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Acute Heart Failure

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ABSTRACT
Heart failure impacts millions of Americans and has an approximate 5-year mortality rate of 50%–55%. Decompensation of this disease state could result in a patient's initial presentation and diagnosis or may reflect a worsening of a chronic condition that is being managed but needs optimization. Secondary to this, it is important for members of the health care team in the emergency department to recognize the presentation of this disease and manage the patient's signs and symptoms appropriately. Patients may be normotensive upon presentation or hemodynamically unstable. Those who are normotensive are often managed with loop diuretics and possibly low-dose vasodilators, whereas those who are hemodynamically unstable require more aggressive, focused care. It is important to note that some patients may present with respiratory failure and with no known history of heart failure. In these cases, a rapid and accurate diagnosis is critical. This article briefly summarizes the common acute clinical presentations of heart failure and the therapies considered first line for treatment based on the primary literature. **Key words:** acute, decompensation, diuretics, heart failure, inotropes, vasodilators

THE MORBIDITY, mortality, and prevalence associated with heart failure bring it to the forefront when it comes to important health care issues in the United States. As the population ages, the incidence of heart failure is expected to increase more than for other cardiovascular disease states secondary to its presence among older Americans (Heidenreich et al., 2013). Acute decompensation of these patients varies, given the diversity associated with the pathophysiology and processes involved. Patients may present with mild signs and symptoms (e.g., pulmonary edema requiring oxygen only) or in a more severe state such as cardiogenic shock. Those who are critically ill must be diagnosed and treated appropriately. Because these patients commonly present to the emergency department, it is imperative that the health care team be familiar with the different presentations and how each should be managed. This article provides a review of the more acute presentations associated with heart failure as

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well as pharmacotherapy options for initial treatment.

ACUTE HEART FAILURE OVERVIEW

Epidemiology and Pathophysiology

In 2012, heart failure cost the United States approximately $30.7 billion, with more than 8 million American adults projected to have the disease by 2030 (Benjamin et al., 2019; Virani et al., 2020). The American Heart Association (AHA) reports that Blacks, followed by Hispanic, Whites, and Chinese Americans, carry the highest risk of developing heart failure, with higher risks associated with the observed greater prevalence of hypertension, diabetes, and lower socioeconomic status among these groups. The AHA also reports that the prevalence of certain types of heart failure may be changing, with increasing rates of hospitalizations for heart failure with preserved ejection fraction (HFpEF). The AHA observed that although there has been some improvement in heart failure survival, 1-year mortality rates remain at almost 30% (Virani et al., 2020). Patient-specific risk factors for heart failure include coronary artery disease, hypertension, diabetes mellitus, obesity (body mass index 30 or more), and smoking. Anemia, advanced age, and female sex are additional risk factors (Virani et al., 2020).

Acute heart failure (AHF) is an umbrella term that encompasses de novo AHF and acute decompensation of chronic heart failure (CHF) (see Table 1). AHF has traditionally been defined as cardiac pump failure resulting in fluid congestion and systemic hypoperfusion, but new insights suggest that the pathophysiology is more complex, involving multiple neurohormonal and hemodynamic interactions (Arrigo, Parissis, Akiyama, & Mebazaa, 2016). In fact, rather than a single disease state, AHF may be more accurately described as a common constellation of clinical findings resulting from a collection of underlying physiological abnormalities (see Figure 1). These underlying abnormalities may include the exacerbation of existing heart failure, as in the case of acute decompensated heart failure (ADHF), or de novo AHF. ADHF is the more common of the two, but de novo AHF still accounts for more than 25% of patients presenting with AHF (Felker, Ellison, Mullens, Cox, & Testani, 2020).

Table 1. Causes of de novo acute heart failure

Acute myocardial infarction	Thyroid storm
Arrhythmias	Pulmonary embolism
Acute valve regurgitation	Cardiac tamponade
Myocarditis	Takotsubo cardiomyopathy
Toxins	Peripartum stress

Venous congestion is apparent in most patients who present with AHF. Fluid accumulation, triggered by hormonal activation, causes an increased capillary pressure gradient. The higher gradient increases the movement of fluid from the capillary into the interstitial spaces. Eventually, this transudation of fluid exceeds drainage capacity of the lymphatic system and edema results (Arrigo et al., 2016).

