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Acute Decompensated Heart Failure: New Strategies for Improving Outcomes

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Abstract

Acute decompensated heart failure is a common emergency department presentation with significant associated morbidity and mortality. Heart failure accounts for more than 1 million hospitalizations annually, with a steadily increasing incidence as our population ages. This issue reviews recent literature regarding appropriate management of emergency department presentations of acute decompensated heart failure, with special attention to newer medication options. Emergency department management and appropriate interventions are discussed, along with critical decision-making points in resuscitation for both hypertensive and hypotensive patients.

CME Objectives

Upon completion of this article, you should be able to:

1. Diagnose acute exacerbations of heart failure using a focused physical examination and appropriate adjunct testing.
2. Identify alternative causes of dyspnea and discuss how to distinguish them from heart failure.
3. Determine the appropriate ED management of acute decompensated heart failure and manage those at risk for rapid deterioration.

Prior to beginning this activity, see "Physician CME Information" on the back page.

Case Presentations

As you arrive for your ED shift, an ambulance pulls in, carrying a patient struggling to breathe. The paramedics quickly brief you: your patient is a 76-year-old woman with a history of heart failure. She has been compliant with all of her medications but has had progressively worsening, difficult breathing. You notice coarse, wet-sounding lungs with poor air movement at the lung bases. You also notice significant pitting edema in both of her legs. She describes orthopnea and states that she has been sitting up in a chair to sleep for "a while." When you examine her medications, you note that she is on a low dose of a beta blocker and an ACE inhibitor, despite a stated history of low blood pressure. She was also prescribed spironolactone and furosemide, and you can feel an implant under the skin of her left chest wall, which she confirms as an AICD. You attach your patient to the cardiac monitor and notice she is tachycardic, with a heart rate of 115 beats/min, and her blood pressure is 80/40 mm Hg. You wonder if she would be best treated with fluids or diuretics, and your medical student asks, "How do we decide?"

While nurses are establishing IV access for your first patient, another nurse pulls you into a nearby room with a patient who just arrived via EMS. The patient is an overweight middle-aged man who is also struggling to breathe. Paramedics report that his blood pressure was 220/130 mm Hg at the scene. You immediately attach the patient to the cardiac monitor and obtain vital signs. His blood pressure is now 240/140 mm Hg. You listen to his lungs and again notice coarse, wet breath sounds. Your patient is tachypneic, leaning forward in bed, and saturating 70% on room air. His oxygen saturation improves to 88% on a 100% nonrebreather mask. His legs are edematous, and he has marked conversational dyspnea. Respiratory failure seems certain unless appropriate action is taken, and you wonder if there is anything that can change this patient's course.

Introduction

The incidence of in-hospital mortality among patients admitted to the hospital for decompensated heart failure is 6.4%.[1] Although there are many management options available, some therapies offer innovative approaches to improve patient outcomes, while others may increase cost without improving outcomes.

In the United States, acute decompensated heart failure (ADHF) is the number one cause of hospital admission in patients over the age of 65 and accounts for more than 1 million hospital admissions and $30.7 billion in healthcare expenditure annually.[2] In individuals aged 65 to 69 years, the prevalence of heart failure is roughly 20 per 1000, and prevalence jumps to more than 80 per 1000 in individuals older than 85 years.[3] The prevalence of heart failure varies by sex and ethnicity, with men demonstrating a higher prevalence than women, and blacks having a higher prevalence than whites.[2] With the aging of the United States population, heart failure is expected to become a more common emergency department (ED) presentation. Projections estimate an increase in the prevalence of heart failure by 46% from 2012 to 2030, with a predicted 8 million adult cases in the United States by 2030.[2]

Not all heart failure is the same. Heart failure with preserved ejection fraction (HFpEF) and heart failure with reduced ejection fraction (HFrEF) represent distinct underlying pathophysiologies that require different approaches in treatment. HFpEF and HFrEF are essentially equal in terms of occurrence, morbidity, and mortality.[4] The underlying volume status of heart failure patients is difficult to assess, yet time is often limited, and the interventions chosen can change the course for better or for worse. In addition, ADHF patients may present with either hypertension or hypotension, which can make management challenging.

This issue of *Emergency Medicine Practice* examines the medical management of ADHF, with a focus on new therapies that may alter conventional management. This issue will enable the emergency clinician to quickly recognize the clinical presentations of the varying types of decompensated heart failure, understand the underlying pathophysiology, and formulate the most appropriate management plan.

Critical Appraisal of the Literature

A literature search was performed via PubMed using the terms *acute heart failure* and *decompensated heart failure*. The search returned 1710 articles; 350 articles from 2014 to present were screened for relevance, and a total of 190 were reviewed based on clinical applicability in the ED. The Cochrane Database of Systematic Reviews was searched for reviews using the terms *decompensated heart failure* and *acute heart failure*, which identified 10 reviews; 108 were identified with the more general search terms of *heart failure*. The majority of these reviews focused on chronic heart failure management and were excluded. Guidelines released jointly by the

American Heart Association and American College of Cardiology Foundation were also reviewed.

The available data that focus on the management of ADHF are, overall, of lower quality than that for chronic heart failure management, and the literature behind newer interventions is even more limited. Studies looking at newer treatment modalities would benefit from the improved generalizability associated with multicenter studies that enroll larger patient populations. The paucity of data on the vasodilator nesiritide is a prime example – the only available studies are small, underpowered, and tend to show nonsignificant trends in terms of efficacy and harm compared with older modalities.[5,6] Even basic interventions (such as nitrate administration) have limited data supporting their use.[7] Less commonly performed rescue therapies, such as extracorporeal membrane oxygenation (ECMO), have even lower-quality data supporting their use, largely due to small sample sizes.[8] The literature behind the management of HFpEF is also weaker than that supporting the management of HFrEF.

Etiology and Pathophysiology

Heart failure occurs when the heart is either unable to pump blood during systole (HFrEF) or to fill with blood during diastole (HFpEF). Heart failure typically begins with injury to the myocardium. The injury may be secondary to a number of causes that develop slowly over time (eg, uncontrolled hypertension, diabetes) or more suddenly (eg, coronary ischemia).

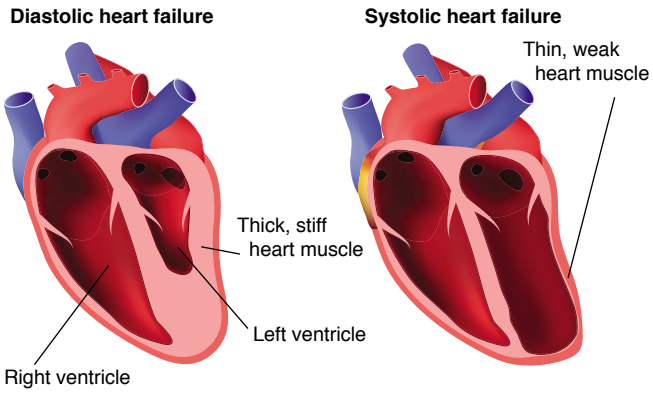
Regardless of the cause, myocardial injury results in structural, electrophysiological, and biochemical remodeling. Over time, structural remodeling produces abnormalities of the ventricular wall that may impair either systolic contraction or diastolic relaxation. **(See Figure 1.)** Electrophysiologic changes may result in impaired conduction, manifested as QRS widening on electrocardiogram (ECG), which can lead to conduction blocks and re-entrant dysrhythmias.[9] Biochemical remodeling is complex and incompletely understood, but it results in impaired myocyte functionality and increased risk of dysrhythmia. These compensatory responses are initially adaptive, but over time, they contribute to the progressively worsening contractility of the cardiac myocytes, leading to impairment of the heart's ability to effectively move blood throughout the circulatory system.

The poor systemic perfusion in patients with severe heart failure results in the release of norepinephrine, activation of the renin-angiotensin-aldosterone axis, and release of arginine vasopressin. Release of these hormones causes systemic vasoconstriction, leading to increases in both preload and afterload, further stressing the ailing heart.

Compared to HFrEF, less is known about the pathophysiology of HFpEF.[10] HFpEF is defined as heart failure with ejection fraction that is either borderline (41%-49%) or normal (> 50%). This constitutes a heterogeneous group of patients that includes well-managed patients with prior diagnosis of HFrEF that showed improvement in their ejection fraction with appropriate therapy, and patients with true diastolic dysfunction. Diastolic dysfunction is characterized by elevated left ventricular filling pressures with impaired myocardial relaxation. Recent studies have suggested that chronic low levels of inflammation and oxidative stress may play a role in the development of diastolic dysfunction via microvascular endothelial inflammation and tissue fibrosis that results in impaired left ventricular relaxation.[11] Due to its distinct pathophysiology, diastolic dysfunction responds differently to traditional heart failure therapies that were intended for the treatment of HFrEF.

Regardless of the underlying etiology, patients presenting with ADHF appear very similar to each other: sodium and fluid are retained, with excess fluid backing up into the lungs, abdomen, and extremities. The result is fatigue, peripheral edema, and dyspnea that is often worse with exertion. In the ED setting, it is challenging to distinguish between the 2 types of heart failure; thus, emergency clinicians generally manage the etiologies in the same way despite the underlying pathophysiological differences.

