Inpatient Evaluation and Management of Acute Decompensated Heart Failure

Case Study and Commentary, Alice Y. Chang, MD, and Jay W. Schneider, MD, PhD

Abstract
Admissions for decompensated heart failure account for 6.5 million hospital days each year. It is increasingly recognized that the inpatient hospitalization is an important opportunity for the implementation of therapies and educational efforts to improve patient outcomes. Inpatient physicians should consider initiation and titration of angiotensin-converting enzyme (ACE) inhibitors; initiation of a low-dose β blocker when clinically stable with appropriate follow-up for further titration as an outpatient; and diuretics to prevent fluid retention. Improving the quality of care during the hospitalization should include patient education regarding medications to improve compliance, recognition of the early symptoms of heart failure (fatigue and edema), and the importance of a low-salt diet. Other risk reduction measures, including lipid management, smoking cessation, and exercise counseling, should also be initiated. Outpatient follow-up and referral to a heart failure management team or specialty clinic is advised for patients with class III and IV symptoms or those who have a history of hospitalizations for heart failure.

Heart failure has become a major medical problem in the United States over the past 10 years. It is estimated that 5 million people in the United States have heart failure, and admissions for decompensated heart failure account for 6.5 million hospital days each year [1]. The greatest expense attributable to heart failure is inpatient hospitalization, which accounts for an estimated $23 billion out of total annual expenditures of $38 billion [2]. Readmissions tend to be recurrent but are potentially preventable through aggressive disease management. The number of people affected by heart failure is anticipated to increase due to the aging of the population, improved survival from acute myocardial infarction, and increasingly successful revascularization strategies and therapies for otherwise life-threatening cardiac arrhythmias.

The treatment of heart failure has come along way historically from the 1939 principles of rotating tourniquets to improve venous return and phlebotomy to relieve volume overload [3]. Although some of the underlying principles regarding volume status and hemodynamics remain the same, modern day treatments include the use of neurohormonal inhibitors that affect hemodynamics as well as the natural history of the disease. Studies have shown that in addition to improving symptoms and well being, several therapeutic agents also reduce mortality, hospitalizations, and cost.

It is increasingly recognized that the inpatient hospitalization is an important opportunity for the implementation of therapies and educational efforts to improve patient outcomes.

From the Brigham and Women’s-Faulkner Hospitalist Program, Division of General Internal Medicine, Brigham and Women’s Hospital, Boston, MA (AYC), and the Division of Cardiology, VA Boston Healthcare System, Boston, MA (JWS).
HEART FAILURE

The following case reviews the evidence regarding the management of decompensated heart failure, in particular, the therapies that can be initiated in the inpatient setting with an eye toward improving quality of life and reducing mortality and readmission to the hospital.

CASE STUDY
Initial Presentation

A 68-year-old man with hypertension presents to the emergency department with a 2-week history of gradually increasing shortness of breath with exertion. He is accompanied by his wife.

History

Last week, his wife noticed that he was mowing very small squares of the lawn, stopping to catch his breath between squares. Lately he has been waking up with shortness of breath and having difficulty sleeping. He was awake all last night, comfortable only when sitting up in a chair. He has not had any chest pain or diaphoresis. He has noticed more swelling in his ankles than usual. His wife remembers that he was admitted for heart failure 3 years ago and had a cardiac catheterization that demonstrated normal coronary arteries. She remembers them saying, “the pump was normal.” He has been treated only for hypertension since then. His medications include verapamil and aspirin. He has smoked 1 pack of cigarettes per day for more than 30 years and drinks 3 or 4 beers on the weekend.

Physical Examination

On physical examination, the patient is a moderately obese male in moderate respiratory distress, with an oxygen saturation of 98% on room air. His pulse is regular, with a rate of 114 bpm, and blood pressure is 170/90 mm Hg. His jugular venous pressure is perceptible at the angle of the jaw when assessed at 45 degrees. His pulmonary examination is significant for fine crackles at the bases. Cardiac examination reveals a laterally displaced point of maximal impulse (PMI) with an S1, S2, S3, but no appreciable S4. A systolic aortic outflow murmur is present but there is no diastolic murmur. No hepatosplenomegaly or ascites are appreciated. Pitting peripheral edema is present in his lower extremities to his calf. There are no carotid or abdominal bruits. Distal pulses are strong.