In ADHF associated with existing heart failure with reduced ejection fraction (HFrEF), persistent sodium accumulation and elevated capillary pressures over time contribute to disruption of the interstitial glycosaminoglycan network. The subsequent dysfunction increases interstitial compliance, which allows even more transudation of fluid and sodium. Sodium caught in the glycosaminoglycan network exerts osmotic pressure to pull fluid out of the capillaries, but it cannot be easily removed from the periphery. Its persistence contributes to the chronic edema that is so often a struggle for HFrEF patients and may also compound an episode of acute decompensation (Arrigo et al., 2016).

Rapid fluid redistribution may be more explanatory of the venous congestion demonstrated in de novo AHF. Acute sympathetic stimulation causes vasoconstriction,

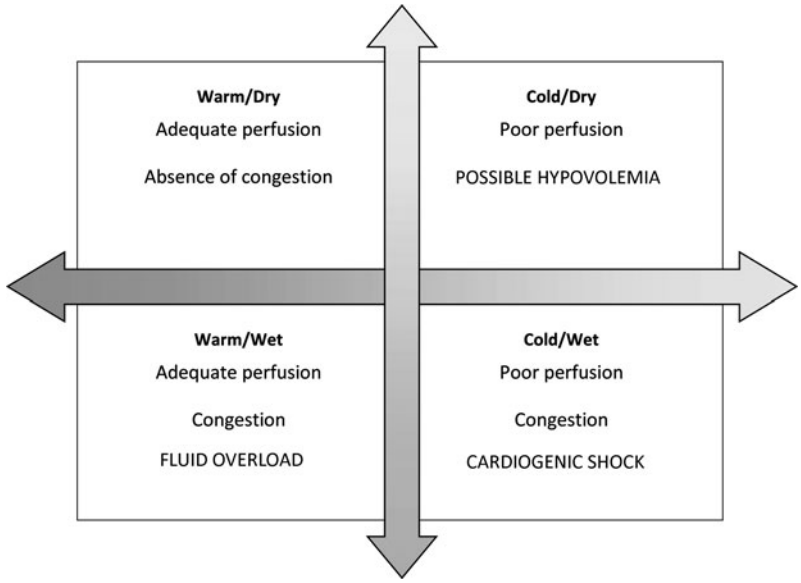

Figure 1. Acute heart failure syndromes.

displacing splanchnic blood into the peripheral venous and pulmonary vessels. This redistribution increases preload even in the absence of significant peripheral fluid accumulation and may be more common in acute exacerbations of HFpEF (Arrigo et al., 2016; Mullens et al., 2019). Regardless of the primary contributing factor, the stretch of vascular endothelium associated with venous congestion in these patients may activate proinflammatory and oxidative cascades that contribute to furthering the cycle of decompensation. This is an area of ongoing research (Arrigo et al., 2016).

AHF Syndromes (Common Presentations)

Warm/Wet: Fluid Overload

"Warm and wet" describes the category of patients with heart failure who present adequately perfused but with fluid overload or congestion. About 60% of AHF patients exhibit this phenotype on presentation. Their fluid overload may progress from subclinical hemodynamic congestion to organ congestion, manifesting as pulmonary edema, intra-abdominal congestion, or renal congestion (Arrigo et al., 2016).

There is no single definitive diagnostic test for clinical congestion, and clinical evidence does not support the use of invasive hemodynamic monitoring in all AHF patients. Instead, laboratory measurements and imaging should be correlated with physical examination findings to establish the presence or absence of fluid overload. Signs and symptoms such as dyspnea, orthopnea, rales, and crackles may indicate pulmonary edema. Intra-abdominal congestion may cause nausea, vomiting, abdominal pain, or hepatomegaly, whereas renal congestion can result in reduced urine output. B-type natriuretic peptide (BNP) is a biomarker released in response to elevated cardiac filling pressures. Other variables may contribute to elevated BNP levels and skew interpretation of positive results, but BNP levels of less than 100 pg/ml or N-terminal pro-BNP (NT-proBNP) levels of less than 300 ng/ml have a high negative predictive value for ruling out a diagnosis of AHF. Chest radiograph (CXR) may be useful for detecting lung congestion, although it is important to remember that up to 20% of patients with congestion will have a normal CXR. Lung ultrasonography may be a more effective imaging alternative for ruling out pulmonary

edema. Increased width and reduced collapsibility of the vena cava on echocardiography may provide additional evidence of elevated right atrial pressures and systemic congestion (Arrigo et al., 2016; Mullens et al., 2019).

