Title: Acute Dyspnea and Decompensated Heart Failure

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Key Words (5–8)

Acute heart failure, Emergency department, risk-stratification, observation units

Abstract/Summary [this will be used for indexing services and does not appear with article]

This is the author's manuscript of the article published in final edited form as:

Pang, P. S., Collins, S. P., Gheorghiade, M., & Butler, J. (2018). Acute Dyspnea and Decompensated Heart Failure. Cardiology Clinics, 36(1), 63–72. https://doi.org/10.1016/j.ccl.2017.09.003

The vast majority of patients hospitalized with acute heart failure (AHF) initially present to the emergency department (ED). Correct diagnosis followed by prompt treatment ensures optimal outcomes. We recommend a 6-axis model to ensure comprehensive initial care of the AHF patient.

Key Points (3–5)

- Use of lung ultrasound significantly improves diagnostic accuracy
- Robust evidence to treat AHF in the ED setting remains poor. However, existing therapies improve symptoms and hemodynamics
- Use a framework to ensure optimal management of the AHF patient. We recommend the 6-axis model

Introduction

Few other acute presentations result in such paradoxical outcomes as acute heart failure (AHF). Patients feel substantially better with current treatment, yet outcomes are dismal – up to 1/3rd of hospitalized patients experience death or re-hospitalization within the next 3 months[1] – suggesting initial improvements fail to address the underlying problem. Akin to treating the fever yet ignoring the abscess. Despite substantial efforts, outcomes have improved little in absolute terms over the last 10 years. With over one million hospitalizations annually in the United States,[2] improving outcomes remains the greatest unmet need in AHF management.

Background

The emergency department (ED) is the beginning of hospital based management for over 80% of AHF patients in the United States.[3-5] Patients most commonly present with shortness of breath or dyspnea.[6-8] Given the broad differential for such a chief complaint, correct diagnosis is paramount yet can be challenging, especially for patients with multiple co-morbidities. Since there is no gold standard – heart failure remains a clinical diagnosis – diagnostic accuracy in the ED ranges from 71 to 86%.[9] At times, the patient's condition mandates treatment before a definitive diagnosis is made. Unfortunately, the evidence base for the pharmacologic management of AHF in the ED is poor, with no guideline recommendations meeting the highest Level 1, Class A grade.[10-12] While modern day management has evolved away from rotating tourniquets and blood-letting, the remaining pharmacologic armamentarium for AHF management is

largely the same as 50 years ago.[13] Lack of evidence does not mean current therapies are ineffective, however.

Diagnosis

As mentioned, patients presenting in extremis requiring simultaneous treatment as the diagnosis is confirmed. However, for most patients, sufficient time exists to form an initial diagnostic and treatment plan.

AHF is a clinical diagnosis. Thus, no single blood test, image, historical feature or exam finding absolutely confirms the presence of HF. In fact, the sensitivity of classic HF exam findings is quite poor. An S3 and jugular venous distention are quite specific, however their sensitivity and inter-rater reliability is poor.[14] With the advent of natriuretic peptides, diagnostic accuracy has improved considerably.[15] However, a recent systematic review examining the clinical utility of natriuretic peptides using interval likelihood ratios – to better account for NP as a continuous variable – suggests the diagnostic 'power' of NP lies more in ruling out AHF. To 'rule-in' AHF, lung ultrasound (LUS) is one of the most useful, easy, and rapid tests to perform at the bedside.[9,16] [FIGURE of US B-LINES] Although multiple scoring systems exist to quantify the number of B-lines, the presence of multiple B-lines in at least 2 areas of each hemi-thorax aids in the diagnosis of AHF in the appropriate clinical setting.

Initial Classification

Perhaps more than any other term, heterogeneity best describes the AHF population. In addition to differences in cardiac structure and function, underlying

etiology of HF, and precipitant of AHF, marked differences in patient characteristics, co-morbid conditions, and background therapies, highlights the challenges of a singular, uniform construct to manage all AHF. As discussed in more detail below, we recommend the importance of thorough knowledge of underlying cardiac substrate, precipitants, and amplifying mechanisms of decompensation to best manage manage, accounting for patient differences. Nevertheless, some baseline characteristics are worth highlighting. Patients are older, with an average age of 73 years, with women comprising approximately half of AHF presentations.[17] Of note, nearly half of AHF patients have a relatively preserved (>40%) ejection fraction. Like AHF, no pharmacologic therapy definitively improves outcomes for patients with HF and a preserved ejection fraction (HFpEF).[10,12] This is a fundamental difference compared to HF with reduced EF (HFrEF). However, mineralocorticoid receptor antagonists are a potential notable exception.[18] Irrespective of a reduced vs. preserved EF, the co-morbid burden for AHF is high, particularly for ischemic heart disease, diabetes, atrial fibrillation, and hypertension as well as COPD.

Given the heterogeneity of the patient population and presentation, we propose a construct to aid the acute care provider in initial management. This is taken from the Braunwald and Gheorghiade 6-axis model.[19] Before in-depth discussion of the model, it is important to highlight what we consider less important in the ED setting.

Preserved vs. Reduced EF

No specific mention of HFpEF vs. HFrEF is mentioned. Although these are the most commonly used terms currently, past terms such as diastolic dysfunction and systolic

dysfunction have also been used to broadly describe patients based on EF. For the acute care physician, with a few exceptions noted below, acute management does not significantly differ based on EF. Blood pressure, for example, better captures this difference in EF; few HFpEF patients will present with marked low SBP due to poor pump function. Where knowledge of EF matters most, is in chronic management.

Although knowledge of EF does not commonly change acute management, bedside or formal ECHO may aid in diagnosing the extent and severity of right sided failure, presence of pericardial fusion, and valvular disease. Such knowledge may also aid in immediate management.

