Advances in the management of acute decompensated heart failure

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ABSTRACT

Heart failure is a common condition that affects millions of people worldwide and is associated with substantial morbidity and mortality. Acute decompensated heart failure refers to the worsening of symptoms that requires changes in drugs or the start of new treatments such as non-invasive positive pressure ventilation. This review summarizes the current data and provides an evidence based approach to the diagnosis and management of acute decompensated heart failure. The review discusses common nomenclature and classifications of the condition, followed by the diagnostic accuracy of medical history, physical examination, electrocardiography, radiographs of the chest, point-of-care ultrasound, and laboratory testing. Current and emerging medical treatments are also discussed.

Introduction

Heart failure affects millions of people worldwide, leading to substantial morbidity and mortality. Progressive worsening of symptoms, referred to as acute decompensated heart failure (ADHF), is associated with a high rate of unplanned emergency department visits, frequent hospital admissions, and increased risk of mortality.[1] The approach used to diagnose and manage ADHF has evolved over time, and there is a need to understand the current state of the science. In this review, we discuss the most important aspects of ADHF care, focusing on epidemiology, nomenclature, diagnosis, management, risk stratification, and disposition. This review is intended for all clinicians who care for patients with heart failure in the acute care setting.

Sources and selection criteria

We searched PubMed and the Cochrane Database of Systematic Reviews for articles published from inception to 1 April 2025 to identify studies reporting on the diagnosis, management, and disposition of ADHF. We evaluated retrospective and prospective studies, randomized controlled trials, systematic reviews and meta-analyses, and guidelines. When available, systematic reviews and meta-analyses were preferentially selected. These were followed sequentially by randomized controlled trials, prospective studies, retrospective studies, and case series.

Epidemiology

Heart failure is a common condition, affecting over 56 million people worldwide.[2] The risk of heart failure increases with age, with a 20-45% lifetime prevalence in people older than 45 years.[3] Although the overall incidence of heart failure has stabilized or declined in some regions,[2] the absolute number of patients with ADHF remains substantial owing to the aging population and improved survival rates from other cardiovascular conditions.[2 4 5] This trend contributes to a growing prevalence of heart failure across Europe and the United States.

In the US, heart failure is the primary or secondary diagnosis for one to three million hospital admissions each year, accounting for up to 2% of the total healthcare budget.[6] By 2030, more than eight million people (one in 33) will have heart failure.[6]

Approximately 1.4% of all emergency department visits are for heart failure, with a rising incidence over time.[7] Among those presenting to the emergency department, nearly three quarters are admitted to hospital.[7] Most people admitted to hospital with ADHF will have a known history of heart failure, with one study reporting an outpatient heart failure diagnosis in 73.4% and treatment for heart failure in 64.9%.[8] Inpatient mortality ranges from 4% to 12% and can be as high as 25% in patients at high risk.[6 9 10] The one year survival rate among those with heart failure is 86.5%, which has been declining over the past decade.[11 12] Among those requiring hospital admission for ADHF, the mortality rate markedly increases.[5] Moreover, there are substantial public health implications, with overall direct costs related to heart failure exceeding $30 billion (£22.3 billion; €25.5 billion) annually and indirect costs (owing to lost economic productivity) exceeding $14 billion in the US.[13 14] Another study reported the global economic burden exceeded $108 billion annually, with approximately $65 billion in direct costs and $43 billion in indirect costs.[15] A systematic review of 16 international studies reported the lifetime cost for patients with heart failure has been estimated to be at least $126 819 per patient.[16]

Importantly, research has shown inequalities in heart failure, with higher rates among women and black patients.[17-21] Among those with ADHF, black patients are more likely to be admitted to hospital and have higher rates of morbidity and mortality.[8 18 22 23] Heart failure has also shown a disproportionate rise in low and middle income countries, with reduced rates of starting goal directed medical treatment after discharge and higher one year mortality rates.[24 25]

Nomenclature

Heart failure is a complex clinical syndrome characterized by inability of the heart to adequately pump sufficient blood to meet the body's metabolic needs, occurring at rest, with exertion, or because of increased filling pressures. Primary symptoms (shortness of breath, edema, and fatigue) are caused by functional or structural cardiac damage. As symptoms progress, patients may notice reduced exercise tolerance or symptoms of fluid retention (eg, pulmonary congestion, splanchnic congestion, peripheral edema).[26] Heart failure can be classified by several different and inter-related categories, including side of involvement, ejection fraction, and acuity. Table 1 presents a list of definitions for several common categories of heart failure. For the purposes of this review, we focus on ADHF with an emphasis on left sided heart failure.

Traditionally, ADHF was defined by three main components: worsening symptoms, treatment location requiring urgent or inpatient care, and the need for escalated care involving intravenous or invasive treatments.[26] However, a new proposed definition emphasizes that it is not the acuity or the location that defines decompensated heart failure, but rather the need for intensified or escalated treatments.[27] Decompensated heart failure is characterized by the active deterioration of heart failure symptoms despite attempts to optimize heart failure treatment, necessitating intensified or rescue treatments beyond standard heart failure treatment. This definition does not specify a location (such as admission to hospital, urgent care, or emergency department visit). This change aims to align the definition with patient profiles and clinical care, providing better guidance in clinical trial design and regulatory approval processes.[27] In this review, we use the term ADHF because it remains the most common terminology; however, it is important to note that timing is not always sudden onset and can include those with more gradual progression.

Although there are several terminologies for heart failure, patients may present with a variety of signs and symptoms, and the European Society of Cardiology classifies these various presentations into several categories, including ADHF, acute pulmonary edema, right ventricular failure, and cardiogenic shock.[28] These categories are based on phenotypes that incorporate peripheral perfusion (normal perfusion or "warm" *v* hypoperfusion or "cold") and congestion (congestion or "wet" *v* no congestion or "dry"). The combination of these includes "warm and wet" (normal perfusion but congested, 70% of patients), "cold and wet" (hypoperfused and congested, 20%), "cold and dry" (hypoperfused but no congestion, <2%), and "warm and dry" (normal perfusion without congestion, <10%).[29]

Patients with ADHF present because of fluid accumulation associated with left ventricular dysfunction with renal sodium and water retention. As cardiac output drops from myocardial injury or stress, a neurohormonal mediated cascade including activation of the renin-angiotensin-aldosterone and sympathetic nervous systems occurs. The clinical effects of this neurohormonal activation are sodium and water retention and increased systemic vascular resistance. These maintain blood pressure and perfusion, but increase myocardial workload, wall tension, and myocardial oxygen demand. The onset is typically gradual (days) with low or normal cardiac output. Patients may present with warm and wet or cold and dry phenotypes.

Table 1 | Common terminology for heart failure[26 27]

Nomenclature	Definition
Left sided heart failure	Failure of the left ventricle to effectively pump blood, leading to pulmonary congestion (eg, rales, dyspnea, orthopnea) and reduced systemic perfusion
Right sided heart failure	Failure of the right ventricle to effectively pump blood, leading to systemic congestion (eg, peripheral edema, hepatomegaly, jugular venous distension)
Biventricular heart failure	Failure of the left and right ventricles, leading to both pulmonary and systemic congestion
Heart failure with preserved ejection fraction (HFpEF)	Left ventricular heart failure characterized by impaired relaxation and filling, but a normal ejection fraction (≥50%)
Heart failure with mildly reduced ejection fraction (HFmrEF)	Left ventricular heart failure characterized by a mildly reduced ejection fraction (41-49%), considered an intermediate category between HFpEF and HFrEF
Heart failure with reduced ejection fraction (HFrEF)	Left ventricular heart failure characterized by impaired contractility and a reduced ejection fraction (≤40%)
Systolic heart failure	Heart failure characterized by reduced myocardial contractility. This is an older term that has been replaced by HFrEF and HFmrEF
Diastolic heart failure	Heart failure characterized by impaired relaxation and filling. This is an older term that has been replaced by HFpEF
Congestive heart failure	Heart failure accompanied by pulmonary or systemic congestion. This is an older term that is no longer recommended
Acute decompensated heart failure (ADHF)	A sudden worsening of chronic heart failure accompanied by pulmonary or systemic congestion
Decompensated heart failure	Active deterioration of heart failure with symptoms necessitating intensified or rescue treatments beyond standard heart failure treatment
Sympathetic crashing acute pulmonary edema	A hyperacute version of ADHF where sympathetic overactivation leads to hypertension, pulmonary edema, and respiratory distress
Cardiogenic shock	Heart failure resulting in tissue hypoperfusion

Box 1: Differential diagnosis for acute dyspnea

Cardiac causes
- Acute coronary syndrome
- Acute decompensated heart failure
- Arrhythmias
- Constrictive pericarditis
- Myocarditis
- Pericardial effusion/cardiac tamponade
- Takotsubo cardiomyopathy
- Valvular dysfunction (eg, aortic stenosis, mitral regurgitation)

Pulmonary causes
- Acute respiratory distress syndrome
- Asthma
- Chronic obstructive pulmonary disease
- Interstitial lung disease
- Pleural effusion
- Pneumonia
- Pneumothorax
- Pulmonary embolism

Other causes
- Anemia
- Foreign body aspiration
- Hypoalbuminemia
- Metabolic disorders (eg, acidosis, sepsis, thyrotoxicosis)
- Neuromuscular disorders (eg, Guillain-Barré, myasthenia gravis, amyotrophic lateral sclerosis)
- Non-cardiogenic pulmonary edema (eg, high altitude pulmonary edema, toxin induced)
- Panic attack
- Toxic inhalation

Acute pulmonary edema is the second presentation category and is caused by fluid redistribution into the lungs from increased afterload, left ventricular diastolic dysfunction, or valvular heart disease. The onset is typically more rapid (hours) with normal cardiac output and normal to high systolic blood pressure. Acute pulmonary edema can be abrupt, severely symptomatic, and rapidly fatal if it goes untreated. Patients present with a warm and wet phenotype.

Isolated right ventricular failure is the third category and presents because of right ventricular dysfunction or precapillary pulmonary hypertension with increased central venous pressure. The onset may be gradual or rapid. Patients present with cold and dry or cold and wet phenotypes.

The final category is cardiogenic shock caused by severe cardiac dysfunction and systemic hypoperfusion, associated with low cardiac output and blood pressure. The onset can be gradual or rapid, and patients typically present with a cold and wet phenotype, though they may also present as cold and dry.[28] Importantly, the blood pressure should be contextualized by their baseline values because some patients may have proportionally low blood pressure despite not meeting a traditional numerical threshold such as mean arterial pressure <65 mm Hg.

One study evaluated these clinical phenotypes and their association with patient management and outcomes, and found most patients had a warm and wet phenotype (76%), followed by cold and wet (17.1%).[30] Patients presenting with cold phenotypes had higher hospital admission and mortality rates compared with other phenotypes. This is consistent with other studies finding warm and wet to be the most common presenting phenotype, while those with cold phenotypes have the highest mortality rates.[9 30-32]

Diagnosis

History and physical examination

The evaluation should begin with a detailed history and focused physical examination. This should include history of heart failure (including ejection fraction and last echocardiogram, if known), drugs taken (including recent changes or missed doses), dietary changes, weight changes, and relevant associated medical conditions (eg, coronary artery disease, hyperthyroidism). Assessment of acute symptoms should include the time course and severity, comparison with previous episodes of ADHF, and any interventions attempted (eg, increases in diuretic dosing). Dyspnea should be quantified using changes in level of orthopnea and distances walked before experiencing dyspnea. Changes in total body weight and urine output should also be noted. Because heart failure can overlap with other conditions (box 1), it is important to use a combination of historical features, physical examination findings, and testing to determine whether ADHF is the cause of the patient's symptoms. Importantly, existing clinical decision tools for ADHF are intended to assess the risk of adverse outcomes to inform admission or discharge decisions, rather than to diagnose heart failure.[33]

There is no single diagnostic test for ADHF; the diagnosis is based on the combination of all clinical data. Table 2 presents a summary of the positive likelihood ratio and negative likelihood ratio for common history and physical examination findings. The presence of a third heart sound or ventricular filling gallop (S3) helps to rule in ADHF (positive likelihood ratio 4.0), while the presence of fever assists with ruling it out (negative likelihood ratio 0.4).[34] The PREDICA trial identified predictive criteria from the history and physical examination to improve the diagnosis accuracy of patients presenting to the emergency department with dyspnea.[35] The trial showed that an emergency physician's gestalt (the clinical judgment about the probability of ADHF) was a strong predictor of correct diagnosis.

