

Congestive Heart Failure in Older Adults

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The frail elderly patient population is characterized not only by age, but also by the inadequacy of the office-based medical model to meet their needs when they can no longer regularly reach the physician's office. Diagnosis and treatment is difficult because we are often called to see new patients with presenting symptoms of congestive heart failure (CHF) that are precipitated by one of many proximate causes.

In 2004, 70% of patients admitted to the hospital for CHF were older than 65.¹ CHF is the leading cause of hospitalization for patients older than 65 and the most costly diagnosis-related group (DRG) in the United States with annual costs in 2003 exceeding \$27 billion.^{2,3} The incidence and prevalence of CHF are rising; prevalence doubles each decade after age 45.³ One-year survival for elder patients with class III and IV CHF is about 60%.³

There is a close and perhaps causative relationship between hypertension and CHF in the elderly population because of the complex neurohormonal reflexes involved. As people age, cardiovascular impedance normally increases, beta-adrenergic responsiveness decreases, and myocardial energy metabolism is altered. The heart's diastolic relaxation and compliance are impaired. The net effect is marked reduction in cardiovascular reserve (see *CHF Classification and Complications*).

Precipitating Events

Precipitating events must be evaluated first, before the patient's CHF can



be addressed. The most common event is patients' failure to take their medications because of financial, social, or cognitive problems. Physicians often choose this time to switch patients from increasingly greater doses of diuretics to use of angiotensin-converting enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARBs) as the primary therapy; the latter two classes are the modern approaches to CHF therapy.³

Other precipitating factors for CHF common to our population include anemia, hyperthyroidism, infection, new-onset arrhythmias such as atrial fibrillation, changes in diet, silent myocardial infarction, worsening valvular disease, inappropriate use of the sodium-retaining nonsteroidal anti-inflammatory drugs (NSAIDs), and environmental

changes such as increased ambient humidity or temperature, and stress. Obviously, these historical and clinical factors must be evaluated too.

When assessing a new patient in the assisted living (AL) setting, medical records, ECG, and x-rays may be unavailable. In that case, many patients, especially those on multiple medications, will be well-served if the physician uses an entirely new approach to assessing their clinical symptoms. Of course, this approach is used only after precipitating causes are addressed.

Physiology Review

The function of the heart is to pump arterialized blood forward under sufficient pressure to meet the metabolic needs of the peripheral tissue. Given adequate oxygenation and a

normal hemoglobin, cardiac output (defined as pulse rate \times stroke volume) should be sufficient to prevent angina (ischemia to the heart), cold knee caps (ischemia to the skin), oliguria or azotemia (ischemia to the kidney), and confusion (ischemia to the brain). Thus, a quick lab check with a portable analyzer and pulse oximetry is required to rule out azotemia, hypoxemia, and metabolic acidosis. A venous sample with a normal base excess can prove the absence of a low perfusion state.

Note that the above definition of inadequate cardiac output (CO) does not include blood pressure. In fact, the only time hypotension is an emergency in CHF is when diastolic pressure is below 60 mm Hg and the patient is experiencing angina or failure symptoms. Because 85% of coronary arterial blood flow occurs during diastole, the aortic root pressure is critical in determining myocardial perfusion. In the emergency department, some patients in shock with angina find that dopamine is the best analgesic.

To evaluate the failing heart, one needs to think in terms of myocardial efficiency, *not* blood pressure. Efficiency of the heart is measured in terms of oxygen demand by the heart balanced against the stroke work index (SWI). Oxygen demand is determined by 5 factors, of which only 3 are clinically relevant: pulse rate, mean arterial pressure (MAP), and wall tension index. Myocardial oxygen demand is linearly increased when the pulse rate exceeds 100 beats per minute (bpm). It is dramatically increased by elevated MAP (often estimated by a value one third the distance between the diastolic and systolic pressures); and it is increased with increasing wall tension index. The wall tension index is impossible to measure on a house call, but can be inferred from an assessment of left ventricular end-diastolic volume (cardiomegaly). For example, the patient with a pulse of 110 bpm, blood pressure of 140/100 mm Hg, and cardiomegaly

CHF Classifications and Complications

New York Heart Association (NYHA) Classification

Class I. No limitation of physical activity. No shortness of breath, fatigue, or heart palpitations with ordinary physical activity.