6-axis Approach to AHF Management

The 6-axis model is a guide to management, establishing a multi-faceted framework to ensure the clinician considers various key aspects of initial AHF management. Certain aspects may be of greater importance depending on the patient. Importantly, the 6-axis model is intended to be dynamic: re-evaluation is essential. At each point of evaluation and re-evaluation, except for de novo vs. chronic HF, each axis should be considered. For example, initial presentation may differ from pre-discharge in regards to the importance of each axis. (See Figure 2) Response to management or failure to respond, as well as results of testing may yield new insights to further tailor management.

Clinical Severity

Initial clinical impression guides urgency of management. Vital signs are an essential and rapid gauge of severity. Patients who present in extremis require immediate stabilization and resuscitation. Examples include flash pulmonary edema and cardiogenic shock. For both, symptomatic management is important but secondary to identification and treatment of the underlying precipitant.

Blood Pressure

Systolic blood pressure is a known prognostic marker in AHF; higher SBP patients have better outcomes.[1] We recommend further sub-classification based on SBP to guide initial therapeutic choices.[20] Although there is considerable overlap, we use cut-points to aid the clinician: (hypotensive = < 100mmHg, normotensive as 100-140, and hypertensive as > 140mmHg). Most AHF patients present 'warm and wet.'[21] Although the SBP is an excellent guide, it is based on this assumption of 'warm and wet.' Patients who are 'cold and wet' – have systemic vasoconstriction but a low cardiac output – may have poor end organ perfusion despite a relatively normal SBP.

Table 1	**Hypotensive**	**Normotensive**	**Hypertensive**
IV loop diuretics		++++	++
Vasodilators		++	++++
Inotropes	++++		

For the hypotensive patient, assessment of volume status is challenging. Patients with a history of severely reduced ejection fraction may have a low SBP at baseline. Thus, reflexive diagnosis of shock is discouraged. Though rarely done by the emergency

physician, decongestive strategies (i.e. vasodilators and IV loop diuretics) may be used to optimize loading conditions for such patients. These patients are amongst the most challenging to manage acutely.

Heart Rate and Rhythm

For patients with severe systolic dysfunction, HR is the major contributor to cardiac output, given the minimal ability to augment stroke volume. Thus, bradycardias, even if minor, may impair cardiac output. On the other hand, in the setting of a stiff left ventricle (or diastolic dysfunction) a very rapid heart rate may be deleterious. For these patients, reduced filling time in the setting of decrease left ventricle compliance may initiate or propagate AHF symptoms.

The most common dysrhythmia to present in the context of AHF is atrial fibrillation (AF). A history of AF is a risk factor for developing HF and vice-versa. For some patients, their first presentation of AHF may be due to AF with rapid ventricular response. For unstable patients, follow guideline recommended protocols; specifically cardioversion. For most patients however, treatment of AF follows a well-established paradigm of asking the following questions: 1) Rhythm control? 2) Rate control? 3) Risk stratify for stroke.

For patients with a known history of AF, management of AHF itself may lower the heart rate by reducing adrenergic drive. In patients with a preserved EF, HR control is particularly important, given the ‘stiffness’ or lack of compliance of the left ventricle. By definition, a faster HR results in relatively less filling time. With decreased LV compliance, a faster HR may result in worsening signs and symptoms of AHF.

Common rate control agents, such as non-dihydropyridines and beta blockers are commonly used to treat AF in the setting of AHF. Although less commonly used, digoxin should also be considered.[22] For patients with a preserved EF, rate control will improve loading conditions and symptoms. For patients with reduced EF, caution is warranted before reflex use of non-dihydropyridines or beta blockers. Permissive tachycardia may be needed to ensure sufficient cardiac output, given these patients may be unable to augment stroke volume.

Precipitants

Identification and treatment of the precipitant of AHF is fundamental to good AHF management. Contrary to tradition, medication non-compliance and dietary indiscretion are not the most common causes of decompensation.[23] Rather, infection and ischemia are more common. Failure to identify the underlying cause is not just an ED phenomenon; a remarkable proportion of patients with new onset heart failure never have an evaluation for underlying ischemic heart disease either during hospitalization or in the following months after presentation.[24]

Co-morbidities

Among the many challenges of managing AHF, consideration of the patients' CV and non-CV conditions is necessary to ensure overall excellent care. At the time of presentation, a COPD exacerbation or uncontrolled diabetes may trigger decompensation. In general, management of both HF and other co-morbidities will occur once the patient is stabilized.

De novo vs. decompensated Chronic HF.

As a general rule, we suggest admission for patients with de novo or new onset HF. In addition to further management, understanding underlying cardiac structure and function, presence or absence of coronary artery disease, as well as education regarding a new chronic disease may be challenging to perform expeditiously in the outpatient setting. A three-prong pathophysiologic approach may be considered: 1) Cardiac substrate 2) Triggers or precipitants 3) Amplifying mechanisms.[25] After stabilization, each should be considered and managed appropriately. For all patients, adherence to guideline recommended therapies are essential. For chronic HF patients, thorough evaluation has ideally, already occurred; hospitalization presents an opportunity for further education and optimization of guideline directed medical therapy.

Risk-stratification and Disposition

Although over 80% of AHF patients are admitted from the ER, not all require hospitalization.[3,26,27] Over a decade ago, one report suggested nearly 50% of AHF patients could be discharged home.[28] Unfortunately, for the small proportion of patients discharged, little is known regarding their outcomes. The best evidence comes from Canadian datasets, suggesting emergency physicians risk stratify poorly, with discharged patients at higher risk for bad outcomes.[29] Existing risk instruments either fail to perform well enough or have yet to be well-validated. Thus, evidence based, universal recommendations regarding who is safe to discharge do not yet exist.

Lack of evidence has not hindered efforts to decrease hospital re-admissions however.[30] Similarly, expert consensus, informed by existing evidence, should inform individual hospital or health care system strategies to reduce hospital admissions from the ED.