Evaluation for precipitating factors could also help diagnosis. These factors include drug or diet non-adherence (excess salt or fluid intake, unable to fill the drug prescriptions or take as recommended), renal failure (especially missed dialysis), poorly controlled hypertension, iatrogenic (recent addition of negative inotropic drugs, starting salt retaining drugs such as non-steroidal anti-inflammatory drugs, steroids, thiazolidinediones, inappropriate treatment reduction, or new dysrhythmic agents),

Table 2 | Pooled likelihood ratios for history and physical examination findings in acute decompensated heart failure[34]

Component	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Past medical history				
Atrial fibrillation	6 (1935)	51.9 (49.8 to 54.2)	2.1 (1.6 to 2.9)	0.82 (0.71 to 0.93)
Arrhythmia	5 (3469)	40.2 (38.6 to 41.9)	2.7 (2.2 to 3.4)	0.75 (0.68 to 0.83)
Coronary artery disease	14 (4983)	42.9 (41.5 to 44.3)	2.0 (1.7 to 2.4)	0.71 (0.64 to 0.79)
Chronic kidney disease	6 (3009)	42.8 (41.0 to 44.6)	3.4 (2.7 to 4.5)	0.75 (0.71 to 0.80)
Diabetes mellitus	19 (7707)	47.3 (46.2 to 48.4)	1.5 (1.3 to 1.7)	0.89 (0.84 to 0.94)
Heart failure	22 (8493)	46.0 (44.9 to 47.0)	2.7 (2.0 to 3.7)	0.58 (0.49 to 0.68)
Hyperlipidemia	5 (2923)	39.8 (38.1 to 41.6)	1.6 (1.3 to 1.9)	0.85 (0.82 to 0.90)
Hypertension	25 (10 137)	45.6 (44.6 to 46.6)	1.3 (1.3 to 1.4)	0.62 (0.53 to 0.73)
No history of COPD	18 (8053)	42.8 (41.7 to 43.9)	1.2 (1.1 to 1.4)	0.7 (0.6 to 0.8)
Previous myocardial infarction	9 (4208)	40.5 (39.1 to 42.0)	2.1 (1.8 to 2.5)	0.82 (0.76 to 0.89)
Renal failure	5 (2840)	40.9 (39.1 to 42.7)	2.3 (1.3 to 3.9)	0.9 (0.73 to 1.11)
Symptoms				
Absence of productive cough	7 (2414)	43.0 (41.0 to 45.0)	1.13 (1.02 to 1.26)	0.6 (0.5 to 0.8)
Dyspnea at rest	4 (2038)	37.9 (35.9 to 40.0)	1.1 (0.9 to 1.4)	0.88 (0.74 to 1.04)
Orthopnea	15 (5430)	45.5 (44.2 to 46.9)	1.9 (1.4 to 2.5)	0.74 (0.64 to 0.85)
Paroxysmal nocturnal dyspnea;	9 (2216)	44.8 (42.8 to 46.9)	1.6 (1.2 to 2.1)	0.79 (0.71 to 0.88)
Physical examination findings				
Absent fever	7 (3197)	43.6 (41.9 to 45.3)	1.14 (1.02 to 1.27)	0.4 (0.3 to 0.6)
Hepatojugular reflux	4 (1209)	60.4 (57.6 to 63.1)	2.2 (1.3 to 3.7)	0.91 (0.88 to 0.94)
Jugular venous distension	23 (8012)	47.8 (46.7 to 48.9)	2.8 (1.7 to 4.5)	0.76 (0.69 to 0.84)
Leg edema	26 (9626)	47.2 (46.2 to 48.2)	1.9 (1.6 to 2.3)	0.68 (0.61 to 0.75)
Murmur	8 (4004)	45.3 (43.8 to 46.8)	1.9 (0.9 to 3.8)	0.93 (0.79 to 1.08)
Rales	22 (8775)	48.2 (47.1 to 49.2)	1.8 (1.5 to 2.1)	0.60 (0.51 to 0.69)
S3	14 (5900)	45.2 (44.0 to 46.5)	4.0 (2.7 to 5.9)	0.91 (0.88 to 0.95)
Wheezing	13 (6970)	44.2 (43.0 to 45.3)	0.6 (0.5 to 0.8)	1.19 (1.10 to 1.30)

ADHF=acute decompensated heart failure; CI=confidence interval; COPD=chronic obstructive pulmonary disease; S3=presence of a third heart sound or ventricular filling gallop.

and substance abuse (cocaine, methamphetamines, ethanol).

Electrocardiography

Although electrocardiogram findings are not effective in confirming or excluding ADHF, all patients with ADHF should undergo electrocardiography to help identify alternative causes (eg, ST elevation myocardial infarction, pericarditis) and evaluate for dysrhythmias requiring targeted interventions for heart rate control. Table 3 includes the likelihood ratios for electrocardiogram findings in ADHF.

Chest radiography

Chest radiographs are commonly ordered in ADHF to identify the presence and degree of pulmonary edema, and to assess for alternative causes (eg, pleural effusion, pneumonia, pneumothorax).[7] The progression of heart failure on chest radiography has been proposed to follow three stages.[36] Stage 1 consists of redistribution of pulmonary vessels, increased cardiothoracic ratio, and a broad vascular pedicle. Stage 2 involves interstitial edema, which includes Kerley B-lines, peribronchial cuffing, hazy contour of vessels, and subpleural edema. Stage 3 involves alveolar edema, which can present with consolidations, a butterfly appearance, cotton wool appearance, and pleural effusions (fig 1). Although these findings are modestly specific for ADHF, their absence does not exclude the diagnosis.[34] Approximately 20% of patients attending the emergency department subsequently diagnosed with ADHF have chest radiographs without evidence of congestion.[37] Table 4 includes the likelihood ratios for chest radiography findings in ADHF. Interstitial edema (positive likelihood ratio 6.4) and Kerley B-lines (positive likelihood ratio 6.5) increase the probability of heart failure.[34] The absence of any of these findings does not significantly decrease the probability of ADHF.

Table 3 | Pooled likelihood ratios for electrocardiogram findings in acute decompensated heart failure[34]

Finding	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Atrial fibrillation	6 (2242)	55.8 (53.7 to 57.8)	2.2 (1.4 to 3.5)	0.88 (0.85 to 0.91)
Ischemic changes	2 (1138)	42.6 (39.8 to 45.5)	2.9 (1.2 to 7.1)	0.78 (0.73 to 0.84)
Normal sinus rhythm	3 (1207)	39.6 (36.9 to 42.4)	0.7 (0.5 to 0.9)	2.88 (1.26 to 6.57)
ST depression	2 (1024)	60.8 (57.8 to 63.8)	2.0 (1.0 to 3.8)	0.97 (0.95 to 1.00)
ST elevation	1 (219)	61.2 (54.6 to 67.4)	0.6 (0.2 to 1.7)	1.03 (0.96 to 1.11)
T wave inversion	1 (709)	69.4 (65.9 to 72.7)	2.4 (1.2 to 4.8)	0.94 (0.90 to 0.98)

ADHF=acute decompensated heart failure; CI=confidence interval.

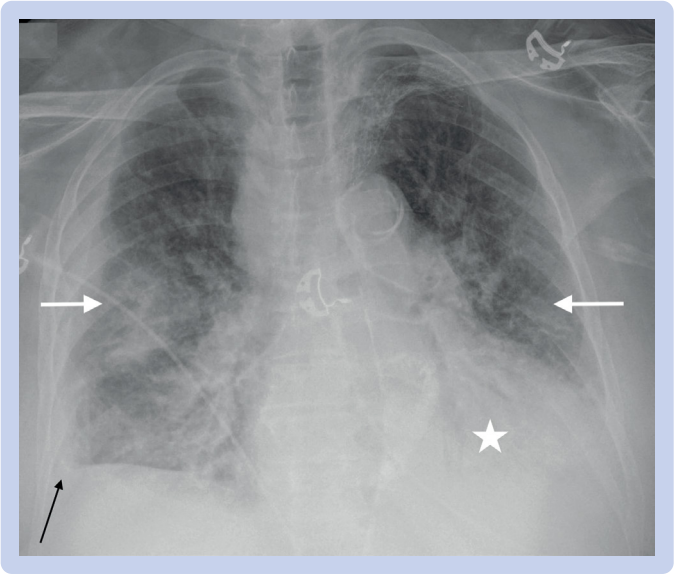

Fig 1 | Chest radiograph with bilateral interstitial infiltrates (white arrows), bilateral pleural effusions (black arrow), and cardiomegaly (star)

Point-of-care ultrasound

Point-of-care ultrasound (POCUS) is a valuable tool allowing for rapid bedside diagnosis. This test can help to reveal emergent causes of dyspnea, such as cardiac tamponade or pulmonary embolism (eg, evidence of acute right heart strain), and can estimate left ventricular function and volume status.[38]

Lung POCUS involves assessment for pulmonary edema, which is defined as the presence of at least three B-lines (hyperechoic imaging artefacts extending two thirds of the length of the ultrasound screen) involving at least two areas bilaterally (fig 2, video 1).[39] A systematic review and meta-analysis showed lung POCUS is more sensitive (91.8% *v* 76.5%) and more specific (92.3% *v* 87.0%) than chest radiography for detecting pulmonary edema in ADHF.[40] Another systematic review of six studies comparing POCUS and chest radiography showed better diagnostic test accuracy of POCUS compared with chest radiography. Chest radiography had a positive likelihood ratio of 7.36 and a negative likelihood ratio of 0.30, while POCUS had a positive likelihood ratio of 8.63 and a negative likelihood ratio of 0.14.[41] Because bilateral B-lines can be found in conditions not caused by pulmonary edema (eg, pulmonary fibrosis, pulmonary contusion, bilateral pneumonia), rapid assessment for raised central venous pressure may follow. An inferior vena cava diameter >2 cm or collapsibility index of <30% is indicative of raised central venous pressure.[42]

Cardiac POCUS can be used in a complementary manner to determine the ejection fraction and diastolic dysfunction, as well as alternative causes such as pericardial effusion or right ventricular dysfunction from pulmonary embolism.[43-45] With training, emergency physicians have reasonable agreement with cardiology interpretations by classifying a visual POCUS estimation of left ventricular ejection fraction into broad categories of normal, moderately reduced, and severely reduced (video 2).[46] More recent data have also shown the role of artificial intelligence to overcome the user dependent nature of POCUS, resulting in high image quality and diagnostic accuracy among more inexperienced users.[47-49] In this capacity, artificial intelligence can assist with image acquisition, image interpretation, and automated measurements. However, while POCUS is valuable in the acute setting, many patients may benefit from subsequent comprehensive transthoracic echocardiography in a delayed fashion. Table 5 includes the likelihood ratios for POCUS findings in ADHF.