• What diagnostic strategy is appropriate?

Physical Findings

Heart failure is a clinical diagnosis and is not synonymous with left ventricular dysfunction or cardiomyopathy. The symptoms of dyspnea, fatigue, orthopnea, and paroxysmal nocturnal dyspnea as well as signs of volume overload are hallmarks of the syndrome. While one study found that a displaced apical impulse was the best predictor of heart failure along with an S4 and elevated jugular venous pressure [4], in another study rales was the most accurate physical finding [5]. Interestingly, elevated jugular venous pressure and the third heart sound have demonstrated prognostic importance. The presence of these findings (in the outpatient setting) was associated with an increased risk for hospitalization, progression of heart failure, and death from pump failure [6].

B-Type Natriuretic Peptide

Difficulties in relying solely on the physical examination to diagnose heart failure led to a search for a quick and accurate diagnostic method analogous to the troponin level for acute myocardial infarction. B-type natriuretic peptide (BNP) is the most promising candidate for this role. BNP is produced and released by the ventricular myocardium in response to increases in end-diastolic volume and pressure. Among patients presenting to the emergency department with shortness of breath, BNP levels were able to distinguish dyspnea unrelated to heart failure (mean BNP level, 110), noncardiac dyspnea in patients with a history of left ventricular dysfunction (mean, 346), and dyspnea due to heart failure (mean, 675) [5]. BNP concentrations can also predict risk for future cardiac events, heart failure readmission, and death. In a VA hospital study, BNP levels greater than 480 pg/mL were associated with a 51% probability of a heart failure–related emergency department visit, hospital admission, or death [7]. While these studies suggest that the BNP test has great promise, there is still some concern about the specificity of BNP levels that may be elevated with other conditions that increase end-diastolic volume and pressure, such as pulmonary hypertension or pulmonary embolus. While a few studies have followed the BNP level during the hospital course, there is little data as to the clinical significance of these levels beyond the initial presentation.

A normal BNP may be more clinically useful for “ruling out” heart failure when a patient has a difficult-to-assess jugular venous pressure inconclusive physical examination findings, and other, noncardiac, diagnoses are a possibility. Certainly, very high BNP levels also make the diagnosis of heart failure more likely, but in these cases some argue that traditional clinical criteria may be just as helpful. The initial BNP level might also be helpful in identifying patients with a higher risk for recurrent events with greater need for educational resources and referrals to heart failure management programs. In the future, BNP levels may offer guidance in tailoring the outpatient treatment of patients with known chronic heart failure with some preliminary study information that this approach improves outcomes [8].
Diagnosis

The history of dyspnea on exertion and paroxysmal nocturnal dyspnea are highly suggestive of congestive heart failure along with the findings of a displaced PMI, pulmonary edema, and volume overload as assessed by jugular venous pressure and peripheral edema. His chest radiograph demonstrates pulmonary vascular redistribution and an enlarged cardiac silhouette consistent with a diagnosis of congestive heart failure. His electrocardiogram (ECG) shows sinus tachycardia at a rate of 110 bpm, voltage criteria for left ventricular hypertrophy with down-sloping ST segment depressions (also seen on an old ECG) consistent with strain and rate-related changes. A BNP level was drawn on admission and is found to be 720 pg/mL; his troponin I level is 0.02 ng/mL.

- What initial treatment approach is appropriate?

Initial management of decompensated heart failure is directed toward control of symptoms and improvement of hemodynamics. In addition to oxygen for shortness of breath and diuretics for volume overload, in this patient with evidence of pulmonary edema, agents focused on lowering blood pressure and filling pressures are important. For mild to moderate hypertension, early initiation of angiotensin-converting enzyme (ACE) inhibitors may be adequate. If blood pressure cannot be controlled with oral agents, the use of nitrates (either topical nitropaste or intravenous nitroglycerin) would be helpful for immediate vasodilation and reduction of preload as well as for blood pressure control. Other intravenous agents for the management of hypertension include labetalol and hydralazine. If nitroprusside is necessary for "malignant" hypertension, transfer to an intensive care unit (ICU) and cardiology consultation is advised. Otherwise, which agents predict admission to an ICU is usually institution-specific.