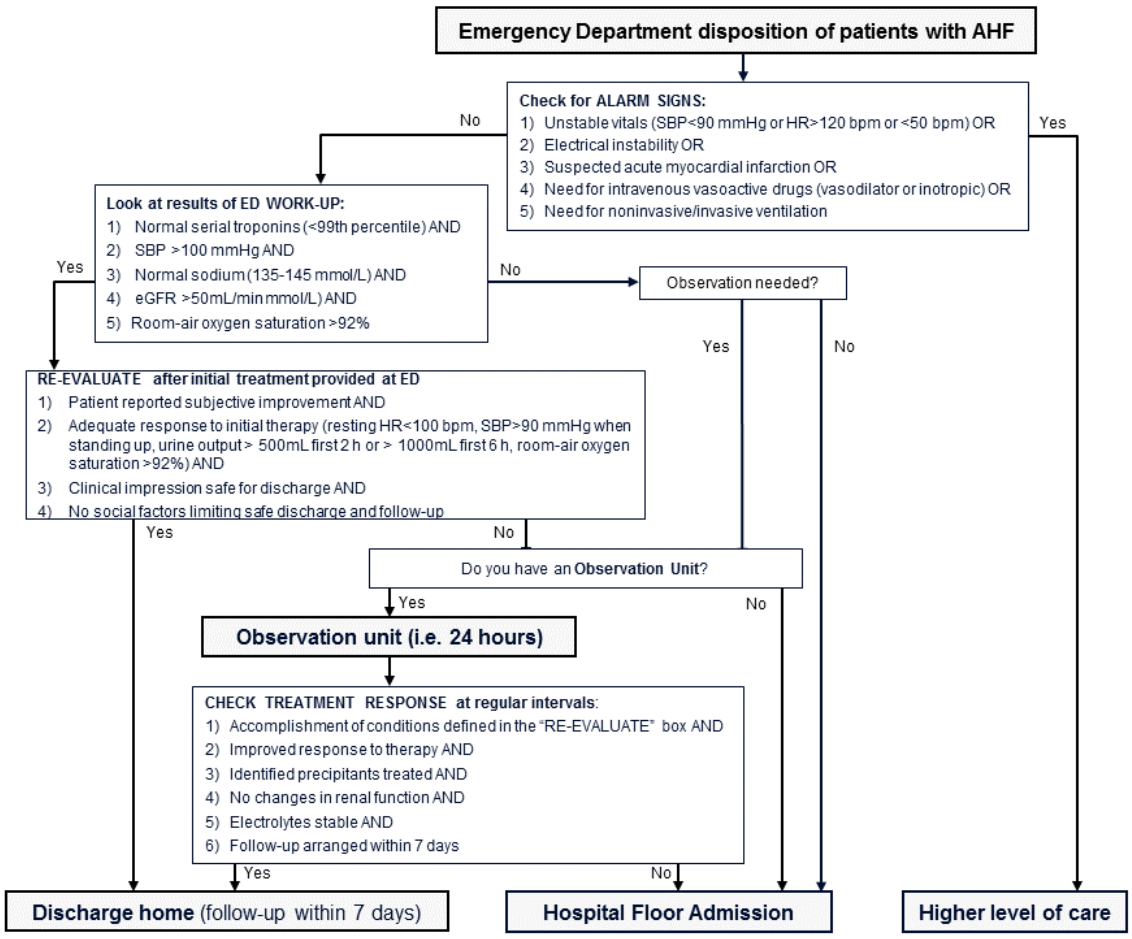
Overall, emergency physicians have a low tolerance for risk.[31] Suggesting ED's move from over 80% admission to 50 or 60% would be difficult, especially absent better evidence. Observation units or short-stay units are a viable alternative to direct discharge.

Observation Units for Heart Failure

Observation for AHF patients has been ongoing for well over a decade.[32,33] Both retrospective analyses and small studies support the safety, effectiveness and potential cost-savings of AHF observation unit (OU) management.[5,34-40] In a retrospective study of 358 patients using Society of Cardiovascular Patient Care guidelines, the authors observed less hospital bed day utilization and similar 30-day readmission rates (12.5% vs 10.0%), respectively after adjustment for age, race, sex, BNP, renal function and ejection fraction.[39] Patients who weren't ready for discharge after SSU management were admitted to the hospital for further treatment.

Compared to low-risk chest pain patients sent to the OU for 'rule-out' ACS, AHF patients are older with significantly more co-morbidities. While follow up after observation is important for all patients, ensuring a seamless handoff after an AHF exacerbation is especially so. Thus, an excellent working relationship with cardiologists

is critical to ensure a successful OU strategy. Figure 3 below is a suggested algorithm for ED disposition.



Observation also affords a luxury generally absent in the ED setting: time. Thus, response to therapy, assessment of functional status, addressing co-morbidities, as well as education, follow up, contact with the primary cardiologist or primary care physicians can occur. Furthermore, time for social work and case management consultation is also possible. As mentioned earlier, the 6-axis model should be considered during each phase of re-evaluation to optimize care.

Other Considerations

Observation (or hospitalization) is an opportunity to optimize (or plan to optimize) care. Figure 4 outlines different facets of AHF to consider prior to discharge. Not all have to be performed during hospitalization; however, each should be considered.

Future Directions

While outcomes from AHF have improved slightly over the last decade, overall, prognosis remains poor. Over a third of patients will die or be re-hospitalized within 90 days after discharge.[1] Attempts to improve outcomes with novel therapies have largely failed. Such failures suggest improving mortality with a short-term infusion during hospitalization is a daunting hurdle.[41] While the reasons for such lack of success is beyond the scope of this article, our limited knowledge of the underlying pathophysiology of AHF – in all its forms – has likely contributed. To this day, an exact agreement on the definition of AHF remains debated.

Lack of success does not mitigate the pressing and growing unmet need. The ED continues to play a critical role in initial diagnosis and management and arguably 'sets the tone' for downstream management. Ideally, the correct path is followed. However, poor upfront management also has potential deleterious downstream consequences. Importantly, the decision to admit or not – one of the costliest healthcare decisions financially – remains an area of ongoing work. Improvements in risk-stratification will decrease the number of hospitalizations for many patients.