Figure 1. Diastolic and Systolic Heart Failure



Diastolic heart failure results in heart failure with preserved ejection fraction; systolic heart failure results in heart failure with reduced ejection fraction.

Differential Diagnosis

The presence of an acute change in a patient's symptoms, presenting either as a new heart failure diagnosis or as an acute change in heart failure severity, should prompt the emergency clinician to look for alternative causes of the patient's decompensation. These precipitating events include "can't

miss" life-threatening diagnoses as well as potentially reversible etiologies. It is crucial to identify these precipitating factors when working up any patient presenting with an acute decompensation to determine whether a treatable inciting event is present. **(See Table 1.)**

The differential diagnosis for patients presenting with dyspnea is extensive, and often patients can present with symptoms that are suggestive of more than one potential cause. In these cases, the emergency clinician must determine the most appropriate tests to tease out the etiology of the patient's dyspnea and to guide the subsequent care. **(See Table 2.)**

Prehospital Care

Prehospital management begins with stabilization of the patient's airway and breathing. The initial rapid assessment should involve measurement of oxygen saturation and application of supplemental oxygen as needed. Patients with acute decompensation without contraindications often respond well to noninvasive positive pressure ventilation (NIPPV) en route to the hospital. Early application of this therapy by paramedics can prevent clinical deterioration and helps to avoid intubation. A meta-analysis involving 5 studies and 1002 patients demonstrated a reduction in both intubations and mortality with the use of continuous positive airway pressure (CPAP) in prehospital patients with acute respiratory failure.[12]

A 12-lead ECG must be obtained to look for cardiac ischemia, since acute coronary syndromes (ACS) can present with the acute onset of heart failure. The presence of an ST-segment elevation myocardial infarction (STEMI) would alter the immediate hospital management and may also change the preferred destination hospital.

Patients with elevated blood pressure and symptoms of heart failure can be started on sublingual nitroglycerin prior to ED arrival. One study looking at the safety of higher-dose sublingual nitroglycerin in the prehospital setting examined 75 patients in an emergency medical services (EMS) system that initiated a high-dose sublingual nitroglycerin protocol involving the administration every 5 minutes, as needed, of 2 tablets of 0.4 mg sublingual nitroglycerin for systolic blood pressure (SBP) > 180 mm Hg, or 3 tablets for SBP > 200 mm Hg. There were only 3 incidents of hypotension, all of which resolved without intervention.[13] This study is limited by its size and the fact that it was not designed to show benefit over alternative protocols. In this particular cohort of hypertensive patients, however, the rare finding of hypotension (3.2%) demonstrated that a higher-dose protocol of nitroglycerin administration was tolerated in the majority of these patients.

One concern regarding the initiation of focused therapy by EMS is the difficulty in differentiating ADHF from other causes of acute respiratory distress. In a retrospective analysis that evaluated 330 patients who received furosemide en route by EMS and/or had an ED diagnosis of heart failure, one-third of the patients who received prehospital furosemide did not end up receiving a final heart failure diagnosis after a more thorough ED evaluation, and more than half of the patients with a final heart failure diagnosis did not receive prehospital furosemide. Patients who received prehospital furosemide had more adverse events and longer length of hospital stays, but the study design precludes drawing definitive conclusions.[14] These findings corroborate an earlier study that found that, of 144 patients receiving furosemide in the prehospital setting, 42% did not receive a subsequent diagnosis of congestive heart failure, and in 17%, the administration of diuresis was deemed potentially harmful.[15]

The difficulty in differentiating heart failure from other causes of acute respiratory distress (eg, pneumonia, chronic obstructive pulmonary disease [COPD], or ACS) limits the utility of initiating focused therapy beyond general stabilization in the prehospital setting. It is our opinion that the prehospital management of most patients with acute heart failure should focus on stabilization of the patient's respiratory status and should avoid targeted medical therapy.

Table 1. Precipitants of Acute Decompensation in Heart Failure Patients

- Acute coronary ischemia
- Valvular dysfunction
- Cardiac arrhythmia
- Pulmonary embolism
- Myocarditis
- Hypertensive emergency
- Pericardial tamponade
- Severe anemia
- Worsening renal failure
- Sepsis
- Drug noncompliance
- Dietary indiscretion
- Medication side effect
- Thyroid dysfunction

Table 2. Differential Diagnosis for Patients Presenting With Dyspnea

Life-Threatening Causes

- Decompensated heart failure
- Chronic obstructive pulmonary disease
- Asthma
- Pneumonia
- Pulmonary embolism
- Acute coronary syndromes
- Aortic dissection
- Pericarditis or pericardial effusion
- Pneumothorax

Emergency Department Evaluation

History

A complete history can sometimes be difficult to obtain in an acutely dyspneic patient. In patients in extremis, attention should focus first on stabilizing respiratory status. Every patient presenting with symptoms concerning for acute heart failure should be asked about the course of their symptoms, including duration, onset, and severity, to determine whether symptoms have been gradually worsening or if some sort of event has caused an acute decompensation. An acute change raises the concern for a precipitating event that may be reversible.

Baseline exercise tolerance should be elicited. Asking a patient how far he or she can walk without feeling short of breath gives the emergency clinician an idea of the severity of the underlying disease. Dyspnea with a patient's activities of daily living is concerning for more advanced heart failure. Recent changes in medications, difficulties with medication compliance, and dietary indiscretion with the ingestion of high-salt foods are important factors to identify, as they may have contributed to worsening symptoms.

Patients should be asked about their medical history and screened for additional risk factors associated with other potential etiologies of their symptoms such as COPD, pulmonary embolism (PE), or pericardial effusion. Family history may offer some clues, particularly in patients with a more extensive family history of cardiac disease. Some patients may have a family history of a dilated cardiomyopathy, while others may have a strong family history of hypertension and coronary artery disease.

Asking about current medications and history of cardiac surgeries can give important information about the severity of the patient's heart failure prior to their ED presentation. Patients with an automatic implantable cardioverter-defibrillator (AICD) in place and those taking medications reserved for more severe cases of heart failure (eg, spironolactone) can be presumed to have more severe baseline disease. Additionally, the dosages of medications can offer clues as to the patient's baseline blood pressure. Patients on very low doses of an angiotensin-converting enzyme (ACE) inhibitor and a beta blocker may have a baseline low or normal blood pressure, while those on much higher doses can be presumed to be hypertensive at baseline. Any acute changes in the patient's blood pressure should be acknowledged and investigated.

Serial weight assessments can be useful in evaluating volume status, but the utility is generally higher in the non-ED setting. Patients in the ED are often not able to provide an accurate weight measurement, and pausing management to weigh an acutely dyspneic patient may not be feasible. However, when it is medically reasonable and when the patient is able to give a reliable baseline weight, comparing the patient's current weight to past measurements can assist in the evaluation of volume status. Weight gain of at least 5 pounds in the preceding 3 days has a high specificity but low sensitivity for detecting ADHF. Smaller amounts of weight gain in the appropriate clinical setting can also be indicative of fluid retention.[16]

Physical Examination

The patient's positioning in bed when you walk in the room can provide useful information on respiratory status. A patient who is sitting forward and struggling to breathe requires urgent intervention, but a patient with poor oxygenation who is no longer struggling but is lying back, fatigued and seemingly comfortable, may be on the verge of immediate respiratory collapse. Delirium in an acute heart failure patient is predictive of worse clinical outcomes and higher short-term mortality.[17]

The physical examination begins with assessment of airway, breathing, and circulation. Heart failure patients can deteriorate rapidly, so blood pressure and oxygenation status should be assessed early and reassessed frequently, especially after the initiation of targeted therapy. The presence of hypotension versus hypertension is a critical distinction that will determine appropriate management strategies. Peripheral pulses should be evaluated for presence, equality, and strength. A narrow pulse, cool extremities, and low blood pressure can all be indicative of a low perfusion state that should prompt more rapid intervention.

Patients suffering from long-term heart failure can develop cardiac cachexia, a syndrome characterized by the loss of both quantity and quality of skeletal muscle.[18] This reduction in muscle mass can produce a deficit in inspiratory muscle strength and function.

Neck veins should be examined for the presence of jugular venous distension. The patient should be examined with the head of the bed at 30° to obtain an accurate assessment, but the presence of jugular venous distension with the patient sitting straight up is also clinically relevant and is suggestive of more severe congestion. Jugular venous distention can be challenging to appreciate in patients with thick or short necks, but in many patients it can be a quick, noninvasive, and easy way to evaluate right-heart pressures.

Lung sounds are important diagnostically but can be difficult to interpret. Patients may have basilar rales or scant breath sounds at the lung bases, which are indicative of fluid collecting in the dependent portions of the lung. More extensive rales can be indicative of more severe pulmonary edema, with fluid collecting throughout the lung. Wheezing is

usually indicative of obstructive lung pathology, but it may also occur as a result of heart failure with pulmonary edema. To hear an example of lung sounds, click the link to an online video demonstrating the lung sounds findings with pulmonary edema (listen with headphones): https://www.youtube.com/watch?v=z4Fu1udzrTw&index=4&list=PLLKSXV1ibO86qgE2y9cMqNFmh6LfOa8RM

Heart sounds should be documented to evaluate for the presence of a new or worsening murmur or distant heart sounds. An abnormal cardiovascular examination may identify the cause of the patient's heart failure. A new murmur may indicate valvular dysfunction, while distant heart sounds may reveal a pericardial effusion. An S3 gallop can sometimes be heard in patients with heart failure and is considered diagnostic.[19] Heart failure with preserved and reduced ejection fraction both present with similar clinical syndromes that are indistinguishable on initial bedside examination. Concerning findings may prompt a more thorough examination that includes bedside cardiac ultrasound to evaluate for effusion or, in the hands of a provider with advanced ultrasound skills, valvular dysfunction or rupture.

