JAMA | The Rational Clinical Examination

# Does This Patient Have Volume Overload? The Rational Clinical Examination

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**IMPORTANCE** Accurate assessment of intravascular volume facilitates management decisions about fluid management in patients with volume overload.

**OBJECTIVE** To identify the most accurate clinical examination, radiographic, and laboratory findings for assessing volume overload in nonintubated patients.

**DATA SOURCES AND STUDY SELECTION** MEDLINE was searched (1946 to January 6, 2026) to identify peer-reviewed English-language studies about the diagnostic accuracy of the clinical examination of spontaneously breathing patients with intravascular volume overload.

**DATA EXTRACTION AND SYNTHESIS** Three authors independently extracted data for each finding and calculated sensitivity, specificity, and likelihood ratios (LRs). A 2-level mixed logistic regression model was used to pool estimates.

**RESULTS** Forty studies, involving 11 490 adult patients, were included, with a prevalence of volume overload of 35% to 69%. Thirty-three of those studies evaluated patients with dyspnea. Prevalence of volume overload was more likely when the physical examination revealed jugular venous distention with the highest point of pulsation more than 3 cm in a vertical line above the sternal angle (LR, 4.1 [95% CI, 2.9-5.6]; specificity, 92%), lower extremity edema (LR, 2.2 [95% CI, 1.5-3.1]; specificity, 80%), or crackles on auscultation (LR, 2.7 [95% CI, 1.7-4.5]; specificity, 81%). Vascular congestion on chest radiography increased the likelihood of intravascular volume overload (LR, 5.9 [95% CI, 2.9-12.0]; specificity, 91%). Point-of-care ultrasonography that identified bilateral pulmonary B-lines suggested volume overload (LR, 4.0 [95% CI, 2.6-6.1]; specificity, 77%), and absence of pulmonary B-lines made volume overload unlikely (LR, 0.09 [95% CI, 0.04-0.23]; sensitivity, 93%). Inferior vena cava collapsibility index of less than 50% increased the likelihood of volume overload (LR, 3.9 [95% CI, 2.5-6.1]; specificity, 79%), and a collapsibility index of at least 50% made it less likely (LR, 0.22 [95% CI, 0.11-0.45]; sensitivity, 82%). Point-of-care ultrasonographic measurement of jugular venous pressure (JVP; >8 cm) also increased the likelihood of volume overload (LR, 2.8 [95% CI, 2.2-3.5]; specificity, 71%), although JVP of 8 cm or less identified patients less likely to have volume overload (LR, 0.26 [95% CI, 0.20-0.33]; sensitivity, 81%). A plasma brain-type natriuretic peptide (BNP) level of 100 ng/mL or higher was the single best test to identify those most likely to have volume overload (LR, 6.9 [95% CI, 2.4-20.4]; specificity, 87%), and a normal value made it less likely (LR, 0.14 [95% CI, 0.08-0.24]; sensitivity, 87%).

**CONCLUSIONS AND RELEVANCE** A BNP level of 100 ng/mL or higher and presence of vascular congestion on chest radiography may be the most useful tests to identify patients with volume overload. Absence of pulmonary B-lines using point-of-care ultrasonography or BNP levels of less than 100 ng/mL may be most useful to exclude volume overload.

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Multimedia

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## Clinical Scenario

A 78-year-old female with atrial fibrillation and a body mass index of 38 (calculated as weight in kilograms divided by height in meters squared) presents with left-leg erythema and edema. Her vital signs reveal a heart rate of 112 beats per minute, temperature 38.5 °C, and oxygen saturation of 86% while breathing room air, which increases to 96% with 3 L of oxygen. Initial laboratory studies are notable for an albumin level of 1.7 g/dL; creatinine, 1.6 mg/dL (141.44 µmol/L); prior baseline creatine of 1.0 mg/dL (88.40 µmol/L); and a brain-type natriuretic peptide (BNP) level of 107 pg/mL (normal, <100 pg/mL). Urinalysis reveals more than 3 g of proteinuria. Chest radiography shows increased vascular markings bilaterally. After 2 days of treatment with loop diuretics for volume overload and antibiotics for cellulitis, her weight decreases by 1 kg, her left leg erythema resolves, and her leg edema has slightly decreased. However, her creatinine level increased to 3.2 mg/dL. Physical examination reveals grade 2 pitting edema to the upper calves and crackles at the lung bases bilaterally with an oxygen requirement of 2 L to maintain an oxygen saturation of 91%. Assessment of jugular venous pressure (JVP) by clinical examination was difficult because of her large body habitus, but her JVP of 11 cm was suggested by point-of-care ultrasonography (POCUS), which also showed an inferior vena cava diameter of 2.5 cm when measured 2 cm from the atrial-caval junction, 10% inferior vena cava collapsibility, and 3 B-lines at each lung base. Has volume overload been confirmed?

