Concise Review for Primary-Care Physicians

Diagnosis and Outpatient Management of Congestive Heart Failure

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Congestive heart failure causes substantial patient morbidity and mortality in the United States. Symptoms and physical findings can be helpful in diagnosis but have limited sensitivity and specificity. Objective measurement of ventricular function is essential in virtually all patients in whom a diagnosis of heart failure is suspected. Reversible causes of heart failure must be sought. Outpatient management includes education and counseling, emphasis on and assessment of compliance with diet, and pharmacologic treatment. Angiotensin-converting enzyme inhibitors are the mainstay of treatment but are underused, and maximal doses are not given apparently because of concern about side effects. Diuretic therapy should be administered only as needed to manage fluid overload. Calcium channel blockers are relatively contraindicated in patients with impaired ventricular function. Patient follow-up should be guided by the results of the medical history and physical examination. Routine serial testing of ventricular function and exercise performance is discouraged.

Congestive heart failure is a clinical syndrome characterized by (1) signs or symptoms of volume overload or (2) manifestations of impaired tissue perfusion such as fatigue or impaired exercise tolerance (or both). It affects an estimated 3 million Americans, and about 400,000 new cases are diagnosed yearly. Patients with congestive heart failure have an impaired quality of life and decreased survival rates. In 1990, the estimated total costs for treatment of heart failure were more than $10 billion.

The syndrome of heart failure may be predominantly left sided, right sided, or biventricular. It is most commonly due to systolic dysfunction, although primary diastolic dysfunction has been reported to cause up to 40% of cases of heart failure.

This review deals exclusively with heart failure due to left or biventricular systolic dysfunction and does not address primary diastolic dysfunction, cor pulmonale, or the treatment of patients with surgically correctable valvular disease. It emphasizes common pitfalls in diagnosis and management, which are potential arenas for improvement in clinical practice. (A more detailed discussion on this topic can be found in the Agency for Health Care Policy and Research Clinical Practice Guideline.) Although general recommendations are presented in this article, management decisions must consider the practitioner's available resources and the specific circumstances of the individual patient.

CLINICAL DIAGNOSIS AND EVALUATION

Symptoms of orthopnea, paroxysmal nocturnal dyspnea, and progressive dyspnea on exertion are more likely to represent heart failure than are edema, decreased exercise tolerance, and fatigue, especially when the three last-mentioned symptoms occur in isolation. Any of these symptoms, however, can be caused by pulmonary diseases, obesity, deconditioning, intermittent cardiac ischemia, or respiratory infections rather than by heart failure.

Results of a physical examination often have limited sensitivity in the diagnosis of heart failure, especially in patients with obesity, barrel chests, or lung hyperinflation. Although findings of increased jugular venous pressure, a third heart sound, or a laterally displaced cardiac apex impulse are fairly specific for heart failure, detection of rales and peripheral edema is not. Leg edema is unlikely to be due to heart failure if the jugular venous pressure is not increased.

In symptomatic patients, cardiomegaly on chest roentgenography is highly suggestive of heart failure, especially when accompanied with pulmonary venous congestion. Normal chest roentgenographic findings, however, do not exclude the possibility of ventricular systolic dysfunction.

Electrocardiography is nonspecific in patients with heart failure; nevertheless, it may detect ischemia, dysrhythmia, infarction, or hypertrophy, conditions that would affect as-
Hospitalization.—Hospitalization is indicated for patients with hemodynamic and electrolyte state, treatment of associated hyperkalemia on the use of ACE inhibitors). And follow-up is inadequate.

Conditions is evident: acute myocardial ischemia, pulmonary edema or severe respiratory distress, severe complications, and initiation of education. More detailed comments about an inpatient treatment strategy are beyond the scope of this review.

At dismissal, the patient, as well as any caregivers, should be well educated in diet, medications, activity and exercise recommendations, and symptoms associated with worsening heart failure (and what to do if they occur). Outpatient follow-up care is important.