The lower extremities should be examined for pitting edema and graded based on how far proximally the swelling spreads. **(See Table 3.)** Patients can appear to be completely comfortable from a respiratory perspective but have edema up to the abdomen. Any asymmetrical swelling that is new or has not been previously investigated may warrant, in the appropriate clinical setting, a lower extremity Doppler ultrasound to evaluate for possible deep vein thrombosis.

Diagnostic Studies

Treatment can be initiated before any diagnostic testing has been completed if a clear case of heart failure has been identified. However, in cases where the diagnosis is unclear, further diagnostic studies can be helpful. All patients, even those believed to have a clear diagnosis, require a basic workup to evaluate for any secondary causes or underlying organ dysfunction.

Audio Recording of Lung Sounds

Scan the QR with a smartphone or go to: https://www.youtube.com/watch?v=z4Fu1udzrTw&index=4&list=PLLKSXV1ibO86qgE2y9cMqNFmh6LfOa8RM (Listen with headphones.)

Electrocardiogram

An ECG should be performed promptly on every patient with suspected decompensated heart failure in order to evaluate for a strain pattern or evidence of acute coronary ischemia and to screen for a dysrhythmia that could explain the deterioration. ADHF may be the presenting picture in STEMI. **(See Figure 2, page 7.)** Vasodilators should not be used among patients presenting with an inferior STEMI (ie, ST elevations in II, III, and aVF), as these patients are often preload dependent, and administration of vasodilators could result in a dangerously low blood pressure.

Diagnostic Imaging

Chest X-Ray

A chest x-ray can help assess the severity of heart failure and may reveal alternative etiologies. The chest x-ray should be evaluated for cardiomegaly (best viewed on a posteroanterior view and lateral chest view, if feasible) and for pulmonary vascular congestion. Pulmonary congestion manifests initially as redistribution of the pulmonary vascularity toward the upper lobes, typically referred to as cephalization. **(See Figure 3, page 7.)** More advanced congestion produces interstitial edema seen as Kerley B-lines, which are thin, 1- to 2-cm lines perpendicular to the pleural surface at the periphery of the lungs. **(See Figure 4, page 7.)** As pulmonary congestion progresses further, patients develop alveolar edema with bibasilar or perihilar consolidations and pleural effusions. **(See Figure 5, page 8.)** Alternative diagnoses (such as pneumonia or pneumothorax) may also be identified on a chest x-ray.

Echocardiography

Echocardiography is the primary imaging modality to evaluate a patient's cardiac function and evaluate for either systolic or diastolic dysfunction. When feasible, a focused bedside echocardiogram can be performed as part of the initial ED evaluation of patients in ADHF to evaluate general cardiac function and screen for gross abnormalities. The echocardio-

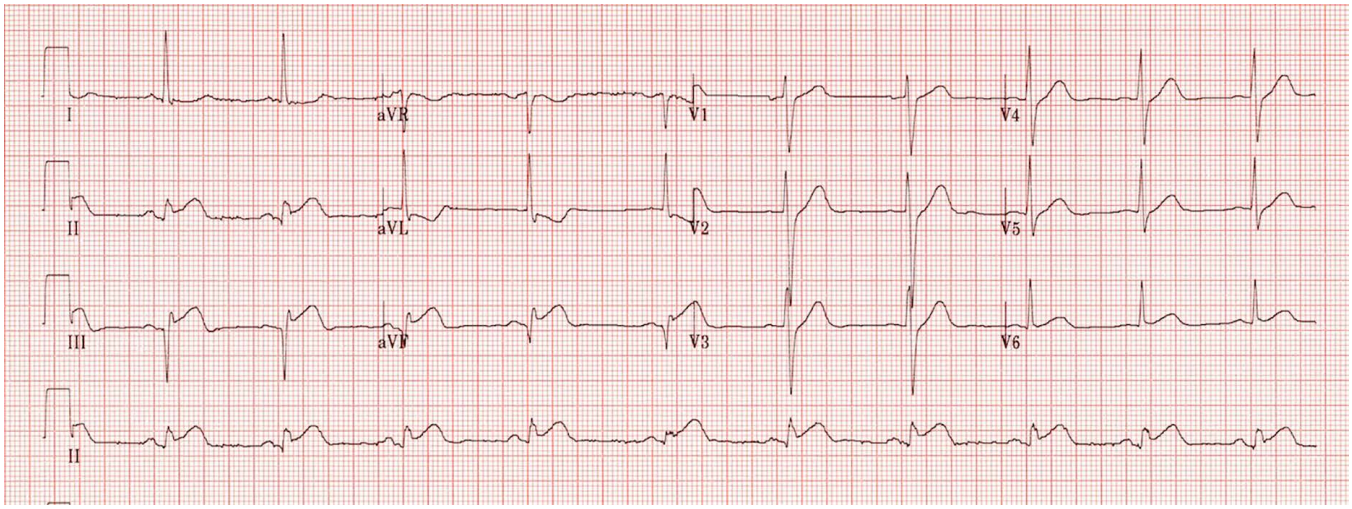
Table 3. Pitting Edema Scale

Score	Severity	Depth	Duration
0	None	0 mm	Not applicable
1+	Mild	2 mm	Rapid disappearance
2+	Moderate	4 mm	10-15 sec
3+	Moderately severe	6 mm	15 sec-2 min
4+	Severe	8 mm	> 2 min

gram is the only part of the initial ED examination where systolic and diastolic dysfunction can be distinguished. Echocardiography is also useful to detect valvular dysfunction and pericardial effusion. The initial ED echocardiogram offers useful information by evaluating approximate left ventricular ejection fraction, diastolic function, wall thickness, focal wall motion abnormalities, and valve function.[20,21]

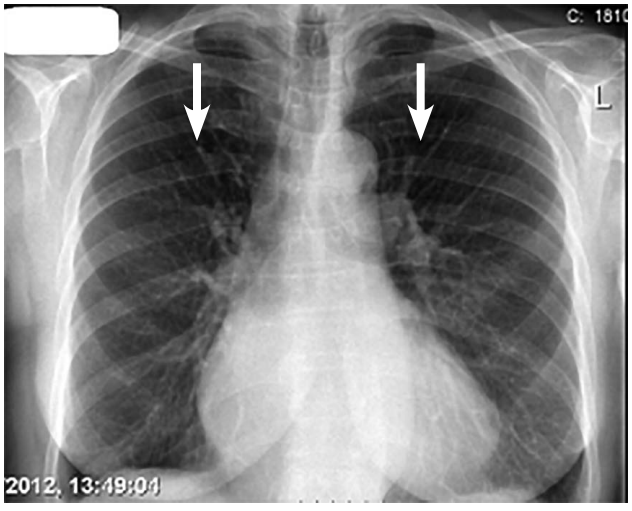
Assessment of left ventricular ejection fraction is generally the first part of a focused cardiac ultrasound examination. For the emergency clinician, this can start with a visual assessment that looks for the general quality of the heart's squeeze.[19] A more precise way to examine left ventricular ejection fraction is via E-point septal separation (EPSS), which measures the smallest distance between the tip of the mitral leaflet and the interventricular septum during diastole. **(See Figure 6, page 8.)** This distance is assessed using M-mode, with the indicator overlying the tip of the mitral leaflet. The larger this distance, the lower the ejection fraction. EPSS > 7 mm is indicative of poor left ventricular

Figure 2. Electrocardiogram Demonstrating an Acute Inferior STEMI



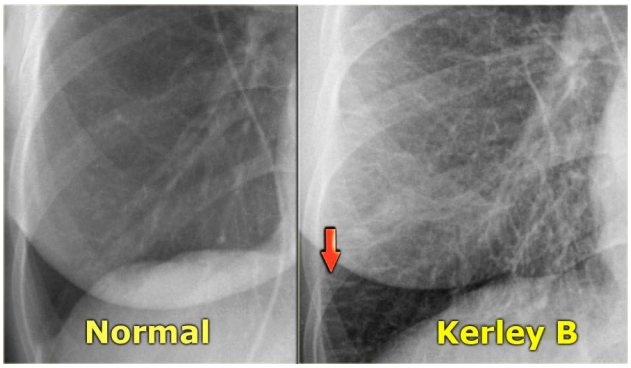
Note ST-segment elevation in II, III, and aVF.
Abbreviation: STEMI, ST-segment elevation myocardial infarction.
Source: http://lifeinthefastlane.com/ecg-library/basics/inferior-stemi/ Used with permission.

Figure 3. Mild Pulmonary Congestion Demonstrating Cephalization



Arrows indicate cephalized blood flow.
Case courtesy of Dr. Usman Bashir, Radiopaedia.org, rID: 18342
Source: http://radiopaedia.org/articles/upper-lobe-pulmonary-venous-diversion
Used with permission.