Management of warm/wet AHF patients depends heavily on appropriate management of fluid overload. Diuretics may be utilized if a net positive fluid balance is suspected. If the fluid overload is deemed to be due to acute redistribution, vasodilators would be more appropriate (Arrigo et al., 2016; Kurmani & Squire, 2017).

Cold/Wet: Cardiogenic Shock

The "cold and wet" category of heart failure describes patients presenting with fluid overload or congestion, as defined earlier, but with poor perfusion from a reduced cardiac output. Signs and symptoms of inadequate perfusion include cool extremities, syncope or dizziness, a narrow pulse pressure, confusion, prolonged capillary fill time, oliguria, and/or pale skin (Ponikowski et al., 2016). Potential causes of this AHF syndrome include myocardial infarction, myocarditis, right ventricle failure, Takotsubo syndrome, cardiomyopathies, and tamponade (Chioncel et al., 2020). When patients fall in the category of this AHF syndrome, they are typically described as being in cardiogenic shock. They will have both increased systemic vascular resistance and pulmonary capillary wedge pressure with a reduced cardiac index (Vahdatpour, Collins, & Goldberg, 2019). Unfortunately, it can be difficult to obtain this objective patient data in the emergency department without the placement of a pulmonary artery catheter. However, there are still some objective measurements that can be obtained in addition to the tests mentioned earlier.

Laboratory signs of inadequate perfusion include a metabolic acidosis as seen on an arterial or venous blood gas, an elevated serum lactate level greater than 2 mmol/L, and an increased serum creatinine level from baseline. In patients with hemodynamic instability, an echocardiogram is also recommended to evaluate cardiac structure and function to determine underlying diagnosis (Chioncel et al., 2020; Ponikowski et al., 2016).

The 1-year mortality rate for this AHF syndrome has been reported as high as 60% (Vallabhajosyula et al., 2020). Therefore, it is essential that patients with this presentation receive an urgent, thorough workup and optimal management of the triggering cause. Pharmacotherapy for this AHF syndrome, in addition to diuretic therapy for congestion as mentioned earlier, is typically dependent on a patient's blood pressure. Patients with a systolic blood pressure (SBP) of greater than 90 mmHg can be treated with vasodilators and inotropes in refractory cases. Hypotensive patients, with an SBP of less than 90 mmHg, though, require inotropic support with vasopressor support as needed (Kurmani & Squire, 2017). These cutoff values should not be considered absolute but rather used as a guide with the patient's baseline blood pressure in mind (Long, Koyfman, & Gottlieb, 2018).

Sympathetic Crashing Acute Pulmonary Edema

Sympathetic crashing acute pulmonary edema (SCAPE) is a life-threatening subset within hypertensive AHF that develops rapidly, over minutes to hours. This presentation occurs as a result of an abrupt release of catecholamines, thereby increasing venous and arterial tone, activation of the renin–angiotensin–aldosterone system, and leads to flash pulmonary edema (Agrawal, Kumar, Aggarwal, & Jamshed, 2016). Diagnosis of this subset is based on clinical assessment of the patient. Here, patients will have rapidly developing dyspnea, restlessness, diaphoresis, hypoxia, tachycardia, and hypertension, usually with an SBP of than 180 mmHg (Agrawal et al., 2016).

Upon examination of these patients, bilateral crepitations are present on chest auscultation (Agrawal et al., 2016). The presence of B-lines over superior anterior lung fields as seen with the use of point-of-care lung ultrasonography can also rapidly diagnose SCAPE (Paone, Clarkson, Sin, & Punnapuzha,

2018). Point-of-care lung ultrasonography is preferred over CXR as it has greater sensitivity and specificity for pulmonary edema (Maw et al., 2019).

