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# Diagnosis of Acute Heart Failure in the Emergency Department: An Evidence-Based Review

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Heart failure is a common presentation to the emergency department (ED), which can be confused with other clinical conditions. This review provides an evidence-based summary of the current ED evaluation of heart failure. Acute heart failure is the gradual or rapid decompensation of heart failure, resulting from either fluid overload or maldistribution. Typical symptoms can include dyspnea, orthopnea, or systemic edema. The physical examination may reveal pulmonary rales, an S3 heart sound, or extremity edema. However, physical examination findings are often not sensitive or specific. ED assessments may include electrocardiogram, complete blood count, basic metabolic profile, liver function tests, troponin, brain natriuretic peptide, and a chest radiograph. While often used, natriuretic peptides do not significantly change ED treatment, mortality, or readmission rates, although they may decrease hospital length of stay and total cost. Chest radiograph findings are not definitive, and several other conditions may mimic radiograph findings. A more reliable modality is point-of-care ultrasound, which can facilitate the diagnosis by assessing for B-lines, cardiac function, and inferior vena cava size. These modalities, combined with clinical assessment and gestalt, are recommended. [West J Emerg Med. 2019;20(6)875-884.]

## INTRODUCTION

Acute heart failure (AHF) is a gradual or rapid decompensation in heart failure (HF) requiring urgent management.[1-4] The condition covers a large spectrum of disease, ranging from mild exacerbations with gradual increases in edema to cardiogenic shock. HF affects close to six million people in the United States (U.S.) and increases in prevalence with age.[6-11] Currently, the emergency department (ED) initiates the evaluation and treatment of over 80% of patients with AHF in the U.S.[12-17] As the population ages, increasing numbers of patients with HF will present to the ED for evaluation and management. However, making the correct diagnosis can be challenging due to the broad differential diagnosis associated with presenting symptoms and variations in patient presentations.

Over one million patients are admitted for HF in the U.S. and Europe annually.[6-11,16-20] In the U.S. population, people have a 20% risk of developing HF by 40 years of age.[21-25] HF is more common in males until the age of 65, at which time males and females are equally affected.[25-28] Patients with HF average at least two hospital admissions per year.[25,29,30] Among patients who are admitted with AHF, over 80% have a prior history of HF, referred to as decompensated heart failure.[20-23] De novo HF is marked by no previous history of HF combined with symptom appearance after an acute event.[3,4,19,23] Mortality in patients with HF can be severe, with up to half of all patients dying within five years of disease diagnosis.[20,21,25] Other studies have found that post-hospitalization mortality rates at 30 days, one year, and five years are 10.4%, 22%, and 42.3%, respectively.[23-27] AHF expenditures approach $39 billion per year, which is expected to almost double by 2030.[31,32]

## METHODS

We searched PubMed and Google Scholar for articles using the keywords "heart failure" and "emergency." We included retrospective studies, prospective studies, systematic reviews and meta-analyses, clinical guidelines, and narrative

reviews focusing on diagnosis of HF including history and physical examination, biomarkers, electrocardiogram (ECG), and imaging. The literature search was restricted to studies published in English. Emergency physicians with experience in critical appraisal of the literature reviewed all of the articles and decided which studies to include for the review by consensus, with a focus on emergency medicine-relevant articles. A total of 124 articles were selected for inclusion in this review.

## DISCUSSION
### Anatomy and Pathophysiology

Normal cardiac physiology is dependent on appropriately functioning ventricular contraction, ventricular wall structural integrity, and valvular competence.[28,33,34] At normal functional status, a person's stroke volume (SV) is approximately one milliliter (mL) per kilogram for every heartbeat.[28,33-36] SV is dependent upon the preload (defined as the amount of myocardial muscle fiber stretch at the end of ventricular filling), afterload (defined as the amount of vascular resistance the ventricle must overcome), and contractility (defined as the strength of the myocardial contraction). In patients with HF, left ventricular (LV) dysfunction can be due to impaired LV contraction and ejection (systolic dysfunction), impaired relaxation and filling (diastolic dysfunction), or a combination of both.[28,33]