Figure 4. Pulmonary Congestion Demonstrating Kerley B-Lines



Source: http://www.radiologyassistant.nl/en/p4c132f36513d4
Used with permission from www.radiologyassistant.nl

ejection fraction.[22] Secko et al demonstrated a high degree of diagnostic accuracy using EPSS to diagnose heart failure using third- and fourth-year emergency medicine residents with minimal prior ultrasound experience.[23]

A more detailed analysis is required to evaluate for HFpEF, which is typically associated with both normal visual assessment of ventricular functioning and normal EPSS. To evaluate for diastolic dysfunction, the mitral annulus velocity is measured using an apical 4-chamber view. Diastolic dysfunction is indicated if there is slowing of the mitral annulus during the initial phase of rapid filling in early diastole.

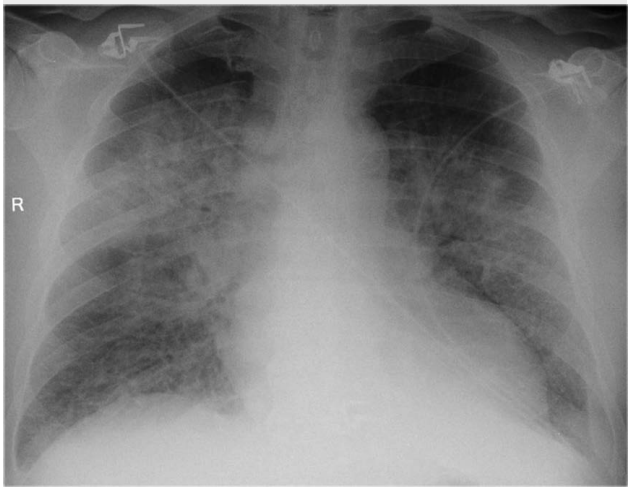
Bedside echocardiography can also be used to rule out alternative etiologies of the patient's acute decompensation, such as cardiac tamponade or valve rupture. Emergency clinicians with more advanced ultrasound techniques can often achieve a high degree of accuracy with echocardiography. Nevertheless, the combination of time constraints that often limit the extent of the examination and the generally lower quality of the smaller ultrasound machines found in many EDs (compared to those used by cardiologists for formal echocardiography) often make it necessary to pursue more complete echocardiography imaging in either radiology or cardiology despite ED bedside imaging. Despite these limitations, the bedside echocardiogram plays an important clinical role in the evaluation of the acutely dyspneic patient, particularly when the diagnosis is unclear.

Pulmonary Ultrasound

Pulmonary ultrasound is a newer application of this imaging modality that has been shown to be very accurate in recognizing pulmonary fluid. Interstitial pulmonary fluid is identifiable on ultrasound as vertical hyperechoic lines that arise from, and run perpendicular to, the pleura. These lines extend into the lung parenchyma and are referred to as B-lines. **(See Figure 7, page 9.)** The presence of 3 or more B-lines in at least 2 bilateral lung zones is indicative of pulmonary edema.[20] Liteplo et al demonstrated that the greater the number of zones demonstrating B-lines, the higher the likelihood of ADHF. This study showed greater sensitivity with an 8-zone ultrasound examination compared to a 2-zone examination, and it also demonstrated that even a more limited ultrasound examination, particularly in conjunction with brain-type natriuretic peptide (BNP) testing, had a high level of diagnostic accuracy.[24]

Compared to chest x-ray, pulmonary ultrasound has a greater degree of both sensitivity and specificity in diagnosing ADHF.[25,26] This modality can be used to distinguish pulmonary congestion from other etiologies (such as pneumonia) that may present with similar findings on chest x-ray.[27] Chiem et

Figure 5. Pulmonary Congestion With Cardiomegaly



Source: http://www.radiologyassistant.nl/en/p4c132f36513d4
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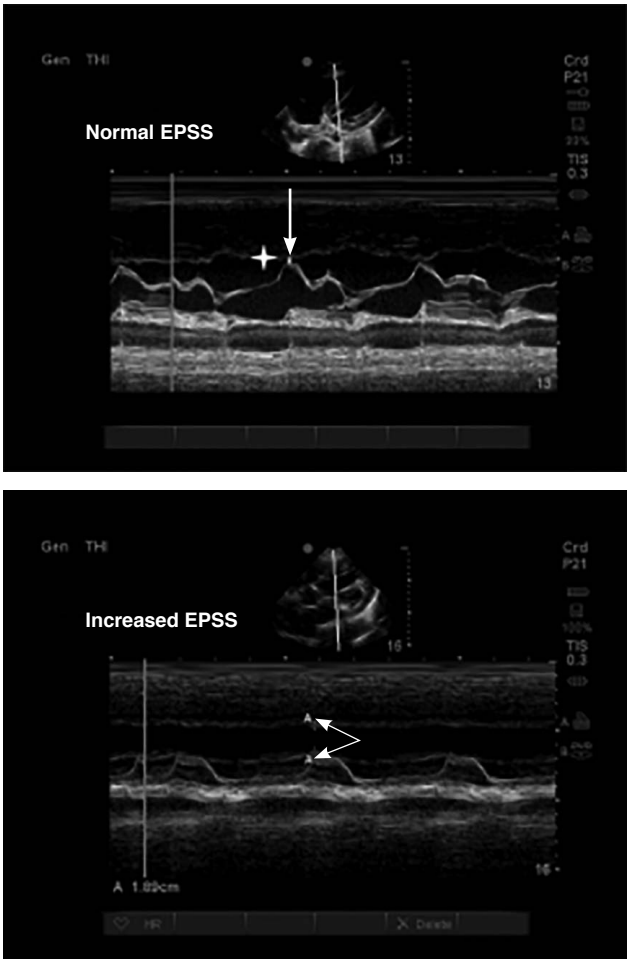
Figure 6. Normal and Increased E-point Septal Separation



Source: NYU/Bellevue EM Ultrasound. Used with permission.
Available at: http://www.nyuemsono.com/archives/1021

al performed a study comparing the findings of novice practitioners, who were provided with only 30 minutes of teaching, with those of expert emergency ultrasonographers. Both groups performed pulmonary ultrasounds looking for B-lines. The study demonstrated a high degree of agreement between the novice and expert practitioners.[28]

When performed by trained providers, the combination of echocardiography with pulmonary ultrasound has a high degree of accuracy in the diagnosis of ADHF. Gallard et al compared the diagnostic performance of bedside cardiopulmonary ultrasound performed by a trained emergency physician with standard diagnostic modalities including chest x-ray and N-terminal prohormone of brain-type natriuretic peptide (NT-proBNP) testing. Cardiopulmonary ultrasound demonstrated a 90% accuracy for the diagnosis of acute heart failure compared to 67% accuracy for clinical judgment alone, and 81% accuracy for clinical judgment combined with NT-proBNP testing and chest x-ray. On average, the cardiopulmonary ultrasound examination took 12 minutes to perform.[29]

Laboratory Testing

Cardiac Biomarkers

BNP is produced in the left ventricle in response to volume or pressure overload and counteracts the renin-angiotensin-aldosterone system, with a diuretic and vasodilatory effect.[30] BNP can be measured as an adjunct test in diagnosing both heart failure with preserved ejection fraction and reduced ejection fraction.

Troponin and BNP are useful to assess both severity and prognosis in cases of ADHF.[31-34] A BNP that is below the reference value has a high negative predictive value and is a useful test to rule out acute heart failure as the source of a patient's dyspnea. Although BNP testing is useful in the acute setting as an adjunct to clinical judgment in cases where the diagnosis is unclear, [35,36] its use alone in guiding therapy is less well established.[37] A higher BNP level in men is associated with a worse long-term prognosis, but this association has not been demonstrated in women.[38]

Troponin is useful for risk stratification of patients in the acute setting.[39] An elevated troponin level is evidence of cardiac myonecrosis and may result from cardiac strain, ischemia, or infarction in the setting of increased ventricular workload. Elevated troponin alone has been associated with increased in-hospital mortality. Patients with elevated troponin have been shown to have an 8% in-hospital mortality, while patients with a normal troponin have an in-hospital mortality of 2.7%.[40] Higher troponin is also associated with increased rates of rehospitalization and increased risk of death at 90 days post hospitalization.[41]

Complete Blood Cell Count

A complete blood cell count (CBC) should be checked on every patient presenting with ADHF. Severe anemia can contribute to the development of acute heart failure and should be addressed acutely with blood transfusion if the hemoglobin level is < 8 g/dL.[42] Diuresis may also be considered during blood transfusion in select patients believed to be at greater risk for acute volume overload.

