care physician, even at home.

References