The newest intravenous agent for the treatment of heart failure is nesiritide, recombinant BNP. It acts as a vasodilator, natriuretic, as well as a neurohormonal inhibitor and has been shown to lower pulmonary capillary wedge pressure with fewer side effects and no tachyphylaxis as compared with nitroglycerin [9]. The potential side effect of hypotension is easily reversed by lowering the infusion rate or stopping the medication. Nesiritide is a vasoactive substance with balanced preload and afterload effects and no observed proarrhythmic effect; therefore, it does not require ICU monitoring [10,11]. There is also some preliminary evidence that compared with dobutamine, nesiritide was associated with lower 21-day readmission rates and lower 6-month mortality rates [12]. Since nesiritide has been demonstrated to inhibit the renin-angiotensin system and lower norepinephrine levels, short-term infusions might confer a long-term hemodynamic or physiologic benefit. In addition, since nesiritide improves renal perfusion, nesiritide has been useful with or without inotropic therapy for patients who develop the cardiorenal syndrome. Currently, the evidence supports use of nesiritide as a vasodilator to lower pulmonary capillary wedge pressure and improve symptoms in acute decompensated heart failure [11,13]. Further study is necessary to demonstrate a cost/outcome benefit.

Indications for ICU monitoring are hypotension unrelated to initiated therapies and not easily reversed, signs of an unstable coronary syndrome, and obtundation or other signs of poor organ perfusion. If unable to maintain organ perfusion due to hypotension or if the patient shows signs of peripheral vasoconstriction, an inotropic agent may be necessary. In particular, when renal failure develops although the patient is still volume overloaded, an inotrope may be necessary to maintain renal perfusion. Cardiology consultation should be obtained in any of these circumstances. In addition to stabilizing the patient’s hemodynamics, the initial evaluation should also look for and treat any coexisting stressors such as ischemia, arrhythmias, or other noncardiac conditions such as anemia or pulmonary disease.

In patients already being treated with a β-blocker, many physicians follow previously held beliefs that acute management with a β-blocker will impair heart function and therefore hold β blockers on admission. However, sudden withdrawal of β blockers can result in worsening contractile function, with loss of protection against high levels of endogenous compensatory adrenergic hormones. When patients already treated with β blockers are admitted with heart failure, American College of Cardiology/American Heart Association (ACC/AHA) guidelines updated in 2001 state that β blockers may be continued unless the patient is in cardiogenic shock or showing clinical signs of systemic hypoperfusion [1]. In addition, β blockers can also be held if β-adrenergic inotrope therapy becomes necessary. In these cases, simultaneous use of β blockers and β agonists would be self-defeating. While it was previously thought that milrinone had a role in the treatment of decompensated heart failure patients concurrently with β blockers, the recent OPTIME-CHF results demonstrated that milrinone had no outcome benefit and was associated with early treatment failure secondary to hypotension and atrial and ventricular arrhythmias. Based on these recent results, there is no evidence to support the use of milrinone for acute decompensated heart failure [14].

If a patient with heart failure is already being treated with an ACE inhibitor and develops renal insufficiency, the ACC/AHA guidelines also recommend continuing the medication unless the creatinine is greater than 3 mg/dL or the patient is hypotensive or shows other signs of clinical instability that would require vasoconstrictors. Adjustments in the
dose or in other medications should be attempted before the abrupt withdrawal of ACE inhibition, which can lead to further clinical deterioration [1].

**Initial Management**

This patient’s symptoms respond to initial management with oxygen, intravenous (IV) nitroglycerin, and IV furosemide 40 mg. At this hospital, stable dose IV nitroglycerin can be managed in a stepdown unit, so he is admitted to the inpatient medical floor with telemetry. His blood pressure falls to 130/80 mm Hg. His verapamil outpatient medication is discontinued.

On the second hospital day, an echocardiogram is obtained that demonstrates marked concentric left ventricular hypertrophy with an estimated ejection fraction of 35%. The left ventricular chamber dimensions are at the upper limits of normal or slightly enlarged. The right ventricle is mildly enlarged and hypertrophied with a slight decrease in contractile function. In comparison to a study 3 years ago, the ventricle appears more dilated with more prominent contractile dysfunction, although there are still no regional wall motion abnormalities. There are no significant left-sided valvular abnormalities, but there is moderate tricuspid regurgitation.

