Approach to Acute Heart Failure in the Emergency Department

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ABSTRACT

Acute heart failure (AHF) patients rarely present complaining of 'acute heart failure.' Rather, they initially present to the emergency department (ED) with a myriad of chief complaints, symptoms, and physical exam findings. Such heterogeneity prompts an initially broad differential diagnosis; securing the correct diagnosis can be challenging. Although AHF may be the ultimate diagnosis, the precipitant of decompensation must also be sought and addressed. For those AHF patients who present in respiratory or circulatory failure requiring immediate stabilization, treatment begins even while the diagnosis is uncertain.

The initial diagnostic workup consists of a thorough history and exam (with a particular focus on the cause of decompensation), an EKG, chest X-ray, laboratory testing, and point-of-care ultrasonography performed by a qualified clinician or technologist. We recommend initial treatment be guided by presenting phenotype. Hypertensive patients, particularly those in severe distress and markedly elevated blood pressure, should be treated aggressively with vasodilators, most commonly nitroglycerin. Normotensive patients generally require significant diuresis with intravenous loop diuretics. A small minority of patients present with hypotension or circulatory collapse. These patients are the most difficult to manage and require careful assessment of intra- and extra-vascular volume status. After stabilization, diagnosis, and management, most ED patients with AHF in the United States (US) are admitted. While this is understandable, it may be unnecessary. Ongoing research to improve diagnosis, initial treatment, risk stratification, and disposition may help ease the tremendous public health burden of AHF.

Abbreviations:

ACC/AHA – American College of Cardiology / American Heart Association

ACEI – ace inhibitor

ACS – acute coronary syndrome

AHF – acute heart failure

ASCEND-HF - Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure

BNP – brain natriuretic peptide

BP – blood pressure

ED – emergency department

EF – ejection fraction

EKG – electrocardiogram

ESC – European Society of Cardiology

HF – heart failure

ICU – intensive care unit

IV – intravenous

IVC – inferior vena cava

IVC-CI – inferior vena cava collapsibility index

LOS – length of stay

NIPPV – non-invasive positive pressure ventilation

NTG – nitroglycerin

SBP – systolic blood pressure

STEMI – ST-elevation myocardial infarction

US – United States

Keywords:
Heart failure; acute heart failure; decompensated heart failure; emergency department

Diagnosis and management begins in the emergency department (ED) for the vast majority of patients hospitalized with acute heart failure (AHF). Unfortunately, the evidence base for ED management is limited.[1,2] This lack of evidence, combined with the heterogeneity of the AHF patient population, results in tremendous variability in clinical practice. The potential impact of ED management is significant, as diagnostic delay or sub-optimal treatment may have significant downstream consequences. Perhaps the costliest ED management decision is deciding who does or does not require hospitalization, the most expensive resource in healthcare.[3,4] As epidemiology and pathophysiology of AHF are covered elsewhere, this review focuses on initial ED management.

Stabilization

Occasionally, patients with possible AHF present in extremis or near respiratory failure. In these instances, the diagnostic work up and management occur in parallel. Importantly, the precipitating cause of the patient's dramatic presentation must be simultaneously identified and treated. Arrhythmias, infection, and acute coronary syndromes are just a few potential precipitants. The classical teaching of ensuring "Airway, Breathing, and Circulation" first is worth reiterating.

Obtunded patients with severe respiratory failure will likely require endotracheal intubation and mechanical ventilation.[5] For patients with respiratory distress who are awake and cooperative, early initiation of non-invasive positive pressure ventilation (NIPPV) significantly decreases mortality and need for intubation.[6] NIPPV can be instituted even if the diagnosis of AHF is in doubt, as evidence suggests benefit even in undifferentiated severe

dyspnea.[7] The increased intra-thoracic pressure from NIPPV may decrease blood pressure (BP), but this is uncommon clinically and more likely with endotracheal intubation. Once the patient has been stabilized, the focus turns to diagnosis.

Initial Diagnosis and Assessment

Fortunately, most AHF patients do not present in extremis. Establishing the diagnosis is the sine qua non of medicine, but is not always easy. It is worth noting the myriad of different patient complaints: Fatigue, dizziness, shortness of breath, chest pain, weakness, exercise intolerance, swelling, and weight gain are all symptoms prompting consideration of AHF as the cause.

