Chapter 53: Acute Heart Failure

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INTRODUCTION AND EPIDEMIOLOGY

Acute heart failure covers a wide spectrum of illness, ranging from a gradual increase in leg swelling, shortness of breath, or decreased exercise tolerance to the abrupt onset of pulmonary edema. While alternative terms such as *decompensated heart failure*, *acute heart failure syndrome*, or *hospitalized with heart failure* have been used nearly interchangeably over the last decade, we refer to patients with either an acute exacerbation of chronic heart failure or a new-onset heart failure as having *acute heart failure*. The term *congestive heart failure* is outdated and describes patients with signs and symptoms of fluid accumulation.

Most ED visits for acute heart failure result in hospital admission.[1] With the aging population, increased survival from acute myocardial infarction, and evidence-based outpatient treatment options, the prevalence of heart failure is expected to increase over the next decade.[2,3,4] ED physicians drive most disposition decisions.[5,6] There have been tremendous advances in outpatient management of heart failure patients. While long-term heart failure management has improved through the use of β-blockers, angiotensin-converting enzyme inhibitors, spironolactone, and cardiac resynchronization therapy,[2,3] acute therapy is largely unchanged. Acute therapies include nitrates, diuretics, and positive-pressure ventilation, the same as in 1974.[7] Only one therapy, nesiritide, has been approved for heart failure treatment in the last three decades, but it is not significantly better than standard treatment.[8]

Heart failure has a poor prognosis, with approximately 50% of patients diagnosed dying within 5 years.[9] Hospitalization also marks an inflection point in a patient's HF trajectory, with those hospitalized having higher mortality than a matched nonhospitalized cohort.[10]

PATHOPHYSIOLOGY

Heart failure is a complicated syndrome manifested by cardinal symptoms (shortness of breath, edema, and fatigue) occurring from functional or structural cardiac damage, impairing the ability of the heart to act as an efficient pump. A clinically useful definition of heart failure is as follows: a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood. The cardinal manifestations of heart failure are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, which may lead to pulmonary and/or splanchnic congestion and/or peripheral edema.[2] There are numerous responsive adaptations in the kidney, peripheral circulation, skeletal muscle, and other organs to maintain short-term circulatory function. Eventually, these responses may become maladaptive, contribute to long-term disease progression, and contribute to acute exacerbations.

Threats to cardiac output from myocardial injury or stress trigger a neurohormonally mediated cascade that includes activation of the renin-angiotensin-aldosterone system and the sympathetic nervous systems. Levels of norepinephrine, vasopressin, endothelin (a potent vasoconstrictor), and tumor necrosis factor-α are increased. Although not measured in routine care, elevated levels of these hormones correlate with higher mortality.

The combined clinical effects of neurohormonal activation are sodium and water retention coupled with increased systemic vascular resistance. These maintain blood pressure and perfusion, but at the cost of increasing myocardial workload, wall tension, and myocardial oxygen demand. Although some patients are initially asymptomatic, a secondary pathologic process called cardiac remodeling begins to occur, eventually triggering more dysfunction.

Natriuretic peptides are the endogenous counterregulatory response to neurohormonal activation in heart failure. Three types are recognized: atrial natriuretic peptide, primarily secreted from the atria; B-type natriuretic peptide, secreted mainly from the cardiac ventricle; and C-type natriuretic peptide, localized in the endothelium. Natriuretic peptides produce vasodilation, natriuresis, decreased levels of endothelin, and inhibition of the renin-angiotensin-aldosterone system and the sympathetic nervous systems. B-type natriuretic peptide is synthesized as N-terminal pre–pro-B-type natriuretic peptide, which is cleaved into two substances, inactive N-terminal pro-B-type natriuretic peptide, with a half-life of approximately 2 hours, and physiologically active B-type natriuretic peptide, with a half-life of about 20 minutes. Assays for both B-type natriuretic peptide and N-terminal pro-B-type natriuretic peptide are available for ED use. Because elevated levels of neurohormones portend a worse prognosis in heart failure, their attenuation provides the basis for most chronic therapies proven to delay heart failure morbidity and mortality. These include treatment with angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, aldosterone antagonists, and β-blockers.

Heart failure may also result from pump dysfunction from acute myocardial infarction. Mechanistically, loss of a critical mass of myocardium r[illegible] is symptomatic hypotension with inadequate perfusion, cardiogenic shock is present (see chapter 50 "Cardiogenic Shock"). Acute pulmonary edema may be precipitous and is the clinical manifestation of a downward spiral of rapidly decreasing

cardiac output and rising systemic vascular resistance on top of underlying cardiac dysfunction. Even relatively small elevations of blood pressure can result in decreased cardiac output. Decreasing cardiac output triggers increasing systemic vascular resistance, which further decreases cardiac output. Acute pulmonary edema can present acutely with severe symptoms, and if not promptly reversed, it may be a terminal event.

ACUTE HEART FAILURE CLASSIFICATION

There are many causes for heart failure (Table 53-1).

TABLE 53-1

Common Causes of Heart Failure and Pulmonary Edema

Myocardial ischemia: acute and chronic*
Systemic hypertension*
Cardiac dysrhythmias (especially atrial fibrillation with rapid ventricular response)*
Valvular dysfunction
Aortic valve disease
Aortic stenosis
Aortic insufficiency
Aortic dissection
Infectious endocarditis
Mitral valve disease
Mitral stenosis
Mitral regurgitation
Papillary muscle dysfunction or rupture
Ruptured chordae tendineae
Infectious endocarditis
Prosthetic valve malfunction
Other causes of left ventricular outflow obstruction
Supravalvular aortic stenosis
Membranous subvalvular aortic stenosis
Cardiomyopathy*
Hypertrophic cardiomyopathy
Dilated†
Restrictive

Toxic: alcohol, cocaine, doxorubicin

Metabolic: thyrotoxicosis, myxedema

Myocarditis: radiation, infection

Constrictive pericarditis

Cardiac tamponade

Anemia

*Seen in the ED with higher frequency.

†Includes idiopathic (see chapter 55, "Cardiomyopathies and Pericardial Disease").

Patients can be categorized into six phenotypes to assist with investigating the causes and precipitants for the acute presentation, as well as directing initial therapy (**Table 53-2**).[11] Those with acute heart failure and hypertension often have a precipitous presentation and may have significant pulmonary edema and hypoxia. Symptoms may be due to fluid redistribution more than fluid overload, and treatment initially focuses on antihypertensive therapy.[12,13] Pulmonary edema may benefit from noninvasive ventilation to decrease the work of breathing and avoid intubation.[14,15] For heart failure accompanied by hypotension or poor perfusion without another cause, think of an ischemic or structural heart trigger creating cardiogenic shock; patients often benefit from inotropic agents and invasive hemodynamic monitoring to guide other therapies.