Laboratory testing

Laboratory testing can be beneficial to identify potential causes and complications of ADHF. Common testing includes a complete blood count, electrolytes, creatinine, liver function testing, troponin, and B-type natriuretic peptide (BNP) or N-terminal proB-type natriuretic peptide (NT-proBNP). The complete blood count can evaluate for anemia as a potential mimic of ADHF. Electrolytes are valuable because many diuretics can cause electrolyte imbalances. Creatinine and liver function testing can identify renal impairment and hepatic congestion, respectively, which can influence the differential diagnosis and inform prognosis. Troponin can be useful for determining whether acute coronary syndrome is present in patients with suggestive symptoms, as well as for prognosis. Notably, troponin can also be raised owing to demand ischemia and should not automatically lead to invasive coronary angiography in the absence

Table 4 | Pooled likelihood ratios for chest radiograph findings in acute decompensated heart failure[34]

Finding	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Alveolar edema	3 (2001)	48.3 (46.2 to 50.5)	5.3 (3.3 to 8.5)	0.95 (0.94 to 0.97)
Cephalization	5 (1338)	54.0 (51.3 to 56.6)	5.6 (2.9 to 10.4)	0.53 (0.39 to 0.072)
Enlarged cardiac silhouette	12 (3515)	51.7 (49.4 to 52.7)	2.3 (1.6 to 3.4)	0.43 (0.36 to 0.51)
Kerley B-lines	2 (814)	46.8 (43.4 to 50.2)	6.5 (2.6 to 16.2)	0.88 (0.69 to 1.33)
Interstitial edema	3 (2001)	48.3 (46.2 to 50.5)	6.4 (3.4 to 12.2)	0.73 (0.68 to 0.78)
Pleural effusion	5 (1326)	55.1 (52.4 to 57.8)	2.4 (1.6 to 3.6)	0.89 (0.80 to 0.99)
Pulmonary edema	15 (4393)	46.6 (45.1 to 48.1)	4.8 (3.6 to 6.4)	0.48 (0.39 to 0.58)

ADHF=acute decompensated heart failure; CI=confidence interval.

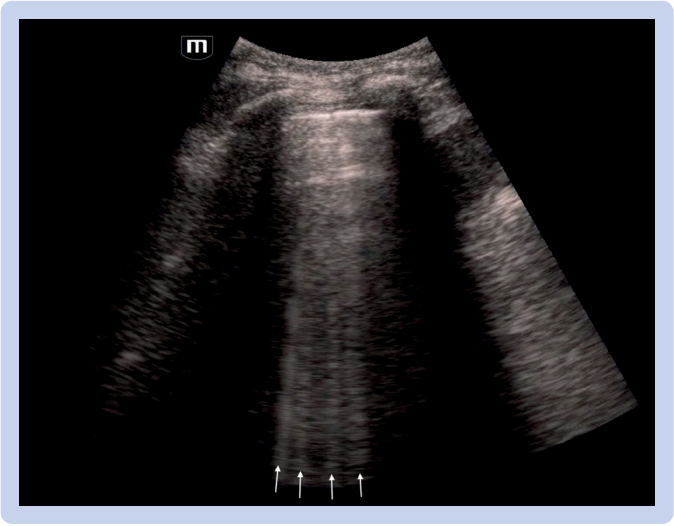

Fig 2 | B-lines (white arrows) on point-of-care cardiac ultrasound suggestive of pulmonary edema

of other findings concerning for acute coronary syndrome.[50]

A raised BNP can suggest volume overload in patients with an unclear clinical picture. However, despite the established use of natriuretic peptide testing, interpretation of results can be challenging in certain patients. Levels can be affected by age, sex, body mass, sacubitril or valsartan treatment, and may have delayed increases in patients with flash pulmonary edema.[51] Dyspnea and modest BNP increases may be seen in pulmonary hypertension, pulmonary embolism, pneumonia, sepsis, and renal failure.[52] As many as 25% of patients fall into the diagnostic "gray zone" (100-500 pg/mL for BNP), confounding test interpretation.[53] Heart failure can largely be excluded in patients with acute dyspnea and NT-proBNP <300 pg/mL or BNP <100 pg/mL. NT-proBNP has age specific cutoffs to further increase accuracy, with cutoff levels of 450, 900, and 1800 pg/mL in patients aged <50, 50-75, and >75 years, respectively.[53] BNP or NT-proBNP testing is best used when diagnostic uncertainty occurs and as an addition to the clinical assessment, rather than in isolation. Results may also be useful for comparison with inpatient testing in a serial fashion.[54 55] Table 6 and table 7 present interval likelihood ratios for BNP and NT-proBNP from a systematic review with individual patient level meta-analysis.[34]

Management

The initial management of ADHF should include hemodynamic stabilization and symptom relief.[32 56] Delays in diagnosis and treatment can worsen morbidity and mortality, with data suggesting an adjusted odds of death increasing by 6.8% for each six hour delay in treatment.[57] Figure 3 summarizes the management of ADHF.

Non-invasive ventilation

Evaluation of oxygenation and respiratory status should be the immediate first step in the emergency department. Non-invasive positive pressure ventilation (NIPPV, including continuous positive airway pressure and bilevel positive airway pressure) should be started rapidly in those presenting with acute respiratory distress to improve oxygenation and reduce work of breathing.[58] Successful NIPPV requires hemodynamic stability, facial anatomy allowing a facemask seal, monitoring, and patient cooperation. NIPPV has been shown to reduce hospital mortality (risk ratio 0.65, 95% confidence interval 0.51 to 0.82; number needed to treat 17) and rates of endotracheal intubation (risk ratio 0.49, 95% confidence interval 0.38 to 0.62; number needed to treat 13), with no difference in adverse events.[59] Randomized trial data have not shown a difference in mortality, endotracheal intubation, myocardial infarction, or length of hospital stay between continuous positive airway pressure and bilevel positive airway pressure.[60] In patients who cannot tolerate NIPPV, high flow nasal cannula may be considered.[61] A recent meta-analysis reported that high flow nasal cannula reduced rates of intubation compared with conventional oxygen treatment (risk ratio 0.31, 95% confidence interval 0.16 to 0.59).[62]

Nitroglycerin

For patients with adequate blood pressure, intravenous vasodilators should be used to reduce afterload and optimize preload, thereby improving symptoms and reducing congestion. These agents are particularly useful in patients with severe hypertension or acute pulmonary edema.[63-65] Nitroglycerin is the drug of choice in patients with ADHF and hypertension (defined as a systolic blood pressure >160 mm Hg).[65-67] An initial dose of 400 µg sublingually (tablets or spray) can be given while obtaining intravenous access. Once intravenous access is established, a nitroglycerin infusion should be started. Studies have shown that an initial high

Table 5 | Pooled likelihood ratios for POCUS in acute decompensated heart failure[34]

Finding	No of studies (No of patients)	% ADHF (95% CI)	Positive likelihood ratio (95% CI)	Negative likelihood ratio (95% CI)
Positive B-lines	8 (1914)	48.2 (46.0 to 50.5)	7.4 (4.2 to 12.8)	0.16 (0.05 to 0.51)
Pleural effusion	2 (155)	40.7 (33.2 to 48.5)	2.0 (1.4 to 2.8)	0.49 (0.22 to 1.10)
Restrictive mitral pattern	1 (125)	43.2 (34.9 to 52.0)	8.3 (4.0 to 16.9)	0.21 (0.12 to 0.36)
Reduced ejection fraction	3 (325)	41.2 (36.0 to 46.7)	4.1 (2.4 to 7.2)	0.24 (0.17 to 0.35)
Increased left ventricular end diastolic dimension	1 (84)	58.3 (47.7 to 68.3)	2.5 (1.5 to 4.2)	0.30 (0.16 to 0.54)

ADHF=acute decompensated heart failure; CI=confidence interval.

Table 6 | Interval likelihood ratios for BNP in acute decompensated heart failure (n=2202)[34]

BNP value (pg/mL)	No of patients (%)	Interval likelihood ratio (95% CI)
0-100	617 (28)	0.14 (0.12 to 0.18)
100-200	308 (14)	0.29 (0.23 to 0.38)
200-300	188 (9)	0.89 (0.67 to 1.17)
300-400	148 (7)	1.34 (0.98 to 1.83)
400-500	148 (7)	2.05 (1.47 to 2.84)
500-600	115 (5)	3.50 (2.30 to 5.35)
600-800	218 (10)	4.13 (3.01 to 5.68)
800-1000	130 (6)	5.00 (3.21 to 7.89)
1000-1500	160 (7)	7.12 (4.53 to 11.18)
1500-2500	105 (5)	8.33 (4.60 to 15.12)
2500-5001	65 (3)	8.91 (4.09 to 19.43)

BNP=B-type natriuretic peptide; CI=confidence interval.

dose bolus of 1000-2000 µg is well tolerated and can lead to improved patient symptoms and oxygen saturation, and reduced rates of intensive care unit admission.[65-68] A starting intravenous infusion dose of 0.5-0.7 µg/kg/min is common and titrated every few minutes up to 200 µg/min based on blood pressure and symptoms. Patients should be closely monitored to prevent hypotension. Flow limiting, preload dependent states such as aortic stenosis, right ventricular infarction, and hypertrophic cardiomyopathy, and patients with volume depletion are at increased risk of vasodilator associated hypotension.[69]

Other vasodilators

If additional arterial vasodilation is needed despite high dose nitroglycerin and NIPPV, intravenous angiotensin converting enzyme inhibitors or dihydropyridine calcium channel blockers may be considered. One retrospective study showed that intravenous enalaprilat 1.25 mg reduced systolic blood pressure by 30 mm Hg within three hours, with less than 2% of patients experiencing hypotension.[70] A separate retrospective study of intravenous nicardipine reported that all patients had a 30 mm Hg reduction in systolic blood pressure in a median of 18 minutes, with only one patient (2.6%) experiencing hypotension.[71] However, these agents should remain second line only after sufficiently titrated doses of nitroglycerin and NIPPV have been administered.[72]

Table 7 | Interval likelihood ratios for NT-proBNP in acute decompensated heart failure (n=2013)[34]

NT-proBNP value (pg/mL)	No of patients (%)	Interval likelihood ratio (95% CI)
0-100	150 (7.5)	0.09 (0.05 to 0.17)
100-300	205 (10.2)	0.23 (0.16 to 0.33)
300-600	212 (10.5)	0.28 (0.20 to 0.39)
600-900	151 (7.5)	0.63 (0.46 to 0.87)
900-1500	249 (12.4)	0.84 (0.67 to 1.06)
1500-3000	273 (13.6)	1.49 (1.19 to 1.86)
3000-5000	225 (11.2)	2.36 (1.81 to 3.08)
5000-10 000	239 (11.9)	2.48 (1.91 to 3.21)
10 000-15 000	112 (5.6)	2.84 (1.90 to 4.23)
15 000-30 000	111 (5.5)	2.93 (1.95 to 4.39)
30 000-200 000	86 (4.3)	3.30 (2.05 to 5.31)

CI=confidence interval; NT-proBNP=N-terminal proB-type natriuretic peptide.

