**Epide**miology

With the aging of the U.S. population and improved survival after myocardial infarction, the prevalence of heart failure is on the rise. At the same time, advances in medical therapy are allowing patients with heart failure to live longer. In 2008, 5.7 million Americans were estimated to have heart failure, with approximately 670,000 new cases diagnosed that year. Heart failure contributes to nearly 300,000 deaths per year, and costs associated with the treatment of heart failure exceed $30 billion annually. Heart failure accounts for nearly 1 million inpatient admissions per year and represents the primary reason for hospitalization in the growing elderly population. Approximately four of every five patients hospitalized for heart failure initially come to the emergency department (ED) for treatment.

Evidence-based literature for ED management of heart failure lags behind that of other emergency conditions, such as acute coronary syndrome and stroke. The number of large, randomized controlled clinical trials is small, and most practice guidelines, such as those from the Heart Failure Society of America and the European Society of Cardiology, rely heavily on consensus statements. A recent American Heart Association scientific statement highlighted the significant gaps in knowledge and the lack of evidence-based approaches to the management of heart failure in the ED. In contrast, data from the Acute Decompensated Heart Failure National Registry (ADHERE) and the Acute Heart Failure Global Survey of Standard Treatment (ALARM-HF) registry have provided important insight into the clinical characteristics and actual patterns of care of these patients.

**KEY POINTS**

- Acute decompensated heart failure can be manifested as volume overload, acute diastolic dysfunction, and low cardiac output.
- Identifying and addressing the precipitants of the decompensation are as important as treating the decompensation itself.
- Biomarkers such as B-type natriuretic peptide and the inactive N-terminal fragment of B-type natriuretic peptide can assist in making the diagnosis, but it is important to understand the limitations of these tests.
- In patients with volume overload, diuretics remain the cornerstone of therapy.
- Not all patients with acute decompensated heart failure are significantly volume-overloaded; overaggressive diuresis risks hypotension and worsening renal function.
- Nitrates are first-line therapy for patients in whom an acute reduction in cardiac preload and afterload is desired.
- Inotropic therapy should not be routinely instituted unless the patient is in cardiogenic shock.
- For patients in respiratory distress, noninvasive support (continuous positive airway pressure or bilevel positive airway pressure) may reduce the need for endotracheal intubation.

**Box 57.1 Causes of Heart Failure**

- Coronary artery disease
- Hypertension
- Valvular disease
- Idiopathic cardiomyopathy
- Alcoholic cardiomyopathy
- Toxic-related cardiomyopathy (e.g., from doxorubicin)
- Postpartum cardiomyopathy
- Hypertrophic obstructive cardiomyopathy
- Tachyarrhythmia-induced cardiomyopathy
- Infiltrative disorders (e.g., amyloid)
- Congenital heart disease
- Pericardial disease
- Hyperkinetic states (anemia, arteriovenous fistula, thyroid disease)
Noncompliance with medications or dietary restrictions and myocardial ischemia are believed to be the most common causes of clinical cardiac decompensation. Other cardiovascular precipitants are arrhythmia (atrial fibrillation in particular), acute valvular dysfunction, and hypertensive crisis, but ADHF can also arise as a consequence of noncardiac conditions such as infections, anemia, alcohol withdrawal, uncontrolled diabetes, and thyroid disease.

**PRESENTING SIGNS AND SYMPTOMS**

The heterogeneity of the signs and symptoms in patients with ADHF reflects, to some extent, the relative contributions of volume overload, acute diastolic dysfunction, and low cardiac output (Table 57.1). Volume overload, which usually occurs in the setting of medication noncompliance or dietary indiscretion (or both), is classically associated with gradually worsening congestive symptoms. Acute diastolic dysfunction can occur in the setting of myocardial ischemia, tachyarrhythmia, or uncontrolled hypertension and is typically manifested as flash pulmonary edema. Nearly half of all patients admitted to the hospital for ADHF have mild or no impairment in systolic function. Overt manifestations of low cardiac output (i.e., hypoperfusion) are not generally seen except in patients with advanced LV dysfunction.

Most patients with ADHF have some degree of dyspnea. However, ADHF can closely mimic many other cardiac, respiratory, and systemic diseases. Historical features such as a history of orthopnea, paroxysmal nocturnal dyspnea, or peripheral edema make the diagnosis of ADHF more likely. The most valuable single piece of historical information to elicit from patients is a previous history of heart failure, myocardial infarction, or coronary artery disease. For example, patients evaluated in the ED because of acute dyspnea are approximately six times more likely to have ADHF if they have previously experienced heart failure (Table 57.2).