The clinical presentation of AHF varies widely, ranging from mildly worsening heart failure, de novo or new onset HF, to overt cardiogenic shock, to hypertensive flash pulmonary edema. Despite the high prevalence of AHF in the ED setting, misdiagnosis occurs in 14-29% of patients[8-11]. Heterogeneous pathophysiology and phenotypic expression, varied underlying causes and precipitants, and substantial co-morbid burden underlie the challenges of diagnosing this syndrome. Nevertheless, timely and accurate diagnosis of AHF is critical to preventing delays in treatment, which have been associated with increased risk of inpatient mortality and longer length of stay (LOS).[12-14].

Unfortunately, no single historical variable, symptom, physical exam finding, biomarker, or imaging modality is sensitive enough to sufficiently exclude the diagnosis of AHF. Dyspnea is the most common symptom prompting patients with AHF to seek care[15], and is most often due

to vascular congestion. Discriminating AHF from other causes of dyspnea, however, remains challenging, especially for those patients without a preexisting diagnosis of heart failure (HF) and those with comorbidities, such as chronic obstructive pulmonary disease. Symptoms classically associated with HF, such as orthopnea and paroxysmal nocturnal dyspnea are reported by only half of patients with AHF and are less than 75% specific for the diagnosis[16]. The physical exam finding with the highest likelihood ratio (LR+) is an S3 gallop (LR+ 4.0 [95%CI 2.7-5.9]), but the absence of this finding has minimal effect on changing the pre-test probability of AHF (LR- 0.91 [95%CI 0.89-0.95])[16]. Jugular venous distension and the hepatojugular reflex are more specific, but are insensitive and dependent on the examiner. Despite knowledge that congestion is the primary underlying cause of patient signs and symptoms, measuring congestion with a high degree of intra and interobserver reliability remains challenging.[17,18]

Natriuretic peptides are the most useful biomarkers for excluding the diagnosis of AHF[19]. Cutoff points of 100 pg/mL and 300 pg/mL for brain natriuretic peptide (BNP) and N-terminal (NT)-proBNP, respectively, substantially reduce the post-test probability of AHF (LR- 0.1) in patients presenting to the ED with dyspnea[16]. Very high BNP values are modestly helpful in ruling in AHF, but intermediate values (100-800 pg/mL for BNP) lack diagnostic specificity. Likelihood ratios associated with even the most elevated NT-proBNP values only modestly favor the diagnosis of AHF[16]. The specificity of these biomarkers above proposed cutoff points are limited by renal dysfunction and advanced age[19]. Other conditions to consider in patients with modestly elevated BNP values are acute respiratory distress syndrome[20], pulmonary embolism[21,22], pulmonary hypertension[23], and valvular heart disease[24]. While natriuretic peptides provide additive diagnostic value beyond clinical and historical variables[25-27], several

studies have failed to demonstrate differences in patient-centered clinical outcomes beyond hospital LOS[28,29] with the addition of diagnostic BNP testing.

In addition to a basic metabolic profile and complete blood count, troponin testing should be considered in AHF patients. Occasionally, troponin testing may uncover occult acute coronary syndrome (ACS), an important precipitant of AHF.[30,31] Importantly, an elevated troponin does not rule in ACS, as many AHF patients may have troponin release.[32] With the recent approval in the US of higher sensitivity assays, a greater proportion of AHF patients will likely be identified with "abnormal" troponin values.[33] Troponins also add prognostic information,[30] but should not be considered a diagnostic test to rule in or out AHF. However, higher sensitivity assays may demonstrate troponin release as an integral part of the AHF syndrome; one recent study demonstrated 98% of AHF patients have measurable troponin, with 81% above the 99th percentile.[34] Both BNP and troponin are recommended in guidelines for the assessment and risk-stratification of the AHF patient.[35,36]

Given the limitations of laboratory testing in isolation, imaging plays a significant role in the diagnostic approach to AHF. The chest radiograph is considered a key component of the diagnostic workup. Chest radiography can identify vascular engorgement, hilar redistribution, interstitial edema, and alveolar edema as well as alternative causes of dyspnea[37]. While highly specific for AHF, these radiographic findings are poorly sensitive.[25,38,39] One series found that cardiomegaly alone had moderate sensitivity (79%) and specificity (80%).[25]