TABLE 53-2

Classification of Acute Heart Failure

Classification	Characteristics
Hypertensive AHF	Signs and symptoms of AHF with relatively preserved left ventricular function, systolic blood pressure >140 mm Hg, typically with a chest radiograph compatible with pulmonary edema and symptom onset less than 48 h
Pulmonary edema	Respiratory distress, rales on chest auscultation, reduced oxygen saturation from baseline, verified by chest radiograph findings
Cardiogenic shock (see chapter 50)	Evidence of tissue hypoperfusion (systolic blood pressure typically <90 mm Hg)
Acute-on-chronic HF	Signs and symptoms of AHF that are mild to moderate and do not meet criteria for hypertensive HF, pulmonary edema, or cardiogenic shock, systolic blood pressure <140 mm Hg and >90 mm Hg, typically associated with increased peripheral edema and symptom onset over several days
High-output failure	High cardiac output, typically with tachycardia, warm extremities, and pulmonary congestion
Right heart failure	Low-output syndrome with jugular venous distention, hepatomegaly, and may have hypotension

Abbrevitions: AHF = acute heart failure; HF = heart failure.

Patients with acute-on-chronic heart failure tend to present with gradual symptoms and weight gain over days to weeks. High-output heart failure is distinguished by a relatively normal ejection fraction and is often caused by anemia or thyrotoxicosis. Isolated right heart failure is c a and jugular venous distension but little or no pulmonary congestion, and the cause is usually from

pulmonary disease, valvular disease such as tricuspid regurgitation, or obstructive sleep apnea. Treatment approaches center on identifying and treating the underlying cause, often without volume removal because low-output states may coexist.

SYSTOLIC AND DIASTOLIC HEART FAILURE

Heart failure is classified as systolic or diastolic by ejection fraction, which is normally 60%. **Systolic dysfunction**, or heart failure with reduced ejection fraction, is defined as an ejection fraction <50%. Mechanistically, the ventricle has difficulty ejecting blood, leading to increased intracardiac volume and *afterload sensitivity.* With circulatory stress (e.g., walking), failure to improve contractility despite increasing venous return results in increased cardiac pressures, pulmonary congestion, and edema.

Diastolic dysfunction, or heart failure with is preserved ejection fraction, is characterized by impaired ventricular relaxation, causing an abnormal relation between diastolic pressure and volume. This results in a left ventricle that has difficulty receiving blood. Decreased left ventricular compliance necessitates higher atrial pressures to ensure adequate left ventricular diastolic filling, creating a *preload sensitivity*. The frequency of diastolic dysfunction increases with age and is more common in chronic hypertension, which leads to left ventricular hypertrophy. Coronary artery disease also contributes, as diastolic dysfunction is an early event in the ischemic cascade.

DIAGNOSIS

Most hospitalized patients with heart failure are admitted through the ED. Commonly, patients will present with dyspnea, which has a large differential diagnosis including heart failure, chronic obstructive pulmonary disease, asthma, pneumonia, and acute coronary syndrome. Misdiagnosis increases mortality, prolongs hospital stay, and increases treatment costs.[16,17,18,19,20] Table 53-3 lists common causes of dyspnea in ED patients. ***There is no single diagnostic test*** for heart failure; it is a clinical diagnosis based on the history and physical examination. Having an understanding of the diagnostic certainty regarding the history, physical examination, and laboratory and radiographic testing is extremely important when caring for ED patients with undifferentiated dyspnea.

TABLE 53-3

Common Causes of Dyspnea

Dyspneic states
Heart failure
Asthma exacerbation
Chronic obstructive pulmonary disease exacerbation
Pleural effusion
Pneumonia or other pulmonary infection
Pneumothorax
Pulmonary embolus
Physical deconditioning or obesity
Fluid retentive states
Dependent edema or deep vein thrombosis
Hypoproteinemia
Liver failure or cirrhosis
Portal vein thrombosis
Renal failure or nephrotic syndrome
Impaired cardiac output states
Acute myocardial infarction
Acute valvular insufficiency
Drug overdose/effect
Dysrhythmias
Pericardial tamponade
Tension pneumothorax
High-output states
Sepsis
Anemia
Thyroid dysfunction

HISTORY AND PHYSICAL EXAMINATION

There is ***no singular historical or physical* examination finding** that achieves both 70% sensitivity and 70% specificity for the diagnosis of acute heart failure.[19] The initial global clinical judgment has a sensitivity of 61% and specificity of 86%. A history of heart failure is the most useful historical parameter, but only has a sensitivity of 60% and specificity of 90% (positive likelihood ratio [LR+] = 5.8; negative likelihood ratio [LR–] = 0.45). Risk factors for acute heart failure sometimes may be helpful, including hypertension, diabetes, valvular heart disease, old age, male sex, and obesity. The symptom with the highest sensitivity for diagnosis is dyspnea on exertion (84%).[19,20] The most specific symptoms are paroxysmal nocturnal dyspnea, orthopnea, and edema (76% to 84%).[19,20] Evaluation for historical precipitating factors (**Table 53-4**) is also useful.

TABLE 53-4

Precipitants of AHF

Nonadherence
Excess salt or fluid intake*
Medication nonadherence*
Renal failure (especially missed dialysis)*
Substance abuse—cocaine, methamphetamines, ethanol
Poorly controlled hypertension
Iatrogenic
Recent addition of negative inotropic drugs (e.g., calcium channel blocker, β-blocker)
Initiation of salt-retaining drugs (e.g., NSAID, steroids, thiazolidinediones)
Inappropriate therapy reduction
New antiarrhythmic agents

*Common in ED patients

Abbreviations: AHF = acute heart failure; NSAID = nonsteroidal anti-inflammatory drug.

On exam, an S_3 has the highest LR+ for acute heart failure (11), but its absence is not useful as a negative predictor (0.88).[19] However, the interrater reliability of an S_3 is not good,[21,22,23] and the ambient noise in a busy ED may interfere with S_3 detection. Abdominojugular reflux (LR+ = 6.4) and jugular venous distension (LR+ = 5.1) are the only other two physical examination findings that have an LR+ greater than 5. Increased neck size, obesity, and rapid breathing may diminish the ability to accurately measure jugular venous distension at the bedside in the ED.