Older patients may lack the typical signs and symptoms of heart failure because they are obscured by the aging process itself or by the presence of coexisting medical conditions.
Table 57.1 Syndromes of Acute Decompensated Heart Failure

<table>
<thead>
<tr>
<th></th>
<th>SYSTEMIC VOLUME OVERLOAD</th>
<th>ACUTE DIASTOLIC DYSFUNCTION</th>
<th>LOW-OUTPUT FAILURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Onset</td>
<td>Gradual</td>
<td>Rapid</td>
<td>Gradual</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Congestive</td>
<td>Congestive/ischemic</td>
<td>Poor perfusion</td>
</tr>
<tr>
<td>Example</td>
<td>Diuretic noncompliance</td>
<td>Hypertensive crisis</td>
<td>End-stage cardiomyopathy</td>
</tr>
</tbody>
</table>

Table 57.2 Sensitivity, Specificity, and Positive Likelihood Ratio of Selected Clinical Findings Associated with Acute Decompensated Heart Failure

<table>
<thead>
<tr>
<th>Past Medical History</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>POSITIVE LIKELIHOOD RATIO (:1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart failure</td>
<td>60</td>
<td>90</td>
<td>4.4</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>40</td>
<td>87</td>
<td>3.1</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>52</td>
<td>70</td>
<td>1.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>POSITIVE LIKELIHOOD RATIO (:1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal nocturnal dyspnea</td>
<td>41</td>
<td>84</td>
<td>2.6</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>50</td>
<td>77</td>
<td>2.2</td>
</tr>
<tr>
<td>Edema</td>
<td>51</td>
<td>76</td>
<td>2.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>POSITIVE LIKELIHOOD RATIO (:1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Third heart sound</td>
<td>13</td>
<td>99</td>
<td>11</td>
</tr>
<tr>
<td>Abdominojugular reflux</td>
<td>24</td>
<td>96</td>
<td>6.4</td>
</tr>
<tr>
<td>Jugular venous distention</td>
<td>39</td>
<td>97</td>
<td>5.1</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>50</td>
<td>78</td>
<td>2.3</td>
</tr>
<tr>
<td>Rales</td>
<td>60</td>
<td>78</td>
<td>2.8</td>
</tr>
<tr>
<td>Wheezing</td>
<td>22</td>
<td>58</td>
<td>0.52</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diagnostic Findings</th>
<th>SENSITIVITY (%)</th>
<th>SPECIFICITY (%)</th>
<th>POSITIVE LIKELIHOOD RATIO (:1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>26</td>
<td>93</td>
<td>3.8</td>
</tr>
<tr>
<td>Pulmonary venous congestion</td>
<td>54</td>
<td>96</td>
<td>12.0</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>74</td>
<td>78</td>
<td>3.3</td>
</tr>
</tbody>
</table>


Nonspecific symptoms such as weakness, lethargy, fatigue, anorexia, and light-headedness may actually be manifestations of decreased cardiac output. Abdominal or epigastric discomfort can be a manifestation of low output or, more commonly, hepatic congestion.

Vital signs provide a sense of the severity of illness and can suggest etiologic factors for decompensation. Hyperthermia or hypothermia may indicate sepsis or thyroid disease. In the absence of rate-controlling pharmacologic agents, tachycardia is nearly universal in patients with decompensated heart
failure. Bradycardia should raise concern for high-degree atrioventricular block, hyperkalemia, drug toxicity (digoxin, calcium channel blocker, beta-blocker), or severe hypoxia. Hypertension is commonly seen in patients with both systolic and diastolic dysfunction. Hypotension may represent baseline blood pressure (BP) in patients with end-stage cardiomyopathy but otherwise raises concern for shock, whether cardiogenic or otherwise.

The diagnostic utility of the physical examination has been well studied in the setting of chronic heart failure but less so for ADHF. It should be recognized that in ADHF, physical findings may be misleading because of the rapidly evolving clinical situation. Generally speaking, jugular venous distention, abdominojugular reflux, pedal edema, and an audible third heart sound are specific but insensitive indicators of heart failure, whereas the presence of pulmonary rales has only moderate specificity for heart failure (see Table 57.2).7

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis in patients with acute respiratory distress is broad and includes ADHF, asthma, chronic obstructive pulmonary disease (COPD), pneumonia, pulmonary embolism, and multiple systemic diseases, including sepsis.

