9. HEART FAILURE

Arleen Brown, MD

The quality indicators for heart failure were developed using the AHCPR Clinical Practice Guideline Number 11, "Heart Failure: Evaluation and Care of Patients With Left-Ventricular Systolic Dysfunction" (Konstam, 1994), and the guideline from a joint task force of the American College of Cardiology and American Heart Association (ACC/AHA Task Force, 1995). These guidelines were supplemented by recent reviews of heart failure (Packer, 1992; Cohn, 1996). Where these core references cited studies to support individual indicators, we have referenced the original sources. We also performed MEDLINE searches of the medical literature from 1985 to 1995 to supplement these references for particular indicators.

IMPORTANCE

Heart failure is a common disease with high associated morbidity and mortality. Over two million people in the United States are affected by this syndrome, and there are over 400,000 new cases of heart failure diagnosed annually (Smith, 1985). In addition it is the only cardiovascular disease that is increasing in prevalence (MMWR, 1994). In the Framingham study, the five-year mortality after congestive heart failure (CHF) developed was approximately 60 percent in men and 45 percent in women (McKee, 1971). In the past two decades, there has been significant improvement in the pharmacologic management of patients with heart failure secondary to left ventricular systolic dysfunction. However, despite these advances, annual mortality exceeds 20 percent overall (Schocken, 1992), and may reach 26 percent over six months for those with severe disease (CONSENSUS, 1987).

Heart failure is also a costly diagnosis. In 1992, over $10 billion were spent on physician visits, medications, nursing home stays, and other health care expenditures, with more than $7 billion of this amount spent on hospitalizations. Because Medicare reimbursements were
only half of the actual charges, these hospitalizations represent an important source of uncompensated medical care.

Heart failure is a clinical syndrome characterized by intravascular and interstitial volume overload and inadequate tissue perfusion. The most common etiology of heart failure in the United States is coronary artery disease, which is the underlying cause in approximately two-thirds of persons with heart failure. Other causes of heart failure are dilated cardiomyopathy (often viral or secondary to toxins such as alcohol), chronic hypertension, valvular disease, and hypertrophic cardiomyopathy. Heart failure may also be divided into systolic dysfunction and diastolic dysfunction. Systolic dysfunction, the impairment of the ventricle’s ability to pump blood forward, is defined by a low ventricular ejection fraction and may result in both congestion and hypoperfusion. Diastolic dysfunction is defined as impaired ventricular relaxation or distention during diastole. It is increasingly recognized as an important cause of heart failure, and may occur in up to 40 percent of patients with heart failure. The left-ventricular ejection fraction may be normal (as in patients with left ventricular hypertrophy) or elevated (as in patients with hypertrophic cardiomyopathy). Combined systolic and diastolic dysfunction is also common.

This chapter will emphasize management of patients with left-ventricular systolic dysfunction. It will not cover guidelines for heart failure due to diastolic dysfunction because there are limited data on therapies known to be effective, nor will it cover heart failure due to valvular disease, aneurysm, or myocardial disease (such as amyloidosis or sarcoidosis).

SCREENING

The prevention of clinical heart failure, through prevention of coronary disease and control of hypertension, is crucial to decreasing the morbidity and mortality associated with this syndrome. Primary prevention measures for coronary disease and control of hypertension are discussed in Chapters 10 and 11, respectively. Additionally, there is a significant role for preventing progression to symptomatic heart failure
in asymptomatic patients with an ejection fraction under 40 percent. These patients have been shown to benefit from the use of angiotensin converting enzyme (ACE) inhibitors. The Studies of Left Ventricular Dysfunction (SOLVD) trial showed that enalapril, titrated to 10 mg twice daily, reduced the development of symptomatic heart failure from 30 percent in the placebo group to 21 percent in the treatment group at the three year follow-up (SOLVD Investigators, 1992). Treatment with ACE inhibitors in this population is discussed further below.