- **Is further diagnostic testing indicated?**

In the workup for first presentation of heart failure with evidence of systolic dysfunction, coronary artery disease must be excluded. Myocardial ischemia due to obstructive coronary artery disease is responsible for two thirds of dilated cardiomyopathies. The ACC/AHA guidelines recommend that all patients with a first presentation of heart failure and angina should be referred to a cardiologist for catheterization and potential revascularization. In patients with dilated cardiomyopathy, noninvasive testing is not usually recommended because these patients often have inhomogeneity of nuclear tracer uptake and regional wall motion abnormalities unrelated to ischemia (eg, left bundle branch block). Even for patients without symptoms of angina or a negative stress test, current ACC/AHA guidelines recommend cardiac catheterization because of the high prevalence of coronary artery disease in patients with heart failure and potential for improved survival with revascularization [15]. More definitive information about the benefits of revascularization are expected with the first randomized controlled trial of coronary artery bypass surgery for patients with systolic dysfunction (STICH). Determining whether catheterization can be deferred to the outpatient setting in patients without symptoms should be based on the clinical situation.

Other etiologies of heart failure to evaluate include hyperand hypothyroidism; atrial or ventricular arrhythmias seen on cardiac telemetry; alcohol or cocaine use; fat pad biopsy if there is any suspicion for infiltrative cardiomyopathy on echocardiography; and iron studies if hypertrophy remains unexplained without the presence of hypertension or valvular disease. If a family history is significant for heart failure or sudden death, a referral to a genetic counselor to test for familial cardiomyopathy is appropriate.

**Further Evaluation**

The echocardiogram confirms a hypertrophic cardiomyopathy that is likely related to his hypertension as there is no evidence of aortic stenosis or family history of heart failure or a hypertrophic cardiomyopathy. Given his fairly recent catheterization and absence of ischemic symptoms, further investigation focuses on other common causes of decompensated heart failure. There is no evidence of dietary indiscretion, and he is compliant with his prescribed medications. When questioned about any recent medication changes, he notes that he had developed a cough thought to be secondary to his lisinopril, which was subsequently switched to verapamil. Review of systems is notable for recent shoulder pain from a rotator cuff tendinitis. He has been taking tablets left over from a previous episode—indomethacin 3 times per day for the past 2 weeks.

- **What are possible reasons for the patient’s subacute decompensation?**

The patient has multiple potential reasons for his subacute decompensation. Nonsteroidal anti-inflammatory drugs (NSAIDs) are often found to be the unsuspected culprits in acute decompensated heart failure. They have significant biologic activity in heart failure patients due to the inhibition of prostaglandin synthesis that alters renal hemodynamics, leading to fluid retention. When patients are also taking ACE inhibitors, NSAIDs are believed to attenuate the kinin-prostaglandin–mediated benefits of ACE inhibitors [1]. Like other NSAIDs, the newer COX-2 inhibitors can also decrease renal blood flow [16]. The cardiodepressant effects of this patient’s calcium channel blocker may have contributed to his decline at the same time that ACE inhibitors were discontinued. Cough is not considered an appropriate reason to stop ACE inhibitors in a heart failure patient unless the symptoms are troublesome and significantly affecting quality of life. Other possibilities to consider in this patient include any binge drinking, with both the acute cardiotoxic effect of alcohol and potential for paroxysmal atrial fibrillation with poorly controlled ventricular response. With the
diagnosis of a hypertensive hypertrophic cardiomyopathy and at least 2 possible explanations for this patient’s subacute decompensation, no further workup is necessary.

- What medications should be initiated in the inpatient setting?

Once the patient’s symptoms and hemodynamics improve, the next phase of the hospitalization is focused on initiating or optimizing a regimen for outpatient management, with an eye toward slowing disease progression, improving survival, and reducing readmissions. While the evidence does not show immediate in-hospital benefit for many of the therapies recommended for the chronic management of heart failure, it has been appreciated that the inpatient setting is an important opportunity for the initiation of these therapies and educational reinforcement that keeps patients compliant with their regimens. The significant percentage of patients with heart failure who are not receiving these potentially lifesaving treatments underscores the importance of starting therapies in the inpatient setting.

