Heart Failure in the 21st Century: A Cardiogeriatric Syndrome

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Chronic heart failure (CHF) is principally a cardiogeriatric syndrome, and it has become a major public health problem in the 21st century due largely to the aging population. Age-related changes throughout the cardiovascular system in combination with the high prevalence of cardiovascular diseases at older age predispose older adults to the development of CHF. Features that distinguish CHF at advanced age from CHF occurring during middle age include an increasing proportion of women, a shift from coronary heart disease to hypertension as the most common etiology, and the high percentage of cases that occur in the setting of preserved left ventricular systolic function. Although the pharmacotherapy of CHF is similar in older and younger patients, the presence of multiple comorbidities in older patients mandates a multidisciplinary approach to care. Manifest CHF is associated with a poor prognosis, especially in elderly persons, and there is an urgent need to develop more effective strategies for the prevention and treatment of this increasingly common disorder to reduce the individual and societal burden of this devastating illness in the decades ahead.

Pathophysiology

Aging is associated with significant alterations in cardiovascular structure and function, which diminish homeostatic reserve and predispose older individuals to the development of CHF (Table 1) (5,11,12). In general, cardiac output is determined by four factors—heart rate, preload, afterload, and contractile state—and age-related cardiovascular changes have a significant impact on each of these parameters. Thus, diminished β-adrenergic responsiveness and degenerative changes in the sinoatrial node impair the heart rate response to stress, impaired myocardial relaxation and decreased compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance compromise ventricular filling and alter preload, increased vascular stiffness and a reduction in vasomotor tone and reduced vascular compliance comprom...
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Table 1. Principal Effects of Aging on Cardiovascular Structure and Function

| Increased vascular “stiffness,” impedance to ejection, and pulse wave velocity |
| Impaired left ventricular early diastolic relaxation and mid-to-late diastolic compliance |
| Diminished responsiveness to neurohumoral stimuli, esp. β1 and β2 adrenergic stimulation |
| Altered myocardial energy metabolism and reduced mitochondrial ATP-production capacity |
| Reduced number of sinus node pacemaker cells and impaired sinoatrial function |

As discussed previously, aging is associated with impaired left ventricular filling due to changes in relaxation and compliance. These alterations lead to a shift in the left ventricular pressure-volume relationship, such that small increments in left ventricular volume result in greater increases in left ventricular diastolic pressure (Figure 1) (16). This increase in diastolic pressure further compromises left ventricular filling and also leads to increases in left atrial, pulmonary venous, and pulmonary capillary pressures, thus predisposing to pulmonary congestion and CHF. “Diastolic” heart failure, as it is often called, accounts for less than 10% of CHF cases in persons under age 65 but more than 50% of cases after age 75 (13–15,17). Diastolic heart failure is also more common in women than in men and accounts for nearly two thirds of all heart failure cases among women over age 80 (13,14).

Clinical Features

Symptoms and Signs

Exertional dyspnea, orthopnea, lower extremity swelling, and impaired exercise tolerance are the cardinal symptoms of CHF at both younger and older age. However, with increasing age, which is often accompanied by a progressively more sedentary lifestyle, exertional symptoms become less prominent (18). Conversely, atypical symptoms, such as confusion, somnolence, irritability, fatigue, anorexia, or diminished activity level, become increasingly more common manifestations of CHF, especially after age 80.

Physical signs of CHF include elevated jugular venous pressure, hepatojugular reflux, an S3 gallop, pulmonary rales, and dependent edema. Each of these features occurs less commonly in older CHF patients, in part because of the increasing prevalence of diastolic heart failure, in which signs of right heart failure are a late manifestation and a third heart sound is typically absent. On the other hand, behavioral changes and altered cognition, which may range from subtle abnormalities to overt delirium, frequently accompany CHF at an elderly age, particularly among institutionalized or hospitalized patients (19).

