Emergency department (ED) presentations for acute decompensated heart failure (ADHF) have increased at a dramatic rate. In the 1990s, ADHF presentations accounted for between 1% and 3% of all ED visits and increased at an average rate of more than 18,000 visits annually.¹² Providing care for these large numbers of patients has significant societal implications because heart failure is the leading diagnosis for Medicare patients older than 65 years and accounts for more bills more than any other disease.³ Hospital charges for 1 week of inpatient treatment of ADHF averaged approximately $10,000 in the 1990s, and is even higher today.⁴ Costs for the treatment of ADHF vary on a daily basis, with nearly 75% of ADHF-related costs occurring in the first 48 hours.⁵

The costs of ADHF are not just an ED problem but begin at its front door. Of patients who have a primary diagnosis of ADHF discharged from the hospital, nearly 80% arrive through the ED.⁶,⁷ On a national level, the direct and indirect costs for treating ADHF were projected to reach more than $34.8 billion dollars in 2008.⁸ The financial impact of ADHF is not stagnant; as the baby boomer generation continues to age, the burden of heart failure on society will continue to increase.⁹,¹⁰

To prepare for this growth, government and hospital administrators are looking for more effective and less costly methods to deliver care to these patients. ED observation units (OU) have been found to be one of the most cost-effective ways to manage patients who have mild to moderately ill presentations of asthma, chest pain, and heart failure.¹¹⁻¹⁸ These units have been associated with a high degree of patient satisfaction and increased diagnostic accuracy.¹⁹⁻²³ When evidence-based protocols are used in observation care for ADHF, return ED visits and readmissions decrease significantly.¹²

OU treatment of ADHF is cost-effective for several rather distinct reasons. OUs are able to provide rapid and focused treatment for patients who have ADHF through specific treatment protocols that follow current American Heart Association (AHA) guidelines and can be implemented immediately. The several-hour delay in care often seen when patients are transferred from the ED to the hospital floor is greatly shortened because observation unit beds are often more available due to greater turnover. The use of protocols can help reduce the number and severity of complications seen with patients who have ADHF.²⁴ Medical therapies are started earlier in OUs than with typical inpatient admission on a hospital floor. A significant secondary benefit is the decrease in ED diversion and delays in care for future patients, because the ED beds are no longer occupied.²⁵,²⁶

The establishment of OUs for the extended care of patients in the ED has evolved over the past 3 decades. Observation medicine initially began as a method to contain costs for conditions with uncertain clinical courses or diagnoses. The most common admissions to the initial OUs were for alcohol intoxication or withdrawal and
overdoses.\textsuperscript{27,28} With little regulation and guidance, more than a decade passed before the idea of observational care caught on. Once the benefits of OUs became more obvious, the model blossomed into a branch of emergency medicine with unique features and goals.

In 1987, more than 10 years after the inception of the OU, the American College of Emergency Physicians Practice Management Committee published a list of features it deemed important in managing observation patients. The committee stated that the goal was “to improve the delivery and quality of medical care to all patients.”\textsuperscript{29} Recommendations included clearly designating the individuals responsible for observation patients at all times, locating units within or adjacent to the ED, and creating protocols for transferring patients in and out of the units.

OU beds are designed for short-term care of mild to moderately ill patients, typically 24 hours and, except in extreme cases, less than 48 hours.\textsuperscript{30} The physical placement of OUs can vary depending on the needs and restraints of the institutions developing them. Most OUs are in dedicated areas within the ED using existing beds,\textsuperscript{31} allowing proximity for physicians and nurses and avoiding the financial expense of building and running a separate facility. Using beds within the ED can contribute to overcrowding, which has become an ever-increasing problem.

As EDs grow and invest in the infrastructure, many will create a separate adjacent clinical area for an OU. The advantages of this include a stable location for patient care and a stable nursing pool composed of staff working solely in the OU. A separate clinical area allows centralization of physical resources required to run an OU and eases education of staff caring for patients in the unit. Physicians caring for these patients are close if emergencies arise, but patients are shielded from the volume and commotion often seen in many EDs.

Some departments may place patients in a virtual observation unit with set protocols but no permanent physical space. This model allows OU beds to be located literally on any floor in a hospital, assuming that the level of care for these patients remains constant. The disadvantages of this type of OU include difficulty maintaining appropriate training and certification of nursing staff caring for patients in the unit, potentially large distances between patients and physicians caring for them, and lack of familiarity of other staff with OU protocols and practice.

The Joint Commission, formerly The Joint Commission on the Accreditation of Healthcare Organizations, requires that OU staffing approximates nurse-to-patient ratios of a hospital floor caring for similar patients. This directive often leads to a nursing-to-patient ratio of 1:4 or 1:5 for the typical OU. Nursing staffing levels can be adjusted throughout the day as the number of patients requiring care in the OU changes, but should not exceed predetermined nurse-to-patient ratios determined by that institution.