An alternate way of defining this would be by the effect on ejection fraction (EF). HF with preserved EF refers to patients with an EF > 50%, while HF with reduced EF refers to patients with an EF < 40%. Borderline preserved EF is defined by HF with an EF of 41-50%.[3,4,17,18,29] The most common form is HF with reduced EF, which is primarily related to a decrease in the functional myocardium (typically associated with ischemic disease or a prior myocardial infarction).[3,4,34] Additional causes include excessive pressure overload from hypertension, valvular incompetence, and cardiotoxic medications. HF with preserved EF occurs due to impaired ventricle relaxation and filling, which accounts for 30-45% of all HF cases.[22,23,33,37,38]

This form of HF results in increased end-systolic and diastolic volumes and pressures and is most commonly associated with chronic hypertension, coronary artery disease, diabetes mellitus, cardiomyopathy, and valvular disease. Both systolic and diastolic HF can present with similar symptoms due to elevated, left-sided intracardiac pressures and pulmonary congestion.[25,28,33-36]

Right ventricular failure most commonly results from LV failure. As the right side of the heart fails, increased pressure in the vena caval system elevates pressure in the venous system of the gastrointestinal tract, liver, and extremities, resulting in edema, jugular venous distension, hepatomegaly, bloating, abdominal pain, and nausea.[25,28,33,34] High-output HF is associated with normal or greater-than-normal cardiac output and decreased systemic vascular resistance.[34-38] The associated decrease in afterload reduces arterial blood pressure and also activates neurohormones, which increase salt and water retention. Diseases that may result in high-output HF include anemia, large arteriovenous fistula or multiple small fistulas, severe hepatic or renal disease, hyperthyroidism, beriberi disease, and septic shock.[36-38]

In AHF, peripheral vascular flow and end-organ perfusion decrease, causing the body to compensate by neurohormonal activation (ie, the renin-angiotensin system), ventricular remodeling, and release of natriuretic peptides.[25,28,34,35] These mechanisms are chronically activated in HF, but worsen during acute exacerbations, resulting in hemodynamic abnormalities leading to further deterioration. Continued progression can result in a critical reduction to end-organ blood flow, leading to severe morbidity and mortality.[3,4,25,28,33-35]

### Heart Failure Classification

Patients with HF are classified into one of four classes, primarily determined by daily function, using the New York Heart Association, American College of Cardiology/American Heart Association, or European Society of Cardiology Guidelines (Table 1).[17,18,39-41] These systems help determine

**Table 1.** Heart failure classification systems.[17,18,39-41]

| NYHA | ACC/AHA | ESC guidelines |
|---|---|---|
| Class I: No symptoms with ordinary activity. | Stage A: Patient is at high risk for developing HF. | 1. Heart failure with reduced ejection fraction (< 40%). |
| Class II: Slight limitation with physical activity. No issues at rest, but physical activity can result in fatigue, palpitations, dyspnea, or angina. | Stage B: Patient has structural heart disorder but no symptoms of HF. | 2. Heart failure with mid-range ejection fraction (40-49%). |
| Class III: Severe limitation in physical activity. Comfortable at rest. However, less than normal physical activity results in fatigue, palpitations, dyspnea, or angina. | Stage C: Patient has past or current symptoms of HF with underlying structural heart disease. | 3. Heart failure with preserved ejection fraction (> 50%). |
| Class IV: Unable to perform physical activity without discomfort. Symptoms may be present at rest. | Stage D: Patient has end-stage disease and requires specialized treatment strategies. | |

*NYHA,* New York Heart Association; *ACC/AHA,* American College of Cardiology/American Heart Association; *ESC,* European Society of Cardiology; *HF,* heart failure.

the appropriate interventions to reduce the likelihood of developing severe LV dysfunction, thereby reducing the patient's potential morbidity and mortality.[3,4,17,18,34] Other means of classification depend on the presence of cardiomyopathy or acute coronary syndrome (ACS). The Nohria-Stevenson classification for decompensated HF in the setting of cardiomyopathy uses perfusion and congestion, while the Killip and Forrester classification systems evaluate AHF in the setting of ACS.[12,17,18,39-45] In general, short-term mortality is low for well-perfused groups and is higher in poorly-perfused patients.[12,17,18,39-45]