Although we recommend the 6-axis model as a framework for initial management, testing and validation of the model is still required. In the future additional components might be added (SEE FIGURE 5) or removed. In this example of an 8-axis model, both CAD and biomarker profiles have been added. At the present, robust assessment of CAD during hospitalization is not routine, nor should it be for every patient. However, ischemia due to CAD may be a direct contributor to decompensation. Whether intervention results in better outcomes is speculative.[42,43] Existing and yet-to-be-discovered biomarkers may also influence management and treatment. One example are high-sensitivity troponin assays. Although a large proportion of AHF patients present with evidence of high-sensitivity troponin release, the exact reasons and how to manage such patients remain areas of active investigation. One potential role of biomarkers would be risk-stratification.[44]

Given the multimorbid burden of AHF patients, decreasing disparities remains a critical area to improve outcomes. Such patients bear a disproportionate burden of health care utilization and worse outcomes.[3,45] Novel ideas such as community paramedicine may have a role.[46,47] From the ED perspective, moving from a transactional health encounter to one that is a part of the longitudinal care of patients will be essential.

Summary/Discussion

Most patients hospitalized with AHF initially present to the ED. Ensuring correct diagnosis and prompt management is critical to ensure good outcomes. We recommend the 6-axis model as a framework for initial management of the ED AHF patient.

Importantly, re-assessment should occur frequently. Rather than hospitalize most patients, use of observation units may provide the necessary and sufficient time to ensure patients are responding appropriately to initial management. Time in observation also allows for more robust transitional care, ensuring good communication with primary care and specialists as well as addressing other co-morbid conditions, education, and social factors..

References

1. Gheorghiade M, Abraham WT, Albert NM, et al. Systolic blood pressure at admission, clinical characteristics, and outcomes in patients hospitalized with acute heart failure. *JAMA : the journal of the American Medical Association.* 2006;296(18):2217-2226.
2. Benjamin EJ, Blaha MJ, Chiuve SE, et al. Heart Disease and Stroke Statistics-2017 Update: A Report From the American Heart Association. *Circulation.* 2017.
3. Storrow AB, Jenkins CA, Self WH, et al. The burden of acute heart failure on U.S. emergency departments. *JACC Heart failure.* 2014;2(3):269-277.
4. Pang PS, Collins SP. Acute Heart Failure in the Emergency Department: Just a One Night Stand? *Acad Emerg Med.* 2017;24(3):385-387.
5. Pang PS, Collins SP, Miro O, et al. The role of the emergency department in the management of acute heart failure: An international perspective on education and research. *Eur Heart J Acute Cardiovasc Care.* 2015.
6. Pang PS, Cleland JG, Teerlink JR, et al. A proposal to standardize dyspnoea measurement in clinical trials of acute heart failure syndromes: the need for a uniform approach. *Eur Heart J.* 2008;29(6):816-824.
7. Pang PS, Collins SP, Sauser K, et al. Assessment of dyspnea early in acute heart failure: patient characteristics and response differences between likert and visual analog scales. *Acad Emerg Med.* 2014;21(6):659-666.
8. Pang PS, Lane KA, Tavares M, et al. Is there a clinically meaningful difference in patient reported dyspnea in acute heart failure? An analysis from URGENT Dyspnea. *Heart & lung : the journal of critical care.* 2017.
9. Martindale JL, Wakai A, Collins SP, et al. Diagnosing Acute Heart Failure in the Emergency Department: A Systematic Review and Meta-analysis. *Acad Emerg Med.* 2016;23(3):223-242.
10. Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J.* 2016;37(27):2129-2200.
11. Yancy CW, Jessup M, Bozkurt B, et al. 2016 ACC/AHA/HFSA Focused Update on New Pharmacological Therapy for Heart Failure: An Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. *Circulation.* 2016.
12. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2013;62(16):e147-239.
13. Ramirez A, Abelmann WH. Cardiac decompensation. *New Eng J Med.* 1974;290:499-501.

14. Wang CS, FitzGerald JM, Schulzer M, Mak E, Ayas NT. Does this dyspneic patient in the emergency department have congestive heart failure? *JAMA : the journal of the American Medical Association.* 2005;294(15):1944-1956.
15. Maisel AS, Krishnaswamy P, Nowak RM, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *The New England journal of medicine.* 2002;347(3):161-167.
16. Gargani L, Pang PS, Frassi F, et al. Persistent pulmonary congestion before discharge predicts rehospitalization in heart failure: a lung ultrasound study. *Cardiovasc Ultrasound.* 2015;13:40.
17. Fonarow GC, Stough WG, Abraham WT, et al. Characteristics, treatments, and outcomes of patients with preserved systolic function hospitalized for heart failure: a report from the OPTIMIZE-HF Registry. *J Am Coll Cardiol.* 2007;50(8):768-777.
18. Pitt B, Pfeffer MA, Assmann SF, et al. Spironolactone for heart failure with preserved ejection fraction. *The New England journal of medicine.* 2014;370(15):1383-1392.
19. Gheorghiade M, Braunwald E. A proposed model for initial assessment and management of acute heart failure syndromes. *JAMA : the journal of the American Medical Association.* 2011;305(16):1702-1703.
20. Collins S, Storrow AB, Kirk JD, Pang PS, Diercks DB, Gheorghiade M. Beyond pulmonary edema: diagnostic, risk stratification, and treatment challenges of acute heart failure management in the emergency department. *Annals of emergency medicine.* 2008;51(1):45-57.
21. Nohria A, Tsang SW, Fang JC, et al. Clinical assessment identifies hemodynamic profiles that predict outcomes in patients admitted with heart failure. *J Am Coll Cardiol.* 2003;41(10):1797-1804.
22. Ambrosy AP, Pang PS, Gheorghiade M. Digoxin for Worsening Chronic Heart Failure: Underutilized and Underrated. *JACC Heart failure.* 2016;4(5):365-367.
23. Fonarow GC, Abraham WT, Albert NM, et al. Factors identified as precipitating hospital admissions for heart failure and clinical outcomes: findings from OPTIMIZE-HF. *Archives of internal medicine.* 2008;168(8):847-854.
24. Doshi D, Ben-Yehuda O, Bonafede M, et al. Underutilization of Coronary Artery Disease Testing Among Patients Hospitalized With New-Onset Heart Failure. *J Am Coll Cardiol.* 2016;68(5):450-458.
25. Braunwald E, Bonow RO, Mann DL, Zipes DP, Libby P, eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 9th Ed.* 9th ed2011; No. 1.
26. Pang PS, Jesse R, Collins SP, Maisel A. Patients with acute heart failure in the emergency department: do they all need to be admitted? *J Card Fail.* 2012;18(12):900-903.
27. Pang PS, Schuur JD. Emergency departments, acute heart failure, and admissions: one size does not fit all. *JACC Heart failure.* 2014;2(3):278-280.
28. Graff L, Orledge J, Radford MJ, Wang Y, Petrillo M, Maag R. Correlation of the Agency for Health Care Policy and Research congestive heart failure admission guideline with mortality: peer review organization voluntary hospital association initiative to decrease events (PROVIDE) for congestive heart failure. *Annals of emergency medicine.* 1999;34(4 Pt 1):429-437.