Nitroprusside dilates venous and arterial vessels, but is less preferred than the other agents because of increased risks of hypotension.[73 74] Owing to their mechanisms, intravenous vasodilators may be more effective than diuretics for patients with acute pulmonary edema caused by increased afterload and fluid redistribution to the lungs, even when there is minimal total body fluid accumulation.[32 64 65 75]

Two recent trials evaluating early intensive and sustained vasodilation showed no difference between intravenous vasodilators and high dose diuretics compared with usual care. The GALACTIC trial included 788 patients randomized to early intensive and sustained vasodilation compared with usual care. The vasodilation strategy included sublingual and transdermal nitrates, low dose oral hydralazine for 48 hours, and rapid up-titration of angiotensin converting enzyme inhibitors, angiotensin receptor blockers, or sacubitril valsartan. There was no difference in their primary endpoint of composite all cause mortality or readmission within 180 days.[76] The ELISABETH trial was a stepped wedge, cluster randomized trial conducted in 15 emergency departments in France that included 503 patients randomized to a care bundle of intravenous nitrate boluses, management of precipitating factors, and intravenous diuretics compared with usual care. There was no difference in the number of days alive and out of the hospital within 30 days, or secondary outcomes of 30 day all cause mortality, cardiovascular mortality, readmission, length of hospital stay, or renal function deterioration.[77]

Diuretics

Among patients with fluid overload, diuretics increase the excretion of water and salt. Intravenous loop diuretics, such as furosemide, are usually administered. In the DOSE trial, there was no significant difference in patient oriented outcomes when furosemide was administered at a lower dose (a dose equivalent to the patient's oral dose) or a higher dose.[78] The high dose strategy was associated with greater diuresis and more favorable outcomes in some secondary measures, but was associated with a transient worsening of renal function. Therefore, if a patient is already on a diuretic regimen, the intravenous equivalent of double their home dose is a reasonable first approach, with adjustment based upon clinical response (table 8). If the patient presents with new onset heart failure or is not on maintenance diuretic treatment, intravenous furosemide 40 mg is an acceptable starting dose.[63]

The timing is controversial because some studies suggested rapid administration of diuresis within the first 60-90 minutes was associated with improved oxygenation and reduced mortality,[80-82] while others reported no difference in mortality.[83 84] A recent systematic review and meta-analysis reported no significant reduction in mortality in hospital, but did identify a 30 day mortality reduction among those receiving early intravenous diuresis (odds

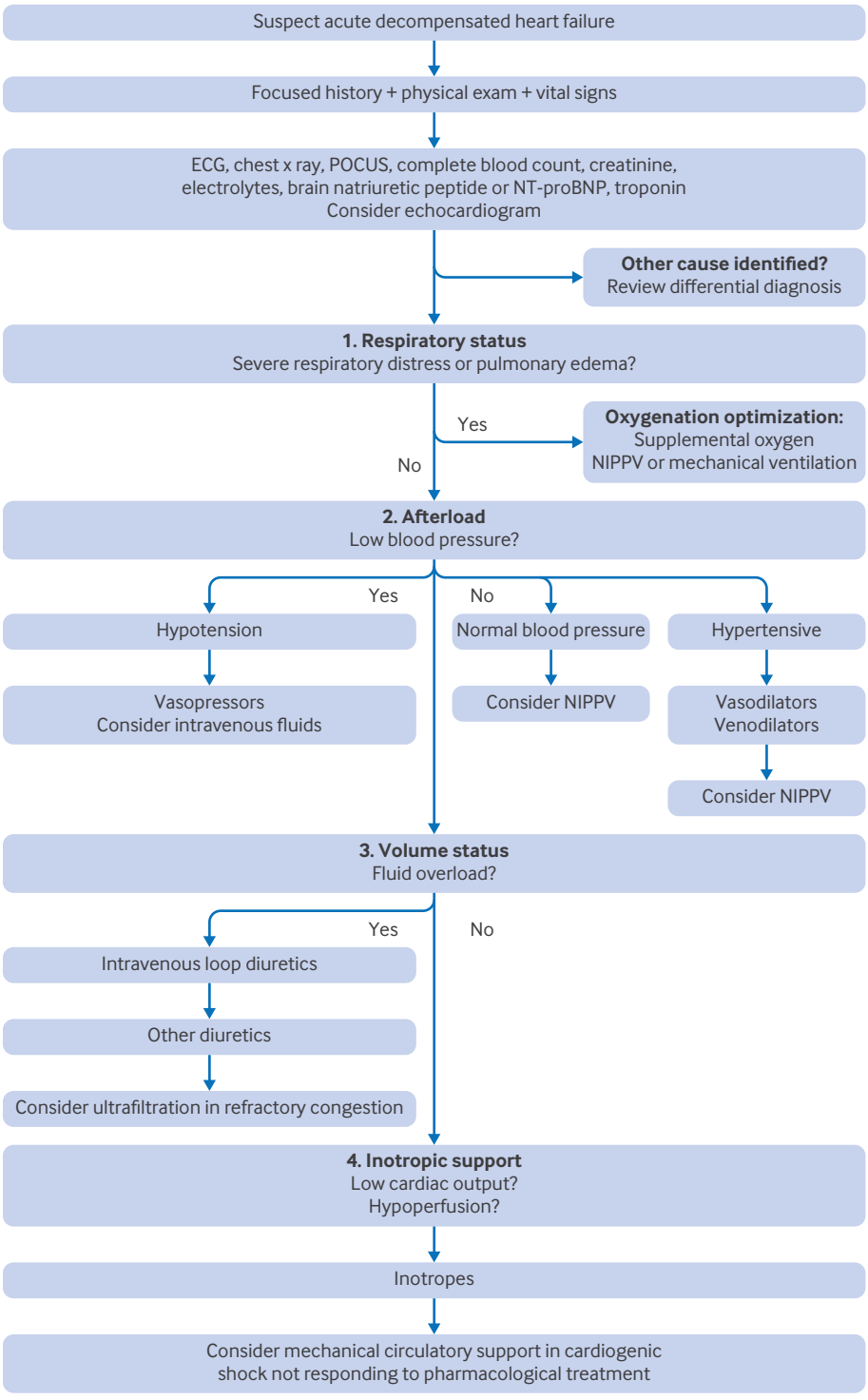

Fig 3 | Management of acute decompensated heart failure. ECG=electrocardiogram; NT-proBNP=N-terminal proB-type natriuretic peptide; NIPPV=non-invasive positive pressure ventilation; POCUS=point-of-care ultrasound

ratio 0.77, 95% confidence interval 0.64 to 0.93).[85] Diuretic response should be evaluated by monitoring urine output or urine sodium with a goal of 100-150 mL/h during the first six hours, or urine sodium content 50-70 mEq/L at two hours.[86 87] If response is inadequate, doses can be doubled, and if still insufficient, additional diuretics acting at different sites of the renal system (eg, thiazides, metolazone, acetazolamide) can be considered with careful monitoring of electrolytes and renal function.[63 88-90] The PUSH-AHF trial found urine sodium guided treatment was associated with improved natriuresis and diuresis, with no difference in adverse events.[91] Studies have also shown a beneficial effect of automated and nursing driven protocols with improved net fluid output and weight loss.[92 93]

Table 8 | Loop diuretic dosing conversion chart[79]

Loop diuretic	Oral (mg)	Intravenous (mg)	Loop diuretic furosemide equivalent
Furosemide	40	20	—
Bumetanide	1	1	Bumetanide 1 mg=furosemide 40 mg by mouth=furosemide 20 mg intravenously
Torsemide	20	20	Torsemide 20 mg=furosemide 40 mg by mouth=furosemide 20 mg intravenously

A recent randomized controlled trial suggested adding acetazolamide 500 mg daily to loop diuretics might increase urine output and expedite systemic decongestion through sequential nephron blockade.[94] The ADVOR trial was a multicenter, randomized, placebo controlled trial of 519 patients with acute heart failure and volume overload, with NT-proBNP levels >1000 pg/mL or BNP >250 pg/mL. Patients received intravenous acetazolamide (500 mg daily) or placebo alongside standard loop diuretics. Successful decongestion within three days was achieved in 42.2% of the acetazolamide group compared with 30.5% of the placebo group (risk ratio 1.46, 95% confidence interval 1.17 to 1.82). Readmission or all cause death occurred in 29.7% of the acetazolamide group and 27.8% of the placebo group (hazard ratio 1.07, 95% confidence interval 0.78 to 1.48). The hospital stay was one day shorter for patients receiving acetazolamide (8.8 days (95% confidence interval 8.0 to 9.5) *v* 9.9 days (9.1 to 10.8)). There were no differences in other outcomes or adverse events.

The CLOROTIC trial enrolled 230 patients with ADHF randomized to oral hydrochlorothiazide (25-100 mg daily) or placebo in addition to intravenous furosemide.[95] Hydrochlorothiazide led to greater weight loss (2.3 *v* 1.5 kg; P=0.002), but there was no difference in dyspnea symptoms. More patients on hydrochlorothiazide had increased serum creatinine (46.5% *v* 17.2%; P<0.001). Heart failure readmission, all cause death rates, and length of stay were similar.

Mineralocorticoid receptor antagonists like spironolactone or eplerenone are commonly used for chronic management because of their role in fluid retention and cardiac remodeling. Mineralocorticoid receptor antagonists can also be added in the acute setting as long as potassium and renal function are not a concern, although the evidence of effectiveness is mixed.[96]

Importantly, a subset of patients with ADHF may be isovolemic with the primary driver of symptoms being sympathetic overactivation resulting in acute hypertension and pulmonary edema. This group, referred to as sympathetic crashing acute pulmonary edema (SCAPE), benefits primarily from vasodilation and NIPPV and may not require routine diuresis.[97]

Ultrafiltration

In the setting of diuretic refractory fluid overload, ultrafiltration or renal replacement therapy may enable more rapid weight loss and reduce readmission for heart failure.[98] The UNLOAD trial compared veno-venous ultrafiltration versus standard intravenous diuresis among 200 patients admitted to hospital for ADHF with evidence of fluid overload and found the ultrafiltration group had more net fluid loss (4.6 *v* 3.3 L; P=0.001) by 48 hours and fewer readmissions for ADHF at 90 days (18% *v* 32%; P=0.037).[99] In contrast, CARRESS-HF randomized 188 patients with ADHF complicated by cardiorenal syndrome and persistent congestion to stepped pharmacological treatment versus ultrafiltration and reported no difference in net fluid loss at 96 hours.[100] Ultrafiltration also carries an increased risk of adverse events, including raised creatinine, bleeding complications, and intravenous catheter related complications.[100] A meta-analysis also found that ultrafiltration was associated with an increased risk of hypotension (odds ratio 2.39, 95% confidence interval 1.20 to 4.76).[98]

Table 9 | Ottawa Heart Failure Risk Score

Input	Points
History	
History of stroke or transient ischemic attack	1
History of intubation for respiratory distress	2
Examination	
Heart rate on emergency department arrival ≥110	2
Room air oxygen saturation <90% on arrival	1
Heart rate ≥110 during 3 minute walk test after emergency department treatment (or too ill to perform)	1
Investigations	
Electrocardiogram with acute ischemic changes	2
Urea ≥12 mmol/L (33 mg/dL)	1
Serum CO_2 ≥25 mmol/L (mEq/L)	2
Troponin I or T raised to myocardial infarction level	2
NT-ProBNP ≥5000 ng/L	1

NT-ProBNP=N-terminal proB-type natriuretic peptide.
Risk of adverse event within 14 days: 0 points: 2.8% (low); 1-2 points: 5.1-9.8% (medium); 3-4 points: 15.9-26.1% (high); 5-9 points: 39.8-89% (very high).