**DIAGNOSIS**

Symptoms that suggest heart failure include paroxysmal nocturnal dyspnea (PND); orthopnea; dyspnea on exertion; lower extremity edema; reduced exercise tolerance; unexplained confusion, altered mental status, or fatigue in an elderly person; and abdominal symptoms associated with ascites and/or hepatic engorgement (such as nausea or abdominal pain). However, most of these symptoms have limited specificity and/or sensitivity for heart failure (Konstam, 1994).

Many patients with a severely reduced ejection fraction will be asymptomatic. One study found that 20 percent of patients with ejection fraction less than 40 percent had no clinical criteria for heart failure (Marantz, 1988), while another found that only 42 percent of patients with an ejection fraction under 30 percent had dyspnea on exertion (Mattleman, 1983).

Of the symptoms that suggest heart failure, dyspnea on exertion is often the presenting symptom, and the combination of orthopnea, paroxysmal nocturnal dyspnea, and progressive dyspnea on exertion are relatively specific for heart failure. In addition, patients with a history of previous myocardial infarction, poorly controlled hypertension, or other heart disease, who also have any of the suggestive symptoms, should be evaluated for heart failure (Konstam, 1994).

Because heart failure may be precipitated or exacerbated by several other clinical conditions, patients with symptoms suggestive of heart failure or with documented new heart failure should be asked about (Kostam, 1994; ACC/AHA Task Force, 1995) (Indicator 2):
• prior myocardial infarction;
• angina or anginal equivalents;
• current complaint of fatigue or dyspnea;
• current complaint of edema or recent weight gain;
• other cardiac history, such as murmur, failure, arrhythmia, pacemaker, rheumatic heart disease, enlarged heart;
• hypertension;
• diabetes;
• renal disease;
• pulmonary disease;
• thyroid disease;
• gastrointestinal disease;
• medications;
• alcohol use.

The physical examination is important, both to the initial evaluation of patients with symptoms suggestive of heart failure and in follow-up evaluation. Many patients with early symptoms of heart failure or with moderate-to-severe left ventricular systolic dysfunction have no physical findings suggestive of heart failure. However, there are several findings on the physical exam that suggest heart failure (Indicator 3):

• Elevated jugular venous pressure or positive hepatojugular reflux;
• A third heart sound;
• Laterally displaced apical impulse;
• Pulmonary rales that do not clear with cough;
• Peripheral edema not due to venous insufficiency.

The third heart sound is the most sensitive physical finding, and in one study was found in 68 percent of patients with EF below 30 percent (Mattileman, 1983). A laterally displaced apical impulse was found in 42 percent of patients, while rales were found in 37 percent of patients (Harlan, 1977). Data on the specificity of the examination in heart failure are limited; however, the most specific signs are elevated jugular venous pressure and the third heart sound. In addition, rales in a patient with other symptoms and no known pulmonary disease are
highly suggestive of heart failure (Konstam, 1994). In studies of the overall utility of the clinical exam for detecting an ejection fraction under 50 percent, sensitivity has ranged from 66 to 95 percent and specificity from 29 to 76 percent (Sanford, 1982; Mattleman, 1983; Cease, 1986; Eagle, 1988; McNamara, 1988; Gadsboll, 1989).

In order to provide a baseline for monitoring treatment response and address potential hypertensive control issues, the initial physical examination of a patient with suspected or documented heart failure should include documentation of weight and blood pressure (Indicator 6).