Unfortunately, these classification systems are not as useful for acute exacerbation of HF, thereby limiting their applicability in the ED setting. In the ED, classification is based upon the patient's hemodynamic status, perfusion, and blood pressure.[3,4,30,42] This differentiation can guide therapy and provides important prognostic information. Most patients are hypertensive or normotensive upon presentation.[16-22] The hypertensive form (associated with a systolic blood pressure > 140 millimeters of mercury (mmHg)) is commonly associated with pulmonary edema, which may occur rapidly (ie, flash pulmonary edema).[46,47] In the normotensive progressive form, systemic edema is predominant.[16-22,30] Hypotensive AHF is associated with end-organ hypoperfusion, while systemic and pulmonary edema is minimal. ACS can occur simultaneously with or exacerbate HF and requires emergent coronary angiography.[48,49] Right-sided HF is associated with right ventricular dysfunction, leading to systemic venous congestion without pulmonary edema if the LV is not involved.[3,4,30]

## History and Physical Examination

Due to the complex pathophysiology involved in HF and multiple phenotypes (eg, low- vs high-output, preserved vs reduced EF, left-sided vs right-sided), the history and physical examination may vary. Patients with HF are heterogeneous in terms of the cardiac structure and function, the etiology of their HF, the precipitant of the AHF exacerbation, comorbidities, and current medications. Early diagnosis is vital, as a delay or misdiagnosis has been associated with an increased risk of adverse outcomes and death.[50-52] Misdiagnosis occurs in up to one-third of patients upon initial presentation.[53-56] While no single historical factor or examination finding can significantly reduce the likelihood of HF in isolation, initial clinical gestalt has been shown to have a sensitivity of 61% and specificity of 86% for the diagnosis.[57,58]

Risk factors for HF include hypertension, renal disease, heart disease, diabetes, male gender, older age, and obesity.[58-61] In particular, advanced age, renal disease, and lower blood pressure are associated with increased mortality in AHF.[60,61] Precipitating factors for AHF exacerbation can include cardiac and non-cardiac causes.[63,64] Cardiac causes include uncontrolled hypertension, dietary or medication noncompliance, aortic dissection, dysrhythmias, and cardiac ischemia.[30,59,63,64] Non-cardiac causes include pulmonary disease, endocrine disease,

infection, worsening renal function, anemia, and medication side effects.[3,4,30,59] Patients who are noncompliant with their diet and medications have been found to have a lower EF, higher brain-type natriuretic peptide (BNP) levels, and greater congestion when compared with their counterparts.[30,63,64] Dysrhythmias are another frequent precipitating cause. Among those, atrial fibrillation is the most common.[17,18,21,29] ACS is more commonly associated with de novo HF.[17,18,29] Components of the history such as weight gain, dyspnea, chest pain, peripheral edema, substance abuse, new medications, past complications, prior hospitalizations, diet changes (eg, salt or fluid intake), and medication compliance are vital to determine the underlying etiology, and an identifiable trigger can be found in approximately 60% of patients.[58-62]

Acutely, the most common symptoms associated with AHF include paroxysmal nocturnal dyspnea (PND), orthopnea, and edema.[16,29,30,57-59] The most common manifestation is dyspnea or edema from elevated LV filling pressures.[4,57-59] However, the classic symptoms such as PND, dyspnea, and orthopnea demonstrate poor sensitivity and specificity (Table 2).[59,65-67]

On examination, an S3 heart sound has the highest specificity, ranging from 97.7–99%, but it has only 12.7% sensitivity.[53,54,57-59] Additionally, an S3 heart sound can be difficult to detect in the ED setting, and inter-rater reliability can be poor.[3,4,59] Hepato-jugular reflux and jugular venous distension possess a specificity of 93.4% and 87% and sensitivity 14.1% and 37.2%, respectively, for HF.[57-59] Lung auscultation is also less reliable, as the presence of rales has a sensitivity of approximately 60% and a specificity approaching 70%.[57-59] Lower extremity edema has a sensitivity of 50% and specificity 78%.[57-59] A meta-analysis evaluating various signs and symptoms in patients with dyspnea found that no single sign or symptom was sufficiently able to rule out AHF, chronic obstructive pulmonary disease, asthma, or pulmonary embolism.[65] However, elevated jugular venous pressure, third heart sound, and lung crepitations were strongly suggestive of a diagnosis of AHF.[65]