When clinicians are 80% confident of the diagnosis of acute heart failure, the "clinical gestalt" outperforms diagnostic tests available in the ED for the diagnosis[19]; however, clinical gestalt may be about 50% accurate in an outpatient setting.[24] Data from the Breathing Not Proper Trial found t[Loading [Contrib]/a11y/accessibility-menu.js]pe natriuretic peptide value had a similar accuracy performance.[25]

CHEST RADIOGRAPHY

Chest radiographs showing pulmonary venous congestion, cardiomegaly, and interstitial edema are most specific for a final diagnosis of acute heart failure,[18,19] but the absence of these does not rule it out, because up to 20% of patients subsequently diagnosed with heart failure have chest radiographs without signs of congestion at the time of prior ED evaluation.[26] Particularly in late-stage heart failure, patients may have few radiographic signs, despite symptoms and elevated pulmonary capillary wedge pressure.[18]

ECG

The ECG is not useful for diagnosis, but it may reveal an underlying cause or precipitant. ECG signs of ischemia, acute myocardial infarction, or dysrhythmias may point to the precipitating cause. The presence of atrial fibrillation has the highest LR+ for a diagnosis of heart failure; however, new T-wave changes were also associated with the diagnosis.[19]

BIOMARKERS

The most widely investigated markers have been the natriuretic peptides, B-type natriuretic peptide, and N-terminal pro-B-type natriuretic peptide. Other novel biomarkers have been explored for both diagnosis and prognosis, such as ST2, galectin 3, and neutrophil gelatinase-associated lipocalin. Their role in the ED is not established; B-type natriuretic peptide and N-terminal pro-B-type natriuretic peptide remain the most important biomarkers in clinical use. Natriuretic peptide tests may add value in the setting of undifferentiated dyspnea in the ED, improving diagnostic discrimination in a variety of settings[25,27] and correlating with cardiac filling pressures and ventricular stretch.[28] As a result, B-type natriuretic peptide or N-terminal pro-B-type natriuretic peptide testing is recommended and helpful when the cause of dyspnea is unclear after standard evaluation (Table 53-5).

TABLE 53-5

Natriuretic Peptide Cut Points for Clinical Decision Making

	Low Cut Point (rule out HF)	High Cut Point (HF likely)		
BNP	100 pg/mL	500 pg/mL		
	Sensitivity 90%	Sensitivity 75%		
	Specificity 73%	Specificity 90%		
N-terminal pro-BNP[29]	300 pg/mL	450 pg/mL if <50 years old	900 pg/mL if 50–75 years old	1800 pg/mL
	Sensitivity 99%	Sensitivity 97%	Sensitivity 90%	Sensitivity 85%
	Specificity 60%	Specificity 93%	Specificity 82%	Specificity 73%

Abbreviations: BNP = B-type natriuretic peptide; HF = heart failure.

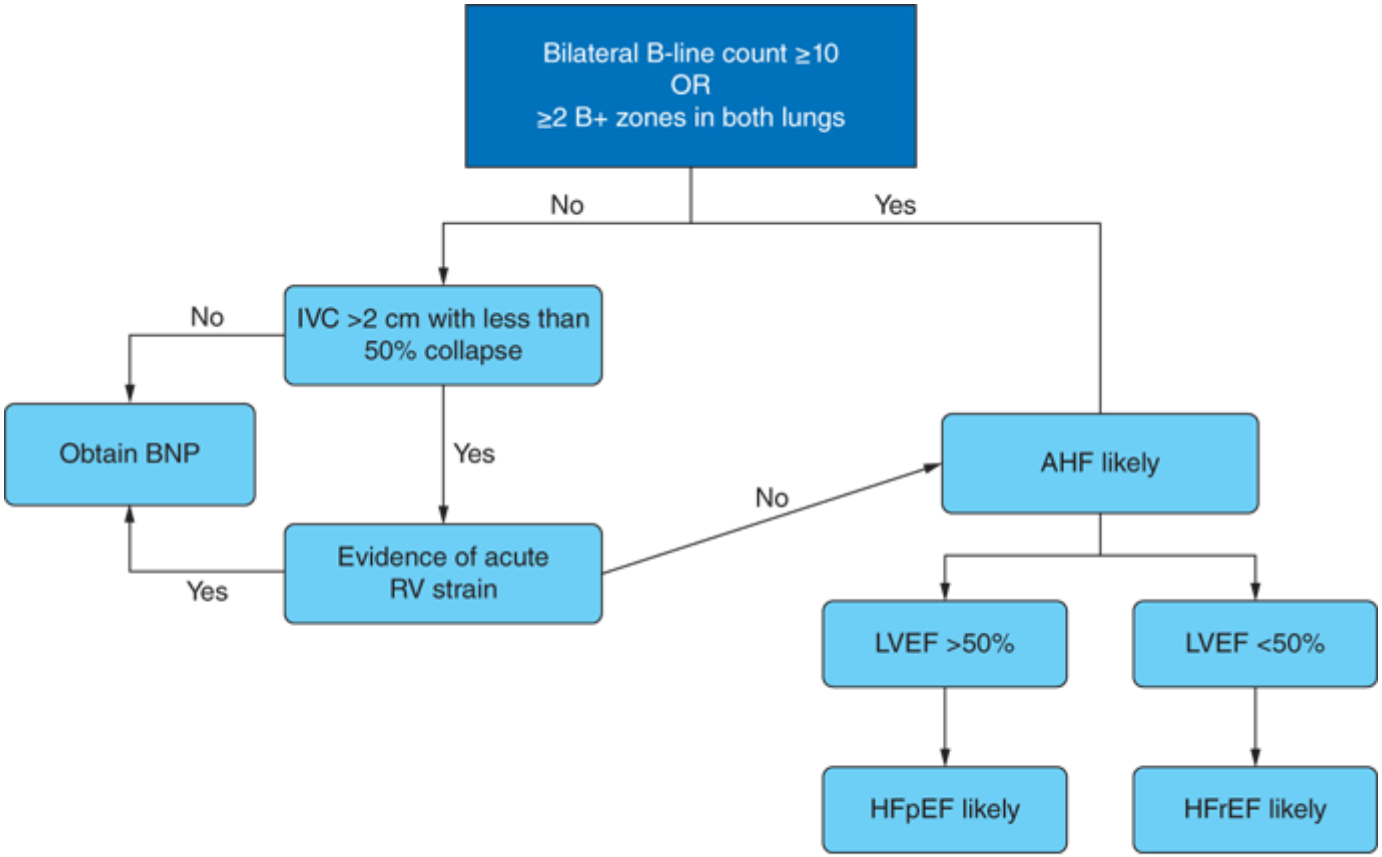
Despite the established value of natriuretic peptide testing, there are many situations where interpretation of results is unclear. Levels can be affected by age, gender, and body mass, and may elevate later in patients who present with flash pulmonary edema.[30] Dyspnea and modest B-type natriuretic peptide elevation are evident in conditions such as pulmonary hypertension, pulmonary embolism, pneumonia, sepsis, and renal failure. As many as 25% of patients will fall into the diagnostic "grey zone" (100 to 500 pg/mL for B-type natriuretic peptide), complicating test interpretation. **B-type natriuretic peptide/N-terminal B-type natriuretic peptide testing is best used when diagnostic uncertainty exists and as an addition to the physician assessment, rather than as a routine measurement.**[27] Similarly, while marked natriuretic peptide elevations are associated with worse short-term outcomes, even low elevations have increased mortality risk, limiting usefulness in bedside prognostication in the ED.[31,32]