Because many patients with heart failure do not manifest any signs, the sensitivity of even careful physical examination is limited. In addition, intra- and inter-rater reliability of the clinical exam in heart failure patients is variable. For the physical examination, interrater agreement ranged from 0.6 for the third heart sound to 0.92 for hepatojugular reflux (Butman, 1993). Agreement was lower, 0.48, for cardiomegaly on chest x-ray. Therefore, some patients with symptoms that are highly suggestive of heart failure, especially those who are treated with medications for heart failure, should undergo echocardiography or radionuclide ventriculography to measure ejection fraction even in the absence of physical examination findings (Indicators 1 and 5) (ACC/AHA Task Force, 1995; Konstam, 1994; Retchin and Brown, 1991). Assessment of left ventricular function helps to evaluate not only the presence of heart failure, but also may indicate its etiology and assist in management of the condition. Echocardiogram and/or radionuclide angiography may help to distinguish systolic from diastolic dysfunction. In addition, these studies may help to distinguish heart failure from other disease states such as pulmonary disease, venous insufficiency, and obesity, with similar signs and symptoms, yet different management strategies. Echocardiography and radionuclide ventriculography are both acceptable means of assessing left ventricular function and can be used to distinguish between systolic or diastolic dysfunction. Although each test has relative advantages and disadvantages in assessing left ventricular function, the proposed indicators do not favor one diagnostic mode over the other (Indicators 1 and 5).
Several other diagnostic tests are recommended by experts for the evaluation of causative, precipitating, or complicating causes of new heart failure. A chest radiograph should be performed to evaluate for cardiomegaly, to distinguish between cardiac and pulmonary causes of dyspnea, and to provide information on chamber size and valvular disease. A chest radiograph without cardiomegaly argues against the diagnosis of heart failure except when pulmonary hyperinflation may mask an enlarged heart (Echeverria, 1983; Dougherty, 1984). An electrocardiogram should also be performed to evaluate the patient for causes of congestive heart failure such as myocardial ischemia, atrial fibrillation, bradyarrhythmias, prior myocardial infarction, low voltage, and left ventricular hypertrophy (Indicator 4).

Several laboratory tests are indicated in the evaluation and management of heart failure. A complete blood count should be performed to evaluate for heart failure due to anemia. A hematocrit below 25 may result in signs and symptoms of heart failure in the absence of underlying cardiac abnormalities. Because renal disease may cause volume overload that can mimic or exacerbate heart failure, an evaluation of renal function should be performed. This should include a urinalysis and serum creatinine. In addition, electrolytes, blood urea nitrogen (BUN), and creatinine should be evaluated to assist in subsequent management, such as determining whether and when an ACE inhibitor should be started (Indicators 4 and 7).

**TREATMENT**

**Diuretic Therapy**

Patients with heart failure and symptoms or signs of volume overload should be treated with a thiazide and/or loop diuretic (Whight, 1974; Kupper, 1986). Symptoms of volume overload include orthopnea, PND, and dyspnea on exertion. The signs of volume overload include pulmonary rales, a third heart sound, jugular venous distention, hepatic engorgement, ascites, peripheral edema, and pulmonary vascular congestion or pulmonary edema on chest radiograph.

Potassium depletion is common in patients treated with diuretics (although patients who are taking ACE inhibitors concomitantly may not
have significant potassium depletion). Even though serum potassium may
be an unreliable indicator of total body potassium, experts recommend
that all patients starting diuretics should have their serum potassium
checked within one week of the start of treatment (Indicator 10). In
addition, heart failure patients in whom diuretic dose is increased
should have a potassium level checked within one week of the increase in
dose (Kostam, 1994) (Indicator 12).

Although diuretics provide hemodynamic and symptomatic benefits in
persons with heart failure with pulmonary or peripheral edema, they have
potential toxicity, due in part to activation of the renin-angiotensin
system, and limited efficacy, as patients on diuretics alone have high
rates of clinical deterioration (Captopril-Digoxin Multicenter Research
Group, 1983). Other medications may be indicated, as reviewed below.

ACE Inhibitors

Mortality benefit has been demonstrated from the use of the ACE
inhibitor enalapril in patients with heart failure. In the SOLVD study,
patients with heart failure who had reduced left ventricular ejection
fractions had a reduction in four-year mortality from 40 percent on
placebo to 35 percent on enalapril, with median survival increased by
six months (SOLVD Investigators, 1992). The CONSENSUS trial of patients
with NYHA Class IV heart failure found a mortality reduction from 52
percent with placebo to 36 percent for those on enalapril (CONSENSUS,
1987). In addition, in the Veterans Affairs Cooperative Vasodilator-
Heart Failure Trial II, enalapril reduced mortality in heart failure
patients more than the combination of isosorbide dinitrate and
hydralazine (Cohn, 1991).