**CENTERS FOR MEDICARE & MEDICAID SERVICES: CODING**

Observation unit stays are billed differently from ED visits or hospitalized in-patient stays. The Centers for Medicare and Medicaid Services (CMS) considers OU visits outpatient observation services to be billed under revenue code 762. The observation period determines whether further inpatient treatment is needed, therefore allowing for its outpatient status. Observation services begin when the nurse notes the arrival of the patient in observation status, because observation is not a physical location. Patients could be accepted into observation status in the ED or any location within the hospital. Observation care continues until physicians order the patients discharged or admitted to the hospital.

Much like ED reimbursement from CMS, rates of professional billing are related to the level of medical complexity of the case and the documentation supporting that complexity. Observational billing is unique in that two separate sets of codes are used, depending on whether the patient is admitted and discharged on the same calendar day. Codes 99234 through 99236 are used for same-day discharges and codes 99217 through 99220 are used for patients undergoing care for two or more calendar days. CMS allows for professional and technical billing for OU care.

CMS allows billable observation services for up to 48 hours for the technical fee but allows exceptions if patients stay longer than 48 hours. However, these extensions cannot be preauthorized. For patients who stay longer than 48 hours and are admitted to a hospital floor, the technical portion of the OU bill often will be bundled into the technical portion of the hospital bill, not allowing for separate collection by the hospital and OU. The professional fee does not change in this scenario.

Until recently, CMS allowed a separate ambulatory payment classification (APC) group payment for technical charges for only three diseases: heart failure, asthma, and chest pain. This rule is changing in 2008 with the adoption of a composite APC, which will provide payment for observation services of all diagnoses. The codes include 8002, which will cover observation care for
patients admitted directly to an OU from a clinic or outpatient setting, and 8003, which will reimburse observation patients admitted after a high-level ED visit signified by documentation supporting a level 4 or 5 ED visit (current procedural terminology [CPT] codes 99284 and 99285). The requirements to meet these codes include a minimum of 8 hours of observational care, a qualifying ED visit, and the absence of any surgical intervention requiring placement in observational care. Patients who have a stay of less than 8 hours can be paid under APC 0604.32

As part of this plan, CMS will bundle technical charges from the ED visit with the observational visit. Although this procedure seems to be an effort to streamline the billing process and improve efficiency, how this change will affect overall reimbursement for institutions is unclear.

STAGES OF OBSERVATION UNIT CARE

Patients presenting with ADHF must be correctly assessed and stabilized in the ED before any disposition decision is made. Although this statement is easy to make in clinical practice, the diagnosis of ADHF can be much more challenging. Using clinical judgment alone to diagnose ADHF resulted in an accuracy rate of 74%. The physicians caring for these patients had a 27% rate of diagnostic uncertainty.33

Using markers such as B-type natriuretic peptide (BNP) and N-terminal pro-BNP increases the diagnostic accuracy of clinicians treating patients who have possible ADHF.34–37 Accurate diagnosis in the ED can be important, because subsequent providers may follow the same care plan until another diagnosis becomes obvious. Other conditions, such as pulmonary embolism, acute coronary syndrome, chronic obstructive pulmonary disease, and pulmonary hypertension, should be considered before patients are placed in the OU. A prominent problem with ADHF is its recidivism; the most common historical finding in a patient admitted for ADHF is a prior history of heart failure.38

After diagnosis, stabilization can begin in the ED. Several medications may be used for initial stabilization. Primary ED medications for ADHF include afterload reducers, such as nitroglycerin, nitroprusside, nesiritide, or hydralazine. These medications can quickly reduce afterload, because approximately 50% of patients present with elevated systolic blood pressure.39,40 Diuretics are common first-line agents used in the acute management of ADHF. Reports have suggested they are used in approximately 70% to 90% of patients who have ADHF.40,41

Other medications used in the ED to treat ADHF include angiotensin-converting enzyme (ACE) inhibitors that block the renin-angiotensin-aldosterone system (RAAS), which is often elevated in patients presenting in acute heart failure. ACE inhibitors can cause hypotension, and therefore should be used carefully or withheld in patients who have borderline hypotension or are also being treated with vasoactive agents, such as nitroglycerin or nesiritide. For patients intolerant of ACE inhibitors because of cough or angioedema, angiotensin receptor blockers (ARBs) can be used. These medications block the RAAS through inhibiting the angiotensin II receptor, and can also cause hypotension.

Another nonpharmaceutical modalities for treatment of ADHF in the ED is noninvasive positive pressure ventilation (NPPV). This means of respiratory assistance is useful in patients who are dyspneic but alert and cooperative and do not have a large oxygen requirement. Forms of NPPV include continuous positive airway pressure and bilevel positive airway pressure. These noninvasive means of ventilatory assistance are used as a bridge to avoid intubation until concurrent medical therapy, such as preload reduction, afterload reduction, and diuretics, has time to work. Rotating tourniquets are not a usual means of modern care, although they were used previously for initial stabilization.