Lung ultrasound has emerged as a useful point-of-care tool for identifying pulmonary edema and diagnosing AHF.[40,41] Sonographic detection of pulmonary edema is based on the identification of vertical artifacts called B-lines, which are thought to result from the

reverberation of sound waves off of fluid-filled pulmonary interstitium. When distributed diffusely in the proper clinical setting, B-lines represent cardiogenic pulmonary edema. A positive lung ultrasound study, defined as two or more bilateral thoracic zones with ≥ 3 B-lines, has good discriminatory value with a LR+ of 7.4 (95% CI 4.2 -12.8). A negative lung ultrasound study substantially lowers the probability of AHF (LR- 0.16 [95% CI 0.05-0.51])[16]. The extent of pulmonary edema can also be semi-quantitatively measured by the sum of the number of B-lines with high inter-rater reliability[42-44]. B-line severity has been shown to correlate with other measures of pulmonary congestion[45] and with the severity of AHF[46,47]. The ESC HF guidelines now includes lung ultrasound as a recommended diagnostic test to confirm pulmonary congestion[37].

Point-of-care ultrasound -determined estimates of intravascular volume and right atrial pressures can be made by measuring the diameter of the inferior vena cava (IVC) and percentage change in IVC diameter during the respiratory cycle. An IVC diameter that fails to substantially decrease with inspiration is considered to have a low collapsibility (or caval) index (IVC-CI), reflecting volume overload and high RAP. The diagnostic performance of different cutoff values for IVC collapsibility index, ranging from 20%-50% have been tested in dyspneic patients presenting to the ED[48-51]. Sensitivities of 80% or greater were achieved in studies that used an IVC-CI cutoff of 33% or greater[49-51]. Specificities associated with these cut-offs ranged from 81%-87%. Alternative causes of a plethoric IVC include tricuspid regurgitation, pulmonary embolism, pulmonary hypertension, and right ventricular infarction.

Echocardiography is integral to the diagnostic workup of HF. While formal echocardiography is rarely available rapidly in the ED, focused cardiac ultrasound in the hands

of trained emergency physicians can be used as a point-of-care tool to assess global systolic dysfunction. Qualitative visual estimations of reduced versus normal ejection fraction (EF) can be made by assessing the inward movement of the interventricular septum and inferior wall of the left ventricle during systole and by observing the degree of excursion of the anterior leaflet of the mitral valve toward the interventricular septum during diastole. These qualitative assessments correlate with more formal, quantitative echocardiographic measures of EF.[52,53] Reduced EF identified by emergency physicians using focused cardiac ultrasound discriminates AHF from other causes of dyspnea with sensitivities ranging from 77-83% and specificities ranging from 74-90% [48,54,55]. However, sonographic assessments of dyspneic patients limited to this single variable would fail to identify HF patients with preserved EF. Identification of a restrictive pattern of diastolic filling using pulsed Doppler analysis of mitral inflow as a surrogate measure of elevated filling pressures assists in the diagnosis of AHF (LR+ 8.3 [95%CI 4.0-16.9])[54]. Acquisition and interpretation of mitral inflow and tissue Doppler data is currently beyond the scope of ED physicians who lack formal fellowship training. Diagnostic approaches that integrate lung, cardiac, and IVC assessments increase the specificity of diagnosing AHF in the ED beyond clinical gestalt, biomarkers, and lung ultrasound alone[48,50,55]. Further research is needed to help delineate the role of focused cardiac ultrasound in the workup of AHF and how different sonographic assessments can be incorporated into diagnostic algorithms. Importantly, point of care US does not replace formal echocardiography.[56]

Initial Management

Once the diagnosis is made, presenting phenotype and cause of exacerbation guides initial treatment. As mentioned earlier, first assuring respiratory and hemodynamic stability is paramount. (See Table 1 for Goals of ED Management) While addressing the patient's respiratory status, the precipitant of AHF should be sought and treated. For example, rapid atrial fibrillation (AF), ACS, pulmonary embolism, underlying infection or dietary indiscretion can all trigger AHF[57]. Often the precipitant is unclear or challenging to identify. Complicating matters, co-morbid conditions may cloud the picture or add challenges to management. A classic example is the patient with both chronic obstructive pulmonary disease and AHF; wheezing may be bronchial or 'cardiac wheezing', and one exacerbation may incite the other. While simultaneous treatment frequently occurs clinically, untoward effects (such as inciting AF with beta agonists) may be detrimental.