The latest guidelines from the AHA/ACC have created a new paradigm for “staging” heart failure patients before symptoms begin. This is a departure from New York Heart Association (NYHA) classification scheme that groups patients by symptoms and that has been the standard used in most studies of heart failure patients (Table 1). Classified by stage (Table 2), heart failure management focuses on preventing or slowing the progression of left ventricular dysfunction, not just improving symptoms. Seen in this light, treatments not only seek to stabilize volume status and hemodynamics but reverse the underlying physiologic process of remodeling that accompanies elevated circulating levels of norepinephrine, angiotensin II, aldosterone, endothelin, vasopressin, and cytokines. The greatest advances in heart failure outcomes come from the class of neurohormonal inhibitors: β blockers, ACE inhibitors, and angiotensin-receptor blockers (ARBs).

Patients admitted to the hospital for decompensated heart failure are by definition in stage C with symptomatic left ventricular dysfunction. Therapies recommended for stage C patients include ACE inhibitors, β blockers, diuretics, and digitalis in addition to salt restriction, treatment of hypertension, smoking cessation, lipid management, encouraging exercise, and discouraging alcohol use [1]. Focusing on outcomes, medications shown to reduce mortality and hospitalizations in patients with systolic dysfunction in large-scale trials include ACE inhibitors [17], β blockers, and spironolactone [18]. Digoxin has been shown to improve symptoms and reduce hospitalizations but not mortality. Diuretics are important for maintaining volume status and have been shown to prevent rehospitalization. Deciding which medications to start, at what point to start them, and the goals of therapy can be guided by the degree of evidence for each agent and extrapolated from the methods used in the trials.

**ACE Inhibitors**

In multiple placebo-controlled trials, ACE inhibitors have been shown to improve symptoms, clinical status, and overall well-being as well as reduce the risk of death and hospitalization [19–21]. Unless a patient has experienced life-threatening reactions such as angioedema or anuric renal failure, every attempt should be made to initiate an ACE inhibitor, even at a low dose if limited by low systemic blood pressures, increased serum levels of creatinine (> 3 mg/dL), renal artery stenosis, or elevated serum potassium levels [1]. Cough, although considered a nuisance, should not be grounds for dismissal of ACE inhibitor therapy. An important part of the intermediate phase of heart failure hospitalizations should be to titrate up the ACE inhibitor dose, as evidence supports a reduction in the combined risk of death and hospitalizations with the higher dose (equivalent to > 30 mg of lisinopril), and trials increased medications to a predetermined goal dose as tolerated [22]. However, if only a low dose is tolerated, it still has proven benefit and should be continued [1].

Alternatives to ACE inhibitors include ARBs and the combination of isosorbide dinitrate/hydralazine. The largest placebo-controlled trial with the ARB valsartan

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**Table 1. New York Heart Association Classification of Cardiac Patients**

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<thead>
<tr>
<th>Class</th>
<th>Limitation of Activity</th>
<th>Appearance of Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>None</td>
<td>None during ordinary activity</td>
</tr>
<tr>
<td>II</td>
<td>Slight</td>
<td>On moderate or normal exertion</td>
</tr>
<tr>
<td>III</td>
<td>Marked</td>
<td>On mild exertion</td>
</tr>
<tr>
<td>IV</td>
<td>Complete</td>
<td>Even at rest, worsened by any exertion</td>
</tr>
</tbody>
</table>

**Table 2. American College of Cardiology/American Heart Association Classification of Chronic Heart Failure (HF)**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At high risk for HF but without structural heart disease or symptoms of HF</td>
</tr>
<tr>
<td>B</td>
<td>Structural heart disease but without symptoms of HF</td>
</tr>
<tr>
<td>C</td>
<td>Structural heart disease with prior or current symptoms of HF</td>
</tr>
<tr>
<td>D</td>
<td>Refractory HF requiring specialized interventions</td>
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</tbody>
</table>
HEART FAILURE