CHOOSING PATIENTS FOR OBSERVATIONAL CARE

When treating patients who have ADHF in an ED that has an OU, deciding which patients are appropriate for observational care is important. Experts have suggested that ED physicians often overestimate the severity of illness in patients who have ADHF and that this has led to excess expense and unnecessary use of critical care hospital beds.42,43 Although this line of thought has some truth, it is difficult to criticize the treatment rendered by ADHF because no good risk stratification tools exist for this patient population.

The absence of a useful risk stratification tool is certainly not cause by a lack of trying. Attempts by emergency physicians and cardiologists have had limited success. Multiple ED and inpatient studies have attempted to risk-stratify patients who have ADHF.44–50 Unfortunately, most of these studies use mortality or complications, including ventricular fibrillation, defibrillation, cardiopulmonary resuscitation (CPR), and intubation, as end points. Other clinically relevant end points for an OU population, such as dyspnea, return visits, length of stay, have not been studied prospectively. These studies have attempted to identify the high-risk
population and assume that the patients not positive for high-risk features are a low-risk population.

Only one study by Auble and colleagues has evaluated multiple decision rules in a head-to-head fashion. In this study, the investigators compared four decision rules, including two by Fonarow and colleagues, using the Acute Decompensated Heart Failure National Registry, a rule developed by Lee and colleagues from the Enhanced Feedback for Effective Cardiology Treatment trial, and the Brigham and Women’s hospital rule. The rules varied from a rather simple decision tree, to more complex point systems, to a rather complicated multivariate logistic regression model using blood urea nitrogen levels, vital signs, and age. Outcomes included death and lifesigns interventions, such as defibrillation, CPR, intubation, and coronary artery bypass graft surgery.

Rates of inpatient death or complications among the low-risk population groups ranged from 6.7% to 9.2%, and rates of 30-day mortality were between 4% and 6%. Most ED physicians would find these rates unacceptably high for admitting a population to an OU.

More recently, a study published by Hsieh and colleagues attempted to validate the acute heart failure index in a retrospective population of more than 8000 patients. In this decision tree, patients who had prior diagnosis of heart failure and no evidence of acute myocardial infarction or ischemia were evaluated for low-risk features. Nineteen other variables, including medical history, laboratory values, vital signs, EKG, and chest radiograph results, were used in a complex decision tree with a total of 14 possible low-risk end points.

The acute heart failure index classified 19.2% of patients as low-risk. Among these patients, death at 30 days occurred in 2.9% (95% CI, 2.1%–3.7%), and 1.7% (95% CI, 1.1%–2.4%) experienced serious complications. These results seem to show a significant improvement over the prior decision rules. The complexity of this rule is problematic and would realistically only allow its use if incorporated into a computerized algorithm as part of an electronic charting program or a program incorporated on a handheld computer. This algorithm, as part of a functional program using “yes” or “no” answers to determine morbidity and mortality risk, can be found at http://www.pitt.edu/~hfpr/. This decision rule still requires prospective validation in multiple geographic regions before it can be supported for widespread use.

Because few singularly useful low-risk characteristics exist, one must know which features may represent a high risk for morbidity and mortality for patients presenting with ADHF. Burkhardt and colleagues retrospectively evaluated 385 patients OU and found that only an elevated blood urea nitrogen (BUN) of greater than 30 mg/dL was associated with OU treatment failure. Elevated serum creatinine levels and hyponatremia were associated with nonsignificant trends toward OU treatment failure.

Dierks and colleagues evaluated 499 patients who had ADHF treated in an ED to determine which characteristics were suitable for OU treatment. Patients were enrolled prospectively and followed up for 30 days. Outcomes included death, myocardial infarction, arrhythmia, and rehospitalization. An initial ED systolic blood pressure of greater than 160 mm Hg and negative troponin I serum value were found to predict successful OU treatment. Other laboratory values associated with a poor prognosis in ADHF include anemia, elevated serum creatinine, and hyponatremia.

Diuretic resistance was also proven to be a powerful predictor of worse prognosis in patients who have ADHF. Patients who have recently noticed decreased efficacy of their diuretics or are taking exceedingly high doses of diuretics may be considered poor OU candidates.

Previously, patients who had a low ejection fraction (EF) were often considered to have a worse prognosis. However, more recent data evaluating patients who had newly diagnosed ADHF suggests that the current rates of mortality at 30 days and 1 year are not affected by the EF. Because no prospective studies have evaluated EF as a risk factor for OU treatment failure, establishing an absolute EF value below which patients would not be considered for OU treatment would be imprudent.