Even before patients reach the hospital, ADHF is associated with significant morbidity and mortality, including malignant arrhythmias and prehospital cardiac arrest. With few exceptions, the safety and efficacy of prehospital interventions have been poorly studied. Prehospital therapy for decompensated heart failure should be undertaken with caution in light of the relatively high number of inaccurate diagnoses made in the field. In as many as 50% of patients with assumed heart-associated respiratory distress, a different condition is diagnosed once they arrive at the hospital. Despite these concerns, evidence suggests that prehospital therapy for presumed heart failure can prevent serious complications and improve survival, particularly for critically ill patients. For example, prehospital use of continuous positive airway pressure (CPAP) in patients with acute pulmonary edema may avert the need for endotracheal intubation.8

Nitroglycerin appears to be the safest and most effective of the prehospital medications used for presumed pulmonary edema.9 The role of other medications for heart failure in the prehospital setting is less clear. Early administration of furosemide appears to have very little benefit and may result in short-term complications. Prehospital use of morphine sulfate for presumed pulmonary edema has been associated with a higher rate of endotracheal intubation, particularly in patients whose condition turns out to have been misdiagnosed in the field.

**EMERGENCY DEPARTMENT EVALUATION**

The approach to patients with ADHF begins with stabilization of respiratory and hemodynamic status and rapid exclusion or treatment of reversible life-threatening conditions. Clinical evaluation and empiric therapy begin simultaneously and consist of supplemental oxygen, cardiac monitoring, pulse oximetry, and intravenous access. Patients with clinical signs of exhaustion or hypoxia despite supplemental oxygen require respiratory support via either invasive or noninvasive means. Those with hypotension, obtundation, cool extremities, or other signs of poor perfusion should be presumed to be in or near cardiogenic shock and be managed accordingly. An electrocardiogram (ECG) should be obtained early to exclude ST-segment elevation myocardial infarction. Once the initial resuscitation is under way, further efforts should be made to establish the diagnosis of ADHF and identify an underlying cause of the acute decompensation.

**DIAGNOSTIC STUDIES**

**LABORATORY TESTS**

The majority of patients with complaints suggestive of ADHF will require laboratory testing. A complete blood count is useful for ruling out anemia as an alternative cause of the dyspnea or as a precipitant of ADHF. An elevated white blood cell count may suggest the presence of an infectious process, especially if bands are present; however, this finding has not been well studied in patients with suspected ADHF. Serum chemistry analysis is important for assessing renal function and overall fluid and electrolyte balance, particularly in patients already receiving diuretic therapy and likely to require additional diuresis.

**MARKERS OF CARDIAC ISCHEMIA**

Elevations in cardiac troponin levels may be found in up to one third of patients with ADHF and can identify patients with a worse short-term prognosis. In any individual case, it remains a clinical determination whether elevations in biomarkers (1) reflect an acute coronary syndrome (i.e., unstable plaque causing ischemia and myocardial cell death and leading to worsening heart failure) or (2) simply reflect the severity of the heart failure (i.e., myocardial cell death with or without underlying ischemia). Information from the history (e.g., onset of symptoms, comparison with previous episodes) and from the ECG may be helpful in this regard.

**B-TYPE NATRIURETIC PEPTIDE AND NT-PROBNP**

B-type natriuretic peptide (BNP) is a counterregulatory hormone produced by cardiac myocytes in response to increased end-diastolic pressure and volume, as occurs in the setting of heart failure. ProBNP is released into the circulation and cleaved into biologically active BNP and an inactive N-terminal fragment, NT-proBNP, which has a half-life three to six times that of BNP. Plasma levels of BNP and NT-proBNP correlate with the degree of LV overload, severity of clinical heart failure, and both short- and long-term cardiovascular mortality.

Plasma levels of BNP and NT-proBNP have been shown to be useful in distinguishing between cardiac and noncardiac causes of dyspnea.10 Acutely dyspneic patients with plasma BNP levels lower than 100 pg/mL or NT-proBNP levels lower than 300 pg/mL are very unlikely to have ADHF (90% to 99% sensitivity), whereas those with BNP levels higher than 500 pg/mL or NT-proBNP levels higher than 1000 pg/mL are very likely to have ADHF (87% to 95% specificity). Intermediate levels must be interpreted in the clinical context (Fig. 57.2).
Interpretation of BNP levels must take into account baseline LV dysfunction and other known or suspected conditions associated with left or right ventricular pressure overload that may result in elevations in BNP. Patients with advanced age or renal insufficiency tend to have higher BNP and NT-proBNP levels, whereas those with a high body mass index tend to have lower levels. Although BNP and NT-proBNP measurements retain discriminatory power in these subpopulations, the optimal cutoff points for diagnosing ADHF may vary. The duration of symptoms also plays a role; for example, in the setting of acute pulmonary edema, these levels may not yet be elevated.