demonstrated a reduction in combined mortality and hospitalization outcomes, particularly when no ACE inhibitor or β blocker was used. However, there was a trend towards an increase in death when combined with a β blocker and ACE inhibitor. The fact that ACE inhibitors are superior to ARBs supports the theory that ACE inhibitor benefits are not mediated by angiotensin II (levels are not reduced after long-term treatment with ACE inhibitors) but by a kinin-prostaglandin effect. Current ACC/AHA guidelines recommend the use of an ARB only if a patient is intolerant to ACE inhibitors because of an intolerable cough or angioedema. Worsening renal function, hyperkalemia, or hypotension can be experienced with ARBs as well. β Blockers should be started preferentially before ARBs, and an ARB should not be added to an ACE inhibitor and β blocker [1]. While the combination of isosorbide dinitrate/hydralazine is an option for patients who cannot take ACE inhibitors due to hypotension or renal insufficiency, they tend to have a greater side effect profile, especially at the high doses necessary to achieve the survival benefit observed in studies [1].

Diuretics

Diuretics initially serve to decrease pulmonary vascular congestion and the volume overload state. In the next phase of the hospital course, diuretics should be adjusted to an oral regimen to achieve and maintain euvolemia with the goal of improving symptoms and avoiding readmission. Volume depletion must be avoided as it stimulates neurohormonal activation and decreases tolerance for ACE inhibitors. This is considered the potential “dark side” of diuretic treatment. Optimally, to avoid volume depletion some patients should be managed with diuretics as needed based on daily weights. There are no large scale trials looking at the benefit of diuretics independent of other agents on mortality and symptoms. A recent meta-analysis of diuretics in heart failure was limited by the small number of studies and small study samples but did note a trend toward reduced mortality, improved symptoms, and increased exercise capacity. The study population included patients with class I to III heart failure and an average ejection fraction of about 46%, and loop diuretics were the most common agents used. A number of the studies also used an active control such as digoxin or ACE inhibitors [23].

Spironolactone likely acts not only as a diuretic but also as a neurohormonal inhibitor, specifically, an aldosterone antagonist. One large-scale, long-term trial of spironolactone use demonstrated a reduced risk for death and hospitalization among patients with NYHA class IV symptoms [18]. Based on this study, ACC/AHA guidelines recommend the addition of low-dose spironolactone if class IV symptoms persist despite an optimized medical regimen including digoxin, diuretics, an ACE inhibitor, and a β blocker [1]. Therefore, spironolactone is less likely to be initiated in the hospital for acute decompensation of heart failure but can be deferred to the outpatient setting. If spironolactone is added during inpatient hospitalization for a patient refractory to maximal therapy, outpatient follow-up of potassium levels is important.

β Blockers

Once taboo in the treatment of heart failure, β blockers are now one of the cornerstones for heart failure management. In addition to improving survival, β-blocker therapy has been shown to decrease the risk for hospitalization and myocardial infarction in patients with systolic dysfunction [24–27]. The β blockers that have been studied include carvedilol, metoprolol XL or CR, and bisoprolol generally in patients with mild to moderate heart failure (class II and III). The standard teaching used to be that initiation of β blockers for heart failure should be deferred to the outpatient setting to avoid impairing the contractility of an already compromised ventricle. However, the medical community has become increasingly concerned that too many patients are going without potentially life-saving therapies. Recently, 3 large-scale studies of patients with left ventricular systolic dysfunction demonstrated that only 25% to 50% of patients were being treated with β blockers on admission to the study [28,29]. The hospital setting is now appreciated as an important opportunity to optimize therapeutic regimens. The Cardiac Hospitalization Atherosclerosis Management Program (CHAMP) studied the effects of an organized multidisciplinary effort to start aspirin, statins, β blockers, and ACE inhibitors prior to discharge. This study showed a dramatic improvement over conventional management in terms of treatment rates at discharge, long-term patient compliance, and outcomes of death and nonfatal myocardial infarction at 1 year [30].