Another pragmatic feature to help determine a patient’s viability for OU treatment is average length of recent hospital admissions. If a patient’s previous four admissions each resulted in a week-long hospital stay, that individual may not be the best candidate for OU treatment. Patients who have other active comorbid conditions that may complicate ADHF treatment, such as renal insufficiency or active pulmonary disease, may more strongly considered for inpatient admission. These decisions should be made on an individual basis, and should include the patient’s primary care physician or cardiologist.

**TREATMENT IN THE OBSERVATION UNIT**

Using processes of care, an effective, evidence-based treatment standard can be created for patients who have ADHF admitted to an OU. As with most effective protocols, this process begins long before any patient is admitted. ED physicians
must meet with the hospital’s cardiology group or groups to discuss which population of patients who have heart failure would best benefit from treatment in an OU setting. Requests for unique exclusions can be discussed and enacted if deemed reasonable.

For example, when creating the ADHF protocol, cardiologists at Metro Health Medical Center requested that all patients who had newly diagnosed ADHF be admitted directly to cardiology. This practice was based on the expectation that patient evaluation would require several days of hospitalization. This process may not be required in all institutions, such as those in which an appropriate rapid workup may still occur for patients who have newly diagnosed heart failure.

OU treatment of ADHF consists largely of afterload reduction and diuretic therapy. Other therapies can be offered, such as nesiritide infusion or ultrafiltration. More nursing-intensive therapies, including inotrope infusions or nitroglycerin drips, often require more care than an OU can provide, and patients taking these medications have a low likelihood of discharge home in 48 hours. The mainstay of treatment is diuretic therapy, which is started in the ED and continued in the OU. Afterload reduction and treatment of the hyperstimulated renin angiotensin system includes ACE inhibitors or ARBs, which could be given in the ED or OU. Second-line agents include vasodilator therapy, such as nesiritide or long-acting nitrates. For patients who have known diuretic resistance, ultrafiltration therapy is a reasonable treatment if available.

Unfortunately, despite optimal medical treatment, some patients may decompensate, requiring additional higher-intensity care. Some signs of deterioration in patients who have ADHF that may be monitored in the OU setting include hypotension; worsening hypoxia; chest pain or symptoms suggesting acute coronary syndrome; anuria or oliguria; and acute dysrhythmias, such as ventricular tachycardia or atrial fibrillation with rapid ventricular response. Orders should include parameters for unstable vital signs to signal physician notification when patients experience clinical deterioration.

Most patients for whom OU treatment of ADHF fails can be admitted safely to a hospital floor for continued treatment. Patients who have symptoms of acute coronary syndrome or dysrhythmias may require cardiac monitoring. Patients who have hypotension may require inotrope therapy and a higher level of nursing care. Systems should be in place to make these transitions as effortless as possible well before patients are admitted to an OU.

Having a realistic idea of the total number of patients who have ADHF who will be admitted to an OU is ideal. This projection will provide the administration with a reasonable estimate of the number of patients who will be placed in the OU. Recent studies have suggested that approximately 30% of patients presenting to the ED with ADHF would be suitable for OU care.44,45

Creating protocols to ease admission and create uniform treatment is ideal in an environment in which physician care may be transferred two or three times a day and dedicated unit coverage may be limited to 8 hours per day. **Fig. 1** provides an example of the heart failure admission protocol used at MetroHealth Medical Center. The orders were created with assistance from cardiologists and were designed to be filled out completely and allow for no ambiguity. Patients should be weighed on arrival to allow comparison when they return in ADHF. Strict monitoring of oral consumption and urinary output is essential to determine the efficacy of diuretic therapy. Parameters for physician contact should be included, because the physician caring for these patients will often not be in the immediate vicinity. Low-salt and diabetic diets should be available. Fluid restriction may be required if the patient is fluid overloaded and unable to comply with dietary demands.

Laboratory tests should include a complete blood cell count to check for anemia and a basic metabolic panel to assess electrolyte status. BNP should be measured at admission and can be followed serially to assess ventricular wall stress response to therapy. In patients treated with nesiritide, a recombinant form of BNP will be measured along with native BNP, and this measurement should be performed 2 hours after the nesiritide infusion is discontinued.

Liver function tests (LFTs) should be measured to determine if cholestatic hepatic dysfunction is occurring as a result of poor perfusion, because more than 40% of patients in ADHF will present with abnormal LFTs.63 Thyroid function should be evaluated but may be withheld if performed in the previous year and the patient is not currently taking thyroid replacement therapy.

Cardiac markers may be ordered at the physician’s discretion for patients presenting with symptoms of acute coronary syndrome and ADHF. For patients who have no prior history of congestive heart failure, cardiac markers should be ordered to rule out myocardial ischemia as a cause of the ADHF episode.