Several studies have also shown improvement in functional status
(SOLVD Investigators, 1992; CONSENSUS, 1987; Bussmann, 1987; Captopril-
Digoxin Multicenter Research Group, 1983; Franciosa, 1985; Jennings,
1984; Kleber, 1991; Magnani, 1986; McGrath BP, 1985; Remes, 1986;
Sharpe, 1984), physical functioning (Chalmers, 1987; Lewis, 1989;
Riegger, 1990; Riegger, 1991), and a reduction in hospitalizations
(SOLVD Investigators, 1992) among patients with heart failure and
reduced left-ventricular ejection fraction who are taking ACE-
inhibitors. It is not clear whether the favorable effects of ACE inhibitors can be attributed to hemodynamic effects, the reduction in the level of angiotensin II in plasma or in tissue, increased plasma concentrations of bradykinin or nitric oxide, and/or inhibition of the central nervous system.

Side effects associated with the use of ACE inhibitors include hypotension, increases in serum creatinine and potassium, cough, and less commonly, dizziness and angioedema (Kjekshus, 1988; Frank, 1989; Packer, 1989; Hasford, 1991). In the SOLVD and CONSENSUS trials, the changes in blood pressure and serum chemistries were relatively small (CONSENSUS, 1987; SOLVD Investigators, 1992), and it has been suggested that relatively low blood pressure, moderate renal insufficiency, and mild hyperkalemia are not absolute contraindications to the initiation or continued use of ACE inhibitors (Konstam, 1992). Because some patients with blood pressure lower than 90 mm Hg can be safely started and maintained on ACE inhibitors, the AHCPR guideline recommends that physicians who are uncomfortable starting or maintaining this therapy in patients with low systolic blood pressure at baseline refer such patients to a clinician with expertise in treating heart failure, rather than discontinue the use of ACE inhibitors or other vasodilators.

Potassium-sparing diuretics should be discontinued in patients on ACE inhibitors, regardless of the serum potassium, and potassium supplements should be withheld unless the patient has a low serum potassium. All patients with renal insufficiency who are on ACE inhibitors should be carefully monitored and doses titrated upward cautiously. Patients with serum creatinine of 3.0 mg/dL or greater were excluded from major trials of these agents, so the risks and benefits of ACE inhibitors in these patients are not known. Because cough is common in heart failure (SOLVD, 1992; Cohn, 1991), patients ACE inhibitors who report coughing should be evaluated for pulmonary congestion as the cause before considering discontinuation of the medication. Dizziness and angioedema are usually mild and do not require discontinuation of the drug; however, angioedema of the oropharyngeal region is an absolute contraindication to further use of the ACE inhibitor (Indicator 10).
The AHCPR clinical practice guidelines recommend that patients with heart failure due to left ventricular systolic dysfunction receive a trial of ACE inhibitors unless they have one or more of the following specific contraindications (Konstam, 1994)(Indicator 8):

1. A history of intolerance or adverse reaction to ACE inhibitors;
2. Serum potassium greater than 5.5 mEq/L that cannot be reduced;
3. Symptomatic hypotension (SBP < 100 mm Hg)

Even after a myocardial infarction and the development of left ventricular systolic dysfunction (EF < 40%), treatment with ACE inhibitors can slow or prevent progression to symptomatic heart failure. The Survival and Ventricular Enlargement (SAVE) trial, which evaluated the use of captopril in patients with myocardial infarction in the preceding three to 16 days and ejection fractions of 40 percent or lower, found 20 percent mortality in captopril-treated patients compared to 25 percent mortality in those on placebo over two to five years follow-up (Pfeffer, 1992). In addition, the proportion of patients hospitalized for heart failure fell from 17 percent to 14 percent. The Cooperative New Scandinavian Enalapril Survival Study (CONSENSUS) II found a reduction in incidence of symptomatic heart failure with enalapril, from 30 percent to 27 percent, but no reduction in mortality (CONSENSUS, 1987).