POINT-OF-CARE ULTRASOUND (US)

Point-of-care cardiopulmonary US can help to determine the cause of dyspnea, including cardiac tamponade, and can determine left ventricular function and volume status, but is not a substitute for comprehensive echocardiography.[31,32] Bedside cardiopulmonary US can also address three questions (**Figure 53-1**): (1) Are there signs of pulmonary congestion? (2) Are there signs of volume overload by measuring the size of the inferior vena cava and its collapsibility? (3) Is the left ventricular ejection fraction low or normal?

FIGURE 53-1.

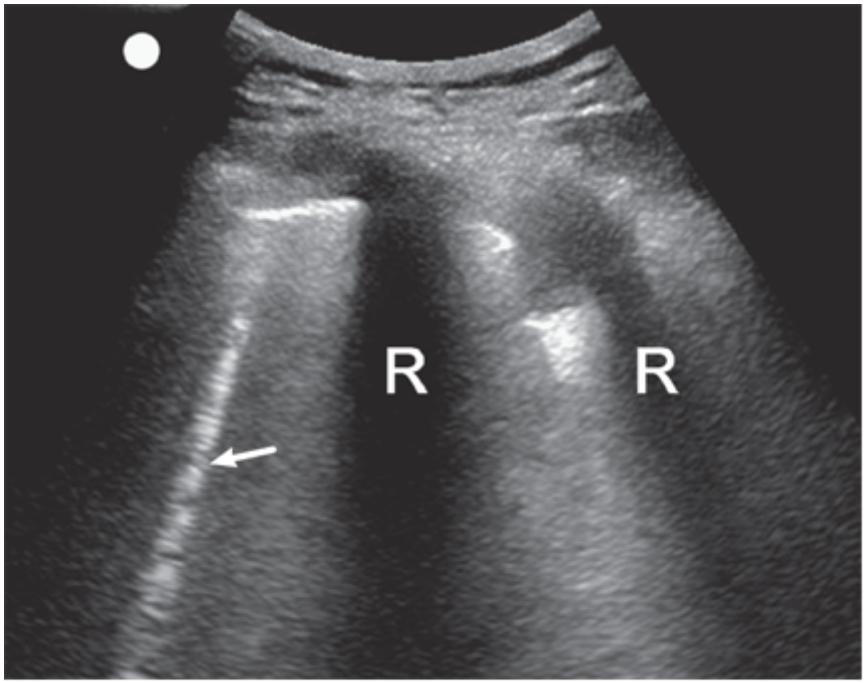
Bedside US use to identify acute heart failure (AHF) in dyspneic ED patients. BNP = B-type natriuretic peptide; IVC = inferior vena cava; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; LVEF = left ventricular ejection fraction; RV = right ventricle.



Pulmonary US is used first to determine if pulmonary congestion is present by looking for B lines. **Sonographic B lines** (Figure 53-2) are ring-down artifacts that arise from the interface of the visceral and parietal pleura when there is swelling of the lung's interlobular septa due to lymphatic congestion as is seen in pulmonary edema.[33] They are the sonographic equivalent of Kerley B lines seen on chest radiography.[34] More than two B lines in any one sonographic window along the anterior and anterolateral chest are pathologic and highly specific for alveolar and interstitial edema.[35]

FIGURE 53-2.

B lines representing thickened inter-alveolar/interlobular septa. R = rib; arrow = B line. [Reproduced with permission from Ma, Mateer, Reardon, and Joing (eds): *Emergency Ultrasound*, 3rd ed. McGraw-Hill, Inc., 2014. Fig. 7-5, Part C only.]



Because bilateral B lines can be present in other conditions not caused by pulmonary edema (e.g., pulmonary fibrosis, pulmonary contusion, bilateral pneumonia), rapid assessment for elevated central venous pressure as a marker of right heart congestion is needed.[36] An inferior vena cava size greater than 2 cm or collapsibility index of <50% is indicative of elevated central venous pressure. In the absence of significant pulmonary disease, these measures are highly correlated with pulmonary capillary wedge pressure and are specific for acute heart failure. One should also use US to look for other clinical conditions that cause an elevation in right heart pressure, including pulmonary embolism or clinically significant tricuspid regurgitation, because both conditions could cause inferior vena cava changes consistent with heart failure.

Determination of left ventricular ejection fraction is the final piece of ED-based bedside ultrasonography. Many of the methods for measuring left ventricular ejection fraction are highly technical and are not compatible with the need for rapid left ventricular ejection fraction determination during the initial ED evaluation of the dyspneic patient. However, with limited training, emergency physicians trained in focused cardiac US have reasonable agreement with expert cardiology interpretations by using a visual estimation of left ventricular ejection fraction into broad categories of normal, moderately reduced, and severely reduced.[37,38] Other markers that have been suggested, such as E-point septal separation a[illegible]ble markers and may be more time-consuming.[39]

TREATMENT

The initial approach is driven by the acuity at presentation, hemodynamics, and volume status. In critically ill patients, airway management is the first priority to ensure adequate oxygenation and ventilation. In those less acutely ill, a focused evaluation ensues next, followed by treatment.

Supplemental oxygen use is guided by pulse oximetry, seeking saturations above 95%. Because hypoxemia is a greater risk than hypercarbia, do not withhold oxygen even when there is concern about carbon dioxide retention. Capnometry and arterial blood gas measurements can later help titrate therapy in the critically ill or if carbon dioxide retention is likely. In those with extreme findings, endotracheal intubation with mechanical ventilation is indicated.

Noninvasive ventilation may improve the symptoms in patients presenting with heart failure or pulmonary edema.[14,40] Successful noninvasive ventilation requires close monitoring, hemodynamic stability, facial anatomy that allows an adequate facemask seal, and patient cooperation. Using either a facemask or a nasal device, noninvasive ventilation can be delivered with continuous positive airway pressure throughout the respiratory cycle or with bilevel positive airway pressure (see chapter 28, "Noninvasive Airway Management"). Noninvasive ventilation plus standard medical therapy appears to reduce the need for intubation and improves respiratory distress and metabolic disturbance versus standard therapy alone.[14,15] Whether it decreases hospital mortality is unclear.[14]

Acute heart failure with hypotension occurs in approximately 3% of patients.[41] Consider acute coronary syndrome, and management may require reperfusion therapy (see chapters 49, "Acute Coronary Syndromes and 50, "Cardiogenic Shock"). Treatment includes the initiation of inotropic therapy (commonly norepinephrine, dopamine, or dobutamine) and admission to an intensive care unit.