Furthermore, the COPERNICUS study offers evidence that carvedilol can be safely initiated with no adverse events in patients with severe heart failure (even ejection fractions below 20%) and with recent or recurrent decompression. Carvedilol has both β1 and β2 effects and acts as an α-blocker and antioxidant. In this study, patients initiated carvedilol only after stability was achieved on oral agents; patients who required intensive care or had been on intravenous inotropic agents in the 4 days prior to trial entry were excluded. The study demonstrated a significant 24% reduction in mortality and hospitalizations after 10 months of treatment [27]. Therefore, a convincing argument can be made that β blockers should be initiated prior to discharge even when presenting in acute decompensated heart failure, and that it can be done safely and with demonstrated survival benefit and reduced readmission rates. The AHA/ACC guidelines call for the initiation of β blockers after a patient has been stabilized. Their specific criteria include management outside of an ICU, no signs of fluid
The patient was restarted on an ACE inhibitor on the second hospital day. Captopril was initiated at a dose of 12.5 mg and titrated up to 50 mg 3 times per day by the third day. When he had no symptoms of dyspnea, signs of pulmonary congestion, or peripheral edema, a low dose of metoprolol XL 25 mg was initiated and tolerated without difficulty. As he became euvoletic, his blood pressure was adequately controlled with the combination of captopril 50 mg 3 times a day and metoprolol XL 25 mg per day. His heart rate was 75 bpm with a blood pressure of 130/85 mm Hg. His serum creatinine initially increased from 1.1 to 1.3 mg/dL but stabilized in this range and tolerated a high dose of ACE inhibitors. His furosemide dose was titrated to an oral regimen of 40 mg once per day. Other interventions included a fasting lipid panel to assess for further risk factor reduction, smoking cessation counseling, exercise, and diet counseling. With an low-density lipoprotein cholesterol of 124 mg/dL, he will be started on simvastatin 10 mg prior to discharge.

Further Management

The patient was restarted on an ACE inhibitor on the second hospital day. Captopril was initiated at a dose of 12.5 mg and titrated up to 50 mg 3 times per day by the third day. When he had no symptoms of dyspnea, signs of pulmonary congestion, or peripheral edema, a low dose of metoprolol XL 25 mg was initiated and tolerated without difficulty. As he became euvoletic, his blood pressure was adequately controlled with the combination of captopril 50 mg 3 times a day and metoprolol XL 25 mg per day. His heart rate was 75 bpm with a blood pressure of 130/85 mm Hg. His serum creatinine initially increased from 1.1 to 1.3 mg/dL but stabilized in this range and tolerated a high dose of ACE inhibitors. His furosemide dose was titrated to an oral regimen of 40 mg once per day. Other interventions included a fasting lipid panel to assess for further risk factor reduction, smoking cessation counseling, exercise, and diet counseling. With an low-density lipoprotein cholesterol of 124 mg/dL, he will be started on simvastatin 10 mg prior to discharge.

Recent studies have indicated that biventricular pacing in patients with class III or IV heart failure and intraventricular conduction delay improved symptoms and reduced the need for hospitalization [33]. Propylactic implantable cardiac defibrillators have also been shown to decrease mortality for patients with severe left ventricular dysfunction (ejection fraction < 30%) and previous myocardial infarction [34]. These studies remain controversial because of the substantial cost for these devices. However, in the case of defibrillators, the possibility of preventing sudden death weighs heavily in any cost/benefit analysis. Future studies hopefully will better define the subgroup of patients that would benefit the most from these treatment devices. At this time, it is unlikely that patients would be referred for these devices during an admission for decompensated heart failure. But the general internist should be aware of the current indications for these devices in heart failure patients.

- Does the specialty of the physician affect care outcomes?

Studies examining differences in outcomes for hospitalized heart failure patients treated primarily by cardiologists versus general internists are somewhat limited by differences in patient populations. Patients cared for by cardiologists tend to be younger, more likely to have a history of ventricular arrhythmias, and to be cared for in an ICU. Nevertheless, an observational study at a tertiary care hospital demonstrated no difference in survival at 30 days, with a trend towards improved survival for patients treated by cardiologists at 1 year. Care by a cardiologist was also associated with greater costs and resource use. While differences in patient populations could have affected these results, cardiologists were more likely to order right heart catheterization, cardiac catheterization, and transfer patients to the ICU [35]. An earlier study shared similar findings regarding tests and procedures and no difference in 6-month mortality rates but found that patients of cardiologists were readmitted less frequently [36]. Cardiac catheterization and subsequent angioplasty or surgical revascularization could explain part of the overall survival benefit along with the initiation of medical therapies proven to impact long-term morbidity and mortality.