Chemistry Panel

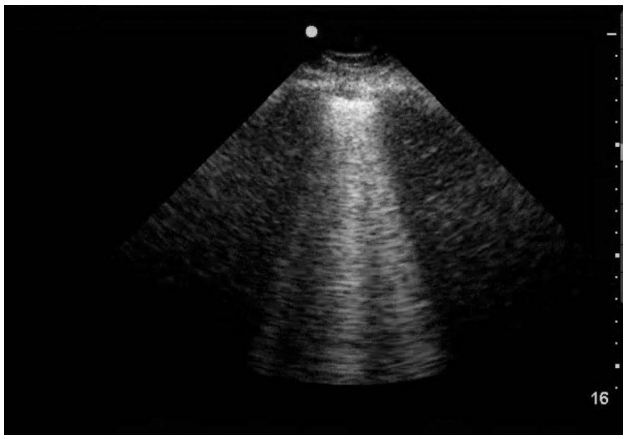
Acutely worsening renal function and acutely increased liver enzymes are both concerning for end-organ dysfunction. Although these findings are neither sensitive nor specific for acute heart failure, they are associated with poorer outcomes.[43-45]

Electrolytes and intravascular hydration status should also be assessed in patients with possible acute heart failure, as derangements of these are not uncommon and can affect outcomes. Low sodium can be a feature of advanced heart failure, and a low chloride level at admission is associated with adverse outcomes.[46]

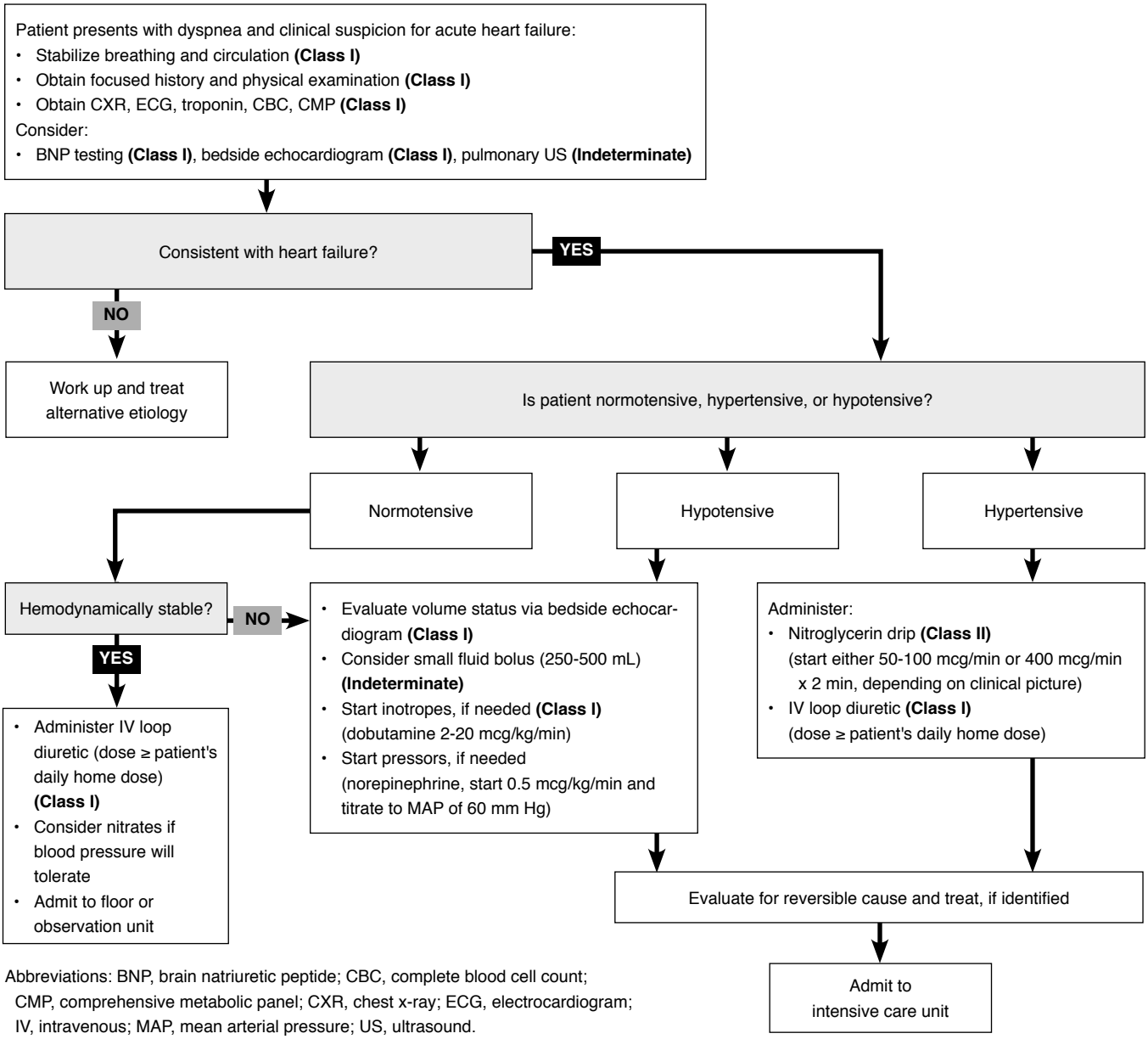
Supplementary Testing

Basic thyroid studies can be included in the evaluation of patients with unexplained or new heart failure to rule out thyroid derangements as the source of the patient's failure. These studies are not necessary in patients with a clear source of decompensation, but they can be useful in cases without an obvious cause. Additional studies for amyloidosis, pheochromocytoma, and rheumatologic diseases may be considered in cases where the cause of a patient's heart failure is unclear; however, these studies are usually performed outside of the ED, and though they should be considered, they do not impact the immediate patient workup and management.

Figure 7. Lung Ultrasound Demonstrating B-Lines



Clinical Pathway for Unstable Patients in Decompensated Heart Failure (Reduced and Preserved Ejection Fraction)



Abbreviations: BNP, brain natriuretic peptide; CBC, complete blood cell count; CMP, comprehensive metabolic panel; CXR, chest x-ray; ECG, electrocardiogram; IV, intravenous; MAP, mean arterial pressure; US, ultrasound.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Treatment

Airway and Ventilation Management

The most important initial management goal in patients with ADHF, regardless of etiology, is to ensure adequate oxygenation and ventilation. This may require supplemental oxygen, NIPPV, or, in severe cases, emergent endotracheal intubation with mechanical ventilation. Patients presenting with a room-air oxygen saturation < 90% should receive supplemental oxygen. Patients who are persistently hypoxemic on supplemental oxygen or who continue to exhibit significantly increased work of breathing require more aggressive intervention via positive-pressure ventilation, either invasive or noninvasive.

If the patient is an appropriate candidate, a trial of NIPPV via CPAP or bilevel positive airway pressure (BiPAP) should be attempted prior to intubation. These patients should be alert enough to participate in the care and delivery of NIPPV and cooperate with the intervention. NIPPV is patient-triggered, and the patient must be able to establish synchrony with the device. This can only be accomplished in an awake patient. In patients who are obtunded or apneic, the clinician should proceed directly to endotracheal intubation. NIPPV helps recruit functional alveoli by both preventing alveolar collapse and by expelling intra-alveolar fluid, thereby reducing the required work of breathing. For more information on the use of NIPPV, including absolute and relative contraindications, see the February 2017 issue of *Emergency Medicine Practice*, "Noninvasive Ventilation for Patients in Acute Respiratory Distress: An Update," at www.ebmedicine.net/NIV.

A retrospective analysis of 2430 patients with ADHF from the ADHERE registry (Acute Decompensated Heart Failure National Registry) showed improved outcomes for patients on NIPPV compared to those receiving endotracheal intubation. Additionally, delaying intubation for a trial of NIPPV did not appear harmful, as there were equivalent outcomes among this group compared with those who received immediate intubation.[47]

A randomized controlled trial by Sharon et al in 2000 that looked at 40 patients with acute cardiogenic pulmonary edema initially raised some concerns over the use of BiPAP. They randomized patients to receive either BiPAP with standard-dose nitrates or high-dose nitrates and standard oxygen administration via face mask. This study showed an increased rate of intubation, myocardial infarction, and death in the BiPAP group; however, because the study did not control for the nitrate dose, it is impossible to draw conclusions about the impact of BiPAP on outcomes.[48] A subsequent randomized controlled trial compared CPAP to BiPAP to standard oxygen therapy in patients with acute cardiogenic pulmonary edema. This trial showed equivalency between CPAP and BiPAP with improved subjective dyspnea, oxygenation, and respiratory rate over oxygen alone, with no increase in myocardial infarction in either the CPAP or BiPAP groups.[49] The use of NIPPV reduced mortality compared with standard therapy, prevented progression to intubation, and decreased intensive care unit (ICU) length of stay by about a day.[50] Current evidence does not reveal a significant difference in outcomes between CPAP and BiPAP, so either can be used in patients with dyspnea resulting from acute heart failure.[51]

If the patient worsens progressively, to the point of exhaustion despite use of NIPPV (as demonstrated by clinical examination or worsening blood gas results), definitive airway management is required via endotracheal intubation. Management of the patient's acute respiratory needs does not require an established diagnosis of acute heart failure and should be initiated regardless of the underlying etiology of the patient's dyspnea.

Drug Therapies

For a summary of drug therapies, including mechanism of action and evidence for use, see **Table 4, page 12.**

Vasodilators

Once the diagnosis of heart failure is made and the patient's respiratory status is stabilized, the subsequent intervention is dependent on whether the patient is hypertensive or hypotensive. Hypertensive patients require management primarily with vasodilators, usually via a nitroglycerin drip, which reduces afterload and improves the patient's cardiac functioning. Other vasodilators that can be considered in these patients include nesiritide and nitroprusside.[52,53] The use of nitroprusside has fallen out of favor in recent years due to the risk for cyanide toxicity and the need for invasive monitoring, but it has a more predictable effect on blood pressure than nitroglycerin.