Management of SCAPE entails emergent initiation of high-dose vasodilators to decrease afterload and noninvasive positive pressure ventilation to redistribute fluid from the pulmonary space. These interventions have been shown to prevent hemodynamic decompensation and endotracheal intubation, intensive care unit (ICU) admission, and mortality (Agrawal et al., 2016; Silvers, Howell, Kosowsky, Rokos, & Jagoda, 2007; Stemple et al., 2021). Loop diuretics are not indicated in this patient population due to their delayed onset of action of 30 min to 2 hr and the fact that most patients with SCAPE are euvolemic (Stemple et al., 2021).

TREATMENT

Diuretics

When AHF patients present with congestion due to volume overload, the only way to solve their underlying problem is either to use ultrafiltration with mechanical renal replacement techniques or to increase renal natriuresis and diuresis with pharmacological agents. Diuretics are a broad class of drugs with varying sites of action that facilitate the renal elimination of sodium and therefore water in an effort to reduce total body fluid volume (Mullens et al., 2019). Experts agree that timely administration of diuretics relieves symptoms and may improve patient outcomes, but whether there are improved clinical outcomes when diuretics are given within a specific door-to-administration time in the emergency department is a topic of current clinical debate (Matsue et al., 2017; Park et al., 2018).

The management of congestion in AHF begins with loop diuretics. Patients with insufficient response to maximal doses of loop diuretics may require a stepped therapy approach that includes the use of other classes of diuretics, such as thiazides or mineralocorticoid receptor antagonists. The use of these additional agents is typically reserved for care of AHF patients beyond the emergency department and is thus beyond the scope of this review (Mullens et al., 2019).

Loop diuretics are the workhorses of fluid management in AHF (see Table 2). They act by inhibiting the reabsorption of sodium in the loop of Henle, where approximately 25% of renal sodium absorption typically occurs. As they also prevent the reabsorption of potassium, these drugs are often referred to as "potassium wasting" diuretics (Mullens et al., 2019). Loop diuretic agents include furosemide, torsemide, bumetanide, and ethacrynic acid. It is important to note that ethacrynic acid should be reserved for use in patients with sulfa-sensitivities, as it is the only loop diuretic that does not contain sulfa but has a higher risk for toxicities than other agents (Somberg & Molnar, 2009).

The clinical activity of loop diuretics is dependent on the ability of the drug to be absorbed (if given orally) and distributed to its site of action in the kidney. Furosemide has a slow and notoriously variable oral absorption. Bumetanide and torsemide are much more rapidly and completely bioavailable, but their absorption is limited by the intra-abdominal congestion often present in AHF. In addition, loop diuretics require a threshold amount of drug to be present before diuresis occurs. Slowed absorption of orally administered drug may prevent efficient attainment of this diuretic threshold and delay clinical benefit. For these reasons, loop diuretics should be administered intravenously. Of note, torsemide is not available in an intravenous preparation in the United States.

It is important to note that certain conditions may predispose patients to diuretic

Table 2. Equivalent loop diuretic doses

	Oral	Intravenous
Furosemide	80 mg	40 mg
Torsemide	20 mg	–
Bumetanide	1 mg	1 mg

resistance. Concomitant nonsteroidal anti-inflammatory drug use, chronic kidney disease, and home diuretic use can, by various mechanisms, decrease patient response to loop diuretics. Thus, some degree of patient-specific dose tailoring may be required. Patients taking loop diuretics at home should be given 2.5 times their home dose intravenously. For example, if a patient takes 40 mg of oral furosemide at home, they should receive 100 mg intravenously. Loop diuretic-naive patients should be given an initial dose of furosemide 40 mg intravenously or bumetanide 1 mg intravenously (see Table 3). Furosemide intravenous bolus doses may be given at a rate of 20–40 mg/min. For furosemide intravenous doses of 160 mg or more, an intermittent infusion should be utilized to reduce risk of ototoxicity. Bumetanide may be administered intravenously at a rate of 0.5–1 mg/min. Onset of action for intravenous furosemide and bumetanide is approximately 5 min (Felker et al., 2020; Mullens et al., 2019).