## Background

Intravascular volume overload, a state of abnormal expansion of the intravascular compartment, is diagnosed clinically when patients have symptoms such as dyspnea, weight gain, or edema and signs of excess volume such as pulmonary crackles, pulmonary and peripheral edema, and jugular venous distention.[1] Conditions that increase the risk of intravascular volume overload include heart failure, kidney insufficiency, cirrhosis, and excess intravenous fluid administration. Accurate assessment and identification of volume overload affect clinical decision-making about hospital admission, medication use, and recognition of clinical decompensation.

Hospitalized patients can be evaluated daily for volume overload by assessment of symptoms (such as dyspnea and orthopnea) and signs (such as peripheral edema, crackles on lung auscultation, and daily weights). However, the criterion standards for confirming volume overload are hemodynamic pressure measurements obtained during right heart catheterization and quantitative blood volume analysis using radioisotope dilution.[1] Physical examination and POCUS findings provide information without delay whereas radiological and laboratory study results take longer to become available. Although achieving an accurate assessment of volume status can be challenging in hospitalized patients, accurate fluid status assessment and management may prevent complications such as pulmonary edema or poor wound healing associated with peripheral edema.[2]

### Key Points

**Question** Which physical examination, ultrasonographic techniques, and laboratory studies have the best evidence for determining volume overload in spontaneously breathing patients?

**Findings** A brain-type natriuretic peptide level of at least 100 ng/mL and the presence of vascular congestion on chest radiography may be the most useful tests to identify patients with volume overload. Point-of-care ultrasonographic findings of inferior vena cava collapsibility, presence of pulmonary B-lines, and ultrasonographically guided jugular venous pressure may also perform better than physical examination maneuvers for determining volume overload.

**Meaning** In addition to brain-type natriuretic peptide levels and chest radiography, point-of-care ultrasonography can be used for clinical decision-making among patients with possible volume overload.

### Pathophysiology

Intravascular volume overload is caused by sodium and water retention, with subsequent plasma volume expansion and increased hydrostatic pressures (**Figure 1**). Intravascular volume overload is driven by arterial underfilling, a process in which the body perceives a low effective circulating volume, even if the total blood volume is normal or high. This process, often associated with heart failure, kidney disease, or cirrhosis, leads to neurohormonal activation of the renin-angiotensin-aldosterone system, sympathetic nervous system, and arginine vasopressin.[3,4]