In general, emergency physicians (EPs) are about 80% accurate in distinguishing between cardiac and noncardiac causes of dyspnea on clinical grounds. Supplementing clinical acumen with routine BNP or NT-proBNP measurement does increase diagnostic accuracy overall, but as demonstrated in the Breathing Not Properly Multinational Study, the improvement is rather marginal. For example, in clear-cut cases, very high or very low values are unlikely to have an effect on diagnosis, whereas in less clear-cut cases, intermediate results are more likely.

BNP and NT-proBNP levels also carry modest prognostic information. Although levels at admission correlate only modestly with short-term outcomes, discharge levels are strong independent predictors of death or readmission.

**ELECTROCARDIOGRAPHY**

The ECG is likely to be abnormal in patients with heart failure. Signs of preexisting conditions such as hypertrophy, myocardial infarction, or dilated cardiomyopathy may be present. Atrial fibrillation or other arrhythmias may be detected. A large proportion of heart failure exacerbations are accompanied by cardiac ischemia, which may be detectable on an ECG.

**CHEST RADIOGRAPHY**

A chest radiograph should be obtained in all patients with suspected ADHF to assess for pulmonary congestion and assist in the differential diagnosis of other lung diseases. Findings on chest radiographs in patients with ADHF include cardiomegaly, vascular redistribution (e.g., cephalization, fullness of the hilar vessels), interstitial edema, and pulmonary edema. Pleural effusions in patients with heart failure tend to be bilateral or localized to the right side. Although the presence of pulmonary congestion is associated with a very high likelihood of ADHF, it should be noted that as many as one in five patients with ADHF do not have evidence of congestion on chest radiographs. In individuals with underlying pulmonary emphysema, congestion may appear atypical or not at all, and patients with long-standing congestive heart failure (CHF) may have scant radiographic evidence of congestion because of well-developed pulmonary lymphatics. Cardiomegaly may be absent in patients with acute heart failure, particularly in those with preserved LV systolic function.

**CARDBAC ECHOCARDIOGRAPHY**

Echocardiography is considered the “gold standard” for assessing the status of LV function, distinguishing between systolic and diastolic failure, and identifying regional wall motion abnormalities. Perhaps more important in the ED setting, echocardiography can assist in diagnosing or excluding potentially reversible causes of acute cardiac decompensation, such as pericardial tamponade, massive pulmonary embolism, ruptured chordae tendineae, and ruptured ventricular septum. As a practical matter, emergency echocardiography is not generally required in most instances of ADHF; particularly if a patient has a history of heart failure and a clear precipitant for decompensation.

**PULMONARY ARtery (SWAN-GANZ) CATHETERIZATION**

Invasive hemodynamic monitoring is not usually necessary for the diagnosis and management of ADHF, and its routine use is not recommended. In the absence of pulmonary disease or disproportionate right heart failure, clinical estimation or measurement of right atrial pressure usually correlates with
left-sided filling pressures. When compared with standard clinical management, hemodynamically guided therapy is not associated with improvement in short- or long-term outcome. Conversely, right heart catheterization remains a reasonable option in patients with cardiogenic shock or when a patient’s hemodynamic status is uncertain after careful clinical evaluation and initial therapy.

**OTHER DIAGNOSTIC MODALITIES**

Elevated end-tidal CO₂ concentrations and reduced peak expiratory flow rates are more commonly seen exacerbations of COPD than of ADHF. However, in neither case is a cutoff value available that can be used to accurately distinguish between cardiac and respiratory causes of dyspnea. Another noninvasive means proposed for aiding in the diagnosis of ADHF is impedance cardiography, in which real-time estimates of cardiac output and pulmonary capillary wedge pressure are derived via dynamic measurement of thoracic bioimpedance. Although the technology has been available for some time, data on the overall utility of this diagnostic tool in the ED setting are limited.

**TREATMENT**

**PRIORITIES OF TREATMENT**

All patients with ADHF who are in respiratory distress should receive supplemental oxygen and be positioned upright, if possible, to improve respiratory dynamics and maximize oxygen delivery to vital organs. Practice guidelines recommend the early application of monitors, such as pulse oximetry, noninvasive BP, and continuous cardiac monitoring, to provide early warning of further decompensation.

Although most patients in respiratory distress can be managed with supplemental oxygen and noninvasive ventilatory support (see the next section), the presence of agonal respirations or profoundly depressed mental status mandates endotracheal intubation. In general, airway management should be accomplished with rapid-sequence intubation because prolonged attempts at intubation risk worsening hypoxia, further cardiac decompensation, and cardiopulmonary arrest. Keeping the patient in an upright position as long as possible before intubation may assist in maximizing pre-oxygenation. Most induction agents (thiopental, fentanyl, and midazolam) are associated with a significant risk for hypotension in patients with ADHF, whereas induction with etomidate is generally considered safe.