Vasopressors and inotropes

A subset of patients will have ADHF with cardiogenic shock, resulting in poor forward flow and hypotension. Cardiogenic shock is defined clinically as a life threatening hypotension with rapidly escalating inotropic or pressor support, and critical organ hypoperfusion (often confirmed by worsening acidosis and lactate levels).[26] Other criteria can include hypotension (systolic blood pressure <90 mm Hg or mean arterial pressure <60 mm Hg), low cardiac output (cardiac index <2.2 L/min/m^2), or pulmonary capillary wedge pressure >15 mm Hg.[26]

These patients often require a combination of vasopressors and inotropic agents.[75] Norepinephrine can be used as a first line agent based on its ability to provide vasoconstriction and inotropic benefits. Epinephrine can also be considered, though

Box 2: Emergency Heart Failure Mortality Risk Grade Criteria

- Age
- Systolic blood pressure
- Heart rate
- O_2 saturation
- Creatinine
- Potassium
- Transport by emergency medical services
- Troponin positive
- Active cancer
- On outpatient metolazone

one randomized trial of norepinephrine versus epinephrine in cardiogenic shock after myocardial infarction reported higher rates of refractory shock with epinephrine.[101] Inotropes may improve hemodynamics, reduce congestion, and increase cardiac output, thereby improving peripheral perfusion. Among patients with low cardiac output and peripheral hypoperfusion, requiring inotropic support, data suggest that milrinone or dobutamine are reasonable, with milrinone showing a slightly lower mortality rate in overall ADHF compared with dobutamine; however, no difference was seen in the subgroup with ADHF and cardiogenic shock.[102] Close communication with a heart failure specialist is recommended in patients with ADHF requiring vasopressors or inotropes.

Implementation

Importantly, implementation to practice remains a persistent challenge. Among patients admitted to hospital, use of guideline directed medical treatment remains underused during the hospital stay and upon discharge.[103] This can be influenced by time constraints, diagnostic uncertainty, resource limitations, or knowledge translation. To address these gaps, some experts have proposed a framework across the care continuum, including patients, clinicians, and public health initiatives.[104]

Box 3: Multiple Estimation of Risk Based on Spanish Emergency Department Score

- Barthel index at emergency department presentation
- Systolic blood pressure
- Age
- NT-proBNP
- Potassium
- NYHA class IV on presentation
- Positive troponin level
- Respiratory rate
- Low output symptoms
- O_2 saturation
- Episode associated with acute coronary syndrome
- Hypertrophy on electrocardiogram
- Creatinine

NT-ProBNP=N-terminal proB-type natriuretic peptide; NYHA=New York Heart Association

Disposition

Although most patients with ADHF are admitted to hospital, several risk stratification tools exist to identify patients at lower risk who may be appropriate for discharge.[7 105] The Ottawa Heart Failure Risk Score was developed to evaluate the risk of 14 day and 30 day adverse events among patients with ADHF who are older than 50 years and attending the emergency department (table 9).[33 106] The Emergency Heart Failure Mortality Risk Grade calculates a seven day mortality risk based on 10 risk factors using an online risk calculator (https://coachcalculator.ices.on.ca/#/; box 2).[107-109] The Multiple Estimation of Risk Based on Spanish Emergency Department Score predicts 30 day mortality in ADHF using an online calculator based on 13 criteria (https://meessi-ahf.risk.score-calculator-ica-semes.portalsemes.org/; box 3).[110 111] The HEARTRISK6 scale was published in 2024 and includes six criteria to predict 30 day adverse events or 14 day return visits (fig 4).[112] The STRATIFY risk stratification tool was developed, externally validated, and owing to its complexity, has been embedded into the electronic health record, requiring no calculation by clinicians.[113- 116] It is designed to predict patients with ADHF at low risk for 30 day complications, and therefore potentially eligible for discharge consideration. Importantly, these tools should only serve as adjuncts to clinical decision making and should not replace it.

The COACH trial was a cross sectional, stepped wedge, cluster randomized trial with 5452 patients enrolled at 10 hospitals in Ontario, Canada, and used the Emergency Heart Failure Mortality Risk Grade 30-Day Mortality-ST Depression score.[117] Patients at low risk were recommended for early discharge (up to three days) with standardized outpatient care, while patients at intermediate and high risk were admitted. Despite similar early discharge rates (57% *v* 58%), the trial showed a 12% reduction in all cause death or admission to hospital for cardiovascular conditions within 30 days in the intervention group compared with the control group (adjusted hazard ratio 0.88, 95% confidence interval 0.78 to 0.99), indicating a positive effect of care after discharge. In the COACH trial, follow-up was with an internist or cardiologist for patients at low risk who were discharged early (median 6 days, interquartile range 3-12) compared with usual care (median 12 days, interquartile range 5-29).

The STRONG-HF trial emphasized the importance of early assessment and up-titration of heart failure treatments in patients discharged after being admitted to hospital for ADHF.[118] The trial included 1078 patients randomized to usual care versus high intensity care. High intensity care included early and rapid intensification of oral heart failure drugs such as angiotensin converting enzyme inhibitors (or angiotensin receptor blockers with or without neprilysin inhibitor), β blockers, and mineralocorticoid receptor antagonists. The primary outcome of heart failure readmission or all cause death at 180 days occurred in 15.2% of patients in

	Points
Initial assessment	
(a) History of valvular heart disease	1
(b) Heart rate	
(i) ≥100 bpm to <120 bpm	2
(ii) ≥120 bpm	3
(c) Treated with non-invasive ventilation	2
Investigations	
(a) Creatinine	
(i) ≥150 µmol/L to <300 µmol/L (≥1.7 mg/dL to <3.4 mg/dL)	2
(ii) ≥300 µmol/L (≥3.4 mg/dL)	3
(b) Troponin	
(i) ≥3x or 4x upper reference limit	1
(ii) ≥5x upper reference limit	2
Fails reassessment after ED treatment (2-6 hours)	
(a) Resting vital signs abnormal (O_2 saturation <90% on room air or usual O_2 heart rate ≥110 bpm, or respiratory rate ≥28) or (b) Unable to start or complete 3 min walk test	1

Risk of short term serious outcomes		
Total score	**Absolute risk (%)**	**Category**
0	6.4	Low
1	8.5	
2	11.3	Medium
3	14.9	
4	19.4	
5	24.8	
6	31.2	High
7	38.3	
8	46.0	
9	53.9	
≥10	61.6	

Fig 4 | HEARTRISK6 scale. bpm=beats per minute; ED=emergency department

the high intensity care group versus 23.3% in the usual care group. Readmissions were reduced in the high intensity group and there was no significant difference in all cause mortality by day 180 or rates of serious adverse events in both groups.

On discharge, it is important to ensure patients have close primary care or cardiology follow-up for re-evaluation and medical optimization. This should include dietary and exercise counseling, drug adjustment (eg, diuretics, antihypertensive agents), and referral for invasive procedures (eg, implantable cardiac defibrillators, cardiac resynchronization treatment) if appropriate. Although beyond the scope of this paper, an in-depth review of modern management of chronic heart failure in the outpatient setting is available from Heidenreich and Sandhu.[119] Before discharge, all patients should have an understanding of the findings, follow-up plan, and indications to return.

Emerging treatments

Sodium-glucose cotransporter 2 inhibitors

Sodium-glucose cotransporter 2 inhibitors (eg, canagliflozin, dapagliflozin, empagliflozin, ertugliflozin) are glucose lowering agents that block the sodium-glucose cotransporter 2 protein located in the proximal convoluted tubule of the nephron for adults with diabetes mellitus. Recent research has proposed a potential role for management in ADHF. EMPULSE was a randomized trial of empagliflozin in ADHF.[120] The primary endpoint was clinical benefit using a composite measure of death, number of heart failure events, and symptom score change at 90 days. Heart failure events included hospital admissions

or urgent, unplanned outpatient visits that required intensification of treatment. Patients were randomized within three days of hospital admission and treated for 90 days. The primary endpoint was achieved more in patients treated with empagliflozin than placebo (stratified win ratio 1.36, P=0.005). Efficacy was independent of ejection fraction and diabetes status. Adverse events rates were similar between groups. A subsequent meta-analysis of randomized controlled trials found early initiation of sodium-glucose cotransporter 2 inhibitors in ADHF was associated with reduced rates of hospital admissions (risk ratio 0.79, 95% confidence interval 0.72 to 0.87) and acute kidney injury (0.76, 0.59 to 0.99).[121]

Soluble guanylate cyclase stimulators

Vericiguat is a new oral soluble guanylate cyclase stimulator, which enhances the cyclic guanosine monophosphate pathway by directly stimulating soluble guanylate cyclase to reduce oxidative stress and improve endothelial dysfunction.[122] A recent meta-analysis of four randomized controlled trials found that the addition of vericiguat 10 mg to those recently admitted to hospital for ADHF had a reduced risk of heart failure related hospital admission (risk ratio 0.92, 95% confidence interval 0.84 to 1.00), but no difference in cardiovascular or all cause mortality.[123]

Cardiac myosin activators

Cardiac myosin activators (eg, omecamtiv mecarbil) are a newer class of myotropes that improve myocardial function by directly augmenting cardiac sarcomere function. A recent randomized controlled trial of cardiac myosin activators in symptomatic chronic heart failure with an ejection fraction less than or equal to 35% reported a reduction in the composite outcome of cardiovascular death, hospital admission for heart failure, or urgent outpatient visit for heart failure at first event (hazard ratio 0.92, 95% confidence interval 0.86 to 0.99).[124]

Guidelines

Several clinical practice guidelines and consensus documents exist for the management of ADHF. Overall, these recommend the use of biomarkers like BNP or NT-proBNP; chest radiographs and echocardiography to assess heart size, pulmonary congestion, and rule out other causes; use of risk scores to estimate mortality risk; maintenance or optimization of guideline directed medical treatment during hospital admission; use of intravenous diuretics for fluid overload; venodilators or vasodilators for afterload; and inotropic support or temporary mechanical circulatory support to maintain systemic perfusion and end organ function among those with cardiogenic shock. A summary of the guidelines is presented in supplementary table 1.[26 28 125 126]

Conclusion

ADHF affects millions of people worldwide and is associated with high morbidity and mortality rates.

QUESTIONS FOR FUTURE RESEARCH

- What is the optimal combination of history, physical examination, and testing to accurately diagnose acute decompensated heart failure (ADHF)?
- Which populations with ADHF will benefit most from angiotensin converting enzyme inhibitors or vasodilators in addition to high dose nitroglycerin and non-invasive positive pressure ventilation?
- What is the optimal combination of elements to identify those with ADHF at low risk to enable safe discharge while reducing hospital admission rates?
- What new pharmacological agents offer advantages in real world settings?

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS MANUSCRIPT

We discussed this review with patients with lived heart failure experiences. They emphasized the importance of clear communication and explaining information in patient centric terminology and language. One patient explained there was a primary focus on their symptoms early on, while no one explicitly told them they had heart failure until they followed up as an outpatient. Patients also emphasized the importance of explaining the consequences of heart failure, risk factors for decompensation, and the role of diet, exercise, and drugs. One patient highlighted the challenges with finding the right combination of drugs and that this led to frequent exacerbations and emergency department visits. There was also fear about the ability to return to work, take care of their family and loved ones, and being able to coordinate care after discharge. Patients stressed the impact on quality of life, including both the physical and psychological impact of heart failure. Finally, they emphasized the role of clear instructions after discharge from the hospital. Based upon this, we expanded our disposition section and added language emphasizing the importance of communication and coordination of care.