### Peripheral Edema

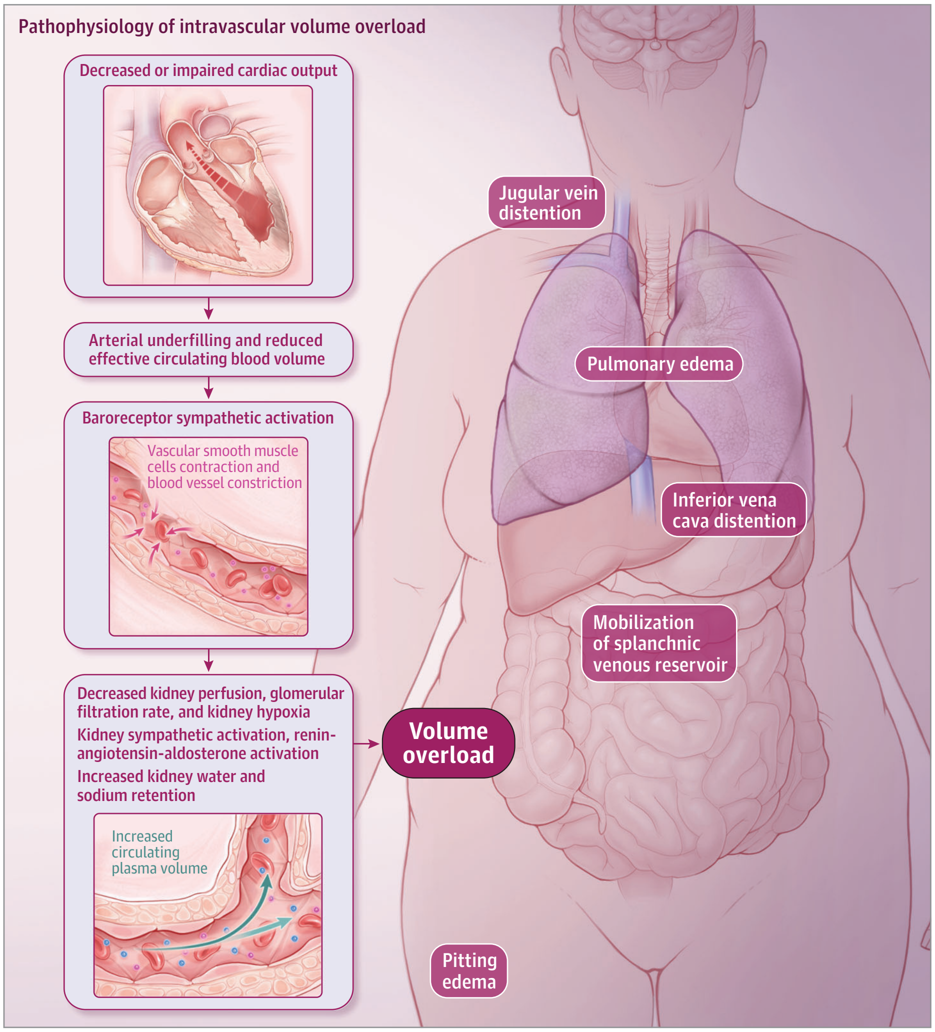
Peripheral edema occurs when capillary filtration exceeds the limits of lymphatic drainage, resulting in accumulation of fluid in the interstitial space. Other factors that contribute to peripheral edema include increased intravascular hydrostatic pressure due to intravascular volume overload, increased vascular permeability, and plasma oncotic pressure.[5] Decreased oncotic pressure associated with hypoalbuminemia may be caused by conditions such as hepatic failure, malnutrition, and proteinuria. Increased intravascular pressure without volume overload may occur in patients with deep vein thrombosis or chronic venous insufficiency.

Peripheral edema is gravity dependent and typically first develops in the feet and ascends up the legs; however, sacral and scrotal edema are also common, particularly in bed-bound patients. Several medications such as calcium channel blockers and gabapentinoids can also cause peripheral edema in the absence of volume overload, primarily through arteriole dilation that increases capillary hydrostatic pressure.

### Pulmonary Interstitial Edema

Pulmonary crackles occur when small airways pop open during inspiration after being obstructed in expiration due to alveolar fluid accumulation or inflammation.[6,7] Pulmonary B-lines identified on POCUS are sonographic artifacts caused by interstitial edema and increased fluid that appear as echogenic vertical lines extending perpendicularly from the pleural line at the top of the ultrasonographic image into the lung parenchyma. Interstitial edema and pulmonary edema also create densities on chest radiographies such as

Figure 1. Intravascular Volume Overload




Kerley B lines, which are horizontal lines in the lung periphery that denote thickened, edematous interlobular septa.[8] Other chest radiographic findings suggesting volume overload include peribronchial cuffing, which is edema of the bronchial walls due to pulmonary vascular congestion, and hazy opacities in the perihilar area caused by pulmonary edema.

## Elevated Right Atrial Pressure

With intravascular volume overload, increased venous fluid return elevates right atrial pressure, which causes jugular venous distension that can be identified on physical examination or with POCUS. Dilation of the inferior vena cava also occurs and can be measured using POCUS.[9] However, jugular venous distention and inferior vena cava dilation may also result from conditions other than volume overload (such as pericardial effusion, pulmonary embolism, and pulmonary hypertension).