**Noninvasive Respiratory Support**

For patients with respiratory distress in whom intubation is not immediately required, noninvasive respiratory support via CPAP or bilevel positive airway pressure (BiPAP) should be instituted (Fig. 57.3). Although the decision to initiate noninvasive respiratory support may depend on a variety of factors, the presumption is that the earlier therapy is instituted, the greater the likelihood of averting intubation. Success also depends on appropriate patient selection. Patients with unstable cardiac rhythms or cardiogenic shock are generally believed to not be candidates for a noninvasive approach. Likewise, in the setting of severe myocardial ischemia or infarction, full ventilatory support may be preferable to decrease myocardial oxygen demand.

CPAP improves lung mechanics by recruiting atelectatic alveoli, improving pulmonary compliance, and reducing the work of breathing. At the same time, particularly in patients with heart failure, CPAP improves hemodynamics by reducing preload and afterload, thereby enhancing LV performance. Pooled data from several randomized, controlled clinical trials suggest that the use of CPAP (at 5 to 10 mm Hg) in patients with respiratory distress caused by ADHF reduces the frequency of endotracheal intubation and may be associated with lower mortality.

BiPAP adds to the physiologic advantages of CPAP during expiration by providing differential positive pressure during inspiration, thereby providing direct assistance with ventilation. However, at present, little evidence suggests an advantage of BiPAP over CPAP in patients with ADHF and pure hypoxemic respiratory failure.

In patients with progressive respiratory failure despite noninvasive support, endotracheal intubation and mechanical ventilation should be instituted.

**PHARMACOLOGIC THERAPY**

The twin objectives of pharmacologic therapy for ADHF are relief of pulmonary congestion and improvement in systemic tissue perfusion. Strategies to achieve these goals involve reducing preload and enhancing LV function while aiming to maintain or even improve myocardial oxygen balance (Table 57.3).

**Diuretics**

Diuretics constitute the mainstay of therapy for patients with volume overload. Although their use is widely recommended as initial therapy for most patients with ADHF, it should be noted that until very recently this practice had not been evaluated in any large, prospective trial. Evidence from in vitro and in vivo experiments suggests that the direct vascular effects of diuretics may also contribute to their mechanism of action. However, studies comparing the acute effects of diuretics and nitrates have tended to emphasize the more favorable overall hemodynamic effects of the latter group (see the next section).

Depending on a patient’s clinical condition and previous use of diuretics, an initial intravenous (IV) dose of...
Nitrates are recommended for the treatment of ADHF, whether of ischemic or nonischemic origin. At low doses, nitroglycerin induces venodilation (preload reduction); at higher doses, nitroglycerin also causes arterial dilation (afterload reduction). Significantly, in patients with severe underlying LV dysfunction, afterload reduction appears to predominate over preload reduction, even with moderate doses of nitroglycerin.

Nitrates have been shown to be both safe and effective for the treatment of ADHF, particularly in the context of acute pulmonary edema. When compared with placebo therapy in the Vasodilation in the Management of Acute Congestive Heart Failure (VMAC) trial, IV nitroglycerin resulted in better dyspnea scores, but the study was not powered to demonstrate differences in morbidity or mortality.

Single doses of sublingual nitroglycerin (0.4 mg) can be given repeatedly every 5 to 10 minutes to provide adequate BP. In the hospital setting, however, continuous IV administration of nitroglycerin is more convenient and allows titration to specific clinical or hemodynamic endpoints (typically starting at 10 to 20 mcg/min and ranging up to 200 mcg/min). The hemodynamic effects of transdermal nitroglycerin are comparable with those of IV nitroglycerin, but this route of administration is less amenable to titration and may be less effective in patients with poor skin perfusion.
The hypotension induced by standard nitrate therapy is generally mild and transient. Severe or persistent hypotension should raise suspicion for hypovolemia, stenotic valvular disease such as aortic stenosis, cardiac tamponade, right ventricular infarction, or recent use of sildenafil (Viagra). If these conditions are known or suspected, nitrates should be avoided or used with extreme caution. Nitrate therapy may not be particularly effective in patients with massive peripheral edema. In such cases, aggressive diuretic therapy is more likely to be of benefit.