Based on these data, two categories of asymptomatic patients should undergo screening for left ventricular dysfunction. Persons who have a history of Q-wave infarction without a record of a post-infarction ejection fraction measurement should have an ejection fraction measured. In addition, patients hospitalized for a myocardial infarction should have an ejection fraction determined unless they are at low risk for moderate to severe systolic dysfunction. Indicators for the determination of the ejection fraction in patients with new myocardial infarction can be found in the Chapter 7.

Patients should be seen within one week of initiation of an ACE inhibitor to monitor blood pressure, renal function, and serum potassium (Indicator 9). Doses of ACE inhibitors should be titrated upward slowly. Treatment should be modified if:
1. There is an increase in serum creatinine of 0.5 mg/dL or more;
2. There is a serum potassium of 5.5 mEq/L or higher;
3. The patient reports symptomatic hypotension (with a documented SBP < 100 mm Hg).

**Digoxin**

Cardiac glycosides have been used to treat heart failure for two centuries, yet their use remains controversial (Konstam, 1994; Cohn, 1996). Although digoxin is the preferred agent in patients with heart failure and atrial fibrillation with rapid ventricular response, it is not clear that it is of benefit in patients in sinus rhythm. A meta-analysis of seven randomized, placebo-controlled trials of digoxin found less research study withdrawal due to clinical deterioration among digoxin-treated patients compared to those on placebo (Jaeschke et al., 1990). The Captopril-Digoxin Multicenter Research Group found a trend toward fewer emergency department visits or hospital admissions for heart failure in patients treated with digoxin compared to those who received placebo and a diuretic (Captopril-Digoxin Multicenter Research Group, 1988). In the RADIANCE trial, patients withdrawn from digoxin while continuing to receive diuretics and an ACE inhibitor were nearly six times more likely to have clinical deterioration than patients who continued to take digoxin (Packer et al., 1993). However, there are still questions about when digoxin should be initiated. In addition, the effects of digoxin on mortality are not clear. Retrospective studies have suggested an adverse effect on survival (Bigger, 1985). However, the Digitalis Investigation Group (DIG, 1997) study, found that, compared to placebo, digoxin had no significant effect on mortality, but was associated with a reduction in hospitalization rates.

**Direct-Acting Vasodilators**

Hydralazine/isosorbide dinitrate has been shown to reduce mortality in heart failure. The absolute mortality was 12 percent for hydralazine/isosorbide dinitrate compared to 19 percent for placebo at one year and 36 percent versus 47 percent respectively at three years (Cohn, 1986). However, in direct comparison with enalapril, there was
similar improvement in exercise capacity but hydralazine/isosorbide dinitrate had a lower absolute mortality reduction than did the ACE inhibitor (Cohn, 1991). The side effects of hydralazine/isosorbide dinitrate led to discontinuation of the agent in 18 to 33 percent of study participants due to headache, palpitations, and nasal congestion.

Based on these data, it has been recommended that hydralazine/isosorbide dinitrate be used in patients with moderate to severe heart failure who are intolerant to or have contraindications to ACE inhibitor use (Konstam, 1994; ACC/AHA Task Force, 1995; Cohn, 1996) (Indicator 13). More recently, angiotensin receptor inhibitors (such as losartan) have been used in some heart failure patients who are unable to take ACE inhibitors, but there are currently no randomized controlled trial data to support this use.

**Beta Blockers**

Although several studies suggest that careful titration of beta-blockers may be beneficial for selected patients with heart failure, the routine use of these agents for heart failure is still experimental (Konstam, 1994).

**Anticoagulation**

There has never been a controlled trial of anticoagulation in heart failure, and routine anticoagulation is controversial (Konstam, 1994).

**Revascularization**

See Chapter 8 for a discussion of revascularization in patients with CAD.