## Laboratory Data

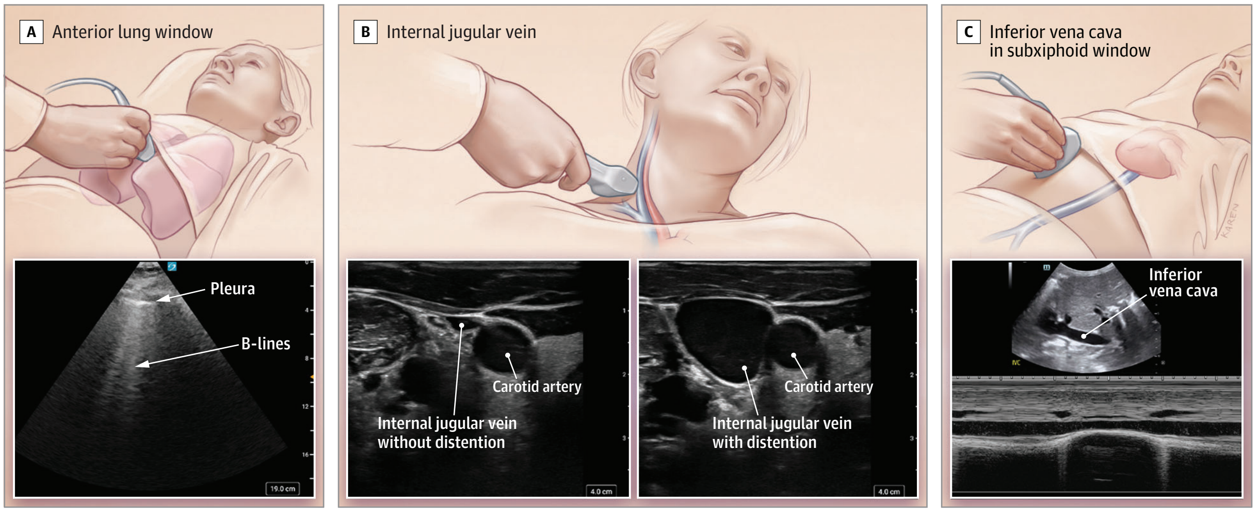
Brain-type natriuretic peptide (BNP) is a hormone secreted by cardiomyocytes in the heart ventricles in response to stretching caused by increased ventricular blood volume; the prohormone of BNP is N-terminal pro-brain natriuretic peptide (NT-proBNP).[10] Both BNP and NT-proBNP are measured in plasma, and laboratory reference values vary by institution. Among euvolemic individuals, BNP and NT-proBNP levels are higher with older age, female sex, kidney failure, and pulmonary hypertension and are lower among individuals with obesity or diabetes (both type 1 and type 2).

## How to Elicit Symptoms and Signs of Volume Overload

### Symptoms

Patients with volume overload often have dyspnea, orthopnea, peripheral edema, weight gain, and paroxysmal nocturnal dyspnea, which involves awakening from sleep due to breathlessness.[11] Orthopnea is best elicited by asking how many pillows are used when sleeping or whether the patient needs to sleep upright.[11] Additional symptoms such as early satiety, abdominal discomfort, and bloating may occur when volume overload causes splanchnic or visceral congestion that leads to abdominal distention. Although rapid weight gain (eg, >2-3 lb [0.45-0.91 kg] in several days) may suggest volume overload (particularly in patients with known heart failure), weight gain could be attributable to other causes such as idiopathic edema or increased caloric intake.

Figure 2. Ultrasonographic Images to Determine Volume Status




A, Hyperechoic B-lines extend perpendicularly and deeply to the pleura in an anterior lung window. The top panel shows probe placement.

B, A cross-sectional view of the internal jugular vein. The top panel shows probe placement.

C, Longitudinal view of the inferior vena cava in the subxiphoid window with M-mode applied 2 cm from the right atrium to assess respiratory variation. The top panel shows probe placement.

### Signs

**Peripheral Edema |** Lower extremity edema is a common physical examination finding of volume overload but is nonspecific because it may occur with other conditions and can be present in patients with hypovolemia, particularly those with hypoalbuminemia and lymphatic obstruction.[12] Evaluation of peripheral edema involves examining the lower extremities for swelling, followed by applying firm pressure to bony prominences (ankles, anterior shins, and feet) for at least 5 seconds, then releasing and observing for persistent indentation or pitting. The degree of pitting (graded from 0, no edema, through 4 based on depth and rebound time), symmetry, and distribution should be recorded. Gravity-dependent areas such as thighs, hips, and sacrum should also be examined. Bilateral, symmetric, pitting edema is most consistent with systemic volume overload.[13]