Diagnosis

Accurate diagnosis of the CHF syndrome at older age is confounded in part by the increasing prevalence of atypical symptoms and signs (18). In addition, exertional symptoms may be attributable to noncardiac causes, such as pulmonary disease, anemia, depression, physical deconditioning, or aging itself. Likewise, peripheral edema may be due to venous insufficiency, hepatic or renal disease, or medication side effects (e.g., calcium-channel blockers), and pulmonary crepitation may be due to atelectasis or chronic lung disease. Despite these limitations, careful clinical assessment for the presence of multiple symptoms and signs should lead to the correct diagnosis in most cases.

Chest radiography is indicated when CHF is suspected, and it remains the most useful diagnostic test for determining the presence of pulmonary congestion. However, chronic lung disease or altered chest geometry (e.g., due to kyphosis) may confound interpretation of the chest radiograph in elderly individuals.

Proper management of CHF is critically dependent on establishing the pathophysiology of left ventricular dysfunction (i.e., systolic vs diastolic), determining the primary and any secondary etiologies (Table 2), and identifying potentially treatable precipitating or contributory factors (Table 3). Differentiating systolic from diastolic dysfunction requires an assessment of left ventricular contractility by echocardiography, radionuclide ventriculography, magnetic resonance imaging, or contrast angiography. Among these, echocardiography is the most widely used and clinically useful noninvasive test for evaluating systolic and diastolic function. In addition, echocardiography provides important information about left ventricular chamber size and wall thickness, atrial size, right ventricular function, the presence and severity of valvular lesions, and pericardial disorders. For these reasons, echocardiography is recommended for all patients with newly diagnosed CHF or unexplained disease progression (20).

Other diagnostic studies that may be indicated in selected patients include an assessment of thyroid function (esp. in the presence of atrial fibrillation), an exercise or pharmacologic stress test to evaluate for the presence and severity of ischemia, and cardiac catheterization if revascularization or another corrective procedure is being contemplated.
Etiology and Precipitating Factors

Systemic hypertension and coronary heart disease account for 70% to 80% of CHF cases at older age (14,21). Hypertension is the most common etiology in older women, particularly those with preserved systolic function. In older men, CHF is more often attributable to coronary heart disease. Other common etiologies include valvular heart disease (esp. aortic stenosis and mitral regurgitation) and nonischemic cardiomyopathy (Table 2). Importantly, CHF in elderly persons is frequently multifactorial, and it is thus essential to identify all potentially treatable causes.

In addition to determining etiology, it is important to identify factors precipitating or contributing to CHF exacerbations (Table 3). Noncompliance with medications and diet is the most common cause of recurrent heart failure exacerbations (22,23), and patients should be closely questioned about their dietary and medication habits. Other common factors contributing to worsening symptoms include ischemia, volume overload due to excess fluid intake (self-mediated or iatrogenic) (24), tachyarrhythmias (esp. atrial fibrillation or flutter), intercurrent infections, anemia, thyroid disease, and various medications or toxins (e.g., alcohol).

Comorbidity

A hallmark of aging is the increasing prevalence of multiple comorbid conditions, many of which have an impact, directly or indirectly, on the diagnosis, clinical course, treatment, and prognosis of CHF in elderly patients (Table 4) (25). Discussion of individual comorbidities is beyond the scope of this article, but it is important to recognize that heart failure in elderly persons virtually never occurs in isolation; diagnosis and proper management must therefore be placed in the context of the patient’s other comorbidities and competing risks.

Management

The principal goals of CHF therapy are to relieve symptoms, maintain or enhance functional capacity and quality of life, preserve independence, and extend survival. Although it is often stated that quality of life is more important than quantity of life in very elderly persons, this in fact is a matter of personal preference. Furthermore, because the elderly heart failure population is characterized by marked heterogeneity in terms of lifestyle, comorbidity, and per-
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sonal goals and perspectives, management of CHF in elderly patients must first and foremost be individualized to each patient’s circumstances and needs.