Table 1

Goals of ED Management (although written sequentially, steps may occur simultaneously)

1. Ensure stability of Airway, Breathing, and Circulation or resuscitate immediately
2. Identify and treat any other potential life threats (i.e. STEMI, dysrhythmias)
3. Ensure diagnosis of AHF and begin treatment.
4. Identify the precipitant of AHF and modify treatment if necessary.
5. Consider the potential contribution of other co-morbid conditions and whether they require urgent treatment
6. Re-evaluate patient to ensure improvement in symptoms, hemodynamics, and clinical impression
7. Risk-stratify patient
8. Disposition planning (admission, observation, discharge)

Initial Classification

As an initial guide, we recommend grouping patients with suspected AHF by systolic BP (SBP).[58,59] As evidenced by registries, SBP is often high (>140mmHg) at the time of presentation.[60] We recommend using cutpoints of > 140mmHg, 100-140 mmHg, and < 100 mmHg to guide initial selection of pharmacologic therapy.[58,59] While there is considerable overlap, simple categorization aids the busy clinician. As such, it is reasonable to assume the predominant pathophysiologic derangement in a patient based on presenting SBP. Notably, not all patients present with total volume overload; the prototypical example is the flash pulmonary edema patient.[61-63] Such patients have also been described as 'vascular failure' or 'volume redistribution' patients.[64-66] These patients most commonly present with elevated SBP. Of note, the latest ESC HF guidelines also support dividing patients based on 'cardiac' (fluid overload predominates) vs. 'vascular' (hypertension predominates) phenotypes.[1]

Initial Therapy

At the present time, no AHF therapy receives a Level I, Class A recommendation from guidelines,[1,2] highlighting the lack of robust evidence from randomized studies. Therapies used today are largely the same as those employed 4 decades ago (Table 2). Rotating tourniquets and phlebotomy are no longer used; whether this represents a major advance is debatable. Importantly, lack of high quality evidence from robust, randomized controlled trials does not equate with ineffectiveness in achieving symptom relief, hemodynamic improvement, and decongestion; all important targets of therapies.

Table 2: Therapeutic Options for the Early Treatment of AHF – A Historical Perspective	
1974[67]	2017
Sit the patient upright	Sit the patient upright
Oxygen	Oxygen
Positive pressure ventilation	Positive pressure ventilation
Morphine	Morphine
Diuretics	Diuretics
Intra-aortic balloon pump	Intra-aortic balloon pump
Phlebotomy	**Inotropes**
Rotating tourniquets	**Vasodilators / Nesiritide**

The Hypotensive AHF Patient

Shock due solely to worsening HF rarely occurs relative to other types of AHF.[68] Given its relatively uncommon presentation combined with the complexity of these patients' underlying pathophysiology, precipitant, cardiac structure, function, and resultant hemodynamic status, management can be challenging. Patients with advanced HF may present with alarmingly low SBP. This may, in fact, reflect their baseline SBP. Even when resuscitating shock, a common mistake is attempting to normalize SBP and HR to values seen in those with baseline normal cardiac structure and function. However, for patients with severely reduced EF, a 'normal' SBP may be unattainable, and tachycardia may be the key contributor to cardiac output.

For patients with low SBP, administering a fluid bolus is nearly a reflexive action. But in the setting of hypoperfusion secondary to heart failure rather than hypovolemia, this may result in worsening pulmonary edema. On the other hand, infection and overdiuresis are common precipitants that may respond quite well to fluid. At the bedside, assessing volume status is challenging, especially in patients with advanced HF. As noted above, ultrasound may be useful, but response to initial treatment will often be the best guide to

subsequent management. Although rarely applied in the ED setting due to concerns of precipitating circulatory failure, AHF patients with low SBP who are congested may require decongestive therapies. Optimizing volume status through diuresis and vasodilation may lead to significant clinical improvement. In some refractory cases, inotropes and vasopressors are required to augment cardiac output and blood pressure.

Inotropes and Vasopressors

Table 3 shows commonly used inotropes and vasopressors. Although inotropes and vasodilators improve hemodynamics, to date, none are associated with better clinical outcomes. In fact, available inotropes have been associated with harm, though the evidence base is small and inconsistent. [69-72] In terms of vasopressors, there is a paucity of robust data to strongly recommend one vasopressor over another. Subgroup analysis from a large randomized trial found increased mortality in patients with cardiogenic shock who were randomized to dopamine compared with those randomized to norepinephrine.[73]