**Pulmonary Congestion |** Auscultation of the lungs in a patient with intravascular volume overload may reveal pulmonary crackles (discontinuous, popping, and nonmusical adventitious sounds during inspiration).[14] Crackles can be heard by placing the diaphragm of the stethoscope over the posterior and lateral thorax, proceeding down the chest and comparing right to left hemithoraces sequentially.[15]

**Elevated Cardiac Filling Pressures |** Examination of JVP is performed with the patient reclined at 30 to 45 degrees, and the head turned at a slight angle to expose the internal jugular venous pulsations between the heads of the sternocleidomastoid muscle. After the highest point of venous pulsation (the meniscus) is identified, the vertical distance is measured relative to the sternal angle. A JVP of more than 3 cm above the sternal angle (>8 cm to the right atrium) suggests elevated right atrial pressure.[16]

### POCUS

POCUS is a noninvasive imaging tool that can be used in the bedside assessment of volume status in patients who are hospitalized or in the emergency department. To determine volume status, POCUS typically assesses the inferior vena cava, internal jugular vein, and lung parenchyma, with 3 commonly used probes: high-frequency linear probes, low-frequency phased-array probes, and low-frequency curvilinear probes. High-frequency probes provide excellent resolution for superficial structures such as the internal jugular vein, and low-frequency probes provide deeper penetration needed to visualize structures such as the inferior vena cava and lung parenchyma. The phased-array probe has a more compact size to fit between ribs and combines multiple ultrasonic signals to optimize temporal resolution for cardiac imaging.

With ultrasonographic imaging, fluid-filled structures appear hypoechoic (dark), whereas air-filled or highly reflective surfaces such as pleura or bone appear hyperechoic (bright). Ultrasound waves penetrate bone and air poorly, often resulting in acoustic shadowing, which limits image acquisition.

**Lung Parenchyma |** Lung POCUS can detect pulmonary congestion and should be performed with patients in a supine or semirecumbent position (**Figure 2**A). A low-frequency phased-array probe is preferred to reduce rib shadowing and increase the penetration of ultrasound waves. Scanning with POCUS is typically performed in longitudinal orientation across multiple anterior and lateral intercostal spaces bilaterally. B-lines can be identified as vertical, hyperechoic lines that arise from the pleural line, extending to the inferior border of the ultrasound screen (with a depth of at least 12 cm; **Video 1**). Pathological B-lines are defined as the presence of 3 or more B-lines within 1 intercostal space (B-pattern) or the summation of the total number of B-lines within the anterolateral thorax (B-line count).[17,18] Lung POCUS does not directly estimate central venous pressure, but visualization of bilateral B-lines in patients with dyspnea suggests pulmonary vascular congestion, typically due to volume overload. However, diffuse B-lines (≥3 B-lines per intercostal space across multiple zones bilaterally) may also occur in conditions such as acute respiratory distress syndrome, and focal B-lines may be seen in patients with pneumonia and pulmonary contusion.

**Ultrasound JVP |** A distended internal jugular vein visible in the upright seated position suggests marked volume overload. For patients with less volume overload, POCUS examination of the internal jugular vein with the patient's trunk reclined at 30 to 45 degrees above the horizontal plane can be performed to estimate central venous pressure (Figure 2B and **Video 2**). A high-frequency linear probe is used in transverse orientation at the base of the neck, just above the clavicle, lateral to the sternocleidomastoid muscle. The internal jugular vein, located adjacent to the carotid artery, can be identified by an increase in diameter with a Valsalva maneuver or visualized with nonpulsatile color Doppler.[19-24]

Measurement of maximal and minimal internal jugular vein anteroposterior diameters during the respiratory cycle (often calculated as a percent variation) can be used, with larger diameters and reduced respiratory variation (<30%) suggestive of elevated right atrial pressure.[22] Other POCUS examination findings suggesting elevated right atrial pressure include reduced collapsibility during a sniff test, when a patient performs a sharp inhalation. Location of the internal jugular vein collapse point (most cephalad point along the vein, which collapses with respiration) can be used as a surrogate for the top of the venous column and is analogous to the meniscus identified in the bedside JVP examination.[25] The POCUS probe is then advanced cranially until the internal jugular vein collapses or becomes smaller than the adjacent carotid artery throughout the respiratory cycle, also known as the collapse point. The vertical height from the collapse point to the sternal angle is measured, and 5 cm is added to estimate the total jugular venous height. A total jugular venous height of more than 8 cm suggests elevated central venous pressure, whereas less than 8 cm suggests normal central venous pressure.