29. Lee DS, Schull MJ, Alter DA, et al. Early deaths in patients with heart failure discharged from the emergency department: a population-based analysis. *Circulation Heart failure.* 2010;3(2):228-235.
30. Butler J, Kalogeropoulos A. Hospital strategies to reduce heart failure readmissions: where is the evidence? *J Am Coll Cardiol.* 2012;60(7):615-617.
31. McCausland JB, Machi MS, Yealy DM. Emergency physicians' risk attitudes in acute decompensated heart failure patients. *Acad Emerg Med.* 2010;17(1):108-110.
32. Peacock WF. Heart failure management in the emergency department observation unit. *Prog Cardiovasc Dis.* 2004;46(5):465-485.
33. Peacock WF. Using the emergency department clinical decision unit for acute decompensated heart failure. *Cardiology clinics.* 2005;23(4):569-588, viii.
34. Peacock WF, Fonarow GC, Ander DS, et al. Society of Chest Pain Centers recommendations for the evaluation and management of the observation stay acute heart failure patient-parts 1-6. *Acute cardiac care.* 2009;11(1):3-42.
35. Collins SP, Storrow AB, Levy PD, et al. Early management of patients with acute heart failure: state of the art and future directions--a consensus document from the SAEM/HFSA acute heart failure working group. *Acad Emerg Med.* 2015;22(1):94-112.
36. Collins SP, Pang PS, Fonarow GC, Yancy CW, Bonow RO, Gheorghiade M. Is hospital admission for heart failure really necessary?: the role of the emergency department and observation unit in preventing hospitalization and rehospitalization. *J Am Coll Cardiol.* 2013;61(2):121-126.
37. Baugh CW, Schuur JD. Observation care--high-value care or a cost-shifting loophole? *The New England journal of medicine.* 2013;369(4):302-305.
38. Baugh CW, Venkatesh AK, Hilton JA, Samuel PA, Schuur JD, Bohan JS. Making greater use of dedicated hospital observation units for many short-stay patients could save $3.1 billion a year. *Health affairs.* 2012;31(10):2314-2323.
39. Schrager J, Wheatley M, Georgiopoulou V, et al. Favorable bed utilization and readmission rates for emergency department observation unit heart failure patients. *Acad Emerg Med.* 2013;20(6):554-561.
40. Storrow AB, Collins SP, Lyons MS, Wagoner LE, Gibler WB, Lindsell CJ. Emergency department observation of heart failure: preliminary analysis of safety and cost. *Congestive heart failure.* 2005;11(2):68-72.
41. Packer M, O'Connor C, McMurray JJV, et al. Effect of Ularitide on Cardiovascular Mortality in Acute Heart Failure. *The New England journal of medicine.* 2017;376(20):1956-1964.
42. Flaherty JD, Bax JJ, De Luca L, et al. Acute heart failure syndromes in patients with coronary artery disease early assessment and treatment. *J Am Coll Cardiol.* 2009;53(3):254-263.
43. Flaherty JD, Rossi J, Fonarow G, et al. Influence of Coronary Angiography on the Utilization of Therapies in Patients with Acute Heart Failure Syndromes: A Report from OPTIMIZE-HF (Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure)(presented as a Late Breaking Clinical Trial). Transcatheter Cardiovascular Therapeutics Conference; October, 2008, 2008; Washington, DC.
44. Pang PS, Teerlink JR, Voors AA, et al. Use of High-Sensitivity Troponin T to Identify Patients With Acute Heart Failure at Lower Risk for Adverse Outcomes: An Exploratory Analysis From the RELAX-AHF Trial. *JACC Heart failure.* 2016;4(7):591-599.

45. Hasegawa K, Tsugawa Y, Camargo CA, Jr., Brown DF. Frequent utilization of the emergency department for acute heart failure syndrome: a population-based study. *Circ Cardiovasc Qual Outcomes.* 2014;7(5):735-742.
46. Choi BY, Blumberg C, Williams K. Mobile Integrated Health Care and Community Paramedicine: An Emerging Emergency Medical Services Concept. *Annals of emergency medicine.* 2016;67(3):361-366.
47. Iezzoni LI, Dorner SC, Ajayi T. Community Paramedicine--Addressing Questions as Programs Expand. *The New England journal of medicine.* 2016;374(12):1107-1109.