### Treatment

#### Hospitalization.

Hospitalization is indicated for patients with known or suspected heart failure if one of the following conditions is evident: acute myocardial ischemia, pulmonary edema or severe respiratory distress, severe complications, and initiation of education. More detailed comments about an inpatient treatment strategy are beyond the scope of this review.

At dismissal, the patient, as well as any caregivers, should be well educated in diet, medications, activity and exercise recommendations, and symptoms associated with worsening heart failure (and what to do if they occur). Outpatient follow-up care is important.

#### General Counseling.

Patients and their families must understand heart failure and the planned management. Education should be presented during multiple sessions (to improve assimilation of information) by physicians with active assistance from dietitians, nurse educators, clinical nurse specialists, pharmacists, and support groups. Visiting or home-health nurses can be especially helpful. The symptoms associated with worsening heart failure and what to do if patients experience them, medications, and diet should be emphasized. Patients should weigh themselves daily and be instructed to seek medical assessment if they note an increase of more than 3 to 5 pounds in 1 to 2 days.

Patients and their families should be advised about the prognosis associated with heart failure. The mean annual mortality rate for patients with class II heart failure is 5 to 10% but increases to as high as 50% for patients with class IV symptoms. Up to half of the deaths are due to sudden arrhythmias, and a fourth occur without preceding worsening of heart failure.

#### Nonpharmacologic Measures.

Regular exercise should be encouraged for patients with stable class I, II, or III heart failure. The need for supervised cardiac rehabilitation should be assessed on a patient-to-patient basis.

Dietary sodium should be restricted. Patients with mild heart failure and no pronounced fluid excess may tolerate a 3-g per day sodium diet (no added salt and avoidance of salty foods). Those with more problematic fluid excess must decrease their dietary intake to 2 g or less per day. The need for fluid restriction should be individualized, although all patients with systolic dysfunction should be advised to limit their intake to less than 2 qt per day. All patients with heart failure should receive specific dietary guidelines; involvement of the spouse and family in this educational process is important.

Alcohol is a myocardial depressant. Patients with alcohol-induced ventricular dysfunction should abstain totally.

### Causes of Heart Failure

<table>
<thead>
<tr>
<th>Cause</th>
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<tbody>
<tr>
<td>Coronary artery disease*</td>
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<tr>
<td>Idiopathic dilated cardiomyopathy</td>
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<tr>
<td>Myocarditis</td>
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<tr>
<td>Hypertension*</td>
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<tr>
<td>Valvular heart disease*</td>
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<tr>
<td>Alcohol*</td>
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<tr>
<td>Chemotherapy</td>
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<tr>
<td>Acquired immunodeficiency syndrome (AIDS)*</td>
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<tr>
<td>Infiltrative disorders (sarcoidosis, hemochromatosis, amyloidosis)*</td>
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<tr>
<td>Peripartum</td>
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<tr>
<td>Muscular dystrophy</td>
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<tr>
<td>Tachycardia-induced heart failure*</td>
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<td>Pheochromocytoma*</td>
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*Heart failure may potentially be reversed with specific treatment.
from alcohol use. Those with ventricular dysfunction but no history of alcohol use should be discouraged from ingesting alcohol; whether complete abstinence is required is unknown.

Noncompliance negatively affects patient symptoms, hospitalization rate, and, possibly, life expectancy. The importance of compliance with diet, medications, and physical activity must be reinforced at all follow-up examinations. An ongoing, coordinated team approach to patient education and supervision of dietary compliance can help minimize noncompliance.

**Pharmacotherapy. Diuretic Treatment.**—Patients with fluid overload should receive diuretic therapy. Although mild heart failure can occasionally be managed with a thiazide diuretic, loop diuretics are necessary for patients with severe fluid overload, renal insufficiency, or refractoriness to thiazides. Patients with heart failure due to systolic dysfunction who do not have volume overload should not be treated with diuretics. In some patients, diuretic therapy is required only intermittently on an “as-needed” basis. Excessive diuresis should be avoided before institution of ACE inhibitor therapy because it potentiates hypotension or renal insufficiency and often leads to an inaccurate diagnosis of intolerance to ACE inhibitors. After ACE inhibitor therapy has been initiated and doses have been increased, decreasing the diuretic dose is often possible or necessary.