The basic approach to CHF management involves identification and treatment of the underlying etiology and contributing factors, implementation of an effective pharmacotherapeutic regimen, and coordination of care through the use of a multidisciplinary team. Each of these components is discussed in detail in the following sections.

Etiology and Precipitating Factors

Although heart failure in the elderly population is rarely "curable," proper treatment of the underlying etiology often improves symptoms and delays disease progression. Thus, hypertension should be treated aggressively (the current recommendation for patients with CHF is a target blood pressure below 130/85 mm Hg) (26), and coronary heart disease should be managed appropriately with medications and/or percutaneous or surgical revascularization. Similarly, therapy for diabetes and dyslipidemia should be optimized, smoking should be strongly discouraged, and a suitable level of regular physical activity should be prescribed. Alcohol intake should be limited to no more than 2 drinks/d in men and 1 drink/d in women, and alcohol use should be strictly proscribed in patients with suspected alcoholic cardiomyopathy.

Severe aortic stenosis is a common cause of CHF in elderly persons, and it can be treated effectively with aortic valve replacement (27). Peri-operative mortality rates are acceptable (less than 10%), and long term results are excellent, even in octogenarians (28). Severe mitral regurgitation may be amenable to surgical therapy (i.e., valve repair or replacement) in selected patients, but the operative results are somewhat less favorable than for aortic valve surgery (29,30). Mitral valve replacement is also effective therapy for severe mitral stenosis; rarely, percutaneous mitral balloon valvuloplasty may be feasible in older patients (30–32).

Atrial fibrillation is a common precipitant of CHF in elderly persons, especially in the setting of diastolic dysfunction. In patients with recent onset atrial fibrillation, most clinicians recommend restoration and maintenance of sinus rhythm if feasible, although the long term benefits of this approach have not been established (33). In patients with chronic atrial fibrillation, the ventricular rate should be well controlled both at rest and during activity. Bradycardia is a less common cause of CHF, for which implantation of a permanent pacemaker provides definitive therapy. Anemia, thyroid disease, and other systemic illnesses should be identified and treated accordingly.

The importance of compliance with medications and dietary restrictions, including avoidance of excessive fluid intake, cannot be overemphasized. Management of these issues is discussed in more detail in the section on multidisciplinary care. Nonsteroidal anti-inflammatory drugs are widely used in older individuals, both by prescription and over the counter. These agents promote sodium and water retention, interfere with the actions of angiotensin converting enzyme (ACE) inhibitors and other anti-hypertensive agents, and may worsen renal function; their use should be avoided whenever possible. Similarly, the use of other medications that may aggravate CHF should be monitored closely.

Pharmacotherapy

The design of an effective therapeutic regimen is based in part on whether the patient has predominantly systolic or predominantly diastolic left ventricular dysfunction. Although these two abnormalities frequently coexist (indeed, virtually all individuals over age 70 have some degree of diastolic dysfunction), for purposes of this discussion patients with an ejection fraction <45% (i.e., moderate or worse left ventricular systolic dysfunction) will be considered as having systolic heart failure, whereas patients with an ejection fraction ≥45% will be considered as having diastolic heart failure.

Systolic heart failure.—In the past 15 years there has been considerable progress in the treatment of systolic heart failure. Although most studies have either excluded individuals over 75 to 80 years of age or have enrolled too few elderly subjects to permit definitive conclusions, the available data indicate that older patients respond as well or better to standard therapies in comparison to younger patients. Furthermore, there is little theoretical basis for suspecting a decremental effect of these therapies in very elderly persons. Therefore, current recommendations are that systolic CHF should be managed similarly in younger and older patients. Specific issues relevant to older patients are discussed in the ensuing paragraphs.

ACE inhibitors.—ACE inhibitors are the cornerstone of therapy for left ventricular systolic dysfunction, whether or not clinically overt CHF is present, and there is strong evidence that ACE inhibitors are as effective in older compared with younger patients, both in terms of reducing mortality and improving quality of life (34,35). On the other hand, older patients are more likely to have potential contraindications to ACE inhibitors (e.g., renal dysfunction, renal artery stenosis, orthostatic hypotension) and may also be at increased risk for ACE-inhibitor–related side effects, such as worsening renal function, electrolyte disturbances, and hypotension. Nonetheless, a trial of ACE inhibitors is indicated in virtually all older patients with documented left ventricular systolic dysfunction.