Other standard initial measures include cardiac monitoring, pulse oximetry, IV access, and frequent vital sign assessments. A urinary drainage catheter may aid in monitoring fluid status in the severely ill or incontinent, but this is best reserved for those with extreme illness or an inability to void (to avoid catheter-related complications later.).

HYPERTENSIVE ACUTE HEART FAILURE

The failing heart is sensitive to increases in afterload, with some patients developing pulmonary edema with a systolic blood pressure as low as 150 mm Hg. Prompt recognition and afterload reduction with vasodilators can avoid the need for intubation.[42]

A short-acting, rapid-onset, systemic venous and arterial dilator, **nitroglycerin** decreases mean arterial pressure by reducing preload and, at high doses initially, afterload. Nitroglycerin may have coronary vasodilatory effects, decreasing myocardial ischemia and improving cardiac function. The routes chosen—IV, sublingual, or transdermal—are often based on severity of symptoms. Sublingual nitroglycerin is easily administered, rapidly bioavailable, and can be given as often as needed to reach a desired clinical end point provided there is adequate blood pressure. An initial approach is repeated sublingual administration of nitroglycerin, 0.4 milligrams, at a rate of up to one per minute, until relief or replacement with IV nitroglycerin. When using the latter (often for those most symptomatic), a starting dose of 0.5 to 0.7 micrograms/kg/min is common and titrated every few minutes up to 200 micrograms/min based on the blood pressure (avoiding large drops) and symptoms (**Table 53-6**; see also Table 53-8). High doses may be beneficial in the acute setting, and adverse events are uncommon.[43] Apply transdermal nitroglycerine (0.5–2 inches to the chest wall based on blood pressure) **only** after initial therapy has improved conditions, or if symptoms are minor, because of the slow onset of action by this route.

TABLE 53-6

Management of Hypertensive Acute Heart Failure*

Stepwise Approach	Comments
Administer oxygen as needed for saturation ≥95%; give sublingual nitroglycerin.	Sublingual nitroglycerin may be repeated up to one per minute.
If severe dyspnea, consider NIV or intubation.	
If BP >150/100 mm Hg, add IV nitroglycerin or nitroprusside; if BP falls below 100 mm Hg, stop nitrates, and monitor for persistent hypotension or symptoms (see chapter 50, "Cardiogenic Shock"). If BP <150/100 mm Hg after sublingual administration and if improved, consider transdermal nitroglycerin.	See chapter 58, "Pulmonary Hypertension"; see text for discussion of these agents.
Start IV loop diuretic (furosemide or bumetanide) in the setting of volume overload.	Initiate nitrates before diuretics.
Assess for severity of illness/high risk: altered mental status persistent, hypoxia despite NIV, hypotension, troponin elevation, ischemic ECG changes, blood urea nitrogen >43, creatinine >2.75, tachycardia, tachypnea, or inadequate urine output.	See chapter 49, "Acute Coronary Syndromes" for ECG criteria.
Admit to intensive care unit if high severity of illness or risk of decompensation.	
Choose discharge or ED observation unit admission if good response to therapy, no high-risk features, and good social support. Admit the rest. Admit to ICU if any ongoing cardiorespiratory compromise or acute ischemia.	Scoring systems may not reliably identify all patients at risk.

*Inclusion: SBP >140 mm Hg

Abbreviations: BP = blood pressure; ICU = intensive care unit; NIV = noninvasive ventilation; SBP = systolic blood pressure.

The most important nitroglycerin complication is hypotension, often only lasting transiently and at times even seen with overall clinical improvement. Hypotension usually resolves after cessation of nitroglycerin. If persistent, think of concomitant volume depletion or right ventricular infarct, and deliver a normal saline fluid bolus (250 to 1000 mL). Headache is frequent, but acetaminophen usually is adequate therapy. Methemoglobinemia is a theoretic possibility but not a concern unless high doses are used for extended intervals. Despite broad uptake into regular clinical practice, nitroglycerin has been subject to surprisingly little prospective study.

Nitroprusside

If further afterload reduction is required (i.e., continued high systemic vascular resistance usually manifested by persistent elevated blood pressure and continued symptoms despite nitroglycerin doses >200 micrograms/min), use IV **nitroprusside**. This drug is a more potent arterial vasodilator than nitroglycerin; its hemodynamic effects include decreased blood pressure, left ventricular filling pressure reduction, and increased cardiac output. The initial dose of nitroprusside is 0.3 micrograms/kg/min, titrated upward every 5 to 10 minutes based on blood pressure and clinical response (maximum 10 micrograms/kg/min). The major complication is hypotension. It is also associated with thiocyanate toxicity, especially with high doses, prolonged (longer than 3 days) use, and hepatic or renal impairment.

The critical end point is rapidly lowering filling pressure to prevent the need for endotracheal intubation. Give IV vasodilators as soon as vascular access is established if the blood pressure remains elevated.

Loop Diuretics

After vasodilator therapy, some patients may require diuretics (see Table 53-8 and next section) based on continued symptoms after blood pressure is controlled. Diuretics (**furosemide** most commonly used) administered alone without vasodilators for hypertensive heart failure may increase mortality[44] and worsen renal dysfunction. Ultimately, successful management of blood pressure and cardiac filling pressure creates marked improvement in respiratory status long before any diuresis.

Contraindications and Alternatives to Vasodilation in Select Settings

Because all vasodilators exert hypotensive effects, do not use if there are signs of hypoperfusion or existing hypotension. **Flow-limiting, preload-dependent states such as right ventricular infarction, aortic stenosis, hypertrophic obstructive cardiomyopathy, or volume depletion increase the r** **ion (Table 53-7).** Combined with acute pulmonary edema, the latter preload-dependent states are very

difficult to manage. Therapy is aimed at decreasing the outflow gradient by slowing heart rate and cardiac contractility. Although this can be accomplished with IV β-blockers, treatment is best done in the intensive care unit with invasive hemodynamic guidance. If there is coexistent shock in the setting of hypertrophic obstructive cardiomyopathy, phenylephrine (40 to 100 micrograms/min IV) is a good choice because it creates peripheral vasoconstriction without increasing cardiac contractility.