Potassium depletion commonly occurs with long-term diuretic therapy. This depletion is offset in varied degrees by concomitant use of ACE inhibitors, potassium sparing diuretic therapy, and dietary salt substitutes that contain potassium. Serum potassium levels should be assessed every 3 to 5 days during initiation, titration, or modification of diuretic or ACE inhibitor therapy and every 2 to 3 months thereafter. Inasmuch as serum potassium can be an unreliable indicator of total body potassium stores, an optimal serum potassium level is 4.0 mEq/L or higher. Hypokalemia refractory to supplementation may signify coexistent hypomagnesemia.

Patients with refractory volume overload despite compliant use of high-dose diuretics and confirmed sodium restriction may require intravenous diuretic therapy, cautious supplemental use of metolazone, addition of spironolactone, or hospitalization for adjudant inotropic or other therapy.

**ACE Inhibitors.**—All patients with ventricular systolic dysfunction regardless of symptomatic state should receive ACE inhibitors unless specific contraindications exist (Table 2). The benefits of ACE inhibitor therapy include the following: reduced mortality, enhanced functional status, and decreased hospitalization rates for congestive heart failure. Patients with asymptomatic left ventricular systolic dysfunction have also been shown to benefit from ACE inhibitor therapy; it slows the progression of left ventricular dilatation and decreases the incidence of heart failure and its associated hospitalizations.

Side effects of ACE inhibitor therapy include hypotension, azotemia, and hyperkalemia, but the importance of such complications in major trials of heart failure was minimal. Use of low doses of ACE inhibitors, carefully supervised slow upward dose titration, avoidance of volume depletion, and attention to other sources of potassium allow most patients to tolerate these agents. A dry, nonproductive cough is a frequent side effect of ACE inhibitors but also a symptom of heart failure, and the distinction is often difficult. Cough is not an absolute indication to discontinue use of ACE inhibitors; rather, a decision must be made on an individual basis whether the benefit of ACE inhibition is outweighed by the patient's distress from the cough.

All ACE inhibitors are likely effective in treating heart failure, although not all have been approved for this indication nor have all been tested in trials of mortality. Data are insufficient for recommending one specific agent over another.

Doses of ACE inhibitors should be titrated upward, with the goal of achieving the doses used in large-scale trials (captopril, 50 mg three times a day; enalapril, 10 or 20 mg twice a day). Patients who cannot take ACE inhibitors should be treated with another vasodilating regimen, as subsequently discussed. In such patients, the role of new angiotensin II inhibitors is not yet defined.

**Digoxin.**—Digoxin is uniquely suited for patients with heart failure due to systolic dysfunction and atrial fibrillation. Data about its role in sinus rhythm have been controversial, although most investigators now believe that patients receiving digoxin have improved physical function, decreased symptoms, fewer examinations in the emergency department, and fewer hospital admissions. Additionally, patients in whom use of digoxin is discontinued are more likely to experience clinical deterioration than are those undergoing sham withdrawal. Accordingly, digoxin is usually initiated along with ACE inhibitors and diuretic therapy in patients with severe heart failure or is added to the treatment program if the diuretic dose is insufficient for mitigating fluid overload.
regimen in patients who remain symptomatic despite opti-

mal management with ACE inhibitors and diuretics. A

large multicenter trial is currently under way to assess the
effects of digoxin on mortality rate.