The management of ADHF requires a comprehensive approach that includes accurate diagnosis, timely intervention, and effective treatment strategies. Key diagnostic tools include history and physical examination, electrocardiography, chest radiography, POCUS, and laboratory testing. Management strategies focus on hemodynamic stabilization, symptom relief, and addressing underlying causes. NIPPV, intravenous vasodilators, and diuretics are essential components of treatment. Among patients with hypotension or those with cardiogenic shock, vasopressors, inotropic agents, and mechanical circulatory support might be necessary. Evidence based clinical practice guidelines should be followed to optimize patient outcomes in ADHF.

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1 Storrow AB, Jenkins CA, Self WH, et al. The burden of acute heart failure on U.S. emergency departments. *JACC Heart Fail* 2014;2:269-77. doi:10.1016/j.jchf.2014.01.006.
2 Martin SS, Aday AW, Almarzooq ZI, et al, American Heart Association Council on Epidemiology and Prevention Statistics committee and stroke statistics subcommittee. 2024 heart disease and Stroke Statistics: a report of US and global data From the American Heart Association. *Circulation* 2024;149:e347-913. doi:10.1161/CIR.0000000000001209.
3 Tsao CW, Aday AW, Almarzooq ZI, et al. Heart disease and stroke statistics-2022 update: a report from the American Heart Association. *Circulation* 2022;145:e153-639. doi:10.1161/CIR.0000000000001052.
4 Rosano GMC, Seferovic P, Savarese G, et al. Impact analysis of heart failure across European countries: an ESC-HFA position paper. *ESC Heart Fail* 2022;9:2767-78. doi:10.1002/ehf2.14076.
5 Bozkurt B, Ahmad T, Alexander K, et al, WRITING COMMITTEE MEMBERS. HF STATS 2024: heart failure epidemiology and outcomes statistics an updated 2024 report from the Heart Failure Society of America. *J Card Fail* 2025;31:66-116. doi:10.1016/j.cardfail.2024.07.001.
6 Hollenberg SM, Warner Stevenson L, Ahmad T, et al. 2019 ACC Expert Consensus Decision Pathway on risk assessment, management, and clinical trajectory of patients hospitalized with heart failure: a report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol* 2019;74:1966-2011. doi:10.1016/j.jacc.2019.08.001.
7 Gottlieb M, Moyer E, Bernard K. Epidemiology of heart failure presentations to United States emergency departments from 2016 to 2023. *Am J Emerg Med* 2024;86:70-3. doi:10.1016/j.ajem.2024.09.059.
8 Chang PP, Chambless LE, Shahar E, et al. Incidence and survival of hospitalized acute decompensated heart failure in four US communities (from the Atherosclerosis Risk in Communities Study). *Am J Cardiol* 2014;113:504-10. doi:10.1016/j.amjcard.2013.10.032.
9 Nieminen MS, Brutsaert D, Dickstein K, et al, EuroHeart Survey InvestigatorsHeart Failure Association, European Society of Cardiology. EuroHeart Failure Survey II (EHFS II): a survey on hospitalized acute heart failure patients: description of population. *Eur Heart J* 2006;27:2725-36. doi:10.1093/eurheartj/ehl193.
10 Follath F, Yilmaz MB, Delgado JF, et al. Clinical presentation, management and outcomes in the Acute Heart Failure Global Survey of Standard Treatment (ALARM-HF). *Intensive Care Med* 2011;37:619-26. doi:10.1007/s00134-010-2113-0.
11 Jones NR, Roalfe AK, Adoki I, Hobbs FDR, Taylor CJ. Survival of patients with chronic heart failure in the community: a systematic review and meta-analysis. *Eur J Heart Fail* 2019;21:1306-25. doi:10.1002/ejhf.1594.
12 Sayed A, Abramov D, Fonarow GC, et al. Reversals in the decline of heart failure mortality in the US, 1999 to 2021. *JAMA Cardiol* 2024;9:585-9. doi:10.1001/jamacardio.2024.0615.
13 Echouffo-Tcheugui JB, Bishu KG, Fonarow GC, Egede LE. Trends in health care expenditure among US adults with heart failure: The Medical Expenditure Panel Survey 2002-2011. *Am Heart J* 2017;186:63-72. doi:10.1016/j.ahj.2017.01.003.
14 Kazi DS, Elkind MSV, Deutsch A, et al, American Heart Association. Forecasting the economic burden of cardiovascular disease and stroke in the United States through 2050: a presidential advisory from the American Heart Association. *Circulation* 2024;150:e89-101. doi:10.1161/CIR.0000000000001258.
15 Cook C, Cole G, Asaria P, Jabbour R, Francis DP. The annual global economic burden of heart failure. *Int J Cardiol* 2014;171:368-76. doi:10.1016/j.ijcard.2013.12.028.
16 Lesyuk W, Kriza C, Kolominsky-Rabas P. Cost-of-illness studies in heart failure: a systematic review 2004-2016. *BMC Cardiovasc Disord* 2018;18:74. doi:10.1186/s12872-018-0815-3.
17 Sandhu AT, Tisdale RL, Rodriguez F, et al. Disparity in the setting of incident heart failure diagnosis. *Circ Heart Fail* 2021;14:e008538. doi:10.1161/CIRCHEARTFAILURE.121.008538.
18 Loehr LR, Rosamond WD, Chang PP, Folsom AR, Chambless LE. Heart failure incidence and survival (from the Atherosclerosis Risk in Communities study). *Am J Cardiol* 2008;101:1016-22. doi:10.1016/j.amjcard.2007.11.061.
19 Bahrami H, Kronmal R, Bluemke DA, et al. Differences in the incidence of congestive heart failure by ethnicity: the multi-ethnic study of atherosclerosis. *Arch Intern Med* 2008;168:2138-45. doi:10.1001/archinte.168.19.2138.
20 Huffman MD, Berry JD, Ning H, et al. Lifetime risk for heart failure among white and black Americans: cardiovascular lifetime risk pooling project. *J Am Coll Cardiol* 2013;61:1510-7. doi:10.1016/j.jacc.2013.01.022.
21 Rethy L, Petito LC, Vu THT, et al. Trends in the prevalence of self-reported heart failure by race/ethnicity and age from 2001 to 2016. *JAMA Cardiol* 2020;5:1425-9. doi:10.1001/jamacardio.2020.3654.
22 Butler J, Yang M, Manzi MA, et al. Clinical course of patients with worsening heart failure with reduced ejection fraction. *J Am Coll Cardiol* 2019;73:935-44. doi:10.1016/j.jacc.2018.11.049.
23 Glynn P, Lloyd-Jones DM, Feinstein MJ, Carnethon M, Khan SS. Disparities in cardiovascular mortality related to heart failure in the United States. *J Am Coll Cardiol* 2019;73:2354-5. doi:10.1016/j.jacc.2019.02.042.
24 Tromp J, Bamadhaj S, Cleland JGF, et al. Post-discharge prognosis of patients admitted to hospital for heart failure by world region, and national level of income and income disparity (REPORT-HF): a cohort study. *Lancet Glob Health* 2020;8:e411-22. doi:10.1016/S2214-109X(20)30004-8.
25 Bragazzi NL, Zhong W, Shu J, et al. Burden of heart failure and underlying causes in 195 countries and territories from 1990 to 2017. *Eur J Prev Cardiol* 2021;28:1682-90. doi:10.1093/eurjpc/zwaa147.
26 Heidenreich PA, Bozkurt B, Aguilar D, et al, ACC/AHA Joint Committee Members. 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation* 2022;145:e895-1032. doi:10.1161/CIR.0000000000001063.
27 Bozkurt B. Proposed new conceptualization for definition of decompensated hf: taking the acute out of decompensation. *JACC Heart Fail* 2023;11:368-71. doi:10.1016/j.jchf.2023.02.001.
28 McDonagh TA, Metra M, Adamo M, et al, ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J* 2021;42:3599-726. doi:10.1093/eurheartj/ehab368.
29 Hollenberg SM, Stevenson LW, Ahmad T, et al, Writing Committee. 2024 ACC expert consensus decision pathway on clinical assessment, management, and trajectory of patients hospitalized with heart failure focused update: a report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol* 2024;84:1241-67. doi:10.1016/j.jacc.2024.06.002.
30 Javaloyes P, Miró Ò, Gil V, et al, ICA-SEMES Research Group. Clinical phenotypes of acute heart failure based on signs and symptoms of perfusion and congestion at emergency department presentation and their relationship with patient management and outcomes. *Eur J Heart Fail* 2019;21:1353-65. doi:10.1002/ejhf.1502.
31 Chioncel O, Mebazaa A, Harjola VP, et al, ESC Heart Failure Long-Term Registry Investigators. Clinical phenotypes and outcome of patients hospitalized for acute heart failure: the ESC Heart Failure Long-Term Registry. *Eur J Heart Fail* 2017;19:1242-54. doi:10.1002/ejhf.890.
32 Chioncel O, Mebazaa A, Maggioni AP, et al, ESC-EORP-HFA Heart Failure Long-Term Registry Investigators. Acute heart failure congestion and perfusion status - impact of the clinical classification on in-hospital and long-term outcomes; insights from the ESC-EORP-HFA Heart Failure Long-Term Registry. *Eur J Heart Fail* 2019;21:1338-52. doi:10.1002/ejhf.1492.
33 Stiell IG, Perry JJ, Clement CM, et al. Prospective and explicit clinical validation of the Ottawa Heart Failure Risk Scale, with and without use of quantitative NT-proBNP. *Acad Emerg Med* 2017;24:316-27. doi:10.1111/acem.13141.
34 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;23:223-42. doi:10.1111/acem.12878.
35 Roncalli J, Picard F, Delarche N, et al. Predictive criteria for acute heart failure in emergency department patients with acute dyspnoea: the PREDICA study. *Eur J Emerg Med* 2019;26:400-4. doi:10.1097/MEJ.0000000000000622.
36 Cardinale L, Volpicelli G, Lamorte A, Martino J, Andrea Veltri. Revisiting signs, strengths and weaknesses of standard chest radiography in patients of acute dyspnea in the emergency department. *J Thorac Dis* 2012;4:398-407. doi:10.3978/j.issn.2072-1439.2012.05.05.
37 Collins SP, Lindsell CJ, Storrow AB, Abraham WT, ADHERE Scientific Advisory Committee, Investigators and Study Group. Prevalence of negative chest radiography results in the emergency department patient with decompensated heart failure. *Ann Emerg Med* 2006;47:13-8. doi:10.1016/j.annemergmed.2005.04.003.
38 Yampolsky S, Kwan A, Cheng S, Kedan I. Point of care ultrasound for diagnosis and management in heart failure: a targeted literature review. *POCUS J* 2024;9:117-30. doi:10.24908/pocus.v9i1.16795.