Although POCUS is commonly used to assess and estimate elevated cardiac filling pressures, ultrasonographic examination of JVP techniques described in the medical literature vary widely.[21-26] This heterogeneity has led to limited evidence about precision and accuracy, and some data suggest that ultrasonographic JVP accuracy with POCUS is training dependent.[26] In addition, obesity may make visualization difficult, but in most cases the internal jugular vein can be adequately assessed with careful technique and minimal probe pressure to avoid vein compression.

**Inferior Vena Cava |** POCUS examination of the inferior vena cava is performed with patients in the supine position (Figure 2C and **Video 3**) because upright positioning may artifactually increase the diameter of the inferior vena cava. A low-frequency phased-array or curvilinear probe is placed 1 cm to 2 cm to the right of the subxiphoid area, with the probe parallel to the head-to-toe axis and angled to face toward the head. After identifying the right atrium and the inferior vena cava, the diameter of the inferior vena cava is measured approximately 2 cm distal to the right atrium junction at the end of expiration. M-mode allows for precise measurement of the diameter with respiratory variation. A dilated inferior vena cava (>2.1 cm) with less than 50% inspiratory collapse suggests elevated central venous pressure. In contrast, a small inferior vena cava (≤2.1 cm) that collapses more than 50% with inspiration suggests normal central venous pressure. Although a diameter may vary with body habitus and may be poorly visualized in patients with obesity or excess bowel gas, the collapsibility index (percentage collapse with inspiration) is interpreted similarly across body types.[27] Additionally, inferior vena cava compliance is decreased in patients with conditions of elevated intra-abdominal pressure such as ascites and pancreatitis, and the inferior vena cava is distended in patients receiving positive pressure ventilation.[28]

## Methods

### Study Selection

The authors conducted a search of MEDLINE (1946 to January 6, 2026) to identify peer-reviewed English-language primary studies about the diagnostic accuracy of components of the clinical examination that identify patients with volume overload. Studies conducted in adults (≥18 years) with conditions that predispose to volume overload (heart failure with preserved or reduced ejection fraction, chronic kidney disease, acute kidney failure, cirrhosis, and pulmonary edema) were included. Studies of patients who were pregnant or had a diagnosis of chronic lymphedema or sepsis were excluded.

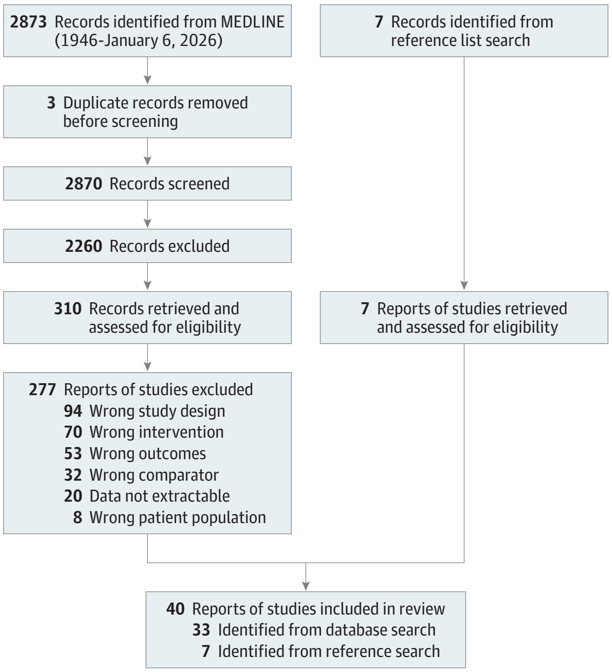
Selected studies evaluated the accuracy of a clinical sign or symptom, laboratory test, chest radiography, and/or POCUS and compared it with the reference criterion standard to assess and diagnose volume overload. Blinded, independent, multiphysician medical record review was most frequently used as the reference standard for comparison; other reference standards included right heart catheterization, central venous pressure measured by central venous catheter, and discharge diagnosis. We used the systematic review program Covidence (Veritas Health Innovation) for study screening and data extraction.