The typical daily dose of 0.25 mg of digoxin should be
decreased to 0.125 mg for patients who have reduced renal
function or baseline conduction abnormalities or who are
small or elderly. After a week of treatment, the serum level
should be assessed and should be reevaluated if heart failure
worsens, renal function deteriorates, additional medications
that affect the digoxin level are added (quinidine, verapamil
hydrochloride, and amiodarone), or signs of toxicity (nausea,
visual change, severe conduction abnormality, or increase in
ventricular arrhythmias) develop.

Other Vasodilators.—Many vasodilators have been
tested in patients with heart failure. Most show no sustained
hemodynamic benefit or decreased morbidity or mortality.
Currently, the only exception is the combination of
isosorbide dinitrate and hydralazine, which has a proven
survival benefit in comparison with placebo, although it is
not as effective as enalapril. Even though side effects are
more common with this combination than with ACE inhibi-
tors, this regimen provides an important alternative for pa-
tients who cannot take ACE inhibitors. Alternatively,
isosorbide and hydralazine may be added (individually or
together) to the treatment regimen of selected patients who
do not receive optimal afterload reduction with maximally
tolerated doses of ACE inhibitors.

The initial dosages are as follows: isosorbide, 10 mg
three times a day, and hydralazine, 25 mg three times a day;
these dosages should be increased weekly as tolerated to 40
mg and 75 mg three times a day, respectively. Of note,
clinical trials used such dosages four times a day, but in 20 to
30% of the patients in these trials, use of one or both drugs
was discontinued because of side effects.

Calcium Channel and β-Adrenergic Blocking
Agents.—First-generation calcium channel blockers
(verapamil hydrochloride, diltiazem hydrochloride, and
nifedipine) are negative inotropic agents and should not be
used in patients with heart failure due to ventricular systolic
dysfunction. Second-generation agents may be safer and
even efficacious due to their greater vasodilating properties.
Data suggest that amiodipine besylate is safe and may pro-
provide survival benefit in patients with heart failure due to idi-
opathic cardiomyopathy, although no such benefit was noted
in patients with ischemic left ventricular dysfunction. Cur-
rently, however, this agent is best reserved for patients who
cannot take ACE inhibitors or the combination of isosorbide
and hydralazine.

In patients with heart failure, β-blockers continue to be
intensively studied and may prove efficacious for certain
patient subgroups. Their use, however, is still considered
experimental and should be confined to centers with experi-
ence and expertise in their administration.

Anticoagulant Therapy.—Patients with heart failure
who have a history of systemic or pulmonary embolism,
atrial fibrillation, or known ventricular or atrial thrombi
should undergo systemic anticoagulant therapy until a target
international normalized ratio of 2.0 to 3.0 is achieved. An-
ticoagulant therapy for other patients with impaired ven-
tricular dysfunction is controversial.

REVASCULARIZATION

In patients with ischemic reduction in left ventricular sys-
tolic function, the goal of revascularization is to prevent
further ischemic injury, to restore function to hibernating
myocardium (that is, viable but nonfunctioning myocardium
due to chronic underperfusion), or both. The three classic
randomized trials that compared coronary artery bypass
grafting to medical management (Coronary Artery Surgery
Study, Veterans Administration Coronary Artery Bypass
Surgery Study, European Coronary Surgery Study) excluded
patients with clinical heart failure or ejection fractions less
than 35%. Cohort studies have demonstrated benefit with
revascularization in patients with both heart failure (or left
ventricular dysfunction) and severe or limiting angina; how-
ever, no randomized controlled studies have evaluated the
outcomes of revascularization in patients with asymptomatic
or minimally symptomatic coronary artery disease and heart
failure or left ventricular dysfunction. Any potential benefit
of surgical revascularization in such patients (with or with-
out large territories of inducible ischemia) must be weighed
against the mortality associated with surgical treatment,
which is higher in patients with lower ejection fractions or
higher class of heart failure (or both). As with coronary
artery bypass grafting, the benefits of percutaneous trans-
luminal coronary angioplasty in patients with heart failure
have not been studied in randomized trials. Because of this
lack of scientific information, the recommendations in Table
3 may be reasonable.