Vasodilators

Vasodilators have become a mainstay of treatment of AHF patients who are hypertensive and dyspneic (Levy, Laribi, & Mebazaa, 2014). In addition, this drug class can be beneficial in reducing systemic vascular resistance (Levy et al., 2014). This is especially important in SCAPE patients as the primary goal of therapy is to reduce afterload to stop the sympathetic surge (Agrawal et al., 2016). Nitrates only trail diuretics as the most frequently given class of medication in AHF patients according to the Acute Decompensated Heart Failure National Registry (ADHERE) and Acute Heart Failure Global Survey of Standard Treatment (ALARM-HF) (Singh, Laribi, Teerlink, & Mebazaa, 2016).

Nitrovasodilators provide an exogenous source of nitric oxide that binds to soluble guanylate cyclase forming cyclic guanosine monophosphate, leading to smooth muscle relaxation. Lower doses have been associated with more venodilation, reducing preload, whereas increased doses lead to arterial vasodilation, reducing afterload and relieving pulmonary congestion (Levy et al., 2014). Based on the 2013 ACCF/AHA Guideline for Management of Heart Failure, if symptomatic hypotension is absent, intravenous nitroglycerin, nitroprusside, or nesiritide (no longer available in the United States) may be considered an adjuvant to diuretic therapy for relief of dyspnea in patients admitted with ADHF (Yancy et al., 2013). Nitrate use is contraindicated with the recent use of phosphodiesterase Type 5 inhibitors (e.g., sildenafil, tadalafil) in hypertrophic obstructive cardiomyopathy and hypotension (Hsieh, Lee, Kao, Hsu, & Chong, 2018; Kuo & Peacock, 2015). These agents will be the focus of this review as other agents such as angiotensin-converting enzyme inhibitors are reserved for refractory cases despite nitrovasodilator use (Long et al., 2018).

Nitroglycerin

Nitroglycerin has become a workhorse for hypertensive, fluid-overloaded AHF and SCAPE patients. It can acutely lower left ventricular filling pressure through venodilation at lower doses, which will reduce pulmonary congestion (Lindenfeld et al., 2010). At higher doses

Table 3. Initial doses of loop diuretics

Loop diuretic at home	2.5 times the oral home dose, given intravenously
Loop diuretic naive	Furosemide 40 mg intravenously or bumetanide 1 mg intravenously or ethacrynic acid 50 mg intravenously[a]

Note. From Felker et al. (2020); Somberg et al. (2009).
[a]Only to be used in sulfa-sensitive patients.

(greater than 100 mcg/min), it will reduce afterload and increase stroke volume and cardiac output (Lindenfeld et al., 2010; Stemple et al., 2021). Nitroglycerin may also improve coronary blood flow, making it an attractive agent in AHF from ischemia or myocardial infarction (Lindenfeld et al., 2010). Dosing of the medication is variable and dependent on the indication of AHF or SCAPE. Adverse effects include headache, abdominal discomfort, and hypotension, which occur more often in volume-depleted patients (Agrawal et al., 2016; Lindenfeld et al., 2010). Tachyphylaxis may also develop within 24 hr in up to 20% of patients (Yancy et al., 2013).

When treating hypertensive, congested AHF patients, intravenous nitroglycerin is preferred over other routes for ease of titration to symptomatic relief. However, a 0.4 mg sublingual nitroglycerin tablet can be administered until intravenous access is obtained or in those without respiratory distress (Long et al., 2018). Starting doses of continuous infusions are variable within the literature. Drip initiation of 5–10 mcg/min and titrations by 5–10 mcg/min every 3–5 min as needed for patient response have been historically reported (Hollenberg, 2007). In more recent literature, there has been support of starting infusion rates at greater than 100 mcg/min with rapid titration (Hunter, Martindale, Abdel-Hafez, & Pang, 2017; Kuo & Peacock, 2015). If not starting an infusion, bolus dosing of 2 mg intravenously every 3 min may decrease endotracheal intubation, need for ICU admission, and mortality (Cotter et al., 1998; Levy et al., 2007; Sharon et al., 2000).