Table 3	Initial Dose	Infusion range	Recommendation Class (Evidence level)
Dobutamine	2-3 ucg/kg/min	2-20 ucg/kg/min	IIB (Level B
Milrinone		0.375-0.75 ucg/kg/min*	IIB (Level B)
Levosimendan		0.05 -0.2 ug/kg/min*	Not available in US
Dopamine	2-5 ucg/kg/min	2-50 ucg/kg/min	IIB (Level B)
Norepinephrine		0.2 – 1.0 ucg/kg/min	
Nitroglycerin	5-20 ucg/min (rapidly titrate to effect)	5-200 ucg/min (rapidly titrate to effect)	IIB (Level A)
Nitroprusside	5-10 mcg/min	0.25 ucg/kg/min – 10 ucg/kg/min	IIB (Level A)
Nesiritide		0.01 ucg/kg/min*	IIB (Level A)
ACE-I (enalaprilat)	1.25-5mg IV bolus q6 hrs	NA	NA
*Consider bolus dosing			

The Hypertensive Patient

Approximately half of patients admitted with AHF present with hypertension (SBP ≥ 140 mmHg).[60,68] In general, these patients tend to be older, have preserved EF,[60] and present with a more acute onset of symptoms, often less than 24 hours. Pulmonary edema in such patients is more likely to be caused by vascular redistribution than by hypervolemia. As such, vasodilators are the mainstay of treatment.

Of the guideline-recommended vasodilator options [nitroglycerin (NTG), nitroprusside, or nesiritide], we recommend NTG as first line.[74] Clinicians and nurses are familiar with NTG; it can be administered rapidly via sublingual and intravenous routes, and it's inexpensive. Bolus doses of up to 2-3 mg are well tolerated and effective, though many clinicians are reluctant to give such large doses.[75,76] Anecdotally, NTG as an IV drip is often withheld due to the requirement for an intensive care unit (ICU)bed. However, the rapid onset/offset of NTG make it an ideal titratable drug to initiate before transitioning to topical NTG or alternative therapies.

Nitroprusside and nesiritide are alternatives to NTG. As with NTG, neither nitroprusside nor nesiritide has been shown to decrease mortality or morbidity in AHF.[77] However, both are effective vasodilators, with nitroprusside being the more potent. While NTG predominantly acts on the venous circulation until at higher doses, nitroprusside acts rapidly on both the arterial and venous circulation. It may precipitously lower BP; thus careful monitoring is required. Nesiritide is one of the most well studied vasodilators in terms of large randomized controlled trials. After initial concerns regarding safety, a large randomized, controlled trial (ASCEND-HF; n= 7,141) found no relative benefit or harm associated with nesiritide in terms of mortality,

hospital readmission, or dyspnea.[78] It may be a reasonable option if a vasodilator is desired but ICU beds are unavailable.

Despite the lack of compelling evidence supporting acute angiotensin converting enzyme inhibitor (ACEI) use in the ED, these agents are sufficiently used in the setting of AHF to be mentioned in the American College of Emergency Physicians [79]. A common misperception: the benefit of ACEI in chronic HF with reduced EF extends to the acute setting. Lack of evidence does not equal a bad therapy; only that sufficiently powered, well-designed trials have not yet been performed.

Morphine's historic use in AHF continues today. Retrospective observational data suggests an increased risk of death in patients treated with morphine for AHF.[80] As it offers no defined benefit, we recommend against routine morphine use in AHF.

The Normotensive Patient (SBP 100-140mmHg)

AHF patients presenting with SBP ranging from 100-140mmHg rarely arrive to the ED in extremis.[60] The prototypical patient reports an indolent course over days or even weeks, and may report significant weight gain. Decongestion with intravenous (IV) loop diuretics is the primary therapy. Bolus or continuous infusion diuretic administration makes no difference.[81] A randomized trial comparing IV doses of the patient's standard oral dose to larger IV doses (2.5 times the standard oral dose) found that larger doses resulted in more diuresis and marginally better dyspnea over the first 72 hours, but also increased the likelihood of creatinine elevation.[81] In addition to diuretic therapy, low-dose vasodilators should be considered in normotensive patients.

A Hospitalist's Perspective in Brief:

From the hospitalist perspective, whether so many AHF patients warrant admission is debatable.[82] Nevertheless, at the present time, most AHF patients are hospitalized. Thus, it is worth highlighting the different clinical framework between hospitalists and ED physicians for the management of AHF. While risking overgeneralization, ED physicians work with limited data in a fast-paced environment where rapid disposition and ensured access to short-term follow-up are paramount. Thorough diagnostic evaluation of cardiac structure, function, and AHF etiology are secondary objectives for ED physicians. Hospitalists tend to expect greater diagnostic clarity and institute management plans that address comorbid conditions and the long-term consequences of cardiac remodeling.