Nitroglycerin

Nitroglycerin drips may be started at much higher doses in hypertensive heart failure patients, compared to patients presenting for chest pain, to rapidly counteract the patient's sympathetic overdrive and improve respiratory function.[54] If a patient presents in extremis with elevated blood pressure, starting doses of 50 to 100 mcg/min can be quickly titrated as high as 400 mcg/min. Alternatively, patients can be started at 400 mcg/min for 2 minutes, followed by a decrease to 100 mcg/min, with titration from there as needed.[54] Emergency clinicians may also choose to use sublingual nitroglycerin in combination with infusion to more rapidly establish therapeutic levels of nitrates and reduce blood pressure.

The emergency clinician should be at the bed-

side while very high doses of nitroglycerin are being administered, and most intravenous (IV) pumps will require manual programing, as they are not designed to give nitroglycerin at doses as high as 400 mcg/min.

Clevidipine

Clevidipine is an IV calcium-channel blocker that has been studied for efficacy in ADHF. In the open-label randomized controlled PRONTO trial, which included 104 patients, clevidipine was shown to achieve the target blood pressure goal faster than the standard of care.[55] However, this drug manufacturer-funded study was limited by its lack of a defined "standard of care," which varied among the participating institutions. Additionally, the doses of nitroglycerin used in the study were far lower than the recommended doses for this indication.

Angiotensin-Converting Enzyme Inhibitors

ACE inhibitors (such as captopril or enalapril) can also be considered in ADHF. These medications suppress the renin-angiotensin-aldosterone system that can ultimately contribute to the development of hypertensive ADHF. Older, small studies have examined the effect of adding an ACE inhibitor to the standard treatment of nitroglycerin with diuretics and have shown more rapid improvement in dys-

Table 4. Treatment Options in Acute Decompensated Heart Failure

Intervention/Agent	Mechanism	Evidence and Use
Vasodilators		
Nitroglycerin	Relaxation of vascular smooth muscle and dilatation of arterial and venous vascular beds	• Safety demonstrated over a wide dosing range with IV infusions • Initial range, 50-100 mcg/min up to brief periods of 400 mcg/min in appropriate patients[54]
Clevidipine	Intravenous calcium-channel blocker	• 1-2 mg/hr initial, max 16 mg/hr (up to 32 mg/hr limited) • Requires dedicated line[55]
Captopril, enalapril, enalaprilat	Angiotensin-converting enzyme inhibitors	• Rapid improvement of dyspnea; however, concern for later hypotension[56,57]
Nesiritide	Relaxation of isolated human arterial and venous tissue preparations that were precontracted with either endothelin-1 or the alpha-adrenergic agonist, phenylephrine	• Use with caution; risk of hypotension and worsening renal function[5,6,58,59]
Inotropes		
Digoxin	Inhibition the Na^+/K^+/ATPase pump, increasing intracellular calcium concentrations	• No improvement over placebo • Not recommended in the acute setting due to slow onset and loading phase[67,68]
Dobutamine	Beta-1 and beta-2 catecholamine agonist	• Increase in myocardial contractility and cardiac output initially but overall increases in tachycardia, ischemia, and dysrhythmias[71-73]
Norepinephrine	Alpha and beta catecholamine agonist	• Increases myocardial oxygen demand • Some benefit over dopamine in select patients with cardiogenic shock[74]
Milrinone	Phosphodiesterase inhibitor	• Increased incidence of dysrhythmias and hypotension; not recommended in the acute setting[75]
Diuretics		
Furosemide	Loop diuretic	• Dosing ≥ patient's normal daily dose in patients with evidence of intravascular overload
Novel Drug Therapies		
Levosimendan	Calcium sensitizer to increase cardiac contractility	• More rapid improvement over placebo, also increased risk of hypotension and dysrhythmias[88-91]
Ularitide	Human atrial natriuretic peptide	• Currently in Phase 2 clinical trials[97-100] • Cenderitide, a human atrial natriuretic peptide in clinical trials, was suspended from testing in February 2017
Omecamtiv mecarbil	Cardiac myosin activator	• Undergoing clinical trials and currently not available
Miscellaneous		
Ultrafiltration	Option among fluid-overloaded patients for whom diuresis fails to achieve adequate resolution of congestion	• The mechanism of fluid removal in ultrafiltration is similar to hemodialysis; however, it focuses on fluid removal rather than solute exchange

pnea secondary to pulmonary edema and improved hemodynamic parameters. However, evidence is limited and there is concern for precipitating hypotension, so use of an ACE inhibitor in the acute setting is not recommended at this time.[56,57]

Nesiritide

Nesiritide should be used with caution, as small studies have shown a nonsignificant trend toward increased risk of death in comparison with the control regimen. These studies have also failed to demonstrate benefit over standard therapy.[5,6] A meta-analysis looked at the dose-dependent effect of nesiritide on renal function and showed a loose association with worsening renal function at low doses but a strong association at high doses.[58] Nesiritide has a significant risk of hypotension and bradycardia in comparison to standard therapy.[59]

Vasodilator Therapy Recommendations

The literature on the use of IV vasodilators in ADHF is limited and most of it fails to examine patients in an acute setting.[60] A fundamental area that needs additional investigation is the use of high-dose IV nitrates in hypertensive patients. Current studies are small and of low quality and have failed to show any significant outcome benefit despite a general clinical consensus of its efficacy. Newer treatment modalities have even weaker evidence supporting their use, but nitrate therapy seems to be most lacking in rigorous study, given its ubiquitous use in EDs. Despite nitrates having yet to be definitively shown to improve mortality, they have been shown to be safe and, anecdotally, to rapidly improve symptoms in hypertensive patients with ADHF. On this basis, IV nitrate therapy is recommended in most clinical practice guidelines, including those offered by the American College of Emergency Physicians.[61]

Inotropes

Hypotensive patients with ADHF offer a unique management challenge. These patients suffer from decreased cardiac contractility at baseline, but at the same time, they can also be intravascularly volume depleted, further contributing to hypotension and decreased perfusion. The decision to add an inotropic agent may seem to be a logical intervention in a hypotensive patient, but studies examining these agents have not produced consistent results.[62-64] In patients with an SBP > 80 mm Hg, inotropes provide no benefit, and pose potential for harm.[65] Most patients admitted with acute heart failure will not require the addition of an inotropic agent and may benefit from small fluid boluses to optimize intravascular volume, with close monitoring for fluid responsiveness. Patients who fail to respond to fluids and remain hypotensive with an SBP < 80 mm Hg are in true cardiogenic shock and may require inotropic support to improve perfusion (American Heart Association Class I, Level C recommendation).[66]

Digoxin

Digoxin is a cardiac glycoside that inhibits the Na^+/K^+/ATPase pump, preventing the movement of sodium into the extracellular space. With a lower transmembrane gradient, the activity of the Na^+/Ca^{2+} pump is reduced, thereby raising the intracellular calcium levels. The increased intracellular calcium is thought to be responsible for not only the inotropic but also the arrhythmogenic effects of the cardiac glycosides. Digoxin provides inotropic support without adverse effects on heart rate or blood pressure. However, the Digitalis Investigation Group demonstrated no improvement in survival over placebo. Because of the lack of demonstrated improved outcomes, combined with the need to load the drug slowly and its slow onset, digoxin does not have any significant role in the acute setting.[67,68]

Dopamine

Dopamine is a catecholamine with a dose-dependent effect. Lower doses (0.5-3 mcg/kg/min) of dopamine cause vasodilation and increase both coronary and renal blood flow. However, despite improved renal blood flow, low-dose dopamine has not been shown to improve renal function in patients with ADHF.[69,70] Intermediate doses of dopamine (3-10 mcg/kg/min) have positive inotropic effects but with a harmful increase in pulmonary capillary wedge pressure, and it should not be used in patients with acute heart failure. Higher doses (10-20 mcg/kg/min) of dopamine result in vasoconstriction that increases afterload, which is harmful in patients with cardiac dysfunction. There is very little evidence to support the use of dopamine, at any dose, in patients with ADHF.

Dobutamine

Dobutamine is a catecholamine with an agonist effect on beta-1 and beta-2 adrenergic receptors. Dobutamine increases myocardial contractility and cardiac output and decreases left ventricular end-diastolic pressure, but may produce a slight increase in heart rate.[71] Studies have demonstrated improvement in heart failure symptoms with dobutamine, but with ultimately increased mortality.[72] The effectiveness of dobutamine is inhibited by beta blockade, so alternative inotropic support is preferred in patients on beta blockers.[73] Infusions of dobutamine over 72 hours are associated with tolerance, tachycardia, ischemia, and dysrhythmias at higher doses.

Norepinephrine

Norepinephrine is used for refractory hypotension despite management with an appropriate inotro-

pic agent, such as dobutamine, or for patients who would not be appropriate candidates for dobutamine, such as those on chronic beta blockade. Norepinephrine has both alpha- and beta-agonist activity and causes both increased inotropic and chronotropic activity as well as peripheral vasoconstriction. Norepinephrine can increase the heart rate, which may be harmful in patients with coronary ischemia due to an increased myocardial oxygen demand. De Backer et al, in a large randomized controlled trial, demonstrated a lower 28-day mortality in patients with cardiogenic shock who were treated with norepinephrine in comparison with dopamine, with a decreased rate of dysrhythmias.[74] However, all inotropes must be used with caution, as they cause increased stress on the already ailing heart.