Sodium nitroprusside is recommended for patients with marked systemic hypertension, severe mitral or aortic valvular regurgitation, or pulmonary edema not responsive to standard nitrate therapy. Nitroprusside profoundly dilates resistance vessels and thereby rapidly reduces BP and afterload. Typically, nitroprusside is started at a dose of 0.1 to 0.3 mcg/kg/min, and the dose is increased as needed to improve clinical and hemodynamic status while maintaining systolic BP above 90 mm Hg or mean arterial pressure above 65 mm Hg. In patients with renal insufficiency, long-term use of nitroprusside carries the potential for cyanide toxicity as metabolites of the agent accumulate.

Nesiritide

Nesiritide (recombinant BNP) is the only pharmacologic therapy for dyspnea associated with ADHF that has been approved by the U.S. Food and Drug Administration (FDA) in recent years. Like other natriuretic peptides, nesiritide has intrinsic vasodilatory as well as mild diuretic and natriuretic properties when administered in supraphysiologic doses (2-mcg/kg bolus, followed by continuous infusion at 0.01 mcg/kg/hr).

A number of trials have shown nesiritide to be more effective than placebo in improving hemodynamic parameters in patients with ADHF, but it is less clear what, if any, clinically important outcomes are improved. In the VMAC trial, pulmonary capillary wedge pressure was lower in patients receiving nesiritide than in those receiving nitroglycerin, but dyspnea scores at 3 and 24 hours were not significantly different between the two randomized groups. Studies have yet to show differences in more durable outcomes, such as of length of hospital stay and hospital costs.

Pooled data from several trials have suggested an association between nesiritide and adverse events, specifically, worsening renal function and death. In contrast, in a more recent multicenter trial, nesiritide demonstrated no excess adverse effects on either renal function or mortality in patients with ADHF. However, in this same trial, nesiritide failed to meet primary efficacy end points with respect to improvement in dyspnea or 30-day outcomes when compared with placebo.

Angiotensin-Converting Enzyme Inhibitors

The beneficial effects of angiotensin-converting enzyme (ACE) inhibitors in patients with chronic heart failure have been appreciated for more than 2 decades. Though not formally recommended by consensus guidelines for the treatment of ADHF, small studies have demonstrated their safety and efficacy as treatment of ADHF.

ACE inhibitors are contraindicated in the context of pregnancy, hyperkalemia, or a history of ACE inhibitor–induced angioedema. Unlike nitrates, most ACE inhibitors have a relatively prolonged duration of action, thus making their dosage less easily titratable.

Inotropic Therapy

Outside the setting of cardiogenic shock (see later), inotropic therapy is not recommended for the routine management of ADHF. Although short-term inotropic therapy may improve hemodynamic performance and acute symptoms, the impact on outcomes is considerably less sanguine. One of the largest randomized placebo-controlled trials ever conducted in patients with ADHF, the Outcomes of a Prospective Trial of Intravenous Milrinone for Exacerbations of Chronic Heart Failure (OPTIME-CHF), showed no difference in mortality or readmission rate in patients receiving inotropic therapy but significantly higher rates of adverse events, particularly sustained hypotension and new atrial arrhythmias.

Nevertheless, in patients with severe signs and symptoms of low-output failure or no response to standard therapy, short-term treatment with inotropic agents may be considered. The goals of therapy must be considered carefully in the context of the patient’s risk for arrhythmia.

Digoxin has a very limited role in the ED management of heart failure. The inotropic effects of digoxin are modest and unpredictable, and they are delayed for at least 90 minutes after intravenous loading.

Morphine

Morphine, one of the oldest drugs still in use for the treatment of ADHF, remains an important adjunct for treating the anxiety and discomfort associated with pulmonary edema. The predominant hemodynamic effects of morphine appear to be mediated through the central nervous system. Morphine can be administered safely to most patients at low IV doses (2 to 4 mg); however, because of its sedative properties and potential to depress respirations, caution should be exercised in administering morphine to patients with chronic pulmonary insufficiency or suspected acidosis. Although a number of retrospective studies have shown an association between administration of morphine to patients with ADHF and higher rates of adverse events, it is not clear that this link is causative.

Investigational Drugs

Over the past decade, a number of newer drugs ranging from various receptor antagonists to calcium sensitizers and novel peptides have been investigated for the treatment of ADHF. Despite early promise with many of these agents, none have been shown in a prospective, placebo-controlled, randomized clinical trial to meet a primary clinical end point with respect to the treatment of ADHF.

In the EVEREST trial, tolvaptan, a vasopressin antagonist, improved short-term signs and symptoms in patients hospitalized with ADHF and receiving standard therapy, but this was not a primary end point of the long-term study. Tolvaptan was, however, approved by the FDA in 2009 for the treatment of hyponatremia associated with heart failure, among other hypervolemic states.