TABLE 53-7

Causes of Hypotension after Vasodilator Use

Excessive vasodilation
Hypertrophic obstructive cardiomyopathy
Intravascular volume depletion
Right ventricular infarction
Cardiogenic shock/myocardial infarction
Aortic stenosis
Anaphylaxis
Unsuspected sepsis

NORMOTENSIVE HEART FAILURE

Shortness of breath, orthopnea, jugular venous distension, rales, and possibly an S_3 may still be evident even in the presence of normal vital signs, oxygenation, and ventilation. In this situation, treat with diuresis first, with further treatment based on response to therapy (Table 53-8).

TABLE 53-8

Medications for Acute Heart Failure

Vasodilators for Acute Heart Failure			
Vasodilator	**Dose**	**Titration End Point**	**Complications**
Sublingual NTG	0.4 milligram every 1–5 min	Blood pressure	Hypotension
IV NTG	0.2–0.4 microgram/kg/min (starting dose)	Symptoms	Headache, hypotension
Nitroprusside	0.3 microgram/kg/min (starting dose), 10 micrograms/kg/min (maximum)	Blood pressure	Hypotension, cyanide/thiocyanate toxicity, coronary steal
		Symptoms	
Diuretics for Heart Failure			
Diuretic	**Dose (IV)**	**Effect**	**Complications**
Furosemide	No prior use: 20–40 milligrams IVP	Diuresis starts within 15–20 min	↓ K+, ↓ Mg^{2+}, hyperuricemia, hypovolemia
	If prior use: total daily IV dose 1 to 2.5 times the patient's previous total daily oral dose, divided in half and given IV bolus every 12 h	Duration of action is 4–6 h	Ototoxicity, prerenal azotemia
	If no effect by 20–30 min, increase subsequent dose		
Bumetanide	1–3 milligrams IV	Diuresis starts within 10 min	Same as above

		Peak action at 60 min	
Torsemide	10–20 milligrams IV	Diuresis starts within 10 min	Same as above
		Peak action in 1–2 h	

Abbreviations: IVP = IV push; NTG = nitroglycerin; ↓ = decreased.

Diuretics

Loop diuretics provide rapid symptomatic relief of congestive symptoms and improve the effects of angiotensin-converting enzyme inhibitors by decreasing intravascular volume. Most ED patients require IV dosing, because bowel wall edema may prevent proper GI absorption. Dosing is guided by symptoms and prior usage (Table 53-8). In general, dose loop diuretics at the lowest possible dose that relieves congestion. Once congestion is resolved, a fixed maintenance dose is continued to prevent recurrence.

Loop diuretics promote water and sodium excretion and are effective except in severe renal dysfunction. Furosemide is inexpensive and effective. Alternatives are bumetanide (1 milligram equivalent to 40 milligrams of furosemide) or torsemide (20 milligrams equivalent to 40 milligrams of furosemide). All trigger rapid diuresis after an IV dose, often within 10 to 15 minutes.

The DOSE trial suggests a total daily IV dose 1 to 2.5 times the patient's previous total daily oral dose, divided in half and administered by IV bolus every 12 hours.[45] For example, if the patient is on furosemide 80 milligrams PO twice a day, then an initial ED dose is 80 to 200 milligrams IV bolus. Higher doses are associated with more rapid symptom improvement but a slight decrease in renal function. For patients who are loop diuretic naïve, a reasonable starting dose is furosemide 40 milligrams IV. Bolus and continuous infusion therapy are equivalent, but the latter is more challenging in the ED and hence often eschewed. Ethacrynic acid (0.5 to 1 milligram/kg; maximum 100 milligrams) is another option. Sulfa allergy is generally not a concern with nonantibiotic drugs such as diuretics that contain a sulfa moiety (see chapter 206 "Antimicrobials" for more d

Diuretics may worsen renal function and create hypokalemia. An increasing QT interval should trigger a search for hypocalcemia, hypokalemia, or hypomagnesemia. Ototoxicity is rare but may occur if diuretics are used in conjunction with aminoglycoside antibiotics. Potassium-sparing diuretics, such as spironolactone (25 to 50 milligrams PO), are generally reserved for advanced chronic heart failure; these are used more for their mortality benefit than diuretic effect.

Urinary diuretic response requires monitoring. With greater symptoms or less response to initial IV diuretics, double the dose and repeat in 30 to 60 minutes or as needed based on urine output. Ongoing congestion or dyspnea after a loop diuretic may signal the need for another therapy, such as a vasodilator.

Other Treatments

Ultrafiltration allows the extracorporeal removal of plasma water from whole blood across a semipermeable membrane with a transmembrane pressure gradient.[46] Ultrafiltration has advantages over diuresis including more precise regulation of fluid removal, avoidance of diuretic-associated electrolyte abnormalities, a higher level of sodium removal for a given amount of volume, and attenuation of significant fluctuations in intravascular volume.[47] While initial studies provided promising safety and efficacy data,[48,49] subsequent study in patients with cardiorenal syndrome and persistent congestion did not demonstrate an advantage of ultrafiltration over bolus diuretic therapy.[50] If all diuretic and medical strategies are unsuccessful, consider ultrafiltration for patients with obvious volume overload to alleviate congestive symptoms and excess weight.[2] Ultrafiltration is unlikely to be deployed in the ED given the need to optimize other approaches first.

Morphine (2 to 5 milligrams IV) relieves congestion and anxiety, but it is associated with adverse events, including the need for mechanical ventilation, prolonged hospitalization, ICU admission, and mortality.[51] If desired for its venodilation properties or pain control, use morphine in small, titrated doses (2 to 4 milligrams IV) and with close monitoring. The trial noting harm did not set out to study morphine use, so selection bias may explain some or much of the findings. Only those failing standard therapy or with severe symptoms received the drug, multiplying the negative outcomes. Nonetheless, morphine has a role secondary to nitrates and loop diuretics and is not needed routinely.

Nesiritide is a vasodilator whose formulation uses recombinant human B-type natriuretic peptide. Several small studies suggested a benefit of adding nesiritide to standard therapy on patient-reported relief of dyspnea, but additional studies, including the pivotal mortality trial ASCEND-HF,[8] found no significant difference in the frequency of rehospitalization or mortality. ASCEND-HF reported an increased risk of both symptomatic and asymptomatic hypotension among patients randomized to nesiritide.[8] Nesiritide does not result in substantial clinical improvement when a[illegible]line agent when nitroglycerin is ineffective or contraindicated.

Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are given for hypertension and chronic heart failure, but there is little data to recommend use in the ED for acute heart failure.