## Laboratory Testing

Laboratory assessment in the patient with suspected AHF can provide important diagnostic and prognostic information.[3,4,30,58,59] Testing should include a complete blood count, basic metabolic panel with renal function testing, liver function testing, troponin, and a BNP level.[30,48-50-47,55,56] Abnormalities in liver function are found in approximately 75% of patients with AHF and are associated with more severe disease.[30,69] If the right ventricle is involved, bilirubin and alkaline phosphatase levels may be elevated, while left-sided disease is more commonly associated with elevated transaminase levels.[30,69] Renal function is an important assessment, as it is a predictor of disease severity and mortality.[15-18,70] Decreased glomerular filtration rate (GFR) is associated with increased length of in-hospital stay, short-term mortality, and long-term mortality.[17,18,70-72] In patients with AHF,

**Table 2.** History and examination findings in acute heart failure.[59]

| Finding | Sensitivity (95% CI) | Specificity (95% CI) | +LR (95% CI) | -LR (95% CI) |
|---|---|---|---|---|
| Orthopnea | 52.1 (50.1–54.0) | 70.5 (68.8–72.1) | 1.9 (1.4–2.5) | 0.74 (0.64–0.85) |
| PND | 46.2 (43.7–48.6) | 73.9 (71.9–75.9) | 1.6 (1.2–2.1) | 0.79 (0.71–0.88) |
| Dyspnea at rest | 54.6 (51.2–58.0) | 49.6 (46.9–52.3) | 1.1 (0.9–1.4) | 0.88 (0.74–1.04) |
| No productive cough | 82.0 (79.6–84.4) | 25.8 (23.5–28.2) | 1.13 (1.02–1.26) | 0.6 (0.5–0.8) |
| History of CHF | 55.5 (53.9–57.1) | 80.2 (79.0–81.3) | 2.7 (2.0–3.7) | 0.58 (0.49–0.68) |
| History of MI | 31.8 (29.7–33.9) | 87.1 (85.8–88.3) | 2.1 (1.8–2.5) | 0.82 (0.76–0.89) |
| History of AF | 30.2 (27.4–33.2) | 85.3 (82.8–87.5) | 2.1 (1.6–2.9 | 0.82 (0.71–0.93) |
| History of CAD | 46.6 (44.5–48.7) | 76.2 (74.6–77.7) | 2.0 (1.7–2.4) | 0.71 (0.64–0.79) |
| History of DM | 28.8 (27.4–30.4) | 81.7 (80.4–82.8) | 1.5 (1.3–1.7) | 0.89 (0.84–0.94) |
| History of CRD | 32.0 (29.4–34.6) | 91.4 (90.0–92.7) | 3.4 (2.7–4.5) | 0.75 (0.71–0.80) |
| History of HTN | 66.9 (65.5–68.3) | 50.7 (49.4–52.1) | 1.3 (1.3–1.4) | 0.62 (0.53–0.73) |
| S3 | 12.7 (11.5–14.0) | 97.7 (97.2–98.2) | 4.0 (2.7–5.9) | 0.91 (0.88–0.95) |
| JVD | 37.2 (35.7–38.7) | 87.0 (85.9–88.0) | 2.8 (1.7–4.5) | 0.76 (0.69–0.84) |
| Hepato-jugular reflex | 14.1 (11.9–16.6) | 93.4 (91.2–95.2) | 2.2 (1.3–3.7) | 0.91 (0.88–0.94) |
| Leg edema | 51.9 (50.5- 53.4) | 75.2 (74.0–76.4) | 1.9 (1.6–2.3) | 0.68 (0.61–0.75) |
| Rales | 62.3 (60.8–63.7) | 68.1 (66.7–69.4) | 1.8 (1.5–2.1) | 0.60 (0.51–0.69) |
| Wheeze | 22.3 (20.9–23.8) | 64.0 (62.5–65.4) | 0.6 (0.5–0.8) | 1.19 (1.10–1.30) |
| No fever | 92.4 (90.9–93.8) | 20.6 (18.8–22.5) | 1.14 (1.02–1.27) | 0.4 (0.3–0.6) |
| Murmur | 27.8 (25.8–29.9) | 83.2 (81.6–84.8) | 1.9 (0.9–3.9) | 0.93 (0.79–1.08) |