Class II. Slight limitation of physical activity. Shortness of breath, fatigue, or heart palpitations with ordinary physical activity, but patients are comfortable at rest.

Class III. Marked limitation of activity. Shortness of breath, fatigue, or heart palpitations with *less than* ordinary physical activity, but patients are comfortable at rest.

Class IV. Severe to complete limitation of activity. Shortness of breath, fatigue, or heart palpitations with *any* physical exertion, and symptoms appear even at rest.

Conditions Associated with Increased Severity in CHF

Weight. Patients who are overweight tend to have more severe CHF.

Impaired Kidney Function. Impaired kidney function is an important indicator of a poor outlook.

Congestion. Two-year survival rates are 87% in those who were free of pulmonary edema compared to 41% to 67% in patients with various signs of the congestion.

Atrial Fibrillation. This abnormal heart rhythm is very dangerous in people with CHF.

Sleep Apnea. Patients with apnea have a higher mortality rate from CHF than those without it.

Depression. Depression may have adverse biologic effects on the immune and nervous systems, blood clotting, blood pressure, blood vessels, and heart rhythms.

Seasonal and Daily Patterns. More emergency department visits and higher mortality occur during winter months and on Mondays in patients with CHF.

Source: University of Maryland Medical Center

on chest x-ray is demanding much more oxygen from the heart than a patient with normal heart size and vital signs. When the presenting symptoms include angina, the approach to CHF is first to treat the increased oxygen demand by using nitrates or calcium channel blockers to lower MAP, slow the pulse, and vasodilate the coronary arteries. Once the patient is stabilized, the CHF may be addressed.

SWI is a complicated measurement that previously required the use of a pulmonary arterial catheter, but the measurement can be thought of in clinical terms quite simply. Remember, the heart does not have to create higher-than-normal pressures to increase CO if the periph-

eral vascular resistance (PVR) is reduced. Left ventricular SWI is calculated basically by multiplying CO by PVR. The human heart is a very efficient flow generator, but a very inefficient pressure generator. Thus, oxygen demand to increase CO is efficiently handled under normal loading conditions. But in hypertension, compensation is difficult. Because of the inefficiencies and maladaptive neurohumoral reflexes of the cardiovascular system, the failing heart simply increases blood pressure without generating an adequate increase in CO. Thus, hypertension is the enemy in both ischemic heart disease and CHF.

In a chronic state of heart failure, physiologic changes in elderly

people are numerous and complex, including:

1. Increased renin levels, which increase the conversion of angiotensin I to angiotensin II, the most potent vasoconstrictor known today
2. Higher renin levels, which also stimulate the kidneys to raise aldosterone levels, thereby leading to increased retention of sodium and water
3. Greater adrenergic tone in the adrenal cortex, which elevates circulating norepinephrine levels and inappropriately increases PVR
4. Decreased levels of adenylyl cyclase, which lowers levels of cyclic AMP that can lead to lower protein kinase levels, calcium entry levels, and calcium reuptake by myofibrils

The net result is a chronic “fight-or-flight” background level of physiologic stress, which ultimately leads to cardiac failure.

I think of my elderly patients as being on an endogenous catecholamine drip that produces more hypertension than forward flow. Since it is unlikely that the patient will ever be “normal,” it is useful to think in terms of functional status when one begins therapy—therapies that maximize forward blood flow at the lowest physiologic cost.

Treatment Principles

To maximize CO, one must think in terms of the 3 major forces we can affect—preload, contractility, and afterload.