In most cases, ACE inhibitor therapy should be initiated at a low dose (e.g., captopril 6.25–12.5 mg TID or enalapril 2.5 mg BID), and the dosage should be gradually titrated upward to the level shown to be effective in clinical trials (captopril 50 mg TID, enalapril 10 mg BID, or lisinopril 20 mg qd) (34–37). Once a maintenance dose has been reached, substituting a once-daily agent (e.g., lisinopril) at equivalent dosage may facilitate compliance. Blood pressure, renal function, and serum potassium levels should be monitored closely during dose titration and periodically during maintenance therapy. In patients unable to tolerate standard ACE-inhibitor dosages due to side effects, dosage reduction is appropriate, as there is evidence that even very low doses of these agents (e.g., lisinopril 2.5-5 mg qd) provide some degree of benefit (38).
Angiotensin receptor blockers.—Angiotensin receptor blockers (ARBs) have a more favorable side effect profile than ACE inhibitors and appear to have similar effects on mortality and hospitalization rates (39,40). However, pending the results of ongoing clinical trials, ARBs should not be considered as first-line therapy for CHF but rather as a reasonable alternative to ACE-inhibitors when the latter agents are not tolerated due to cough or other side effects.

Hydralazine and isosorbide dinitrate.—The combination of hydralazine 75 mg QID and isosorbide dinitrate 40 mg QID was associated with decreased mortality in a small trial of CHF patients less than 75 years of age (41). Although ACE inhibitors are superior to hydralazine-nitrates in improving survival (42), the combination provides an additional alternative for patients intolerant of ACE inhibitors. Side effects are common with both hydralazine and high-dose nitrates, and the QID dosing schedule is a further disadvantage of these agents.

Beta blockers.—Beta blockers, once widely viewed as contraindicated in patients with CHF, have now been shown to improve left ventricular function and to decrease mortality in a broad population of heart failure patients, including those up to 80 years of age (43–45). As a result, beta blockers are now recommended as standard therapy for clinically stable patients without major contraindications. Use of beta blockers in older patients may be limited by a higher prevalence of bradycardia and severe chronic lung disease, and older patients may also be more susceptible to the development of fatigue and impaired exercise tolerance during long-term beta-blocker administration.

Carvedilol, metoprolol, and bisoprolol have all been shown to improve outcomes in patients with systolic CHF, but only carvedilol has been approved for the treatment of CHF in the United States. Beta-blocker treatment should be initiated at low dosages in stable patients upon a background of ACE inhibitor and diuretic therapy. Recommended starting dosages are carvedilol 3.125 mg BID, metoprolol 6.25 to 12.5 mg BID, and bisoprolol 1.25 mg QD. The dose should be gradually increased at 2- to 4-week intervals to achieve maintenance dosages of carvedilol 25 to 50 mg BID, metoprolol 50 to 100 mg BID (or sustained release metoprolol 100–200 mg QD), or bisoprolol 5 to 10 mg QD. Lower dosages and a slower titration protocol may be appropriate in patients over 75 years of age. Contraindications to beta blockade include marked sinus bradycardia (resting heart rate <45–50 BPM), PR interval ≥0.24 seconds, heart block greater than first degree, systolic blood pressure <90 to 100 mm Hg, active bronchospastic lung disease, and severe decompensated CHF.

Digoxin.—Digoxin improves symptoms and reduces hospitalizations in patients with symptomatic systolic heart failure treated with ACE inhibitors and diuretics but has no effect on total or cardiovascular mortality (46). These effects are similar in younger and older patients, including octogenarians. Digoxin is thus indicated for the treatment of systolic heart failure in patients of all ages who remain symptomatic despite standard therapy.