The quality of included studies was graded as described for the Rational Clinical Examination (eTable 1 in the Supplement).[29,30] The patient population, intervention or exposure, comparator, and outcome of included studies were evaluated to assess the risk of bias and applicability of the results to this review (eFigures 1 and 2 in the Supplement).

### Statistical Methods

Three authors (B.D., M.K., and A.L.) independently extracted data from the included articles into 2 × 2 tables for each clinical measure of volume overload. Sensitivity, specificity, and likelihood ratios (LRs) with 95% CIs were recalculated from extracted data for each study (eTable 2 in the Supplement). Pooled sensitivities, specificities, and LRs from included studies were estimated using the metan (multienvironment trials analysis) command, which estimates effect sizes with corresponding standard errors and CIs for binary or continuous measures using Stata version 19.5 (StataCorp). Random-effects models with log transformation of LRs (eform option) were employed. Publication bias was not formally tested due to the small number of studies available for each clinical measure of

Figure 3. PRISMA Flow Diagram for Selection of Articles




PRISMA indicates Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

volume overload (ie, <10 studies).[31] We highlighted findings with an LR of 2.0 or higher as an increasing the likelihood of volume overload or an LR of 0.5 or less for a decreasing the likelihood.

## Results

A total of 2870 articles were initially screened, of which 310 articles underwent full text review and 33 articles were included in this review. An additional 7 articles identified from citations of the articles in the original search were included, for a total of 40 included articles (**Figure 3**). Based on the Rational Clinical Examination criteria, 2 were level 1 quality, 28 were level 2 quality, and 10 were level 3 quality (eTable 1 in the Supplement). Among these 40 articles, which included 11 490 patients (**Table**), independent medical record review by a physician blinded to the result of the clinical examination was the most frequent reference standard and heart failure was the most common diagnosis.

### Prevalence of Volume Overload

The study populations consisted primarily of patients presenting to emergency departments with dyspnea (26 studies) or had been admitted to inpatient wards or intensive care units (8 studies), followed by patients presenting prior to right heart catheterization (inpatient or outpatient; 4 studies), outpatient clinic (1 study), and urgent care settings (1 study). The prevalence of volume overload was 28% to 83% in the emergency department settings, 43% to 69% in inpatient or intensive care unit settings, 35% to 43% in combined inpatient and outpatient settings, 62% in outpatient heart failure settings, and 39% in the urgent care setting.

### Clinical Examination

Ten articles involving 5041 patients described the test characteristics of jugular venous distention for predicting volume overload.[8,10,13,30,32-37] Eight studies evaluated patients in the emergency department and 2 studies in the urgent care setting. The presence of jugular venous distention was highly specific (specificity, 92%; LR, 4.1 [95% CI, 2.9-5.6]) but much less sensitive (sensitivity, 35%; LR, 0.72 [95% CI, 0.60-0.87]).

Auscultation of crackles was evaluated by 3 studies involving 2363 patients: 2 in the emergency department involving 482 patients and 1 involving 1881 patients in the outpatient setting.[13,34,38] The presence of crackles had relatively high specificity (specificity, 81%; LR, 2.7 [95% CI, 1.7-4.5]), but lower sensitivity (sensitivity, 56%; LR, 0.56 [95% CI, 0.32-1.0]).[22,27,30,33]

Nine studies involving 5335 patients evaluated the utility of lower-extremity edema for predicting volume overload.[8,13,30,33-37,39] Of these articles, 7 evaluated 3314 patients in the emergency department, 1 evaluated 140 patients in the hospital, and 1 evaluated 1881 outpatients. Lower-extremity edema had similar diagnostic accuracy compared with auscultated crackles (specificity, 80%; LR, 2.2 [95% CI, 1.5-3.1]; sensitivity, 44%; LR, 0.67 [95% CI, 0.52-0.87]). Although frequently used in clinical practice, the grading system for edema (0 to ≥4) is subjective and lacks reliability and reproducibility.[40] No studies assessed the diagnostic utility of the grade of peripheral edema, beyond the dichotomous description of present or absent.