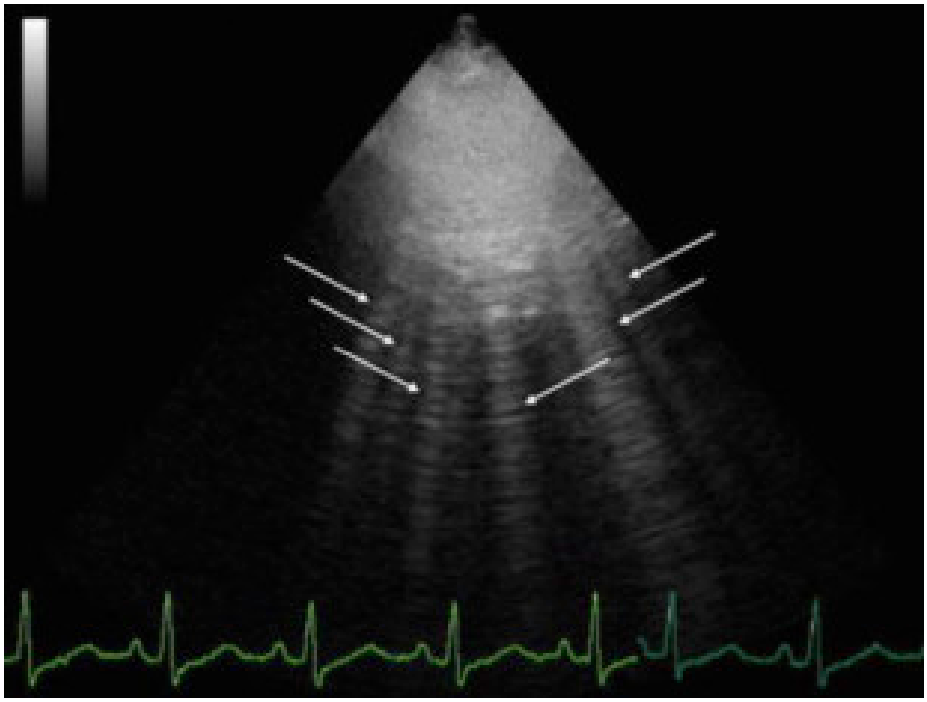


Fig. 1
Lung ultrasound. Arrows point to B-lines.

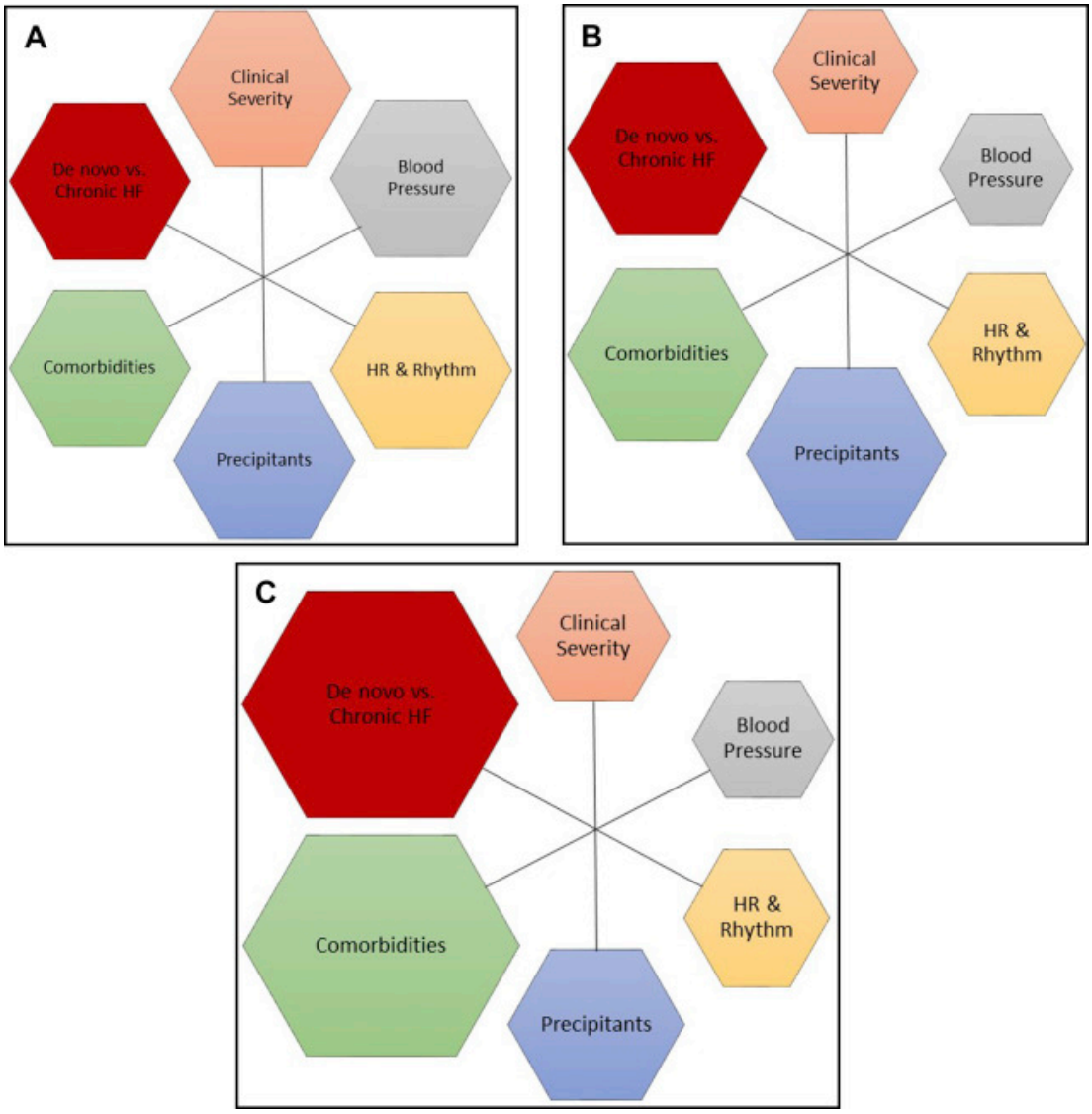


Fig. 2
The 6-axis model. (A) Represents initial ED presentation and management. (B) Highlights how certain components of the 6-axis model may increase or decrease in importance during hospitalization. (C) Further highlights the dynamic nature of AHF. By discharge, ensuring comorbidities were addressed is critical. Whether or not the patient had de novo vs chronic HF might significantly impact further disease management.
(From Gheorghiade M, Braunwald E. A proposed model for initial assessment and management of acute heart failure syndromes. JAMA 2011;305(16):1703; with permission.)