The volume of distribution and renal clearance of digoxin decline with age, and the therapeutic range for digoxin is lower in older patients (0.5–1.3 ng/ml after age 70) (47). Higher concentrations of digoxin are associated with increased toxicity but no greater efficacy (48). In most older patients with preserved renal function (est. creatinine clearance ≥50 ml/min), digoxin 0.125 mg daily will provide a therapeutic effect. Lower dosages should be used in patients with underlying renal insufficiency. Although routine monitoring of serum digoxin levels is not recommended, a level should be obtained whenever digoxin toxicity is suspected.

Digoxin side effects include arrhythmias, heart block, gastrointestinal disturbances, and altered neurological function (e.g., visual disturbances). Although older patients are often thought to be at increased risk for digitalis toxicity, this was not confirmed in the recently completed Digitalis Investigation Group trial (Rich MW, unpublished data).

Diuretics.—Diuretics are an essential component of therapy for most patients with CHF and are the most effective agents for relieving congestion and maintaining euvolemia. Some patients with mild CHF can be effectively controlled with a thiazide diuretic, but the majority will require a loop diuretic such as furosemide or bumetanide. In patients with more severe CHF or significant renal dysfunction (serum creatinine ≥2.0 mg/dl), the addition of metolazone 2.5 to 10 mg QD may be necessary to achieve effective diuresis. Recently, the addition of spironolactone 12.5 to 50 mg daily to standard CHF therapy has been shown to reduce mortality in patients with New York Heart Association (NYHA) class III-IV CHF, and benefits were similar in older and younger patients (49,50).

In general, diuretic dosages should be titrated to eliminate signs of pulmonary and systemic venous congestion. Common side effects include worsening renal function (often due to overdiuresis) and electrolyte disorders. To minimize these effects, renal function and serum electrolyte levels (sodium, potassium, and magnesium) should be monitored closely during the initiation and titration phase of diuretic use and periodically thereafter. Spironolactone is contraindicated in patients with severe renal insufficiency or hyperkalemia; painful gynecomastia may occur in up to 10% of patients taking this agent.

Approach to treatment.—Figure 2 provides a suggested approach to the pharmacologic treatment of systolic CHF. All patients with left ventricular systolic dysfunction, whether asymptomatic or symptomatic, should receive an ACE inhibitor (or alternative vasodilator if ACE inhibitors are contraindicated or not tolerated). Patients with stable symptoms and no contraindications should also receive a beta blocker, and diuretics should be administered in sufficient doses to maintain euvolemia. Digoxin should be added for patients who remain symptomatic despite the above regimen, and spironolactone should also be used in patients with persistent NYHA class III-IV symptoms.

Diastolic heart failure.—Despite the fact that over 50% of elderly CHF patients have preserved left ventricular systolic function (13–15), none of the major CHF clinical trials
has specifically targeted this disorder. As a result, treatment of diastolic CHF remains largely empiric. As with systolic CHF, the underlying cardiac disorder and associated contributing disorders should be treated appropriately. In particular, hypertension and coronary heart disease should be managed aggressively. Diuretics should be used judiciously to relieve congestion while avoiding overdiuresis and pre-renal azotemia. Topical or oral nitrates may be beneficial in reducing pulmonary congestion and orthopnea. On the basis of the results of the Heart Outcomes Prevention Evaluation (51), an ACE inhibitor such as ramipril 2.5 to 5 mg BID is rational therapy for most older adults with vascular disease, but the value of ACE inhibitors in the treatment of diastolic heart failure per se has not been established. Similarly, beta blockers are indicated in patients with coronary heart disease (esp. prior myocardial infarction), but the long-term effects of these agents in diastolic CHF are unknown. Angiotensin receptor blockers and calcium channel blockers are effective antihypertensive agents in elderly patients, and these drugs may provide symptomatic palliation in selected patients with diastolic CHF (52,53). Digoxin, in addition to its inotropic effect, also facilitates diastolic relaxation and may improve symptoms and reduce hospitalizations in patients with CHF and preserved systolic function (46). In summary, the physician treating diastolic CHF is presented with an array of therapeutic options, none of proven benefit, and therapy should be individualized and guided by prevalent comorbidities and the observed response to specific therapeutic interventions.