*CI,* confidence interval; *PND,* paroxysmal nocturnal dyspnea; *CHF,* congestive heart failure; *MI,* myocardial infarction; *AF,* atrial fibrillation; *CAD,* coronary artery disease; *DM,* diabetes mellitus; *CRD,* chronic respiratory disease; *HTN,* hypertension; *JVD,* jugular venous distension.

every 10 mL/minute decrease in GFR is associated with an increase in mortality of 7%.[71,72]

Troponin testing can assist in prognostication and in the detection of underlying ischemia as a potential inciting event for AHF. Elevated troponin levels are associated with higher re-hospitalization rates and 90-day mortality.[17,18,48,49] Troponin elevation is common in AHF, as one study found elevated troponin levels in 98% of patients with diagnosed AHF, with 81% of the levels above the 99th percentile.[73] Other studies have suggested that this may be closer to 30-50%.[3,4,30] However, an elevated troponin is not specific for ACS and may be seen with a variety of other causes, including demand ischemia and renal dysfunction.[17,18,48-50]

Natriuretic peptides (ie, BNP and NT-proBNP) may be a valuable adjunct when the provider is unclear of the diagnosis.[57-59,74-77] BNP is produced by cardiac myocytes when exposed to significant myocardial stretch. Use of BNP and NT-proBNP may be sensitive, but not specific for the diagnosis of AHF. Levels less than 100 picograms (pg) per milliliter (mL) for BNP have demonstrated a sensitivity and specificity of 93.5% and 52.9%, respectively, with negative likelihood ratio (LR-) of 0.2.[57-59] Using a 300 pg/mL cut-off for NT-proBNP demonstrates a LR- of 0.09.[59] However, elevated levels only moderately increase the likelihood of AHF, as specificity improves to 72.9% with a value of 1550 pg/mL for NT-

proBNP.[59,74-79] A BNP level > 400 pg/mL or a NT-proBNP level > 900 pg/mL is consistent with AHF; however, in patients over the age of 75 years, the NT-proBNP level should be increased to 1800 pg/mL.[3,4,30,74-77] Obesity can falsely lower the natriuretic peptides levels,[3,4,30,74-76,79] while renal disease may falsely elevate levels (especially with GFR < 60 mL/min).[74,75,80,81]

Other conditions associated with elevations in natriuretic peptide levels include pulmonary embolism, pulmonary hypertension, valvular heart disease, and acute respiratory distress syndrome. BNP levels of 100-400 pg/mL and NT-proBNP levels of 300-900 pg/mL are non-specific and may require further testing.[74-77,82-87] Although these biomarkers may assist in differentiation of other conditions, studies have not demonstrated improved patient-centered outcomes with use of natriuretic peptides .[86-88] Observational trial data suggest natriuretic peptides demonstrate sensitivity over 90%, but specificity is poor.[80,88-92] Data from randomized, controlled trials found that knowledge of the BNP levels did not significantly change the ED treatment, mortality, or readmission rates; however, it may decrease hospital length of stay and total cost.[76,93-99]

**Electrocardiogram**

An ECG should be rapidly obtained to evaluate for the etiology or precipitating factors (eg, ACS, atrial fibrillation with rapid ventricular response, ventricular dysrhythmia).[3,4,26,57,59]

An ECG is unlikely to diagnose or exclude AHF in isolation.[57,59,100,101] Prolonged QRS and junctional rhythms are associated with worse patient outcomes.[100,101] Table 3 demonstrates ECG findings in AHF.[57,100,101]