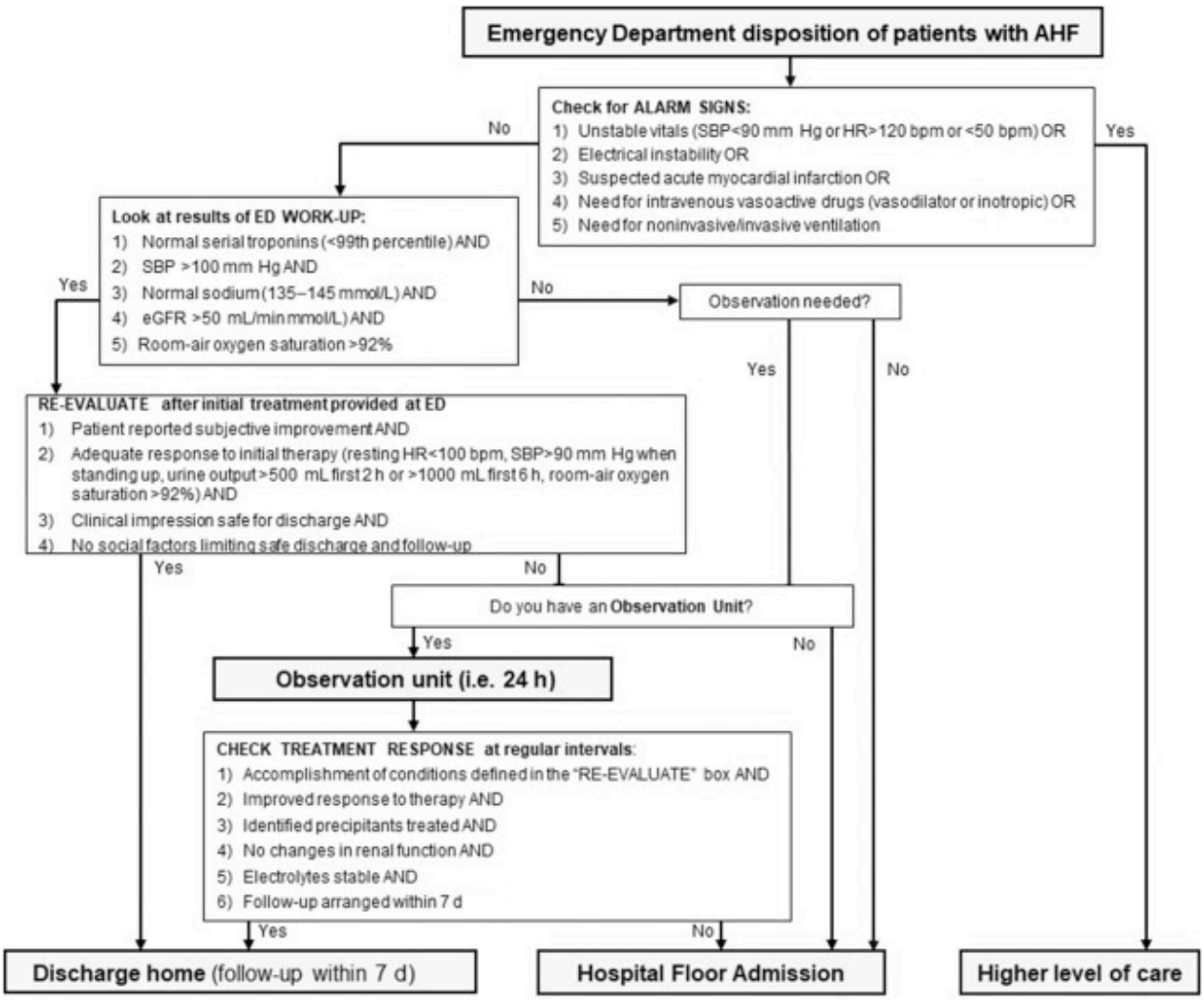


Fig. 3
ED disposition of patients with AHF.
(From Miro O, Levy PD, Mockel M, et al. Disposition of emergency department patients diagnosed with acute heart failure: an international emergency medicine perspective. Eur J Emerg Med 2017;24(1):2–12; with permission.)