Multidisciplinary Care

The management of CHF in older patients is frequently complicated by the presence of multiple comorbid conditions, polypharmacy, dietary concerns, and a host of psychosocial and financial issues. Moreover, these factors often contribute to poor outcomes in older patients, including frequent hospitalizations (23,54). To address these issues and to provide comprehensive yet individualized care for older CHF patients, a coordinated multidisciplinary approach is recommended. Several recent studies have documented the efficacy of multidisciplinary CHF disease management programs in reducing hospitalizations and improving quality of life in older patients, and these interventions have also been reported to lower overall medical costs (55,56).

Elements of an effective CHF disease management program include patient and caregiver education, enhancement of self-management skills, optimization of pharmacotherapy (including consideration of polypharmacy issues), and close follow-up. The structure of a CHF disease management team is similar to that of a multidisciplinary geriatric assessment team and typically includes a nurse coordinator or case manager, a dietitian, a social worker, a clinical pharmacist, a home health representative, a primary care physician, and a cardiology consultant. Specific goals of disease management are to improve patient compliance with medications, diet, and exercise recommendations by enhancing education and self-management skills in each of these areas; to provide close follow-up and improved healthcare access through telephone contacts, home health visits, and nurse or physician office visits; and to optimize the medication regimen by promoting physician adherence to recommended CHF treatment guidelines (20), simplifying and consolidating the regimen when feasible, eliminating unnecessary medications, and minimizing the risks for drug–drug and drug–disease interactions.

End of Life

The overall 5-year survival rate for older patients with established CHF is less than 50% (i.e., the prognosis is worse than for most forms of cancer) (57,58). Clinical features portending a less favorable outcome include older age, more severe symptoms and functional impairment, lower left ven-
tricular ejection fraction, underlying coronary heart disease, and impaired renal function (59). Older patients with advanced CHF, as evidenced by NYHA class III-IV symptoms, have a 1-year mortality rate of 25% to 50%; for these patients, CHF can properly be considered a terminal illness. In addition, all CHF patients are at risk for sudden arrhythmic death, which may occur during periods of apparent clinical stability. For these reasons, it is appropriate to address end-of-life issues early in the course of CHF and to reconsider these issues periodically as the disease progresses or when changes in clinical status occur.

Although discussing end-of-life issues is often challenging for healthcare providers as well as patients and families, specific measures should be undertaken to plan for and facilitate end-of-life care (60). These include the development of an advance directive and appointment of durable power of attorney. The advance directive should be as explicit as possible in defining circumstances under which the individual patient would not want to be hospitalized, intubated, subjected to other life-sustaining interventions (e.g., a feeding tube), or resuscitated. Because it is well established that patients often change their minds about these issues as clinical circumstances evolve, it is important to maintain open communication throughout the disease process.

End-stage CHF is frequently accompanied by considerable discomfort and anxiety, and data from the SUPPORT study indicate that most patients and families express concerns about the quality of end-of-life care (61,62). A cardinal principal of end-of-life care is to provide adequate relief of pain and suffering through the judicious use of conventional therapies in conjunction with narcotics (e.g., morphine), sedatives (e.g., benzodiazepines), and other comfort measures. Equally important is the provision of emotional support for the patient and family, assisted by nurses, members of the clergy, social service representatives, and other qualified healthcare professionals.