For the treatment of SCAPE in the emergency department, utilization of high-dose nitroglycerin along with noninvasive positive pressure ventilation has been shown to reduce endotracheal intubation and ICU admission (Paone et al., 2018; Stemple et al., 2021). Sublingual form use has been reported until intravenous access is gained (Agrawal et al., 2016). Although earlier studies reported an infusion with starting rates between 10 and 20 mcg/min, more recent literature displayed successful patient outcomes when starting rates were as high as 400 mcg/min (Agrawal et al., 2016; Paone et al., 2018; Stemple et al., 2021). Paone et al. (2018) reported a successful patient case in which intubation was avoided after utilization of their emergency department protocol for high-dose nitroglycerin. The protocol included an infusion starting rate of 400 mcg/min in adult patients presenting to the emergency department and one of the following criteria: tachypnea (respiratory rate [RR] more than 30 breaths/min), significant dyspnea (use of accessory muscles of respiration or air hunger), significant hypoxia (SpO_2 90% or less on room air or 95% on supplemental oxygen), presentation with pulmonary rales or B-lines over superior anterior lung fields on bedside ultrasound scan, and/or SBP of greater than 160 mmHg or mean arterial pressure (MAP) of 120 or more (Paone et al., 2018). Titration by 50 mcg/min every 5 min occurred until symptomatic improvement, defined as at least two of the following: resolution of tachypnea (RR reduction by 25% of initial reading or RR 24 or less), resolution of dyspnea (use of accessory muscles/air hunger), resolution of hypoxia (SpO_2 90% or more on room air or 95% on supplemental oxygen), and SBP of less than 160 mmHg or MAP of less than 120 mmHg (Paone et al., 2018). Stemple et al. (2021) more recently reported four successful patient cases where nitroglycerin infusions were started at 200–400 mcg/min and titrated up to a maximum of 800 mcg/min until symptom improvement was achieved. The infusions were slowly titrated off with no adverse effects occurring (Stemple et al., 2021).

Nitroprusside

Nitroprusside has a balanced effect on ventricular preload and afterload reduction. This then leads to a reduction in left ventricle filling pressure and increase in stroke volume. AHF patients with left ventricle dysfunction or those with mitral regurgitation may benefit from the use of this agent (Lindenfeld

et al., 2010). For AHF, the initial infusion rate can range between 5 and 10 mcg/min, with dose increases every 5 min as needed up to 300 mcg/min (Lindenfeld et al., 2010; Rhoney & Peacock, 2009). Prolonged use of this agent in patients with renal dysfunction and at doses exceeding 400 mcg/min for over 72 hr should be discouraged because of the potential risk of thiocyanate toxicity (Lindenfeld et al., 2010; Long et al., 2018). Nitroprusside could be a potential alternative to nitroglycerin in the management of SCAPE; however, there are currently no data to support its use (Stemple et al., 2021).

Inotropes

Approximately 10% of hospitalized AHF patients will have hypotension and organ hypoperfusion secondary to significantly decreased cardiac output (Abraham et al., 2005). In these patients, inotropic agents may be used to increase cardiac output and restore perfusion if patients are unresponsive to other treatments. Signs and symptoms seen with hypoperfusion include hypotension, tachycardia, fatigue, decreased urine output, and cool extremities. In a state of hypoperfusion, patients will likely be unresponsive to diuretic therapy secondary to decreased blood flow to the kidneys.

Inotropes are far from benign therapies, with a host of possible complications associated with their use. As a result, inotropes should not be used routinely for all patients presenting with AHF. These agents are associated with tachyarrhythmias, myocardial ischemia, and possible worsening of hypotension. According to the ADHERE, patients receiving vasoactive therapy have higher rates of in-hospital mortality as well as increased hospital and ICU/coronary care unit length of stay (Abraham et al., 2005).

The most commonly recommended inotropic agents in AHF are dobutamine and milrinone, though patients may require therapy with dopamine or other vasoactive agents such as norepinephrine or epinephrine based on their clinical presentation and etiology. Dobutamine and milrinone may have similar physiological responses, but their mechanisms and pharmacokinetics are quite different and should be selected on the basis of specific patient parameters (see Table 4).