In regards to clinical management, the use of diuretics is often a major point of contention between hospitalists and ED physicians. IV loop diuretics are the cornerstone of acute therapy for AHF patients. Nearly 90% of patients hospitalized with AHF receive IV loop diuretics in the [83],[68]ACC/AHA (American College of Cardiology/American Heart Association) and ESC guidelines, though large randomized controlled trials have yet to be performed (and it is doubtful they ever will be). Given the absence of other therapies to readily decongest patients, why IV diuretics are withheld or underdosed appears perplexing. Although retrospective studies suggest harm associated with early aggressive IV loop diuretic use, no prospective evidence supports this hypothesis.[84,85]. Withholding IV diuretics in the ED may be perceived by the inpatient teams as delaying patient care.

If diuretics are given in the ED, they are often underdosed. By their very mechanism of action,[86] loop diuretics must be secreted via active transport in the proximal tubules of the kidney. Rather than minimize doses, especially in patients with impaired renal function, higher doses are required to reach the dose-response threshold.[86]

Disposition and Outcomes

Contrary to commonly held belief, most patients who visit the ED are sent home; only 9.3% of the annual 130 million ED visits in the US result in hospitalization. However, nearly all ED patients with AHF are hospitalized. From 2006 to 2011, the annual US hospitalization rate for AHF patients in the ED has consistently been around 85%.[87] Given financial penalties tied to excess re-hospitalization, this admission rate warrants scrutiny.

Administrative data analyses suggest up to 50% of patients with AHF could be discharged or observed briefly and released.[88] AHF is a progressive illness and the short-term prognosis following hospitalization is unacceptably poor. This makes the concept of a low-risk AHF patient difficult for the emergency physician to embrace. Yet within the spectrum of risk, some are lower than others. Identifying patients safe to be sent home from the ED remains challenging, as the majority of risk-stratification work in AHF focuses on defining and characterizing high risk in-patients, making extrapolation to the ED setting challenging.[89] While some risk instruments, such as the AHF Index, EMHRG, STRATIFY, or the Ottawa Heart Failure Index are promising,[90-92] none have gained widespread acceptance, either due to the need for further validation, differences in patient populations, or limited information on outcomes for

discharged patients identified as lower risk.[93] Identifying patients low enough risk for safe discharge from the ED remains a key focus for research.

The absence of high risk features (i.e. low BP, high BNP levels, worsening renal function, elevated troponin, and hyponatremia) does not equal no risk,[89,94,95] but it does equate to lower risk. In the future, absence of myocardial injury by high sensitivity troponin assays may help identify low risk patients.[96] As risk-stratification improves, appropriate selection of patients for treatment in observation or short-stay units in AHF may become easier.[97,98] These units may provide more time to risk stratify patients, gauge response to therapy, provide education, engage case management and social work as needed, reconcile medications, and facilitate close follow up. These tasks are often challenging to complete during a brief ED stay. Furthermore, given the reluctance to discharge lower-risk AHF patients from the ED, the use of observation medicine as a 'bridge' may be more clinically feasible and acceptable to ED physicians.[3,94]

For higher risk patients, hospitalization may offer benefit to improve symptoms, optimize volume status, and ensure initiation of guideline directed chronic medical therapy. AHF pharmacologic therapies, on their own, have not been proven to affect post-discharge outcomes.[2] However, hospitalization may help higher risk patients achieve symptomatic relief, euvolemia/complete decongestion, and medical optimization. Patients with new onset or de novo HF should also be admitted, as potentially reversible or modifiable causes may be identified. These patients will also need education about self-managing their new chronic illness. Overall however, indiscriminate admission is unlikely to translate into patient-centered benefit or justifiable cost. As mentioned previously, identifying who can be safely observed or discharged from the ED remains an unmet need.

Conclusion:

The ED management of AHF centers around diagnosis, stabilization, identification of the precipitant of AHF, initial treatment, and risk-stratification. We recommend initial ED treatment be guided by presenting phenotype but treatment largely centers around diuretics and vasodilators. Although currently available therapies improve symptoms, none definitively improve outcomes. Identification of life saving therapies for the early treatment of AHF remains an unmet need, though whether a short-term treatment can influence longer term post-discharge outcomes remains unclear. As US EDs continue to admit nearly all AHF patients, identifying appropriate low-risk patients for discharge and close follow-up would result in tremendous value to the health care system.

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