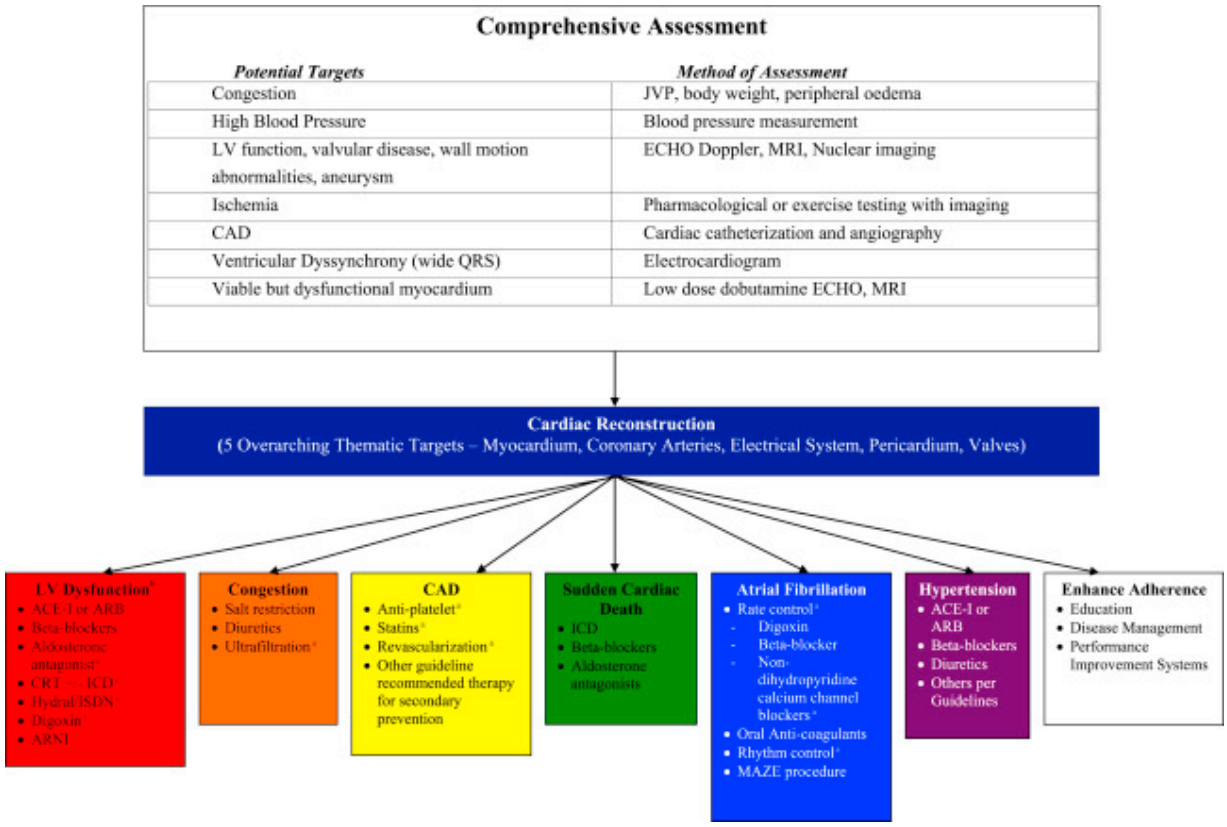


Fig. 4
Comprehensive assessment and targeted implementation of evidence-based therapies. ACE-I, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; CRT, chronic resynchronization therapy; Hydral, hydralazine; ICD, implantable cardiac defibrillator; ISDN, isosorbide dinitrate; JVP, jugular venous pulse; LV, left ventricle. a Select patients. b Viable but dysfunctional myocardium.
(Adapted from Pang PS, Komajda M, Gheorghiade M. The current and future management of acute heart failure syndromes. Eur Heart J 2010;31(7):784–93; with permission.)

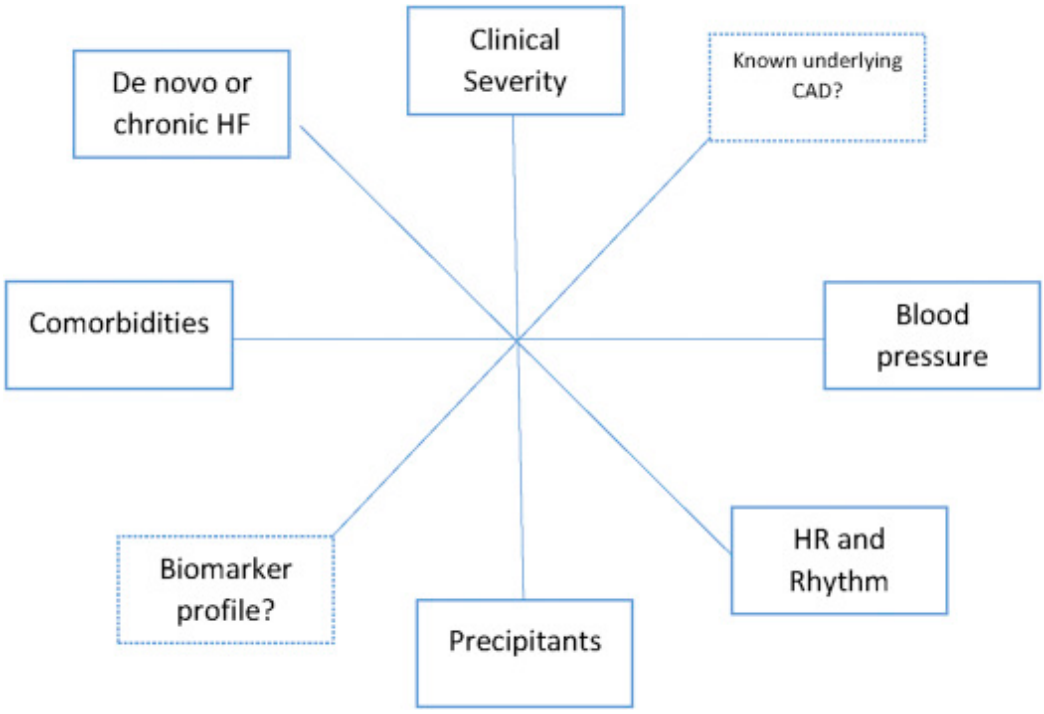


Fig. 5
Potential to Expand the 6-axis Model.
(Data from Gheorghiade M, Braunwald E. A proposed model for initial assessment and management of acute heart failure syndromes. JAMA: the journal of the American Medical Association 2011;305(16):1702–3.)

Table 1

Initial management based on systolic blood pressure

	Hypotensive (SBP <100 mm Hg)	Normotensive (SBP 100–140 mm Hg)	Hypertensive SBP >140 mm Hg
IV loop diuretics	…	++++	++
Vasodilators	…	++	++++
Inotropes [a]	++++	…	…

Abbreviations: +, strength of the recommendation; IV, intravenous; SBP, systolic blood pressure.