<table>
<thead>
<tr>
<th>Table 3.—Guidelines for Evaluation of Ischemia in Patients With Heart Failure</th>
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<tr>
<td>Patients with contraindication to revascularization (including unwillingness to consider such a procedure) should not undergo evaluation of an ischemic contribution to their ventricular dysfunction</td>
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<tr>
<td>Patients with heart failure and angina should undergo coronary artery angiography</td>
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<tr>
<td>Patients with heart failure and a history of myocardial infarction but no current angina should undergo physiologic testing for ischemia and possibly angiography</td>
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<tr>
<td>Decision of whether and how to search for ischemia in patients with heart failure but no angina or evidence of prior myocardial infarction should be individualized</td>
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FOLLOW-UP
The results of the medical history and physical examination should provide the cornerstone of patient follow-up. Information from family members and other caregivers is often a useful supplement to the patient's history. Findings on a physical examination may be more helpful in monitoring patients with heart failure than in making the initial diagnosis. Routine serial testing of ejection fraction or exercise tolerance is usually not justified. Nonetheless, these factors should be reassessed if a pronounced change is clinically suspected or if a major change in management strategy is being contemplated.

REFERRAL
Patients whose condition remains limited despite maximal management with traditional agents should be referred to centers that have experience with severe heart failure, access to experimental medical therapy and devices, and a successful transplantation program.

REFERENCES
2. Rihal CS, Davis KB, Kennedy JW, Gersh BJ. The utility of clinical, electrocardiographic, and roentgenographic variables in the prediction of left ventricular function. Am J Cardiol 1995; 75:220-223

Questions About Congestive Heart Failure (See article, pages 1080 to 1084)

1. A 73-year-old man has exertional fatigue but minimal dyspnea. He has an ejection fraction of 29% and is currently being treated with digoxin, 0.125 mg every other day; furosemide, 80 mg three times a day; and captopril, 6.25 mg twice a day. His blood pressure is 90/60 mm Hg, and his pulse is 96 beats/min. The neck veins are flat when he is reclined at 30°. His lungs are clear, an S3 gallop is evident but no murmurs, and his legs are free of edema. Pertinent laboratory findings are as follows: hemoglobin, 14.5 g/dL; potassium, 3.7 mEq/L; creatinine, 1.9 mg/dL; blood urea nitrogen, 92 mg/dL; and digoxin level, 1.6 ng/mL. The most appropriate management at this time would include which one of the following?
  a. Decrease furosemide dosage to 80 mg twice a day and increase captopril dosage as tolerated after 2 to 3 days
  b. Add spironolactone, 25 mg twice a day, to the regimen to increase the serum potassium level
  c. Increase digoxin dosage to 0.125 mg 2 of every 3 days
  d. Consolidate furosemide dosage to 120 mg twice a day to decrease the hypokalemia
  e. Refer patient to a medical center that has a protocol for β-blocker therapy for patients with heart failure

2. A 59-year-old man has severe exertional dyspnea, new three-pillow orthopnea, and paroxysmal nocturnal dyspnea but denies having edema, exertional distress in his chest, neck, arm, or epigastric area, or a history of myocardial infarction. His only risk factor for premature coronary artery disease is diabetes mellitus, for which insulin was recently instituted. Physical examination reveals bilateral rales, a displaced apical impulse, and an S3 gallop. Electrocardiography discloses a nonspecific conduction delay but no Q waves. Chest roentgenography shows pulmonary edema. The ejection fraction by rest MUGA is 23%, and the ventricular dysfunction is symmetric. Which one of the following statements about his management is most appropriate?
  a. Best initial treatment is as an outpatient; thus, the diuretic and ACE inhibitor doses could be optimized while the patient receives his usual diet
  b. Hospitalization to undergo an endomyocardial biopsy; myocarditis, sarcoidosis, hemochromatosis, or amyloidosis should be sought
  c. Hospitalization for diuretic therapy, initiation of ACE inhibitors, collection of cardiac enzymes, and some assessment of myocardial perfusion
d. Assessment of his coronary circulation is unnecessary because his ejection fraction is too low to consider revascularization, no data support revascularization in the absence of angina, and his symmetric ventricular dysfunction on MUGA precludes a diagnosis of coronary disease
e. Decision to assess his coronary circulation should not be made until he has received medical treatment because the issue will be moot if he becomes asymptomatic after receiving therapy