Preload: The hypovolemic patient may be hypotensive or in shock simply because of fluid depletion. This is easily discerned by taking a history and physical and using portable laboratory testing equipment. The hypervolemic patient is often much harder to evaluate because chronic rales, interstitial lung disease, emphysematous changes, and obesity may all affect our confidence level. Therefore, some simple in-home testing may be useful. A

chest x-ray that shows cardiomegaly is strong support for a chronic hypervolemic state and elevated left ventricular filling pressures. The presence of left ventricular hypertrophy (LVH) or left ventricular strain pattern on the ECG (R waves in V5 and V6 add up to more than 25 mv) is often associated with volume overload. A simple test, done only in the hypertensive patient, is the so-called Chatterjee test, in which one monitors the pulse before and after a single dose of sublingual nitroglycerine (NTG). Since NTG predictably lowers the pulmonary capillary wedge pressure (PCWP; left ventricular filling pressure), the hypervolemic patient will *not* have a

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tachycardic change in pulse with NTG. The normal or hypovolemic patient will show an increased pulse rate of 10% or more. Thus, the absence of tachycardia to NTG is *de facto* evidence of increased PCWP and requires treatment.

The use of nitrates alone to control preload and subsequent CHF symptoms is complicated by the tolerance that all patients develop to the longer-acting versions. The use of nitrates in elderly patients is most often beneficial during the acute house call when the patient has suddenly decompensated and has hypertension in addition to CHF symptoms. One must be careful to place the patient in a recumbent position before administration, especially in the home setting, be-

cause nitrate syncope is much more likely to occur in these patients.

After preload is reduced, diuresis is the next strategy. Diuretics have been the mainstay of treatment for both hypertension and CHF for some 40 years despite the fact that they have yet to be shown to increase survival rates in controlled prospective clinical trials. Currently, 35% to 40% of all seniors over the age of 65 are taking diuretics in some form. However, cumulative studies now clearly show a number of adverse effects from the use of diuretics:

1. Sixty percent of all toxic drug reactions result from diuretic use in elderly patients.
2. Diuretics cause the most common drug reactions requiring hospitalization.
3. Both loop and thiazide diuretics cause increased serum cholesterol, although subsequent cardiac disease is unproven.
4. All diuretics can cause increased uric acid levels with subsequent tophaceous gout.
5. Both loop and thiazide diuretics can cause glucose intolerance in patients with type 2 diabetes, which is not reversible when diuretic therapy is stopped. (This is less true of Bumex than Lasix.)
6. Both types of diuretics cause deafness, even in chronic oral dosing regimens (Bumex less so than Lasix).
7. Both diuretic types can cause decreased calcium absorption and worsen osteoporosis (loop diuretics more so than thiazides).
8. Both types can cause behavioral risks with incontinence in women and urine retention in men.
9. Both can lead to hyperkalemia and hypokalemia in elderly patients.
10. Both can cause severe hyponatremia in elderly patients.

Thus, the use of diuretics is now considered second- or third-line therapy, after more contemporary treatment for both hypertension and/or CHF has failed.

Contractility: The state of in-

otropy of the heart in CHF patients is compromised by many of the maladaptive neurohormonal changes discussed previously and by loss of cardiac tissue from current ischemic heart disease and past infarcts. Unfortunately, the use of phosphodiesterase inhibitors in elderly persons to increase inotropy has been clinically unsuccessful. Use of adrenergic agents such as dopamine and dobutamine requires monitoring and infusion therapy. However, although stage IV CHF patients are doing well on home Dobutrex drips, studies have shown such therapy does not prolong life. And many patients believe a chronic IV decreases the quality of life.

The essential difference between the two adrenergic agents in common use today is important. Whereas dopamine causes a more potent increase in inotropy and blood pressure, it has little or no effect on PCWP, increases ventricular irritability, and requires much more care. Dobutamine, on the other hand, not only has less toxicity at the same cardiac output, but lowers PCWP dramatically, thereby restoring the normal cardiac anatomy and reducing myocardial oxygen demand.

To reduce the oxygen demand of the heart, beta-adrenergic blockers were increasingly used in CHF patients during the '80s and early '90s. Unfortunately, the use of beta blockers to control hypertension in elderly patients creates much more toxicity and has been shown to increase mortality due to side effects and increased cholesterol levels in all age groups. The current new enthusiasm for selective beta blockade in elderly patients needs to be analyzed more closely with ambulatory cardiac output monitoring to improve patient selection.