## Imaging

Imaging is an important component in the patient with suspected heart failure. The most common modality used is the chest radiograph (CXR). Several findings suggest the diagnosis of heart failure on CXR, including cardiomegaly, central vascular congestion, and interstitial edema (Table 4).[17,18,41,102] However, a normal CXR should not be used to exclude the diagnosis of AHF, as up to 20% of CXRs may appear normal in AHF.[4,102-106] Studies evaluating physician accuracy with identifying AHF on CXR have demonstrated sensitivities of 59-74.5% and specificities of 86.3-96%.[59,103-105] While CXR should not be used to exclude AHF, it can be valuable for identifying alternate disease processes that may mimic AHF.[3,4,102-105]

Bedside ultrasound can be valuable for diagnosing AHF, with high specificity and positive likelihood ratios (Table 5). Ultrasound can be used to evaluate for B-lines, pleural effusions, inferior vena cava size and respiro-phasic variability, and cardiac contractility.[59,106-108] B-lines are vertical artifacts that result from sound wave reverberation through fluid-filled pulmonary interstitium. The presence of greater than three B-lines in two bilateral lung zones defines a positive lung ultrasound examination.[56,106-113] The number of lung zones examined varies in the literature, with eight thoracic lung zones used in the initial lung ultrasound protocols, while newer studies have used four or six lung zones. B-lines demonstrate high sensitivity and specificity for interstitial edema,[59,107,108] while the identification of pleural effusions is not as helpful.[59]

Assessment of EF on ultrasound may be assessed with visual assessment or quantitative measurements. Qualitative visual estimation is made by assessing the inward movement of the interventricular septum and inferior wall of the LV during systole.[59,106-113] E-point septal separation (EPSS) is a quantitative measurement assessing the distance between the anterior mitral valve leaflet and ventricular septum. An EPSS measurement > 7 mm is suggestive of an EF < 50%.[111-114] Ultrasound can also estimate intravascular volume through the measurement of inferior vena cava diameter and percentage change during the respiratory cycle. However, diagnostic performance is controversial, with many confounding factors and a wide range of sensitivities and specificities.[115-117] One study found that by using a combination of lung, cardiac, and inferior vena cava ultrasound, the authors were able to improve diagnostic accuracy by 20%.[118] Others have suggested that combining CXR with ultrasound may increase the sensitivity and specificity for diagnosing AHF.[103]

**Table 3.** Electrocardiogram findings in acute heart failure.[59]

| Finding | Sensitivity (95% CI) | Specificity (95% CI) | +LR (95% CI) | -LR (95% CI) |
|---|---|---|---|---|
| Ischemic changes | 34.0 (29.8–38.4) | 84.2 (81.2–86.9) | 2.9 (1.2–7.1) | 0.78 (0.73–0.84) |
| T-wave inversion | 10.0 (7.5–13.0) | 95.9 (92.3–98.1) | 2.4 (1.2–4.8) | 0.94 (0.90–0.98) |
| ST depression | 5.6 (3.9–7.7) | 96.5 (94.2–98.1) | 2.0 (1.0–3.8) | 0.97 (0.95–1.00) |
| ST elevation | 5.2 (2.1–10.5) | 91.8 (83.8–96.6) | 0.6 (0.2–1.7) | 1.03 (0.96–1.11) |
| Atrial fibrillation | 20.5 (18.3–22.9) | 89.9 (87.9–91.7) | 2.2 (1.4–3.5) | 0.88 (0.85–0.91) |
| Normal sinus rhythm | 55.4 (50.9–60.0) | 17.8 (15.1–20.8) | 0.7 (0.5–0.9) | 2.88 (1.26–6.57) |

*CI,* confidence interval; *LR,* likelihood ratio.