Use of cardiac markers in patients who have known congestive heart failure is more controversial. Although many institutions, including MetroHealth Medical Center, often admit patients who...
**Fig. 1.** The heart failure admission protocol for MetroHealth Medical Center. (*Courtesy of MetroHealth Medical Center, Cleveland, OH.*)

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<th>DATE</th>
<th>HOUR</th>
<th>ORDERS</th>
<th>NURSE'S NOTATION</th>
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<th>NURSE'S SIGNATURE</th>
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<td>Diagnosis: Congestive Heart Failure Exacerbation <em>(Page 1 of 2)</em></td>
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<td>Condition: Stable</td>
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<td>Nursing: ☑ Call MD for worsening dyspnea or increasing O2 requirement</td>
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<td>☑ Measure fluid input and output, patient weight at admission</td>
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<td>☑ B type natriuretic peptide</td>
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<td>☑ LFTs</td>
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<td>☑ TSH if not obtained within last year.</td>
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<td>☑ Repeat B type natriuretic 2 hrs after nesiritide is discontinued</td>
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SO-CDU CHF 05/05
have known ADHF to hospital floors without a “rule-out” period in a telemetry bed, whether this is an optimal practice is unknown. The current AHA guidelines for management of ADHF do not even mention cardiac markers or troponin. What is known is that the rate of positive cardiac markers in patients who have ADHF approaches 20%. A positive troponin test is an independent risk factor for death if seen in the ED presentation, which has led some authors to propose...
testing cardiac markers on all patients presenting with ADHF.67 The rate of coronary artery disease in patients who have known heart failure is more than 50%. Baseline elevations in troponin I do not differ between patients who have idiopathic and ischemic cardiomyopathy.68,69

Echocardiography is recommended in the 2005 AHA guidelines on the management of chronic heart failure.64 However, whether this study must be repeated in subsequent admissions is unclear. The authors’ practice is to obtain an echocardiography study if the patient has not been evaluated in the prior year, or if new findings on physical examination suggest structural cardiac changes that may influence outcome, such as mitral regurgitation or aortic stenosis not previously noted or worse than prior examinations.

Medical treatment of ADHF in the OU should continue the treatment plan begun in the ED. For patients placed on diuretics, two approaches are available. Many patients receive intravenous boluses of furosemide to encourage fluid loss. If the initial dose is not completely effective, a second or third dose may be given. Several studies have suggested that continuous intravenous infusions of furosemide result in greater diuresis and less ototoxicity, particularly if begun early in the hospitalization.70–73 Continuous infusion medications present a risk for excessive diuresis, and therefore stop-gap measures should appear in the OU orders instructing the nursing staff to discontinue the medication once a desired level of diuresis is reached. Electrolytes should be monitored and replaced as needed when aggressive diuresis is being performed.

B-blockers have become a mainstay of heart failure treatment because they have been shown to decrease morbidity and mortality.74–77 Patients who have known heart failure should take a daily B-blocker. Currently approved formulations include metoprolol, bisoprolol, or carvedilol. B-blockers help decrease the heart’s exposure to chronic adrenergic stimulation and decrease ventricular remodeling.

Because most patients who have heart failure are currently undergoing this therapy, the question arises of what to do if they are currently in ADHF and being admitted to an OU; should the B-blocker be held? Prior dogma has instructed physicians to stop B-blockers in ADHF episodes. Although B-blockers can depress cardiac contraction, suddenly stopping them may expose the heart to increased endogenous catecholamines, which could produce tachydysrhythmias and myocardial ischemia.

Experts have suggested that patients in mild to moderate ADHF and who were not hypotensive should be maintained on B-blockers.78–80 Evidence supports that patients who have acute myocardial infarction experiencing ADHF benefit from B-blocker treatment.81 More recent studies have determined an increase in morbidity and mortality and increased length of stay may be associated with decreasing or stopping B-blocker therapy for patients in ADHF.82,83 The most recent published American College of Cardiology (ACC) guidelines suggest that patients who have been taking B-blockers long-term decrease or stop them only if evidence of hypoperfusion is present.64 Table 1 lists the starting dosages of B-blockers approved for use in the treatment of heart failure. Doses may be adjusted if bradycardia or hypotension develops.

Like B-blockers, ACE inhibitors and ARBs have shown direct long-term mortality benefits to patients who have heart failure.84–87 Patients diagnosed with systolic heart failure and no contraindication should be placed on an ACE inhibitor or ARB. These medications should be started at low doses and gradually titrated upward. Patients who have a history of renal insufficiency or hypokalemia should avoid these medications unless closely follow by their physician. For patients being started on them, serum potassium levels should be checked at 3 days and 1 week initially to check for hyperkalemia. If the patient seems unreliable or unable to return for blood draws, it may be more prudent for the primary care physician to begin this medication. Table 1 lists the starting doses for ACE inhibitors and ARBs.