**Future Directions**

In light of the high prevalence and poor prognosis associated with CHF in elderly persons, it is evident that more effective means for the prevention and treatment of this disorder are needed. At present, the most effective preventive strategies involve aggressive treatment of established risk factors for the development of CHF (i.e., hypertension and coronary heart disease). Several studies have shown that even modest reductions in blood pressure are associated with substantial reductions in incident CHF among elderly hypertensive patients (Table 5) (63–68). Likewise, treatment of elevated cholesterol levels with an HMG-CoA reductase inhibitor (“statin”) has been shown to decrease incident CHF following an acute coronary event (69). Similarly, it is likely that smoking cessation, weight control in obese patients, and aggressive control of diabetes will all lead to a reduction in CHF. Finally, thrombolysis and coronary angioplasty reduce infarct size and the subsequent risk for CHF in patients with acute myocardial infarction (MI), and the more widespread application of reperfusion therapies in elderly patients with acute MI should be strongly encouraged.

Asymptomatic left ventricular systolic dysfunction is associated with a high rate of progression to clinical CHF, and ACE inhibitors have been shown to reduce the incidence of CHF in these patients (36,70). Therefore, documented systolic dysfunction mandates ACE inhibitor therapy even in the absence of symptoms. Although routine screening for left ventricular dysfunction is not justified at the present time, it has been argued that screening echocardiography may be worthwhile in high-risk older patients, such as those with known coronary heart disease or multiple coronary risk factors (71).

In addition to existing therapies, several new treatments for CHF, both pharmacological and technological, are currently under active investigation (Table 6). Although it is difficult to project which of these new therapies will ultimately come into clinical use, it is likely that the management of CHF will undergo substantial evolution over the course of the next several decades; indeed, we may be on the threshold of radical changes in the paradigm of CHF prevention and therapy.

### SUMMARY AND CONCLUSIONS

At the dawn of the twenty-first century, chronic heart failure has become a major public health problem that threatens to consume astronomical resources in the years

| Table 5. Effect of Antihypertensive Therapy on Incident Heart Failure in Older Adults |
|-----------------------------------|-------|-------------|-----------------|
| Trial                             | n     | Age Range (y) | Reduction in CHF |
| EWPHE(63)                         | 840   | ≥60         | 22%             |
| Coope(64)                         | 884   | 60–79       | 32%             |
| STOP-HTN(65)                     | 1627  | 70–84       | 51%             |
| SHEP(66)                          | 4736  | ≥60         | 55%             |
| Syst-Eur(67)                     | 4695  | ≥60         | 36%             |
| STONE(68)                        | 1632  | 60–79       | 68%             |

*Note: CHF = chronic heart failure; EWPHE = European Working Party on Hypertension in the Elderly; SHEP = Systolic Hypertension in the Elderly Program; STONE = Shanghai Trial of Nifedipine in the Elderly; STOP-HTN = Swedish Trial in Old Patients with Hypertension; Syst-Eur = Systolic Hypertension in Europe Trial.*

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**END-OF-LIFE ISSUES**

Asymptomatic left ventricular systolic dysfunction is as-
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ahead as the number of older adults at risk for developing CHF continues to rise. A multitude of age-related changes in cardiovascular structure and function, coupled with the increasing prevalence of cardiovascular diseases at older age, serve to perpetuate an exponential increase in both the incidence and prevalence of CHF with advancing age. As a result, CHF is largely a disorder of elderly persons, and, in context of the marked heterogeneity of the older adult population, chronic heart failure warrants designation as a true cardiogeriatric syndrome. As such, optimal management of CHF in the elderly population necessitates a multidisciplinary approach that effectively addresses all aspects of patient care, both pharmacologic and nonpharmacologic, as well as relevant comorbidities, in a comprehensive, coordinated, and personalized manner.

Current treatment of CHF in elderly patients is characterized by widespread underutilization of proven therapies (72), insufficient evidence to guide treatment in major patient subgroups (e.g., octogenarians and beyond, nursing home residents, patients with advanced comorbidities, and individuals with diastolic CHF), and inattention to critically important psychobehavioral issues (e.g., compliance, personal preferences, and end-of-life care). Clearly there is a need for substantial additional research aimed at developing more effective approaches to the prevention and treatment of chronic heart failure in older adults.

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