39 Pivetta E, Goffi A, Nazerian P, et al, Study Group on Lung Ultrasound from the Molinette and Careggi Hospitals. Lung ultrasound integrated with clinical assessment for the diagnosis of acute decompensated heart failure in the emergency department: a randomized controlled trial. *Eur J Heart Fail* 2019;21:754-66. doi:10.1002/ejhf.1379.
40 Chiu L, Jairam MP, Chow R, et al. Meta-analysis of point-of-care lung ultrasonography versus chest radiography in adults with symptoms of acute decompensated heart failure. *Am J Cardiol* 2022;174:89-95. doi:10.1016/j.amjcard.2022.03.022.
41 Maw AM, Hassanin A, Ho PM, et al. Diagnostic accuracy of point-of-care lung ultrasonography and chest radiography in adults with symptoms suggestive of acute decompensated heart failure: a systematic review and meta-analysis. *JAMA Netw Open* 2019;2:e190703. doi:10.1001/jamanetworkopen.2019.0703.
42 Darwish OS, Mahayni A, Kataria S, Zuniga E, Zhang L, Amin A. Diagnosis of acute heart failure using inferior vena cava ultrasound: systematic review and meta-analysis. *J Ultrasound Med* 2020;39:1367-78. doi:10.1002/jum.15231.
43 Unlüer EE, Karagöz A, Akoğlu H, Bayata S. Visual estimation of bedside echocardiographic ejection fraction by emergency physicians. *West J Emerg Med* 2014;15:221-6. doi:10.5811/westjem.2013.9.16185.
44 Mandavia DP, Hoffner RJ, Mahaney K, Henderson SO. Bedside echocardiography by emergency physicians. *Ann Emerg Med* 2001;38:377-82. doi:10.1067/mem.2001.118224.
45 Alerhand S, Sundaram T, Gottlieb M. What are the echocardiographic findings of acute right ventricular strain that suggest pulmonary embolism?*Anaesth Crit Care Pain Med* 2021;40:100852. doi:10.1016/j.accpm.2021.100852.
46 Labovitz AJ, Noble VE, Bierig M, et al. Focused cardiac ultrasound in the emergent setting: a consensus statement of the American Society of Echocardiography and American College of Emergency Physicians. *J Am Soc Echocardiogr* 2010;23:1225-30. doi:10.1016/j.echo.2010.10.005.
47 Baloescu C, Bailitz J, Cheema B, et al. Artificial intelligence-guided lung ultrasound by nonexperts. *JAMA Cardiol* 2025;10:245-53. doi:10.1001/jamacardio.2024.4991.
48 Gottlieb M, Patel D, Viars M, Tsintolas J, Peksa GD, Bailitz J. Comparison of artificial intelligence versus real-time physician assessment of pulmonary edema with lung ultrasound. *Am J Emerg Med* 2023;70:109-12. doi:10.1016/j.ajem.2023.05.029.
49 Gottlieb M, Schraft E, O'Brien J, Patel D. Diagnostic accuracy of artificial intelligence for identifying systolic and diastolic cardiac dysfunction in the emergency department. *Am J Emerg Med* 2024;86:115-9. doi:10.1016/j.ajem.2024.10.019.
50 Bellolio F, Gottlieb M, Body R, Than MP, Hess EP. Evaluating patients with chest pain in the emergency department. *BMJ* 2025;388:r136. doi:10.1136/bmj.r136.
51 Maisel AS, Clopton P, Krishnaswamy P, et al, BNP Multinational Study Investigators. Impact of age, race, and sex on the ability of B-type natriuretic peptide to aid in the emergency diagnosis of heart failure: results from the Breathing Not Properly (BNP) multinational study. *Am Heart J* 2004;147:1078-84. doi:10.1016/j.ahj.2004.01.013.
52 Burke MA, Cotts WG. Interpretation of B-type natriuretic peptide in cardiac disease and other comorbid conditions. *Heart Fail Rev* 2007;12:23-36. doi:10.1007/s10741-007-9002-9.
53 Januzzi JLJr, Chen-Tournoux AA, Christenson RH, et al, ICON-RELOADED Investigators. N-Terminal Pro-B-Type Natriuretic Peptide in the Emergency Department: The ICON-RELOADED Study. *J Am Coll Cardiol* 2018;71:1191-200. doi:10.1016/j.jacc.2018.01.021.
54 Logeart D, Thabut G, Jourdain P, et al. Predischarge B-type natriuretic peptide assay for identifying patients at high risk of re-admission after decompensated heart failure. *J Am Coll Cardiol* 2004;43:635-41. doi:10.1016/j.jacc.2003.09.044.
55 Di Somma S, Magrini L, Pittoni V, et al. In-hospital percentage BNP reduction is highly predictive for adverse events in patients admitted for acute heart failure: the Italian RED Study. *Crit Care* 2010;14:R116. doi:10.1186/cc9067.
56 Long B, Koyfman A, Gottlieb M. Management of heart failure in the emergency department setting: an evidence-based review of the literature. *J Emerg Med* 2018;55:635-46. doi:10.1016/j.jemermed.2018.08.002.
57 Maisel AS, Peacock WF, McMullin N, et al. Timing of immunoreactive B-type natriuretic peptide levels and treatment delay in acute decompensated heart failure: an ADHERE (Acute Decompensated Heart Failure National Registry) analysis. *J Am Coll Cardiol* 2008;52:534-40. doi:10.1016/j.jacc.2008.05.010.
58 Gray A, Goodacre S, Newby DE, Masson M, Sampson F, Nicholl J, 3CPO Trialists. Noninvasive ventilation in acute cardiogenic pulmonary edema. *N Engl J Med* 2008;359:142-51. doi:10.1056/NEJMoa0707992.
59 Berbenetz N, Wang Y, Brown J, et al. Non-invasive positive pressure ventilation (CPAP or bilevel NPPV) for cardiogenic pulmonary oedema. *Cochrane Database Syst Rev* 2019;4:CD005351. doi:10.1002/14651858.CD005351.pub4.
60 Li H, Hu C, Xia J, et al. A comparison of bilevel and continuous positive airway pressure noninvasive ventilation in acute cardiogenic pulmonary edema. *Am J Emerg Med* 2013;31:1322-7. doi:10.1016/j.ajem.2013.05.043.
61 Doshi P, Whittle JS, Bublewicz M, et al. High-velocity nasal insufflation in the treatment of respiratory failure: a randomized clinical trial. *Ann Emerg Med* 2018;72:73-83.e5. doi:10.1016/j.annemergmed.2017.12.006.
62 Yang S, He Y, Liu S, Zhang Y. High-flow nasal cannula oxygen therapy is superior to conventional oxygen therapy but not to non-invasive mechanical ventilation in reducing intubation rate in hypoxia and dyspnea due to acute heart failure: a systematic review and meta-analysis. *Chin Med J (Engl)* 2023;136:479-81. doi:10.1097/CM9.0000000000002227.
63 Mebazaa A, Yilmaz MB, Levy P, et al. Recommendations on pre-hospital & early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine. *Eur J Heart Fail* 2015;17:544-58. doi:10.1002/ejhf.289.
64 Cotter G, Metzkor E, Kaluski E, et al. Randomised trial of high-dose isosorbide dinitrate plus low-dose furosemide versus high-dose furosemide plus low-dose isosorbide dinitrate in severe pulmonary oedema. *Lancet* 1998;351:389-93. doi:10.1016/S0140-6736(97)08417-1.
65 Levy P, Compton S, Welch R, et al. Treatment of severe decompensated heart failure with high-dose intravenous nitroglycerin: a feasibility and outcome analysis. *Ann Emerg Med* 2007;50:144-52. doi:10.1016/j.annemergmed.2007.02.022.
66 Wilson SS, Kwiatkowski GM, Millis SR, Purakal JD, Mahajan AP, Levy PD. Use of nitroglycerin by bolus prevents intensive care unit admission in patients with acute hypertensive heart failure. *Am J Emerg Med* 2017;35:126-31. doi:10.1016/j.ajem.2016.10.038.
67 Patrick C, Fornage L, Ward B, et al. Safety of prehospital intravenous bolus dose nitroglycerin in patients with acute pulmonary edema: a 4-year review. *J Am Coll Emerg Physicians Open* 2023;4:e13079. doi:10.1002/emp2.13079.
68 Wang K, Samai K. Role of high-dose intravenous nitrates in hypertensive acute heart failure. *Am J Emerg Med* 2020;38:132-7. doi:10.1016/j.ajem.2019.06.046.
69 Khot UN, Novaro GM, Popović ZB, et al. Nitroprusside in critically ill patients with left ventricular dysfunction and aortic stenosis. *N Engl J Med* 2003;348:1756-63. doi:10.1056/NEJMoa022021.
70 Ayaz SI, Sharkey CM, Kwiatkowski GM, et al. Intravenous enalaprilat for treatment of acute hypertensive heart failure in the emergency department. *Int J Emerg Med* 2016;9:28. doi:10.1186/s12245-016-0125-4.
71 Ibarra FJr, Holzmann S, Shah S, et al. Utility of nicardipine in the management of hypertensive crises in adults with reduced ejection fractions. *Am J Emerg Med* 2024;75:79-82. doi:10.1016/j.ajem.2023.10.041.
72 Harrison N, Pang P, Collins S, Levy P. Blood pressure reduction in hypertensive acute heart failure. *Curr Hypertens Rep* 2021;23:11. doi:10.1007/s11906-021-01127-8.
73 Sharon A, Shpirer I, Kaluski E, et al. High-dose intravenous isosorbide-dinitrate is safer and better than Bi-PAP ventilation combined with conventional treatment for severe pulmonary edema. *J Am Coll Cardiol* 2000;36:832-7. doi:10.1016/S0735-1097(00)00785-3.
74 Publication Committee for the VMAC Investigators (Vasodilatation in the Management of Acute CHF). Intravenous nesiritide vs nitroglycerin for treatment of decompensated congestive heart failure: a randomized controlled trial. *JAMA* 2002;287:1531-40. doi:10.1001/jama.287.12.1531.
75 Mebazaa A, Motiejunaite J, Gayat E, et al, ESC Heart Failure Long-Term Registry Investigators. Long-term safety of intravenous cardiovascular agents in acute heart failure: results from the European Society of Cardiology Heart Failure Long-Term Registry. *Eur J Heart Fail* 2018;20:332-41. doi:10.1002/ejhf.991.
76 Kozhuharov N, Goudev A, Flores D, et al, GALACTIC Investigators. Effect of a strategy of comprehensive vasodilation vs usual care on mortality and heart failure rehospitalization among patients with acute heart failure: the GALACTIC randomized clinical trial. *JAMA* 2019;322:2292-302. doi:10.1001/jama.2019.18598.
77 Freund Y, Cachanado M, Delannoy Q, et al. Effect of an emergency department care bundle on 30-day hospital discharge and survival among elderly patients with acute heart failure: the ELISABETH randomized clinical trial. *JAMA* 2020;324:1948-56. doi:10.1001/jama.2020.19378.
78 Felker GM, Lee KL, Bull DA, et al, NHLBI Heart Failure Clinical Research Network. Diuretic strategies in patients with acute decompensated heart failure. *N Engl J Med* 2011;364:797-805. doi:10.1056/NEJMoa1005419.