Oral angiotensin-converting enzyme inhibitors decrease mortality and hospitalizations in patients with reduced ejection fraction[2]; these are often used after observation care if no contraindications exist after contact with a primary care physician or cardiologist. Oral angiotensin receptor blockers are alternatives to or can be added to angiotensin-converting enzyme inhibitors in select heart failure patients with reduced ejection fraction.[2] Their use may also be considered after consultation and the conclusion of treatment. Treatment of angiotensin-converting enzyme inhibitor–induced angioedema is outlined in the chapter 14, "Anaphylaxis, Allergies, and Angioedema."

β-Blockers are not usually initiated in the acute setting, except perhaps to control rate-related heart failure. They are generally reserved for stable patients. The rationale for β-blockers rests on the fact that norepinephrine levels are elevated in heart failure, contribute to myocardial hypertrophy, increase afterload and coronary vasoconstriction, and are associated with mortality. β-Blockers reduce sympathetic nervous system activity and are used for mortality reduction and symptom relief.

Drugs to Avoid in Acute Heart Failure

Oral **calcium channel blockers** have myocardial depressant activity and are not routine treatment for acute heart failure, with trials demonstrating either no benefit or worse outcomes. If necessary, amlodipine may be used for compelling clinical reasons (e.g., as an antianginal agent despite maximal therapy with nitrates and β-blockers).

Avoid selective or nonselective **nonsteroidal anti-inflammatory drugs** in patients with acute heart failure. They can cause sodium and water retention and blunt the effects of diuretics,[2] and may increase morbidity and mortality.

DISPOSITION DECISIONS

While risk-stratification tools are commonplace in other ED disease processes such as chest pain and pneumonia, we lack a readily available and validated ED-based risk-stratification tool that has been compared to physician judgment.

Thus, disposition decisions in ED patients with acute heart failure are often based on physician judgment, a physiologic risk assessment, and an assessment of barriers to successful outpatient care such as caregiver support, access to medications, and timely follow-up (**Figure 53-3**). High-r[redacted]with acute heart failure associated with morbidity and mortality (**Table 53-9**)[52] include renal dysfunction, low blood pressure, low serum sodium, and elevated natriuretic peptides or cardiac troponin. Unfortunately, high-risk markers are not present in

up to 50% of ED patients, limiting the impact in disposition decisions.[53] Prospective testing of four acute heart failure prediction rules suggests they would not be useful in the ED.[54]

TABLE 53-9

Selected ED-Based Risk-Stratification Studies from the Last 8 Years that Examine Events within 30 Days or Less of Index ED Presentation

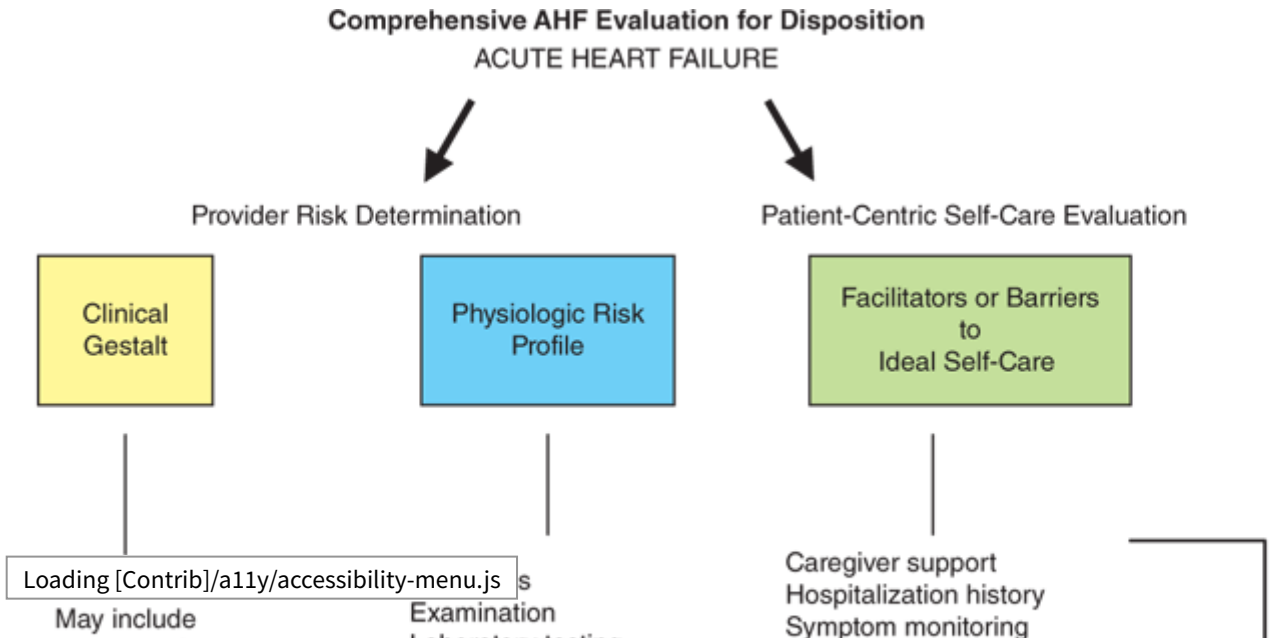
Author/Year	N	Predicted Outcome	Variables in Final Model	Low-Risk Markers
Lassus/2013	441–4450 (pooled analysis, total n varied by biomarker evaluated)	30-d and 1-y mortality	ST2, MR-proADM, CRP, NT-proBNP, BNP, MR-proANP in addition to clinical model (age, gender, blood pressure on admission, estimated glomerular filtration rate <60 mL/min/1.73 m^2, sodium and hemoglobin levels, and heart rate)	No
Stiell/2013	559	30-d death and 14-d serious nonfatal events	History of TIA/CVA, vital signs, ECG and lab findings	No
Lee/2012	15,164	7-d mortality	Creatinine, BP, O_2 saturation, Tn, history of cancer, home metolazone, EMS transport	Yes
Hsieh/2008	8384	Inpatient mortality or serious medical complications, 30-d mortality	pH, pulse, renal function, WBC, glucose, sodium	Yes
Lee/2003	2624/1407	30-d mortality	Age, RR, BP, BUN, sodium, cerebrovascular disease, dementia, COPD, cirrhosis, cancer, hemoglobin	Yes