**Weight Loss and Other Non-Pharmacologic Management**

Recent studies show that regular exercise can improve the functional status and symptoms of patients with heart failure. Therefore, experts recommend that all patients with stable heart failure should be encouraged to exercise (Sullivan, 1989; Coats, 1990; Kellermann, 1990; Konstam, 1994; ACC/AHA Task Force, 1995; Cohn, 1996). Expert consensus also supports dietary sodium restriction, though
evidence for the efficacy of this recommendation is lacking (Konstam, 1994) (Indicator 14).

**FOLLOW-UP**

Follow-up for patients on diuretics or ACE inhibitors was discussed in the section on treatment.

Because readmission for heart failure is common and costly, attention has focused on interventions to reduce rehospitalization (Gooding, 1985; Rich, 1995). A recent randomized controlled trial consisting of a multidisciplinary, nurse-directed intervention demonstrated improved quality of life, fewer hospitalizations, and a trend toward improved survival in elderly patients with heart failure (Rich, 1995). Additional analyses showed an improvement in medication adherence (Rich, 1996).

Management after hospitalization should include a visit in the outpatient clinic or a home visit no later than one week after discharge (Retchin and Brown, 1991). This visit should include a check of electrolytes, BUN, and creatinine unless they have been done since hospital discharge. Other goals of the post-discharge visit should be to assess adherence to medications and to dietary restrictions, if any; to ensure that the patient’s weight is stable; to adjust medication dosages as indicated; and to ensure that patient, family, and caregivers understand how and when to contact the practitioner (Kostam, 1994; ACC/AHA Task Force, 1995) (Indicators 15, 16 and 17).
REFERENCES


### RECOMMENDED QUALITY INDICATORS FOR HEART FAILURE

These indicators apply to men and women age 18 and older.

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Quality of Evidence</th>
<th>Literature</th>
<th>Benefits</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td><strong>Diagnosis</strong></td>
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<tr>
<td>1. Patients newly diagnosed with heart failure who are beginning medical treatment should receive an evaluation of their ejection fraction within 1 month of the start of treatment.</td>
<td>III</td>
<td>Konstam, 1994; Marantz, 1988</td>
<td>Improve function; reduce symptoms.</td>
<td>No data for the time interval.</td>
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<td>2. Patients newly diagnosed with heart failure should have a history at the time of the diagnosis documenting the presence or absence of all of the following:</td>
<td>III</td>
<td>Retchin, 1991; Konstam, 1994; ACC/AHA Task Force, 1995</td>
<td>Reduce symptoms; reduce mortality.</td>
<td>Rule out reversible/treatable causes or precipitants of heart failure.</td>
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<td>a. Prior myocardial infarction or cardiac disease;</td>
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<td>b. Current symptoms of chest discomfort or angina;</td>
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<td>c. History of hypertension;</td>
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<td>d. History of diabetes;</td>
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<td>e. Current medications; and</td>
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<td>f. Alcohol use.</td>
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<td>3. Patients with a new diagnosis of heart failure should have the following elements of the physical examination documented at the time of presentation:</td>
<td>III</td>
<td>Retchin, 1991</td>
<td>Reduce symptoms.</td>
<td>Physical exam findings are of limited sensitivity and specificity.</td>
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<td>a. Weight;</td>
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<td>b. Blood pressure;</td>
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<td>c. Lung exam;</td>
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<td>d. Cardiac exam;</td>
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<td>e. Abdominal exam; and</td>
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<td>f. Lower extremity examination.</td>
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<td>Indicator</td>
<td>Quality of Evidence</td>
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<td>4. Patients with a new diagnosis of heart failure should be offered all of the following studies within 1 month of the diagnosis (unless performed within the prior 3 months):</td>
<td>III</td>
<td>Retchin, 1991; Kostam, 1994</td>
<td>Detect underlying causes for heart failure; reduce symptoms.</td>
<td>There is no evidence for the time periods for any of these tests. The choice of tests is based more on clinical judgment and opinion than on trials showing improved outcomes</td>
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<td>a. Chest x-ray;</td>
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<td>b. EKG;</td>
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<td>c. Complete blood count;</td>
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<td>d. Serum sodium, potassium, and bicarbonate;</td>
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<tr>
<td>e. Serum creatinine; and</td>
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<td>f. Urinalysis.</td>
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<td>5. Patients with a diagnosis of heart failure who are being treated with medications for their heart failure should have one of the following documented in the medical record at least every two years:</td>
<td>III</td>
<td>Retchin, 1991; Konstam, 1994; ACC/AHA Task Force, 1995</td>
<td>Decrease mortality.</td>
<td>Assessment of left ventricular function may indicate the etiology of the heart failure and assist in condition management.²</td>
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<td>a previously measured ejection fraction, or</td>
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<td>a new evaluation of their ejection fraction.</td>
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<td>6. Patients who are hospitalized for symptoms of heart failure should have all of the following elements of the physical examination documented on the day of hospitalization:</td>
<td>III</td>
<td>Retchin, 1991</td>
<td>Guide management decisions; reduce symptoms.</td>
<td>No data on time interval. This indicator applies only to patients admitted primarily for heart failure.</td>
</tr>
<tr>
<td>a. Weight;</td>
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<td>b. Blood pressure;</td>
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<td>e. Abdominal exam; and</td>
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<td>f. Lower extremity examination.</td>
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</table>
### Indicators