The use of digoxin remains controversial in patients who do not have atrial fibrillation. Although digoxin is the oldest and most proven inotropic agent, and the only such drug that also decreases wall tension index (cardiomegaly),

its toxicity and need for periodic serum measurements makes it risky for some patients.

A *New English Journal of Medicine* study in 1997 of class II or III CHF patients showed no change in mortality with digoxin therapy added to ACE inhibition therapy, but demonstrated a significant improvement in functional status and decrease in hospital days.⁴ Most patients with an ejection fraction (EF) of more than 45% will do well on digoxin alone for control of symptoms of CHF.

Patients with cardiomegaly and/or an S3 gallop should have a trial of digoxin alone before using multiple drug combinations. Finally, no one argues about the use of



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digoxin to control rapid ventricular response in patients with atrial fibrillation and CHF.

Afterload: If SWI can be reduced by reducing MAP, one might be able to both increase CO and decrease myocardial oxygen demand. This result would be the best of both worlds! Indeed, the newer studies show that peripheral vasodilatation causes less morbidity and better survival rates with better functional status than conventional beta blocker/diuretic combinations. The geriatric literature offers many studies confirming the observation that ACE inhibitors alone can control hypertension and should be used for patients at risk for CHF (such as LVH or cardiomegaly on chest x-ray) even when they are asymptomatic. By using low-dose ACE inhibitors at

night, one can often avoid orthostatic hypotension and preserve the peripheral vasomotor reflexes so important to preventing side effects. Many patients can be converted from 2 to 4 older medications (digoxin, furosemide, potassium chloride, and beta-blockers such as Tenormin) to a single dose of benazepril 10 mg at bedtime. Titrating the benazepril up to 80 mg daily may control blood pressure while relieving the symptoms of CHF as well. Therapeutic effects must be evaluated by the patient's subjective reports of exercise tolerance or orthopnea, as well as objective measurement of weight. If CHF is to be controlled on ACE inhibitors alone, the patient will lose weight on it.

In addition, the Cooperative North Scandinavian Enalapril Survival Study^{5,6} showed that ACE inhibition improves symptoms and exercise tolerance, while affording significant survival benefits among patient with severe CHF. These agents not only decrease afterload, but preload as well, and deter activation of the neuroendocrine system, leading to reduced risk of hypertensive episodes, stress reduction, less fluid retention, and perhaps less arrhythmogenicity.

A newer class of drugs similar to ACE inhibitors is the angiotensin-receptor blockers (ARBs) such as losartan. These drugs have been shown in several trials (ELITE, LIFE) to provide better than or equal survival to ACE inhibitors, and they do not cause the chronic cough that plagues some patients who take ACE inhibitors.^{7,8}

Some patients require diuretic therapy, and adding a calcium channel blocker may be useful to improve inotropy by increasing coronary blood flow and further decreasing the peripheral vascular resistance. In addition, calcium channel blockers such as amlodipine have been shown to increase end-diastolic ventricular relaxation, thereby promoting ventricular filling and increasing stroke volume at the

same filling pressures. Once hypertension is under control, SWI is decreased enough to require no further pharmacological therapy for heart failure. Should such therapy be required, a diuretic should be cautiously introduced because its effect is additive to both ACE inhibition and calcium channel blockade.

What has been lacking in both the critical care and outpatient care of patients with CHF is an ability to measure systemic vascular resistance (SVR). Thus, we still base our clinical decisions on nonphysiologic parameters, ie, MAP. Although MAP may be a useful guideline, many patients with borderline or low CO are normotensive while their SVR is markedly elevated. We cannot know this without measuring CO, which heretofore has been invasive and expensive with use of the pulmonary arterial catheter. The advent of noninvasive, inexpensive cardiac impedance studies in the outpatient and home settings has opened up dramatic opportunities to not only titrate therapies toward maximum lowering of SVR to improve CO, but also to titrate therapies both acutely and chronically toward optimal cardiac efficiency since impedance can track the preejection period and minimize isovolumetric contraction time.