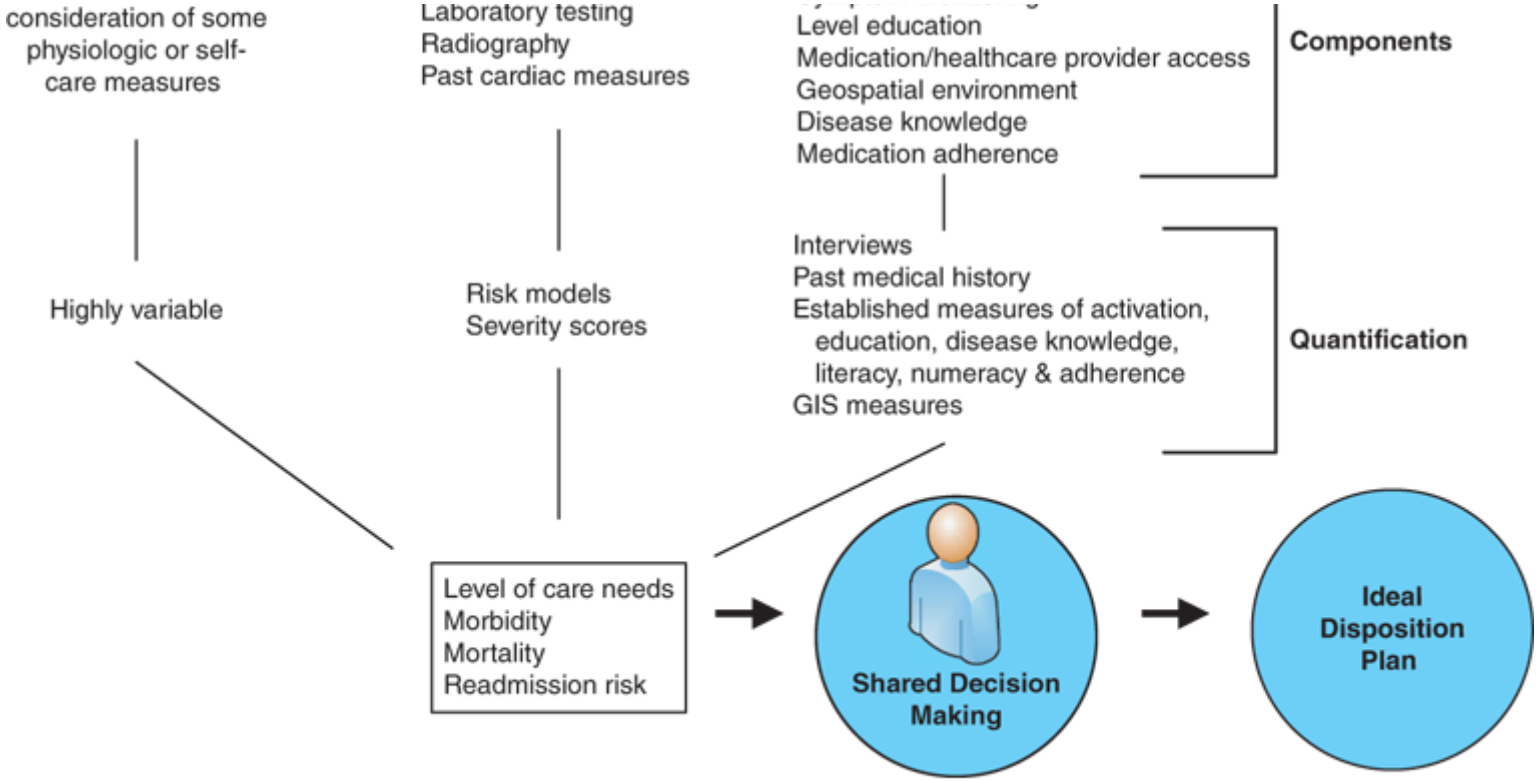
Author/Year	N	Predicted Outcome	Variables in Final Model	Low-Risk Markers
Auble/2005	33,533	Inpatient mortality or serious medical complications, 30-d mortality and AHF readmission	pH, pulse, renal function, WBC, glucose, sodium	Yes
Fonarow/2005	65,275	In-hospital mortality	BUN, systolic BP, creatinine	No

Abbreviations: AHF = acute heart failure; ANP = atrial natriuretic peptide; BNP = B-type natriuretic peptide; BP = blood pressure; COPD = chronic obstructive pulmonary disease; CRP = C-reactive protein; CVA = cerebrovascular accident; RR = respiratory rate; TIA = transient ischemic attack; Tn = troponin.

FIGURE 53-3.

Factors impacting disposition decisions in ED patients with acute heart failure (AHF).





Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

The unpredictability of postdischarge behavior and care limits, coupled with the elevated overall risk of harm or repeated care events, limits the ability to discharge patients directly from the ED. Admit patients with high-risk features to the hospital (Table 53-10).[55] Those who require invasive monitoring or procedures require intensive care unit admission. Others may be appropriate for non–intensive care unit level care. Observation unit management is an option in others with lower risk features. Many patients do not have high-risk features at initial ED evaluation and experience improvement in dyspnea during their ED stay as a result of standard therapy.[56] Many have complete symptom resolution within 12 to 24 hours of initial therapy, a typical time period of observation. The monitoring of blood pressure, heart rate, urine output, and body weight is easily accomplished in the observation setting, and any diagnostic testing (labs, echocardiography) needed can occur. Finally, an extended observation interval allows patients to receive heart failure education, confirm outpatient medications, and arrange follow-up prior to discharge. Ideally, outpatient follow-up within 5 days can decrease readmissions.[57] Prior studies suggest 75% of patients will respond to therapy, will have no identifiable high-risk features, and will be discharged home. Their rates of readmission are similar to or better than those who are managed in a [redacted] an inadequate response to initial therapy or with high-risk features identified during their observation stay

are admitted to the hospital for further management. An observation unit strategy can help reduce costs while delivering quality care for select lower risk ED patients with acute heart failure.[58]

TABLE 53-10

Heart Failure Observation Unit/Short Stay Exclusion Criteria

Recommended Exclusions
Positive troponin
Blood urea nitrogen >40 milligrams/dL
Creatinine >3.0 milligrams/dL
Sodium <135 mEq/L
New ischemic changes on ECG
New onset of acute heart failure*
IV vasoactive infusions being actively titrated
Significant comorbidities requiring acute interventions
Respiratory rate ≥32 breaths/min and/or requiring noninvasive ventilation at the time of OU consideration
Signs of poor perfusion at the time of OU consideration
Suggested Exclusions
Poor social support
Poor follow-up

*Although part of the published guidelines, many institutions admit patients to OUs with new-onset heart failure.

Abbreviation: OU = observation unit.

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USEFUL WEB RESOURCES

American College of Cardiology/American Heart Association—http://content.onlinejacc.org/article.aspx?articleid=1127651

Canadian Cardiovascular Society—http://dx.doi.org.lproxy.nymc.edu/10.1016/j.cjca.2012.10.007

European Society of Cardiology: http://www.escardio.org/guidelines-surveys/esc-guidelines/GuidelinesDocuments/Guidelines-Acute%20and%20Chronic-HF-FT.pdf

Heart Failure Society of America: http://www.heartfailureguideline.org/

Silverchair