Digitalis is a cardiac glycoside that has been used in the treatment of ADHF for more than 2 centuries. Digitalis blocks myocardial sodium potassium adenosine triphosphatase (Na-K-ATPase). Enzymatic blockade in the kidney causes natriuresis, which increases sodium excretion, and decreases renin release. Although once a mainstay of heart failure treatment, digitalis has been relegated to a second-line therapy by some groups and was changed from class I to class IIa classification in the most recent AHA update.64,88,89 Other studies have suggested that it may still be useful but only at a lower serum concentration than used previously.90,91 Digitalis is still recommended for patients who have persistent heart failure symptoms who have not responded to treatment with B-blockers, diuretics, or ACE inhibitors (or ARBs). It is not indicated as a medication to stabilize an ADHF episode. Therefore, its usefulness in the OU setting is limited, and it may be best newly prescribed under the guidance of a consulting cardiologist.

Long-acting nitrates, such as isosorbide dinitrate, have been recommended for select patients...
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<th>Initial Dose</th>
<th>Target Dose</th>
<th>Half-Life (h)</th>
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<tbody>
<tr>
<td><strong>ACE inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lisinopril</td>
<td>5 mg qd</td>
<td>20 mg qd</td>
<td>12</td>
<td>$12.99</td>
<td>Hypotension, hyperkalemia, angioedema, cough, renal insufficiency (need to monitor potassium, check 1–2 wk after initiating therapy, monitor creatinine with impaired renal function)</td>
</tr>
<tr>
<td>Captopril</td>
<td>6.25 mg TID</td>
<td>50 mg tid</td>
<td>1.9</td>
<td>$9.99</td>
<td></td>
</tr>
<tr>
<td><strong>Angiotensin receptor blockers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Losartan</td>
<td>50 mg qd</td>
<td>100 mg qd</td>
<td>2</td>
<td>$85.51</td>
<td>Hypotension, hyperkalemia, angioedema, cough, and rarely rhabdomyolysis (need to monitor potassium, check 1–2 wk after initiating therapy, monitor creatinine with impaired renal function)</td>
</tr>
<tr>
<td>Valsartan</td>
<td>20 mg bid</td>
<td>40 mg bid</td>
<td>6</td>
<td>$114.36</td>
<td></td>
</tr>
<tr>
<td><strong>Loop diuretics</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>10 mg qd</td>
<td>Variable</td>
<td>2</td>
<td>$2.85</td>
<td>Hypotension, hypokalemia, hypomagnesemia, hyponatremia, hyperlipidemia, ototoxicity, myelosuppression (patients may require electrolyte supplementation)</td>
</tr>
<tr>
<td>Bumetanide</td>
<td>0.5 mg qd</td>
<td>Variable</td>
<td>1.5</td>
<td>$23.52</td>
<td></td>
</tr>
<tr>
<td><strong>Thiazide diuretics</strong></td>
<td></td>
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</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>25 mg qd</td>
<td>Variable</td>
<td>6–15</td>
<td>$2.39</td>
<td>Dizziness, headaches, muscle cramps, hypotension, hypokalemia, hypomagnesemia, hyponatremia, hypercalcemia (start at lowest dose, may require electrolyte monitoring)</td>
</tr>
<tr>
<td>Metolazone</td>
<td>0.5 mg qd</td>
<td>Variable</td>
<td>14</td>
<td>$37.37</td>
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<tr>
<td><strong>Beta-blockers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Carvedilol</td>
<td>3.125 mg bid</td>
<td>25 mg bid</td>
<td>7–10</td>
<td>$31.98</td>
<td>Hyperglycemia, hypotension, bradycardia, dizziness, fatigue (avoid stopping drug abruptly)</td>
</tr>
<tr>
<td>Metoprolol XL</td>
<td>12.5 mg qd</td>
<td>Variable</td>
<td>7</td>
<td>$35.99</td>
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</tr>
<tr>
<td>Bisoprolol</td>
<td>2.5 mg qd</td>
<td>Variable</td>
<td>9–12</td>
<td>$31.00</td>
<td></td>
</tr>
<tr>
<td><strong>Digitalis</strong></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.125 mg qd</td>
<td>Variable</td>
<td>40</td>
<td>$12.99</td>
<td>Anorexia, vomiting, diarrhea, and dizziness (avoid in patients who have atrioventricular block, such as Wolff-Parkinson-White syndrome, and those who have hypokalemia)</td>
</tr>
<tr>
<td><strong>Nitrates</strong></td>
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<tr>
<td>Isosorbide dinitrate</td>
<td>20 mg qd</td>
<td>Variable</td>
<td>1</td>
<td>$5.65</td>
<td>Hypotension, headache, syncope (often used in combination with hydralazine, not to be used with impotence medications)</td>
</tr>
<tr>
<td><strong>Combination medications</strong></td>
<td></td>
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</tr>
<tr>
<td>Isosorbide dinitrate/</td>
<td>20–37.5 mg tid</td>
<td>Variable 1–15</td>
<td>$191.97</td>
<td>Hypotension, headache, syncope, hypokalemia, hypomagnesemia, hyponatremia, hypercalcemia (not to be used with impotence medications)</td>
<td></td>
</tr>
<tr>
<td>hydrochlorothiazide</td>
<td></td>
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</tbody>
</table>

*a* Prices listed assume 1-month supply. *(Data from Drugstore.com. Accessed February 1, 2008.)*
who have ADHF. These patients include those who are symptomatic on maximal therapy and those who have exertional or nocturnal dyspnea. Long-acting nitrates have been shown to offer better survival than placebo but not ACE inhibitors and therefore should not be given in their place. Side effects include headache and hypotension. Tolerance is common with extended use, and therefore they are not prescribed around the clock. Because these are second-line agents for heart failure, cardiology guidance may be useful in choosing to which patients oral nitrates should be prescribed.