**Table 4.** Chest radiograph findings in acute heart failure.[59]

| Finding | Sensitivity (95% CI) | Specificity (95% CI) | +LR (95% CI) | -LR (95% CI) |
|---|---|---|---|---|
| Kerley B lines | 9.2 (6.5–12.5) | 98.8 (97.3–99.6) | 6.5 (2.6–16.2) | 0.88 (0.69–1.13) |
| Interstitial edema | 31.1 (28.2–34.2) | 95.1 (93.6–96.3) | 6.4 (3.4–12.2) | 0.73 (0.68–0.78) |
| Cephalization | 44.7 (41.1–48.4) | 94.6 (92.6–96.3) | 5.6 (2.9–10.4) | 0.53 (0.39–0.72) |
| Alveolar edema | 5.7 (4.7–6.9) | 98.9 (98.4–99.3) | 5.3 (3.3–8.5) | 0.95 (0.94–0.97) |
| Pulmonary edema | 56.9 (54.7–59.1) | 89.2 (87.9–90.4) | 4.8 (3.6–6.4) | 0.48 (0.39–0.58) |
| Pleural effusion | 16.3 (13.7–19.2) | 92.8 (90.4–94.7) | 2.4 (1.6–3.6) | 0.89 (0.80–0.99) |
| Cardiomegaly | 74.7 (72.9–76.5) | 61.7 (59.4–63.9) | 2.3 (1.6–3.4) | 0.43 (0.36–0.51) |

*CI,* confidence interval; *LR,* likelihood ratio.

**Table 5.** Bedside ultrasound findings in acute heart failure.[59,107]

| Finding | Sensitivity (95% CI) | Specificity (95% CI) | +LR (95% CI) | -LR (95% CI) |
|---|---|---|---|---|
| Positive B lines | 94.1 (81.3-98.3) | 92.7 (90.9–94.3) | 12.4 (5.7-26.8) | 0.06 (0.02–0.22) |
| Pleural effusion | 63.5 (50.4–75.3) | 71.7 (61.4–80.6) | 2.0 (1.4–2.8) | 0.49 (0.22–1.10) |
| Reduced EF | 80.6 (72.9–86.9) | 80.6 (74.3–86.0) | 4.1 (2.4–7.2) | 0.24 (0.17–0.35) |
| Increased LV end-diastolic dimension | 79.6 (65.7–89.7) | 68.6 (50.7–83.1) | 2.5 (1.5–4.2) | 0.30 (0.16–0.54) |
| Restrictive mitral pattern | 81.5 (68.6–90.7) | 90.1 (80.7–95.9) | 8.3 (4.0–16.9) | 0.21 (0.12–0.36) |

*CI*, confidence interval; *LR*, likelihood ratio; *EF*, ejection fraction; *LV*, left ventricular.

## Disposition

Due to the heterogenous nature of heart failure, disposition may be challenging. The majority of patients presenting to the ED in the U.S. with AHF are admitted.[12-14] Patients with hemodynamic instability or critical illness should be admitted to an intensive care unit, and patients with newly diagnosed HF may benefit from admission for further evaluation and management.[17,18,21,119] Other patients who may require admission include those with poor response to medical treatment or inability to obtain follow-up, significant electrolyte abnormalities, elevated blood urea nitrogen or creatinine, or ischemia on ECG or biomarker testing.[120] In those with prior history of HF and the absence of the aforementioned items, risk stratification tools such as the Emergency Heart Failure Mortality Risk Grade or the Ottawa Heart Failure Risk Score may be able to identify a select subset of low-risk patients, but these scoring systems require further validation.[120-124]

## CONCLUSION

Heart failure is a common presentation to the ED, which can be confused with other clinical conditions. Acute heart failure refers to the gradual or rapid decompensation of heart failure, resulting from either fluid overload or maldistribution. Typical symptoms can include dyspnea, orthopnea, or edema. The physical examination may reveal pulmonary rales, an S3 heart sound, or extremity edema. Laboratory studies should include an electrocardiogram, complete blood count, basic metabolic profile, coagulation studies, troponin, brain natriuretic peptide, and a chest radiograph. Point-of-care ultrasound can facilitate the diagnosis by assessing for B-lines, cardiac function, and inferior vena cava size. Understanding the diagnostic approach can improve the diagnostic accuracy and allow for more rapid initiation of the correct intervention.

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