Dobutamine

Dobutamine is a synthetic catecholamine that acts on β_1, β_2, and α_1 receptors. The primary effect seen in AHF is increased contractility of the heart secondary to β_1 stimulation within the myocardium. Dosing for dobutamine in AHF ranges from 2.5 to 20 mcg/kg/min. At lower doses, the stimulation of β_2 and α_1 receptors within the vasculature produces vasodilation. The combined effect seen is increased cardiac output and reduced afterload. At increased doses of dobutamine (greater than 5 mcg/kg/min), vasoconstriction will begin to occur as α_1 stimulation increases (Yancy et al., 2013). A decreased effect may be seen in patients on chronic β-blocker therapy, which is standard in the management of

Table 4. Comparison of commonly used inotropes

Inotrope	Mechanism	Dosing	BP effect	Inotropy	Vasoconstriction	Vasodilation
Dobutamine	β_1 greater than β_2 greater than α_1	2.5–20 mcg/kg/min	+	++	+[a]	+
Milrinone	PDE3 inhibition	0.125–0.75 mcg/kg/min	−	+	Neutral	+

Note. BP = blood pressure; PDE3 = phosphodiesterase-3.
[a]Effect seen at doses of greater than 5 mcg/kg/min.

CHF (Metra et al., 2002). Dobutamine should only be considered for short-term use, given an increased risk of mortality compared with other treatment modalities, such as diuretics and vasodilators (Wang, Zhu, & Shan, 2015). In addition, patients may develop tolerance to dobutamine therapy even during short courses (Metra et al., 2002).

Milrinone

Milrinone inhibits the phosphodiesterase-3 enzyme (PDE3). By inhibiting PDE3, intracellular cyclic adenosine monophosphate (cAMP) is not degraded by the enzyme, causing an accumulation. As cAMP accumulates, protein kinase A is activated, which allows an influx of calcium ions into myocardial cells. This influx of calcium is what produces the positive inotropic effect of milrinone, a mechanism that is completely independent of β receptors. It is worth noting that this same mechanism occurs within the smooth muscle cells of the pulmonary and peripheral vasculature, leading to vasodilation. Dosing for milrinone in AHF ranges from 0.125 to 0.75 mcg/kg/min. As with dobutamine, data from the ADHERE indicate an increased mortality risk with milrinone compared with other treatment options (Abraham et al., 2005). The Outcomes of a Prospective Trial of Intravenous Milrinone for Exacerbations of Chronic Heart Failure (OPTIME-CHF) looked at milrinone in addition to standard care. The authors concluded that milrinone was associated with arrhythmias and prolonged hypotensive episodes with no significant benefit on mortality or hospitalizations (Cuffe et al., 2002). An additional subgroup analysis of these data concluded that there was even an increased risk of mortality in patients with an ischemic etiology of their heart failure (Felker et al., 2003).

MONITORING

As is the case with other critically ill patients who present to the emergency department, monitoring should start as soon as possible. Vital signs, an electrocardiogram, and urine output are essential initially and may be frequently reassessed on the basis of the patient's signs, symptoms, diagnosis, and treatment plan. The initial laboratory assessment should include BNP and troponin levels to assess for other cardiac complications, such as ischemia. A CXR should be obtained in patients presenting with dyspnea. Point-of-care ultrasonography (POCUS) is becoming a staple within the emergency department and is beginning to gain utility in the diagnosis of AHF in patients with dyspnea. POCUS may also be useful in assessing the efficacy of diuretic therapy (Qaseem et al., 2021). Arterial and/or central lines may be warranted on the basis of hemodynamic status, requested blood samples for monitoring, and prescribed treatments (e.g., inotropes, vasopressors) (Vazir & Cowie, 2012). Pulmonary artery catheterization is no longer recommended; however, there may be clinical scenarios in which it may be useful such as when congestion and perfusion are difficult to assess and the patient is not adequately responding to treatment (Binanay et al., 2005; Shah et al., 2005).

CONCLUSION

AHF often has a rapid onset of signs and symptoms and is associated with high morbidity and mortality. For patients who present to the emergency department, rapid assessment and management are essential. Clinical scenarios will vary from patient to patient, and treatment for each patient should be based on the presentation (i.e., severity of signs and symptoms). In mild cases, the primary treatment is usually intravenous diuretics. For those who present with flash pulmonary edema, hypotension or other more severe signs and symptoms, vasodilators, inotropes, or vasopressors may be warranted.

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