Hydralazine is a direct vascular smooth muscle relaxant used often in combination with long-acting nitrates. It is not as effective as ACE inhibitors in reducing mortality in patients who have heart failure but may be used in those intolerant of ACE inhibitors and ARBs. When used in combination with long-acting nitrates and ACE inhibitors or β-blockers, hydralazine was found to be particularly effective at reducing mortality in African Americans. The results of this research led to the creation of BiDil, a combination medication containing isosorbide dinitrate and hydralazine. This medication was the first to receive approval from the U.S. Food and Drug Administration (FDA) for treating disease in a specific race. Current guidelines do not suggest this combination as a first-line agent in African Americans who can tolerate ACE inhibitors and β-blockers.

Nitroglycerin is a potent vasodilator with both venous and arteriole vasodilator properties. It is effective at reducing preload and afterload rapidly in patients presenting in ADHF. The half-life of intravenous nitroglycerin is 1 to 4 minutes, requiring that it be given in a continuous intravenous drip or topical form. The intravenous form is often used for flash pulmonary edema or ADHF presentations with elevated blood pressure. However, because this form requires frequent titration, it is not ideal for OU use. A sublingual tablet and spray forms are also available, with physiologic affects lasting from 1 to 6 hours after administration. The tablet and topical forms can be used in an OU setting but should not supplant other more effective therapies, such as ACE inhibitors. Nitrates should be avoided in patients taking medications for erectile dysfunction, such as sildenafil, because the combination can cause severe hypotension.

Nesiritide is a recombinant form of BNP that is given intravenously and produces both natriuresis and vasodilation. Nesiritide has distinct advantages over other intravenous vasodilators, including nitroglycerin and nitroprusside, because it does not require intravenous titration or invasive hemodynamic monitoring, making it more practical for OU use. Studies suggest that it provides more rapid improvement of symptoms and decreases mortality and hospital admissions compared with dobutamine, and facilitates more rapid decrease in pulmonary capillary wedge pressure compared with nitroglycerin.

Nesiritide is given intravenously with an initial loading dose and then a maintenance infusion. It can cause hypotension, which may be potentiated by the use of other medications affecting pressure, and therefore ACE inhibitors are typically withheld. In the OU setting, nesiritide was shown to decrease the duration of hospitalization by 4 days and showed nonsignificant trends in decreased hospitalization after OU treatment and 30-day hospital readmission.

Although nesiritide was becoming widely used for the treatment of ADHF in ED and OU areas, this practice changed after two published meta-analyses suggested that it may worsen renal function and increase mortality. These findings led to a rapid decrease in the use of nesiritide for ADHF. Multiple editorials have supported and refuted the use of nesiritide in ADHF. More recent studies have suggested that nesiritide does not affect renal function or that it may be a dose-related phenomenon. A large-scale prospective study is currently addressing the safety and efficacy of nesiritide in ADHF. Information from this and other prospective studies will be needed to determine the usefulness of nesiritide and which patients it would benefit most. Currently, nesiritide remains a second-line agent for the treatment of ADHF.

Inotropes, including dobutamine, dopamine, milrinone, and amrinone, may be used to increase cardiac output and renal blood flow. They are often reserved for patients who have severely depressed cardiac output not responding to afterload reduction and are administered to fewer than 15% of patients who have ADHF. Inotropes are administered as a constant infusion and are not used in OU care because they are often given over several days.

Ultrafiltration recently became a treatment option for patients in ADHF. Ultrafiltration is a type of membrane filtration using hydrostatic pressure to force liquid and small solutes through a semipermeable membrane. One company has received FDA approval for use of an ultrafiltration device in ADHF. The Aquadex Flexflow requires two 18-gauge or larger peripheral intravenous catheters or a double-lumen central venous catheter to draw off blood and return blood, minus the filtered portion, to the patient. The patient must be anticoagulated with heparin before use to prevent clotting of the filter. Patients who have
contraindications to anticoagulation would not be candidates for this therapy. The device can remove up to 500 mL/h of ultrafiltrate, which consists of water and sodium. The rate of fluid removal is improved with the use of larger-gauge intravenous lines or catheters. Patients may be maintained on ACE inhibitors, β-blockers, and even diuretics, but intravenous vasodilators should be avoided.