Relaxin, a natural peptide structurally similar to insulin that has unique vasodilator properties, is currently in phase III trials as a potential treatment option for ADHF.
**Beta-Blockers**

Long-term beta-blocker therapy affords an important survival benefit for patients with systolic heart failure. In contrast, administration of beta-blockers to patients with acute systolic dysfunction has been associated with life-threatening clinical deterioration. For this reason, institution of beta-blocker therapy is not recommended in the setting of ADHF, and long-term beta-blocker therapy is generally administered cautiously or at a reduced dose.

**ULTRAFILTRATION**

A novel approach to the problem of volume overload involves ultrafiltration of peripheral blood to remove excess fluid and electrolytes. Though typically reserved for patients with significant renal failure or volume overload unresponsive to diuretics, evidence from the Ultrafiltration vs IV Diuretics for Patients Hospitalized for Acute Decompensated CHF (UNLOAD) trial demonstrated that as an alternative to diuretic therapy, ultrafiltration results in greater fluid loss and lower rates of rehospitalization. In the ED, a major limitation of this approach is the feasibility of securing the necessary equipment and intravenous access.

## SPECIAL CIRCUMSTANCES

### CARDIOGENIC SHOCK

Heart failure with cardiogenic shock can be the initial manifestation of acute ST-segment elevation myocardial infarction. Although mortality remains high in this setting, referral for emergency cardiac catheterization and revascularization is of proven benefit. Noncardiac causes of shock, such as hypovolemia, sepsis, poisoning, and massive pulmonary embolism, must also be considered.

Aside from addressing reversible causes of shock, the overarching goal in treating patients with cardiogenic shock should be to restore and maintain perfusion of vital organs. Patients who are initially seen in shock with normal BP or only mild hypotension often have a favorable response to dobutamine (starting at 2 to 3 mcg/kg/min). When compared with dopamine, dobutamine is associated with a lower incidence of arrhythmias, less peripheral vasoconstriction, and more consistent reduction in LV filling pressure for a comparable rise in cardiac output. Dopamine is required for patients who have severe hypotension (systolic BP of approximately 70 to 80 mm Hg) in the presence of volume overload or after bolus administration of saline. At moderate doses (4 to 5 mcg/kg/min), dopamine improves cardiac output without causing excessive systemic vasoconstriction. If the patient can be stabilized with dopamine, dobutamine can then be added and the dose of dopamine lowered, with the goal of reducing myocardial oxygen demand. In extreme cases, norepinephrine can be added to increase systolic pressure to acceptable levels (≈80 mm Hg). However, because of the adverse effects on renal and mesenteric perfusion, use of high-dose dopamine or norepinephrine should be considered only as a temporizing measure until definitive therapy can be substituted.

It is important for the EP to distinguish patients with acute cardiogenic shock from those with low BP or other signs of systemic hypoperfusion in the setting of preexisting severe or end-stage systolic heart failure. Assessment and treatment of these patients can be extremely challenging, and optimal management may require the involvement of a heart failure specialist. Attempts to aggressively treat these patients can lead to rapid decompensation. Frequently, the key to management is identifying the cause of the decompensation.

### ATRIAL FIBRILLATION

Atrial fibrillation is seen in approximately one third of patients with ADHF. Although loss of synchronized atrial contractions is of minimal hemodynamic significance in patients with normal ventricular function, in those who have abnormal LV systolic or diastolic function, loss of the atrial kick can have profound consequences. This is particularly evident when atrial fibrillation is accompanied by a rapid ventricular response, thereby reducing filling time.

When assessing a patient with rapid atrial fibrillation and ADHF, it is often difficult to attribute cause and effect. New-onset rapid atrial fibrillation may be the precipitant of ADHF, but more commonly, rapid atrial fibrillation is a response to worsening heart failure (e.g., via neurohormonal activation). This distinction can sometimes be difficult to make clinically, but regardless, attention must be paid, to some degree, to managing both conditions.

Management of atrial fibrillation in the context of ADHF should focus on treating the underlying precipitants of decompensation (e.g. volume overload) while also controlling the ventricular rate. However, caution should be exercised in the use of a beta-blocker or calcium channel blocker for rate control because of the potential negative inotropic effects. Digoxin, diltiazem, and amiodarone are considered acceptable agents for rate control, even in patients with LV systolic dysfunction. Cardioversion, whether electrical or chemical, is a reasonable treatment alternative for truly unstable atrial fibrillation, but sinus rhythm may not be achieved or maintained if the underlying heart failure is not addressed.