Referrals to heart failure disease management programs also might explain improved outcomes from specialty care. Common causes of decompensated heart failure requiring admission are potentially avoidable with appropriate education and follow-up (progressive volume overload, diet and medication noncompliance, failure to seek care) [1,37]. Enrollment in a disease management program offers ongoing patient education, enlists patients to become more active participants in their care, and provides close follow-up of patients via phone or home visits to address changes in symptoms before hospitalization is necessary. Studies of heart failure management programs demonstrate patients have improved quality of life and fewer hospitalizations, with subsequent reduction in cost of care [38,39]. High-risk patients (older, previous history of heart failure hospitalizations, class III or IV symptoms) were targeted to make the programs cost-effective.

Based on the current data, however, there is no measurable short-term benefit to inpatient care provided primarily by a cardiologist versus a general internist. Given the prevalence of heart failure in the population, management must be expected
to be in the hands of general internists as well as cardiologists. The focus of this question should turn instead to ways that internists can learn effective treatment strategies to improve outcomes with care delivered or initiated in the inpatient setting and when cardiology referral is appropriate. Further studies will need to examine whether closing the gap in medical therapy prescribed or improving patient follow-up and education will affect long-term outcomes for patients treated by general internists.

Summary
Heart failure admissions not only account for a significant number of hospitalizations but also result in considerable costs to the health care system. Given the impact of heart failure in our society, management during the hospitalization should not only treat the acute problem but also include interventions that will improve long-term outcomes and reduce readmission rates.

Based on the evidence, inpatient physicians should consider the following measures in heart failure patients: initiation and titration of ACE inhibitors; initiation of a low-dose β blocker once clinically stable with appropriate follow-up for further titration as an outpatient; and diuretics to prevent fluid retention. An ARB should be tried only if an ACE inhibitor is not tolerated because of angioedema or very troublesome cough. Spironolactone should be reserved for class IV heart failure patients with an already optimized regimen that includes an ACE inhibitor, β blocker, and diuretics. It may, therefore, be more appropriate for initiation by the outpatient health care provider. Similarly, digoxin should be added only after the other agents have been optimized and may also be deferred to the outpatient setting.

Finally, improving the quality of care during the hospitalization should include patient education regarding medications to improve compliance, recognition of the early symptoms of heart failure (fatigue and edema) as well as the importance of a low-salt diet. Other risk reduction measures, including lipid management, smoking cessation, and exercise counseling, should also be initiated. Outpatient follow-up and referral to a heart failure management team or specialty clinic is advised for patients with class III and IV symptoms or who have a history of hospitalizations for heart failure.

This review does not include discussions about diastolic dysfunction. There are no additional methods for diagnosing diastolic dysfunction, and few clinical trials included or addressed patients with heart failure but preserved systolic function. In the absence of data, the ACC/AHA guidelines recommend attention to physiologic parameters, including control of hypertension and tachycardia as well as reduction of volume and preload with diuretics [1].

Corresponding author: Alice Y. Chang, MD, Brigham and Women’s Hospital, 75 Francis St., Boston, MA 02115, achang@partners.org.

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Part 1. Please respond to each statement.

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<thead>
<tr>
<th>Statement</th>
<th>Strongly Agree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>I was provided with new information pertinent to my practice.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I reaffirmed a specific skill or knowledge.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>This article will help with clinical decision making.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relevant clinical outcomes are addressed.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The case is communicated in a manner that kept my interest.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The case presentation is realistic and effective.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I could easily interpret the tables and figures.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My attitude about this topic changed in some way.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Additional comments: ______________________________________________________________________________________
________________________________________________________________________________________________________

Part 2. Please complete the following sentence.

As a result of reading this case study, I . . .

☐ see no need to change my practice.
☐ will seek more information before modifying my practice.
☐ intend to change the following aspect(s) of my practice: (Briefly describe)
________________________________________________________________________________________________________
________________________________________________________________________________________________________


Signature: ____________________________ Date: ____________________________

Part 4. Identifying information: Please PRINT legibly or type the following:

Name: ____________________________ Fax number: ____________________________
Address: ____________________________ Telephone number: ____________________________
Social Security number: ____________________________

Medical specialty: ____________________________

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Detroit, MI 48201

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