79 Konerman M, Bozaan D, Adie S, et al. *Michigan Medicine Inpatient Diuretic Guideline for Patients with Acute Decompensated Heart Failure.* [Internet] Michigan Medicine University of Michigan, 2022.
80 Matsue Y, Damman K, Voors AA, et al. Time-to-furosemide treatment and mortality in patients hospitalized with acute heart failure. *J Am Coll Cardiol* 2017;69:3042-51. doi:10.1016/j.jacc.2017.04.042.
81 Mikami T, Ishii M, Yamamoto N, et al. Association of early administration of furosemide with improved oxygenation in patients with acute heart failure. *ESC Heart Fail* 2021;8:3354-9. doi:10.1002/ehf2.13379.
82 Marques P, Brito MT, Vasques-Nóvoa F, et al. Door-to-furosemide time and clinical outcomes in acute heart failure. *Eur J Emerg Med* 2023;30:85-90. doi:10.1097/MEJ.0000000000001006.
83 Park JJ, Kim S-H, Oh I-Y, et al. The effect of door-to-diuretic time on clinical outcomes in patients with acute heart failure. *JACC Heart Fail* 2018;6:286-94. doi:10.1016/j.jchf.2017.12.017.
84 Ouwerkerk W, Tromp J, Cleland JGF, et al. Association of time-to-intravenous furosemide with mortality in acute heart failure: data from REPORT-HF. *Eur J Heart Fail* 2023;25:43-51. doi:10.1002/ejhf.2708.
85 Sampaio Rodrigues T, Garcia Quarto LJ, Nogueira SC, et al. Door-to-diuretic time and mortality in patients with acute heart failure: A systematic review and meta-analysis. *Am Heart J* 2024;269:205-9. doi:10.1016/j.ahj.2023.12.012.
86 Mullens W, Damman K, Harjola V-P, et al. The use of diuretics in heart failure with congestion - a position statement from the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail* 2019;21:137-55. doi:10.1002/ejhf.1369.
87 Damman K, Ter Maaten JM, Coster JE, et al. Clinical importance of urinary sodium excretion in acute heart failure. *Eur J Heart Fail* 2020;22:1438-47. doi:10.1002/ejhf.1753.
88 Mullens W, Verbrugge FH, Nijst P, et al. Rationale and design of the ADVOR (Acetazolamide in Decompensated Heart Failure with Volume Overload) trial. *Eur J Heart Fail* 2018;20:1591-600. doi:10.1002/ejhf.1307.
89 Cox ZL, Hung R, Lenihan DJ, Testani JM. Diuretic strategies for loop diuretic resistance in acute heart failure: the 3T trial. *JACC Heart Fail* 2020;8:157-68. doi:10.1016/j.jchf.2019.09.012.
90 Felker GM, Ellison DH, Mullens W, Cox ZL, Testani JM. Diuretic therapy for patients with heart failure: JACC state-of-the-art review. *J Am Coll Cardiol* 2020;75:1178-95. doi:10.1016/j.jacc.2019.12.059.
91 Ter Maaten JM, Beldhuis IE, van der Meer P, et al. Natriuresis-guided diuretic therapy in acute heart failure: a pragmatic randomized trial. *Nat Med* 2023;29:2625-32. doi:10.1038/s41591-023-02532-z.
92 Meegan G, Sangkachand P, O'Brien M, et al. Safety and efficacy of an automated nurse-driven diuretic titration protocol: the Yale Diuretic Pathway. *J Card Fail* 2020;26:S22. doi:10.1016/j.cardfail.2020.09.070.
93 Rao VS, Ivey-Miranda JB, Cox ZL, et al. Natriuretic equation to predict loop diuretic response in patients with heart failure. *J Am Coll Cardiol* 2021;77:695-708. doi:10.1016/j.jacc.2020.12.022.
94 Mullens W, Dauw J, Martens P, et al, ADVOR Study Group. Acetazolamide in acute decompensated heart failure with volume overload. *N Engl J Med* 2022;387:1185-95. doi:10.1056/NEJMoa2203094.
95 Trullàs JC, Morales-Rull JL, Casado J, et al, CLOROTIC trial investigators. Combining loop with thiazide diuretics for decompensated heart failure: the CLOROTIC trial. *Eur Heart J* 2023;44:411-21. doi:10.1093/eurheartj/ehac689.
96 Schuermans A, Verbrugge FH. Decongestion (instead of ultrafiltration?). *Curr Opin Cardiol* 2024;39:188-95. doi:10.1097/HCO.0000000000001124.
97 Long B, Brady WJ, Gottlieb M. Emergency medicine updates: sympathetic crashing acute pulmonary edema. *Am J Emerg Med* 2025;90:35-40. doi:10.1016/j.ajem.2024.12.061.
98 Shi X, Bao J, Zhang H, Wang H, Li L, Zhang Y. Patients with high-dose diuretics should get ultrafiltration in the management of decompensated heart failure: a meta-analysis. *Heart Fail Rev* 2019;24:927-40. doi:10.1007/s10741-019-09812-2.
99 Costanzo MR, Guglin ME, Saltzberg MT, et al, UNLOAD Trial Investigators. Ultrafiltration versus intravenous diuretics for patients hospitalized for acute decompensated heart failure. *J Am Coll Cardiol* 2007;49:675-83. doi:10.1016/j.jacc.2006.07.073.
100 Bart BA, Goldsmith SR, Lee KL, et al, Heart Failure Clinical Research Network. Ultrafiltration in decompensated heart failure with cardiorenal syndrome. *N Engl J Med* 2012;367:2296-304. doi:10.1056/NEJMoa1210357.
101 Levy B, Clere-Jehl R, Legras A, et al, Collaborators. Epinephrine versus norepinephrine for cardiogenic shock after acute myocardial infarction. *J Am Coll Cardiol* 2018;72:173-82. doi:10.1016/j.jacc.2018.04.051.
102 Biswas S, Malik AH, Bandyopadhyay D, et al. Meta-analysis comparing the efficacy of dobutamine versus milrinone in acute decompensated heart failure and cardiogenic shock. *Curr Probl Cardiol* 2023;48:101245. doi:10.1016/j.cpcardiol.2022.101245.
103 Zheng J, Sandhu AT, Bhatt AS, et al. Inpatient use of guideline-directed medical therapy during heart failure hospitalizations among community-based health systems. *JACC Heart Fail* 2025;13:43-54. doi:10.1016/j.jchf.2024.08.004.
104 DeVore AD, Bosworth HB, Granger BB. Improving implementation of evidence-based therapies for heart failure. *Clin Cardiol* 2022;45(Suppl 1):S52-9. doi:10.1002/clc.23845.
105 Rider I, Sorensen M, Brady WJ, et al. Disposition of acute decompensated heart failure from the emergency department: An evidence-based review. *Am J Emerg Med* 2021;50:459-65. doi:10.1016/j.ajem.2021.08.070.
106 Stiell IG, Clement CM, Brison RJ, et al. A risk scoring system to identify emergency department patients with heart failure at high risk for serious adverse events. *Acad Emerg Med* 2013;20:17-26. doi:10.1111/acem.12056.
107 Lee DS, Stitt A, Austin PC, et alPrediction of heart failure mortality in emergent care: a cohort study. *Ann Intern Med* 2012;156:767-75. doi:10.7326/0003-4819-156-11-201206050-00003
108 Lee DS, Lee JS, Schull MJ, et al. Prospective validation of the Emergency Heart Failure Mortality Risk Grade for acute heart failure. *Circulation* 2019;139:1146-56. doi:10.1161/CIRCULATIONAHA.118.035509.
109 Sepehrvand N, Youngson E, Bakal JA, McAlister FA, Rowe BH, Ezekowitz JA. External validation and refinement of Emergency Heart Failure Mortality Risk Grade risk model in patients with heart failure in the emergency department. *CJC Open* 2019;1:123-30. doi:10.1016/j.cjco.2019.03.003.
110 Miró Ò, Rossello X, Gil V, et al, ICA-SEMES Research Group. Analysis of how emergency physicians' decisions to hospitalize or discharge patients with acute heart failure match the clinical risk categories of the MEESSI-AHF Scale. *Ann Emerg Med* 2019;74:204-15. doi:10.1016/j.annemergmed.2019.03.010.
111 Wussler D, Kozhuharov N, Sabti Z, et al. External validation of the MEESSI Acute Heart Failure Risk Score: a cohort study. *Ann Intern Med* 2019;170:248-56. doi:10.7326/M18-1967.
112 Stiell IG, Perry JJ, Eagles D, et al. The HEARTRISK6 scale: predicting short-term serious outcomes in emergency department acute heart failure patients. *JACC Adv* 2024;3:100988. doi:10.1016/j.jacadv.2024.100988.
113 van Dam PMEL, van Doorn WPTM, van Gils F, et al. Machine learning for risk stratification in the emergency department (MARS-ED) study protocol for a randomized controlled pilot trial on the implementation of a prediction model based on machine learning technology predicting 31-day mortality in the emergency department. *Scand J Trauma Resusc Emerg Med* 2024;32:5. doi:10.1186/s13049-024-01177-2.
114 Collins SP, Jenkins CA, Harrell FEJr. Identification of emergency department patients with acute heart failure at low risk for 30-day adverse events: the STRATIFY decision tool. *JACC Heart Fail* 2015;3:737-47. doi:10.1016/j.jchf.2015.05.007.
115 Stolldorf DP, Storrow AB, Liu D, et al. A mixed-methods observational study of strategies for success in implementation science: overcoming emergency departments hurdles. *BMC Health Serv Res* 2025;25:147. doi:10.1186/s12913-024-12102-9.
116 Christensen M, Reale C, Anders S, et al. Special Issue on CDS Failures: "We'll blow right by it": barriers to uptake of the STRATIFY-CDS for acute heart failure. *Appl Clin Inform* 2025; [forthcoming].
117 Lee DS, Straus SE, Farkouh ME, et al, COACH Trial Investigators. Trial of an intervention to improve acute heart failure outcomes. *N Engl J Med* 2023;388:22-32. doi:10.1056/NEJMoa2211680.
118 Mebazaa A, Davison B, Chioncel O, et al. Safety, tolerability and efficacy of up-titration of guideline-directed medical therapies for acute heart failure (STRONG-HF): a multinational, open-label, randomised, trial. *Lancet* 2022;400:1938-52. doi:10.1016/S0140-6736(22)02076-1.
119 Heidenreich P, Sandhu A. Advances in management of heart failure. *BMJ* 2024;385:e077025. doi:10.1136/bmj-2023-077025.
120 Voors AA, Angermann CE, Teerlink JR, et al. The SGLT2 inhibitor empagliflozin in patients hospitalized for acute heart failure: a multinational randomized trial. *Nat Med* 2022;28:568-74. doi:10.1038/s41591-021-01659-1.
121 Laborante R, Paglianiti DA, Bianchini E, et alSafety and efficacy of early initiation of sodium-glucose co-transporter inhibitors 2 in patients hospitalized for acute heart failure: A meta-analysis of randomized controlled trials. *Eur J Intern Med* 2025;135:p55-63. doi:10.1016/j.ejim.2025.01.014
122 Armstrong PW, Pieske B, Anstrom KJ, et al, VICTORIA Study Group. Vericiguat in patients with heart failure and reduced ejection fraction. *N Engl J Med* 2020;382:1883-93. doi:10.1056/NEJMoa1915928.
123 Yang H, Luo C, Lan W-Q, Tang YH. Vericiguat treatment of heart failure: a systematic review and meta-analysis. *World J Clin Cases* 2023;11:8330-42. doi:10.12998/wjcc.v11.i35.8330.

124 Teerlink JR, Diaz R, Felker GM, et al, GALACTIC-HF Investigators. Cardiac myosin activation with omecamtiv mecarbil in systolic heart failure. *N Engl J Med* 2021;384:105-16. doi:10.1056/NEJMoa2025797.
125 Silvers SM, Gemme SR, Hickey S, et al, American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Acute Heart Failure Syndromes. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute heart failure syndromes: approved by ACEP Board of Directors, June 23, 2022. *Ann Emerg Med* 2022;80:e31-59. doi:10.1016/j.annemergmed.2022.05.027.
126 Ponikowski P, Voors AA, Anker SD, et al, ESC Scientific Document Group. 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;37:2129-200. doi:10.1093/eurheartj/ehw128.

Web appendix: Supplementary table 1
Video 1: B-lines on point-of-care thoracic ultrasound suggestive of pulmonary edema
bmj-2025-084242-vid1The BMJ Video Player
Video 2: Decreased ejection fraction on point-of-care cardiac ultrasound
bmj-2025-084242-vid2The BMJ Video Player