Milrinone

Milrinone is a phosphodiesterase inhibitor that has a similar effect to dobutamine, but it bypasses beta blockade. It was initially investigated as a newer potential therapy for patients in decompensated heart failure; however, a multicenter randomized controlled trial of 951 patients demonstrated an increased incidence of cardiac dysrhythmias and hypotension without any improvement in length of hospitalization, in-hospital mortality, or 30-day mortality, or risk of readmission. Use of milrinone is not recommended in the acute setting.[75]

Inotrope Therapy Recommendations

Based on the best available evidence, patients with ADHF and an SBP < 80 mm Hg that does not respond to fluids should be started on either dobutamine or norepinephrine. Lower dosing of inotropic agents is advocated to ensure adequate peripheral perfusion and prevent end-organ dysfunction.[63,76,77]

Diuretics

Once the acute heart failure patient's airway and overall respiratory status has been stabilized, IV loop diuretics, such as furosemide, may be administered with the goal to improve the patient's pulmonary vascular congestion. Bumetanide can be considered in the place of furosemide for patients who are known to be resistant or allergic to furosemide. No clear, best dose has been identified, despite studies looking at lower versus higher doses and drips versus bolus dosing.[78-80] Patients with heart failure who demonstrate symptoms of fluid overload should be treated with IV diuretics early in their ED stay.[81] The dose of IV diuretic given should be either equal to or greater than the patient's daily oral dose of loop diuretic (if the patient is already receiving a loop diuretic).[78] Diuretics do not take effect as quickly as the other interventions discussed previously, but they do play an important role in the treatment of failure-induced fluid overload.

Mechanical Circulatory Support

If cardiogenic shock persists despite the administration of inotropes and pressors, mechanical circulatory support can be considered, if available. Options include an intra-aortic balloon pump, ECMO, and left ventricular assist device placement. These options are not well studied in randomized controlled trials due to the small populations available to study, but they may be considered as rescue therapy in critically ill patients in consultation with cardiology. Mechanical circulatory support has been shown, through small cohort studies, to be a feasible option for salvage therapy in very sick patients.[82,83]

Further Management

ACS may cause ADHF and should be treated with urgent medical management and revascularization.[84] Unfortunately, it can be difficult to distinguish demand ischemia secondary to heart failure from true ACS. For the emergency clinician, consulting cardiology early in the management of cases with ischemic ECG changes is crucial to ensuring that patients with coronary vascular insufficiency are appropriately considered for endovascular interventions.

For patients with cardiogenic shock secondary to ACS, the Shock Trial showed a 67% relative improvement in long-term survival, measured at 6 years, for patients managed with rapid revascularization.[85] The role of revascularization is not clear for patients presenting with failure without obvious acute ischemia. An observational study looked at patients presenting with acute heart failure who received coronary angiography. It showed that, in their group, 27% of patients who received angiography required revascularization, and the intervention reduced rates of both mortality and rehospitalization.[86] This study is limited by its observational design and lack of randomization, but it offers an interesting perspective in terms of the potential utility of angiography in patients presenting with heart failure.

Inpatient management for patients with ADHF focuses on maximizing medical management, including lipid control and initiating an ACE inhibitor, a beta blocker, and a diuretic. Interventions that are considered for patients with more severe heart failure include aldosterone antagonists (eg, spironolactone) and AICD placement.[87]

Controversies and Cutting Edge

Novel Drug Therapies

Levosimendan

Levosimendan is a new medication being studied that is considered a "calcium-sensitizer," and is infused intravenously. It has been approved for use in Europe, but not yet in the United States. Levosimendan works by increasing myocardial contractility by sensitizing the cardiac myocytes to calcium and

causing vasodilatation. In the REVIVE study that examined 600 patients with ADHF, levosimendan, when added to standard therapy, resulted in a more rapid symptomatic improvement when compared to placebo with standard therapy; however, there was an increased risk of hypotension and dysrhythmias associated with its administration.[88] This study was funded by the drug manufacturer, and subsequent studies have not demonstrated improvement in mortality or rehospitalization when levosimendan was compared to dobutamine.[89-91]

Jia et al studied the combination of levosimendan with nesiritide in 120 patients and demonstrated that the combination provided benefit over either therapy alone and over placebo in terms of clinical effectiveness. Unfortunately, combining the therapies made it difficult to determine whether a single agent or only the combination was primarily responsible for the improvement.[92] A more in-depth investigation to compare levosimendan to more traditional lower-cost interventions is needed. However, levosimendan is currently a consideration in patients in whom dobutamine is not indicated (ie, patients on chronic beta blockade).

Serelaxin

Another new pharmaceutical under investigation is serelaxin (RLX030), which is composed of recombinant human relaxin-2. Relaxin is a vasoactive peptide that serves as a vasodilator, in addition to stimulating cardiac remodeling.[93] Serelaxin is administered via 48-hour infusion. In the RELAX-AHF study (a 1161-person drug manufacturer-funded study), serelaxin was shown to improve symptomatic dyspnea when added to standard therapy, but showed no effect on hospital readmission or cardiovascular death.[94,95] It has shown similar effectiveness on patients with reduced ejection fraction (versus preserved ejection fraction).[96] The United States Food and Drug Administration rejected fast-track approval for serelaxin in 2014, citing the need for additional studies to evaluate the drug's efficacy. The large follow-up RELAX-AHF-2 study recently failed to meet its primary endpoints. It is unclear whether additional studies will be performed, but this drug is unlikely to play any significant role in the management of ADHF in the foreseeable future.

Human Natriuretic Peptides

Human atrial natriuretic peptide has also been examined through several randomized controlled trials. While outcomes demonstrated an improvement in hemodynamic parameters, they have shown no improvement in mortality.[97] Ularitide, a synthesized human natriuretic peptide similar in properties to nesiritide, is undergoing Phase 2 clinical trials, but studies are not yet far enough along to determine any beneficial effects.[98-100]

Omecamtiv Mecarbil

Omecamtiv mecarbil, a cardiac myosin activator, is also undergoing clinical trials to determine whether its initially demonstrated effect on cardiac contractility will be reflected in any improvement in symptoms or outcomes among patients with ADHF.[102]

Ultrafiltration

Ultrafiltration is an option for fluid-overloaded patients for whom diuresis fails to achieve adequate resolution of congestion. The mechanism of fluid removal in ultrafiltration is similar to hemodialysis; however, it focuses on fluid removal rather than solute exchange. Ultrafiltration can be accomplished through a smaller-diameter catheter than hemodialysis, but it generally requires a peripherally inserted central catheter (PICC) line. Ultrafiltration is mainly beneficial when patients have become resistant to diuretic therapy, but the presence of diuretic resistance is often not known during initial management in the ED. The UNLOAD trial evaluated ultrafiltration versus IV diuretic therapy in patients with functioning kidneys, and it demonstrated that ultrafiltration removes a larger volume of fluid and is associated with a greater reduction in 90-day resource utilization compared to diuretic therapy. In this study, ultrafiltration was also determined to be safe, with no increased incidence of adverse outcomes.[103-106] Nonetheless, ultrafiltration is more invasive and more expensive than medical diuresis and is often unnecessary in patients with functioning kidneys unless attempted diuresis has failed. For this reason, ultrafiltration is generally initiated after admission once a patient's responsiveness to the diuretic administered in the ED has been assessed.

Novel Biomarkers

Novel biomarkers are being actively investigated to determine what role they may play for both diagnosis and prognosis of patients with ADHF. Syndecan 1, which is still experimental and not yet available in hospitals for laboratory testing, was shown in a small single-center study to be predictive of both acute kidney injury and in-hospital mortality,[107] but larger multicenter trials are needed to more accurately determine its utility.

Disposition

Patients presenting with ADHF generally require admission to the hospital for hemodynamic optimization, volume regulation, and adjustment of their outpatient medications. Only patients with mild symptoms and reliable outpatient follow-up are considered for discharge home following treatment in the ED. Patients with any significant vital sign abnormalities or respiratory difficulties as well as those with evidence of cardiac strain are generally

admitted to the hospital for cardiac monitoring and diuresis. These patients are typically admitted to a telemetry bed, and they may require admission to a floor with greater monitoring by nursing, depending on the severity of their disease.

Due to their risk of further deterioration, patients with peripheral hypoperfusion, hypotension, significant acute renal dysfunction, respiratory failure requiring NIPPV or intubation, need for inotrope infusions or nitroglycerin drips, and those with presumed ACS typically require more intensive monitoring on admission in an ICU.

Time- and Cost-Effective Strategies

- Rapid diagnosis followed promptly by initiation of appropriate therapy is the best way to reduce both costs and length of stay. Heart failure is largely a clinical diagnosis. Adjunctive tests are mainly helpful in patients whose diagnosis is unclear and to look for any underlying cause of the patient's decompensation. BNP testing can help pinpoint the diagnosis in patients with an unclear etiology of their dyspnea, thereby reducing the length of hospitalization and costs.[109]
- By starting appropriate care as soon as possible (and often immediately on arrival), deterioration that requires longer stays and more invasive and expensive interventions may be prevented.
- Using NIPPV can save on costs by avoiding the more invasive intervention of intubation. In appropriate patients, avoidance of intubation helps reduce hospital stay.
- The mainstays of medication management of ADHF are generally the older, less expensive medications. While newer medications are on the horizon, starting management with older medications and then moving on to the more expensive interventions, if necessary, is a good way to reduce costs of care.