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Quality of Evidence</th>
<th>Literature</th>
<th>Benefits</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients who are hospitalized for heart failure should have the following performed within one day of hospitalization: a. Serum electrolytes; and b. Serum creatinine.</td>
<td>III</td>
<td>Mattleman, 1983; Echeverria, 1983; Dougherty, 1984; Aguirre, 1989; Aronow, 1990; Godsboll, 1989; Eagle, 1988</td>
<td>Improve symptoms; decrease complications of hyper- or hypo-kalemia and renal insufficiency.</td>
<td>No data on time intervals</td>
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### Treatment

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Quality of Evidence</th>
<th>Literature</th>
<th>Benefits</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with a diagnosis of heart failure who have an ejection fraction of less than 40% and no contraindications to ACE inhibitors should be receiving an ACE inhibitor.</td>
<td>I</td>
<td>Captopril-Digoxin Multicenter Research Group, 1983; Jennings, 1984; Sharpe, 1984; McGrath, 1985; Magnani, 1986; Remes, 1986; Chalmers, 1987; CONSENSUS, 1987; Bussman, 1987; Lewis, 1989; Rieger, 1990; Rieger, 1991; Cohn, 1991; SOLVD Investigators, 1992.</td>
<td>Reduce mortality; decrease symptoms; improve functional status.</td>
<td>SOLVD trial showed that enalapril (titrated to 10 mg bid) reduced the development of symptomatic heart failure from 30% in the placebo group to 21% in the treatment group at 3 year follow-up.</td>
</tr>
<tr>
<td>Patients with the diagnosis of heart failure who are started on an ACE inhibitor should have a potassium checked within 1 week of starting the ACE inhibitor</td>
<td>III</td>
<td>Konstam, 1994; ACC/AHA Task Force, 1995; Cohn, 1996;</td>
<td>Reduce complications of hyperkalemia.</td>
<td>Serum potassium must be monitored closely in patients on ACE inhibitors. There are no data for the time interval.</td>
</tr>
<tr>
<td>Patients with the diagnosis of heart failure who are on an ACE inhibitor should have the following checked every year: a. Serum potassium; and b. Serum creatinine.</td>
<td>III</td>
<td>Konstam, 1994; ACC/AHA Task Force, 1995; Cohn, 1996;</td>
<td>Reduce complications of hyperkalemia and renal failure.</td>
<td>There are no data for the time interval.</td>
</tr>
<tr>
<td>Indicator</td>
<td>Quality of Evidence</td>
<td>Literature</td>
<td>Benefits</td>
<td>Comments</td>
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<td>11. Patients with the diagnosis of heart failure who are started on a diuretic should have a potassium level checked within 1 week of the start of treatment</td>
<td>III</td>
<td>Konstam, 1994</td>
<td>Reduce hypokalemic complications.</td>
<td>There are no data for the time interval.</td>
</tr>
<tr>
<td>12. Patients with the diagnosis of heart failure in whom diuretic dose is increased should have a potassium level checked within 1 week of the increase in dose.</td>
<td>III</td>
<td>Konstam, 1994</td>
<td>Reduce hypokalemic complications.</td>
<td>There are no data for the time interval.</td>
</tr>
<tr>
<td>13. Patients with the diagnosis heart failure and an ejection fraction of less than 40% who are not on ACE inhibitors should be on hydralazine/isosorbide dinitrate, in the absence of contraindications.</td>
<td>I</td>
<td>Cohn, 1986; Cohn, 1991; Konstam, 1994; ACC/AHA Task Force, 1995; Cohn, 1996</td>