### RENAL DYSFUNCTION

In part because of common preconditions such as diabetes and hypertension and in part because of the effects of diuretics and low cardiac output on renal function, heart failure and renal insufficiency frequently coexist. Approximately 1 in 5 patients with ADHF have creatinine levels higher than 2.0 mg/dL. In patients with ADHF, preexisting renal insufficiency is associated with greater morbidity and mortality, and worsening of renal function over the course of treatment is associated with poorer outcomes.

In patients undergoing hemodialysis, heart failure is the most common reason for ED visits. Not surprisingly, ADHF is frequently a result of volume overload between dialysis treatments. Although hemodialysis is the obvious treatment of choice for these patients, it may not always be immediately available, and supplemental oxygen, CPAP, and nitrates are generally effective in stabilizing patients until hemodialysis can be performed.

## FOLLOW-UP AND NEXT STEPS IN CARE

The vast majority of patients with ADHF evaluated in the ED are admitted to the hospital. Discharge from the ED without
adequate treatment may be associated with recurrent visits and short-term morbidity and mortality. ADHF is often a dynamic entity: one patient may appear dramatically ill at initial evaluation but respond rapidly to treatment, whereas another patient may experience serious complications after a period of apparent stability. For any individual patient, identifying and addressing the precipitant of the decompensation is critical to making the correct disposition.

The Heart Failure Society of America has established criteria for discharging patients with heart failure from the ED (Box 57.3). However, these guidelines have not been prospectively studied. It should be noted that previously published criteria from the U.S. Agency for Health Care Policy and Research failed to account for more than 30% of 30-day mortality. Thus, although published guidelines can assist with triage, the significant rate of morbidity mandates that clinical judgment be incorporated into the decision-making process.

For patients with ADHF admitted to the hospital, inpatient mortality is approximately 4%, and the median length of stay exceeds 4 days. In those admitted with advanced stages of heart failure, inpatient mortality approaches 10%. Clinical correlates of major complications or death during hospitalization include hypotension; tachypnea; ECG abnormalities; hyponatremia; renal insufficiency; elevations in troponin, BNP, and NT-proBNP; and poor initial diuresis. However, even patients without any of these risk factors have measurable rates of in-hospital morbidity and mortality. A risk stratification tool derived from the ADHERE registry has been developed to help clinicians determine the risk for mortality in patients with ADHF (Fig. 57.4).

For a patient discharged home from the ED, consultation with the patient’s primary care physician or cardiologist is important. For example, it is likely that the patient’s outpatient medication regimen will require adjustment to prevent a return to the ED. In some studies, intensive outpatient

![Fig. 57.4](image-url)  
**Fig. 57.4** Risk stratification of patients hospitalized for acute decompensated heart failure (ADHF). **BUN**, Blood urea nitrogen; **Cr**, [serum] creatinine; **SBP**, systolic blood pressure.

**Box 57.3 Heart Failure Society of America Recommendations for Discharge of Emergency Department Patients with Heart Failure**

**Admission is recommended for:**
- Severe acute decompensated heart failure: Hypotension
- Worsening renal function
- Altered mental status
- Dyspnea at rest
- Arrhythmia with hemodynamic compromise, including new-onset atrial fibrillation
- Acute coronary syndrome

**Admission should be considered for:**
- Worsening congestion (pulmonary or systemic)
- Significant electrolyte disturbance
- Associated comorbid conditions:
  - Pneumonia
  - Pulmonary embolism
  - Diabetic ketoacidosis
  - Transient ischemic attack, cerebrovascular accident
- New-onset heart failure

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**Admission for ADHF**

- **BUN < 43**
  - **SBP ≥ 115 mg Hg**: Low risk
  - **SBP ≤ 115 mg Hg**: Intermediate risk

- **BUN > 43**
  - **SBP ≥ 115 mg Hg**: Intermediate risk
  - **SBP ≤ 115 mg Hg**: Renal
    - **Cr ≤ 2.75**: Intermediate risk
    - **Cr ≥ 2.75**: High risk
follow-up has been shown to be successful in preventing recurrent ED visits and hospitalizations.

**OBSERVATION UNITS**

ED observation units have been advanced as a safe and cost-effective means of treating a subset of ADHF patients, thereby avoiding the need for hospital admission. Admission to an observation unit allows the ED physician to assess a patient’s response to diuretic (or other) therapy over time. Although interest in this field is growing, no randomized studies have been performed to date to substantiate the use of such units.²⁶

**REFERENCES**

*References can be found on Expert Consult @ www.expertconsult.com.*
REFERENCES