Summary

ADHF presents with a variety of clinical symptoms ranging from lower extremity swelling to frank respiratory distress, and patients presenting in extremis require rapid and aggressive intervention to prevent deterioration and death. The management of ADHF begins with airway management and respiratory support, with the level of intervention dependent on the patient's severity. These interventions may range from supplemental oxygen via nasal cannula up to NIPPV or intubation with mechanical ventilation.

Medication management is dependent on the patient's symptoms and hemodynamics. Patients believed to be experiencing volume overload are candidates for diuresis using a loop diuretic, often in combination with other therapies. Hypertensive patients with respiratory distress require aggressive vasodilation with a high-dose nitroglycerin drip to lower afterload and improve cardiac functioning. Hypotensive patients can be given a trial of small fluid boluses to evaluate whether intravascular volume may actually be depleted. Hypotensive patients who are unresponsive to fluids require blood pressure support via inotropes to increase the heart's ability to pump. Dobutamine is the first-line agent for patients in cardiogenic shock, but it is ineffective for any patients with baseline beta blockade. Patients on beta blockers should be started on norepinephrine as their first-line agent for blood pressure support.

Workup should focus on determining potentially reversible causes of the patient's acute decompensation, such as cardiac ischemia or valvular dysfunction, and on evaluating for end-organ dysfunction. Patients requiring aggressive interventions are admitted to the hospital for close monitoring, and some will require admission to the ICU.

Case Conclusions

Your first patient, the 76-year-old woman with hypotensive heart failure, was initially given a small fluid bolus without any response; her blood pressure remained low and her breathing remained labored. A bedside echo showed a poor ejection fraction, and her chest x-ray demonstrated bibasilar infiltrates with cephalization. A central line was placed, and you started her on dobutamine, with improvement in her peripheral perfusion and respiratory status. She was admitted to the cardiac ICU for her decompensated heart failure, and prior to hospital discharge, she was given a left ventricular assist device to support her cardiac function.

Your second patient, the middle-aged man with hypertensive decompensated heart failure with acute pulmonary edema, was started immediately on BiPAP to support his breathing, and he responded well. Bedside pulmonary ultrasound showed B-lines, confirming the diagnosis of pulmonary edema. He was started on a high-dose nitroglycerin drip, which resulted in a significant improvement in his respiratory symptoms. He received IV diuresis and was admitted to the ICU for further management.

Risk Management Pitfalls for Management of Acute Decompensated Heart Failure

1. **"The patient looked comfortable, so I didn't immediately check his pulse oximetry."**
   Vital signs are truly vital in these patients. Blood pressure and pulse oximetry must be checked immediately. A low pulse oximetry level requires immediate intervention with supplemental oxygen or respiratory support.

2. **"I wasn't sure what was going on, but I didn't know how to use the ultrasound machine."**
   Bedside echocardiogram is a crucial diagnostic tool to help confirm the diagnosis of heart failure. The onus is on the emergency clinician to learn to use ultrasound as a diagnostic tool for diagnosis of heart failure and other ED presentations.

3. **"The patient was short of breath, so I started treatment for heart failure."**
   There are many diagnostic entities that can cause dyspnea and mimic the symptoms of heart failure. Particularly in obese patients who are poorly conditioned at baseline, many other causes of dyspnea can be mistaken for heart failure. Examples include pulmonary embolism, pneumonia, pericardial effusion, and COPD. Treatment can be started quickly if the diagnosis is clear, but alternative etiologies should be actively sought.

4. **"The patient was wheezing, so I knew it was COPD and did not worry about heart failure."**
   While heart failure traditionally presents with rales on the pulmonary examination, cardiac wheezes can also occur and are not necessarily indicative of obstructive pulmonary disease as the primary etiology. Additionally, patients may have coexisting obstructive pulmonary disease and heart failure, which can complicate the clinical picture.

5. **"There was a focal infiltrate on the chest x-ray, so I knew it couldn't be heart failure."**
   Patients can present with multiple coexisting etiologies, and heart failure can be exacerbated by secondary assaults, such as sepsis. Additionally, pulmonary congestion can present as bilateral consolidation on chest x-ray.

6. **"The patient had chest pain and obvious heart failure, so we treated for heart failure but did not do any additional diagnostic testing."**
   Don't miss secondary causes of heart failure. Coronary ischemia, pulmonary embolism, and pericardial effusion can all contribute to the heart's inability to pump effectively. These secondary reversible or treatable causes are crucial to identifying and effectively managing these patients.

7. **"The patient was hypotensive but had a history of heart failure, so I did not give any IV fluids."**
   Patients with heart failure can be intravascularly depleted and may require gentle fluid resuscitation in the setting of hypotension. The fluids should be given in smaller aliquots, but fluid should not be withheld in these patients, particularly in hypotensive patients who do not look grossly fluid overloaded.

8. **"I wasn't sure what to do for my unstable patient, so I just gave furosemide and waited."**
   These patients respond well to aggressive, early interventions. Failure to quickly intervene can allow clinical deterioration, requiring more-invasive airway and circulatory support. Furosemide is an inadequate sole intervention in an unstable patient.

9. **"My patient was having difficulty breathing, so I immediately intubated."**
   While the decision to intubate is always a clinical one, a trial of NIPPV is often appropriate in patients with ADHF. NIPPV is a useful temporizing measure that can stabilize the patient until more definitive interventions have taken effect. The appropriate contraindications for NIPPV should always be considered, but in the appropriate patient, it can be a useful adjunct.

10. **"My patient was looking much better on the nitroglycerin drip, so I weaned her off and discharged her home."**
    Heart failure patients have a high risk of clinical deterioration and require close observation. Patients requiring nitroglycerin or pressor drips require ICU-level care and close monitoring. Any patient with respiratory symptoms should be admitted for diuresis and close observation.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study is included in bold type following the reference, where available. The most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. **Which of the following would be indicative of an alternative cause of dyspnea rather than heart failure?**
   a. Fever
   b. Fatigue
   c. Dyspnea with exertion
   d. Lower extremity swelling

2. **Which of the following is the most severe sign of fluid overload on a chest x-ray?**
   a. Kerley B-lines
   b. Cephalization
   c. Bilateral perihilar consolidation
   d. Cardiomegaly

3. **Which of the following can be a laboratory finding in advanced heart failure?**
   a. Hyponatremia
   b. Hypernatremia
   c. Hypokalemia
   d. Hyperkalemia

4. **Regarding noninvasive positive-pressure ventilation, which of the following is TRUE?**
   a. It has not been shown to reduce in-hospital mortality.
   b. It reduces the rate of progression to intubation.
   c. BiPAP is more effective than CPAP.
   d. It should be started on every patient presenting with respiratory distress.

5. **Which of the following interventions has been clearly demonstrated in the medical literature to improve outcomes for patients with ADHF?**
   a. Furosemide
   b. Nitroglycerin
   c. Dobutamine
   d. Extracorporeal membrane oxygenation

6. **The use of IV nitroglycerin in hypertensive patients with acute heart failure is:**
   a. Poorly studied and not recommended
   b. Poorly studied but recommended
   c. Well-studied and not recommended
   d. Well-studied and recommended

7. **Which following medications is commonly used in patients with heart failure?**
   a. Dopamine
   b. Sulfa antibiotic
   c. ACE inhibitor
   d. Albuterol

8. **Which of the following drugs can be used in patients with acute decompensated heart failure with low blood pressure?**
   a. Nitroprusside
   b. Nesiritide
   c. Nitroglycerin
   d. Dobutamine

9. **A patient with decompensated heart failure with edema and elevated jugular venous distension presents to the ED. He is prescribed furosemide 40 mg daily at home. What dose of diuretic should be initiated?**
   a. Furosemide 40 mg orally
   b. Furosemide 20 mg IV
   c. Furosemide 60 mg IV
   d. Furosemide should not be used, as the patient is resistant to the medication

10. **A patient with decompensated heart failure with underlying chronic kidney disease is unresponsive to diuretic therapy. What adjunctive therapy should be used to eliminate excess fluid?**
    a. IV nitroglycerin
    b. Left ventricular assist device placement
    c. Cardiac transplantation
    d. Ultrafiltration

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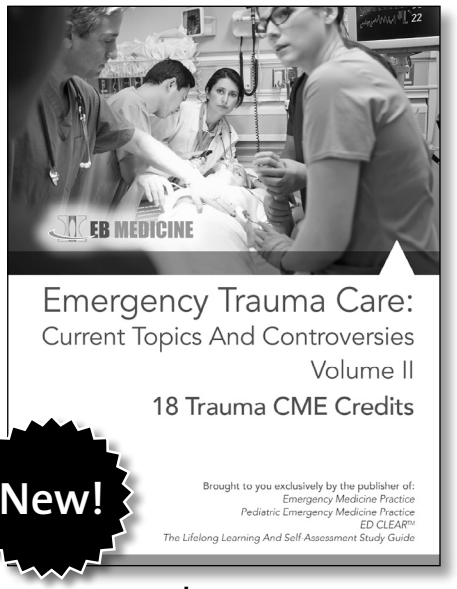

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