3. A 68-year-old woman is referred because of a 2-month history of progressive exertional dyspnea. She has a history of borderline hypertension and has received long-term treatment with hydrochlorothiazide, 25 mg per day. Physical examination reveals mild rhonchi that clear incompletely with cough, a grade 2 (on the basis of 1 to 6) systolic ejection murmur at the left sternal border, and mild pitting edema. Chest roentgenography discloses mild cardiomegaly, and electrocardiography reveals mild left ventricular hypertrophy. Which one of the following is the most appropriate next step in her management?

a. Double diuretic dose and assess potassium level in 3 days
b. Order an echocardiogram
c. Initiate therapy with captopril (6.25 mg), assess dose, and increase to 12.5 mg three times a day if tolerated
d. Refer patient to a dietitian and nurse educator for heart failure instructions and then initiate therapy with a long-acting ACE inhibitor
e. Discontinue use of the thiazide diuretic; determine potassium level and chemistry panel; initiate furosemide, 20 mg per day; and reassess her condition in 4 days

4. A 63-year-old man is referred 3 weeks after hospitalization because of an anterior wall myocardial infarction complicated by hypertension. His ejection fraction is 33%, he is asymptomatic and participating in cardiac rehabilitation, and a recent dipyridamole thallium scan, including reinjection and delayed images, showed a fixed anterior, septal, and apical flow reserve abnormality. Current daily medications include furosemide, 40 mg; potassium chloride, 20 mEq; enalapril, 5 mg; nifedipine, 60 mg; doxazosin mesylate, 4 mg; and warfarin sodium, 5 mg. Which one of the following would be most appropriate in his clinical management?

a. Discontinue use of warfarin in the absence of an indication for its use
b. Recommend an angiogram to assess his candidacy for revascularization
c. Discontinue use of furosemide and potassium while assessing for evidence of fluid accumulation
d. Discontinue use of nifedipine, maximize dosage of enalapril as tolerated by renal function and potassium status, and discontinue use of doxazosin if blood pressure control allows
e. Six weeks after the infarction, schedule MUGA to reassess ventricular function, a graded exercise test to establish exercise tolerance, and plasma catecholamines to assess status of neurohormonal system

5. A 57-year-old man has severely limiting exertional dyspnea but no other symptoms. Recent echocardiography disclosed an ejection fraction of 23%. He is taking digoxin, 0.25 mg per day; enalapril, 5 mg twice a day; and furosemide, 40 mg daily. On physical examination, his pulse is irregularly irregular at 104 beats/min, and his blood pressure is 105/65 mm Hg. His lungs are clear, and an S₃ gallop and trace lower extremity edema are evident. Laboratory findings are as follows: potassium, 4.2 mEq/L; creatinine, 1.2 mg/dL; hemoglobin, 13.8 g/dL; sensitive thyroid-stimulating hormone, 0.05 mIU/L; total cholesterol, 287 mg/dL; and low-density lipoprotein cholesterol, 168 mg/dL. Which one of the following would be the most appropriate in his management?

a. Decrease dosage of furosemide to 20 mg daily for 3 days and then increase enalapril dosage to 10 mg twice a day
b. Perform transesophageal echocardiography and electrical cardioversion if no thrombus is present
c. Refer patient to a dietitian for a low cholesterol, low sodium diet and initiate therapy with a 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitor such as lovastatin
d. Assess digoxin level and increase dosage to 0.375 mg daily if the level allows
e. Further evaluate his thyroid function

Correct answers:

1, 2, 3, 4, 5, 6