Atrial Fibrillation

No discussion of CHF or hypertension in elderly patients can be complete without mention of the importance of anticoagulation for patients with atrial fibrillation (AF). Warfarin (Coumadin) has severe toxicity problems; therefore, international normalized ratio (INR) measurements should be coordinated with home health agencies to prevent significant events. Studies have not been done to show the use of warfarin in addition to vitamin E and/or baby aspirin daily, but they clearly have shown that warfarin is preferable to vitamin E or aspirin alone.

In one study of 651 patients with AF published in *Stroke* (1997),⁹ a

significant bias against using warfarin in patients older than 45 years existed. This bias was still present when risk factors such as diabetes, heart disease, history of prior cerebrovascular accident (CVA), or GI bleeding were accounted for. Fully 42% of the AF patients were discharged from the hospitals with no specific anticoagulation therapy. Of the patients with no contraindication to anticoagulation, 40% went untreated, although patients between the ages of 45 and 54 were twice as likely to be treated as those between ages 75 and 84.

The clinical benefit of anticoagulation in younger CHF patients is now a mainstream concept. A meta-analysis

of 5 prospective clinical trials of stroke prophylaxis in nonvalvular AF published in 1994 demonstrated that anticoagulation reduced stroke rates by 68% and overall mortality by 33%. The overall stroke rate was 4.5% for controls and 1.4% yearly for the warfarin-treated group. In these studies, aspirin reduced stroke rate by 3.6% but had no effect on mortality rates.¹⁰

The Framingham and other studies have clearly shown that stroke risk in patients with AF increases with advancing age. Predictors of increased risk besides age include prior stroke, history of hypertension, mitral valvular disease, diabetes, history of myocardial infarction (MI), and CHF. In data pooled from numerous trials, the risk of intracranial hemorrhage in patients older than 80 was 0.3%, about the same as in the over-65 group.

Given that warfarin anticoagula-

tion reduces deaths and strokes significantly, that CHF increases the risk for stroke and MI, and that recent data suggest there is no greater complication rate in older patients with anticoagulation, it seems prudent to offer anticoagulation to patients with AF and discuss the relevant issues of quality of life, safety of medication compliance, and supportive care in the home. ALC

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References

1. Rosamond W, Flegal K, Friday G, et al. Heart disease and stroke statistics—2007 update. A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2007;115:e69-e171.
2. *AHRQ Electronic Newsletter*. September 16, 2005, Issue #178. Agency for Healthcare Research and Quality, Rockville, MD. AHRQ Web site. <http://www.ahrq.gov/news/enews/enews178.htm>. Accessed August 28, 2007.
3. Hunt SA, Abraham WT, Chin MH, et al. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). *Circulation*. 2005;112:154-235.
4. The effect of digoxin on mortality and morbidity in patients with heart failure. The Digitalis Investigation Group. *N Engl J Med*. 1997;336(8):525-533.
5. Swedberg K, Kjeksus J. Effects of enalapril on mortality in severe congestive heart failure: results of the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS). *Am J Cardiol*. 1988;62(2):60A-66A.
6. Effects of enalapril on mortality in severe congestive heart failure. Results of the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS). The CONSENSUS Trial Study Group. *N Engl J Med*. 1987;316(23):1429-1435.
7. Pitt B, Segal R, Martinez FA, et al. Randomised trial of losartan versus captopril in patients over 65 with heart failure (Evaluation of Losartan in the Elderly Study, ELITE). *Lancet*. 1997;349:747-752.
8. Dahlöf B, Devereux RB, Kjeldsen SE, et al. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. *Lancet*. 2002;359(9311):995-1003.
9. Munschauer FE, Priore RL, Hens M, Castilone A. Thromboembolism prophylaxis in chronic atrial fibrillation. *Stroke*. 1997;28:72-76.
10. Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation. Analysis of pooled data from five randomized controlled trials. *Arch Intern Med*. 1994;154(13):1449-1457.