<td>Reduce mortality.</td>
<td>Hydralazine/isosorbide dinitrate has been shown to reduce mortality in heart failure, though the absolute mortality reduction is less than that for ACE inhibitors. There is similar improvement in exercise capacity compared to ACE inhibitors.</td>
</tr>
<tr>
<td>14. Patients with a new diagnosis of heart failure who are started on medical treatment for heart failure should have dietary counseling within 1 month of the start of medical treatment.</td>
<td>II-1, II-2</td>
<td>Konstam, 1994</td>
<td>Reduce symptoms; improve medical management.</td>
<td>Recent studies show that regular exercise can improve the functional status and symptoms of patients with heart failure. No studies have evaluated a specific dietary sodium restriction.</td>
</tr>
<tr>
<td><strong>Follow-up</strong></td>
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<td>15. Patients who have been hospitalized for heart failure should have follow-up contact within 4 weeks of discharge.</td>
<td>I, III</td>
<td>Retchin, 1991; Rich, 1995</td>
<td>Prevent worsening of symptoms; prevent toxicity from medications.</td>
<td>There are no data on the time interval. Recent randomized controlled trial data showed improved quality of life, fewer hospitalizations, and a trend toward improved survival in elderly patients with heart failure who participated in a multidisciplinary nurse-directed intervention.</td>
</tr>
<tr>
<td>16. Patients who have been hospitalized for heart failure should have the following physical examination elements performed during the first post-discharge visit: a. Weight; b. Blood pressure; c. Lung exam; d. Cardiac exam; e. Abdominal exam; and f. Lower extremity examination.</td>
<td>III</td>
<td>Retchin, 1991</td>
<td>Prevent worsening of symptoms; prevent toxicity from medications.</td>
<td></td>
</tr>
</tbody>
</table>

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Patients who have been hospitalized for heart failure should have the following laboratory tests performed within 4 weeks of discharge:
- Creatinine;
- Potassium.

Prevent worsening of symptoms; prevent toxicity from medications.

<table>
<thead>
<tr>
<th>Definitions and Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Medical treatment for congestive heart failure: medications prescribed to treat symptomatic systolic dysfunction, including diuretics (e.g., thiazide and loop diuretics), ACE inhibitors, hydralazine/isosorbide dinitrate, and digoxin.</td>
</tr>
<tr>
<td>2 Evaluation of ejection fraction: Tests evaluating ejection fraction include echocardiogram, MUGA and left heart catheterization.</td>
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<tr>
<td>3 Contraindications to hydralazine/isosorbide:</td>
</tr>
<tr>
<td>• Systolic blood pressure &lt; 100 mm Hg</td>
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<tr>
<td>• Allergy to hydralazine/isosorbide</td>
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<tr>
<td>• Lupus</td>
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<tr>
<td>4 Dietary counseling: Any mention in the medical record of counseling regarding diet, fluid intake, or salt intake, or a referral to a dietician or nutritionist.</td>
</tr>
</tbody>
</table>

Quality of Evidence Codes

| I | Randomized Controlled Trial (RCT) |
| II-1 | Nonrandomized controlled trials |
| II-2 | Cohort or case analysis |
| II-3 | Multiple time series |
| III | Opinions or descriptive studies |