The device seems to be effective in treating patients resistant to diuretics who are fluid overloading. One study showed an average fluid removal of more than 8 L with no change in sodium, potassium, creatinine, or BUN. The Ultrafiltration versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated Congestive Heart Failure trial compared ultrafiltration with standard care for ADHF in hospitalized patients and showed a greater amount of weight and net fluid loss in the ultrafiltration group. Even more impressive was the 50% reduction in rehospitalizations for heart failure and return ED or clinic visits within 90 days of discharge.

Although this new technology seems promising, particularly for patients resistant to diuretics, its usefulness in the OU setting is unclear. The need for larger venous access and anticoagulation make it less desirable for patients familiar with diuretic treatment. Further studies are needed to determine the usefulness of ultrafiltration in the OU population.

The long-term risk for sudden cardiac death in patients who have severe systolic dysfunction is significant, with one third to one half of patients who have heart failure dying unexpectedly. Several trials have shown benefit for patients who have ischemic heart failure and ejection fractions less than 35%. Placement of implantable cardiac defibrillators (ICD) has now become standard care, largely replacing antiarrhythmic therapy, and the cost of placement is currently covered by Medicare for patients who meet eligibility requirements. These criteria include EF of 35% or less, New York Heart Association class II or III disease for greater than 9 months, and inducible or sustained ventricular tachycardia.

Patients who seem eligible for ICD placement or who have a history of ventricular tachycardia should be referred to a cardiologist for electrophysiologic study. Patients who have a history of palpitations, syncope, or near-syncope may be referred for outpatient continuous cardiac monitoring, such as a Holter or event monitor, to determine if significant arrhythmias are occurring.

Some medications should likely be avoided or at least not started while patients are being treated for ADHF, including nonsteroidal anti-inflammatory agents, calcium channel blockers, and diabetic agents, such as metformin, thiazolidinedione, and cisapride.

Criteria for determining discharge are often based on a patient’s clinical presentation after a period of treatment. Several authors have provided more explicit discharge criteria for patients in the OU treated for ADHF. Most of these recommendations have not been prospectively studied but are pragmatic assessments of the patient’s clinical status. Evidence suggests that urine output less than 1 L after treatment predicts treatment failure. Some findings that may suggest that the patient has not improved include the presence of a third heart sound, increased BUN, hypotension, hypoxia, and dyspnea at rest or with minimal exertion. Having patients ambulate through the department is often prudent to show that they will be able to do so if discharged. Some suggested discharge criteria include:

Clinically improved dyspnea
Ability to ambulate back to baseline
Urine output greater than 1 L
Resting heart rate less than 100 beats/min and systolic blood pressure greater than 80 mm Hg
Pulse oximetry greater than 91% if not normally on supplemental oxygen
No clinical or EKG evidence of cardiac ischemia
No significant increase in serum BUN or creatinine
No new or significant dysrhythmias

The most recent AHA/ACC guidelines for the treatment of ADHF do not provide discharge criteria but recommend that patients be euvolemic before discharge.

It is extremely important to start recommended heart failure medications and provide prescriptions to patients discharged after OU therapy, because studies have shown that providing appropriate long-term heart failure medications, such as β-blockers and ACE inhibitors, reduces mortality.

Discharge education is an important aspect of care that is often overlooked but can have a large beneficial effect if administered properly. Education concerning smoking and medication and dietary compliance can reduce recidivism and decrease medical expenses for these patients.

The importance of smoking cessation education and therapy deserves additional mention. Simply by quitting smoking, patients can reduce their mortality by 30% within 2 years. This mortality benefit is similar to that seen with the use of β-blockers or ACE inhibitors in the treatment of heart failure. Having patients receive the
advice directly from the treating physician is very influential and can increase rates of smoking cessation greater than 60%. If resources are available, a multidisciplinary team, including cardiologist dietitians, social workers, and heart failure educators such as nurse practitioners, would be ideal to provide patient information on the importance of medication and dietary compliance, exercise, smoking cessation, and following up with future tests and appointments. Other areas for patient education include monitoring dyspnea symptoms and weight and notifying the physician for worsening conditions. The AHA Get with the Guidelines program offers Internet-based educational tools to educate patients who have heart failure who are being discharged. Ensuring that patients are taking appropriate medications, such as β-blockers and ACE inhibitors, is extremely important. Patients who leave with prescriptions for cardiac medications are more likely to adhere to that medication regimen.

SUMMARY

ADHF is a common illness presenting to the ED that is amenable to OU treatment. As the number of baby boomers continues to grow and the incidence of heart failure increases, the financial implications of ADHF treatment will become more prominent. Obtaining institutional support and developing a good working relationship with cardiology colleagues is vital to creating workable ADHF protocols for whichever type of OU an institution decides to use.

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Acute Decompensated Heart Failure

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