### Radiographic Findings

Pulmonary vascular congestion on chest radiographies (eg, interstitial edema or peribronchial cuffing) was evaluated in 7 studies involving 2584 patients,[10,32,33,35,41-43] although there was no consistent definition of pulmonary vascular congestion. Four studies evaluated 1750 patients in the emergency department, 2 studies evaluated 513 patients in the hospital, and 1 study evaluated 321 patients at an urgent care facility. Pulmonary vascular congestion on chest radiography was highly specific (specificity, 91%; LR, 5.9 [95% CI, 2.9-12]) for volume overload, although it was less sensitive (sensitivity, 51%; LR, 0.53 [95% CI, 0.37-0.76]).

### POCUS Examination

Eleven studies of 1182 patients evaluated pulmonary B-lines as a predictor of pulmonary vascular volume overload, with a positive test defined as greater than 2 B-lines in at least 2 different lung zones bilaterally.[38,41,42,44-51] Among these studies, 9 evaluated 918 patients in emergency departments, 1 evaluated 130 patients in the hospital, and 1 evaluated 134 patients in the intensive care unit. The presence of pulmonary B-lines increased the likelihood of intravascular volume overload (LR, 4.0 [95% CI, 2.6-6.1]; specificity, 77%), and the absence of B-lines made intravascular volume overload substantially less likely (LR, 0.09 [95% CI, 0.04-0.23]; sensitivity, 93%).

The inferior vena cava collapsibility index was evaluated with POCUS in 4 studies with 377 patients[9,19,49,52]: 3 involving 253 emergency department patients and 1 involving 124 patients treated in both an inpatient and outpatient setting. A normal inferior vena cava collapsibility index (≥50%) was associated with decreased likelihood of volume overload (LR, 0.22 [95% CI, 0.11-0.45]; sensitivity, 82%), whereas an abnormal index (<50%) was associated with a

Table. Summary of Diagnostic Accuracy for Tests of Volume Overload[a]

| Measures of volume overload | No. of studies | Prevalence of volume overload, No. (% range) of patients | Cutoff for measures | % (95% CI) | | Likelihood ratio (95% CI) | |
|---|---|---|---|---|---|---|---|
| | | | | Sensitivity | Specificity | Positive | Negative |
| **Clinical examination** | | | | | | | |
| Jugular venous pressure or distention | 10 | 5041 (9-53) | >3 cm above sternal angle | 35 (26-44) | 92 (89-95) | 4.1 (2.9-5.6) | 0.72 (0.60-0.87) |
| Auscultation of crackles | 3 | 2363 (12-63) | Present | 56 (15-97) | 81 (60-100) | 2.7 (1.7-4.5) | 0.56 (0.32-1.0) |
| Lower extremity edema | 9 | 5335 (17-55) | Absent | 44 (33-55) | 80 (73-87) | 2.2 (1.5-3.1) | 0.67 (0.52-0.87) |
| **Chest radiography** | | | | | | | |
| Intravascular congestion | 7 | 2584 (35-62) | Various[b] | 51 (35-68) | 91 (87-95) | 5.9 (2.9-12) | 0.53 (0.37-0.76) |
| **Point-of-care ultrasonography** | | | | | | | |
| Pulmonary B-lines | 11 | 1182 (28-62) | >2 B-lines in 2 zones bilaterally | 93 (83-97) | 77 (67-85) | 4.0 (2.6-6.1) | 0.09 (0.04-0.23) |
| Inferior vena cava collapsibility index (<80%) | 4 | 377 (39-59) | <50% | 82 (65-92) | 79 (66-88) | 3.9 (2.5-6.1) | 0.22 (0.11-0.45) |
| Ultrasonographic jugular venous pressure | 6 | 592 (35-64) | >8 cm above right atrium | 81 (76-87) | 71 (65-78) | 2.8 (2.2-3.5) | 0.26 (0.20-0.33) |
| Laboratory findings | | | | | | | |
| Brain-type natriuretic peptide[c] | 6 | 3494 (42-69) | ≥100 ng/mL | 87 (78-93) | 87 (66-96) | 6.9 (2.4-20) | 0.14 (0.08-0.24) |

[a] See eTable 2 in the Supplement for results from individual studies.

[b] Chest radiography cutoffs included peribronchial cuffing, presence of interstitial edema, and pulmonary congestion.

[c] N-terminal brain-type natriuretic peptide cutoff ranged from 283-817 for single-point cutoff and 450-1800 for age-related cutoff.

higher likelihood of volume overload (LR, 3.9 [95% CI, 2.5-6.1]; specificity, 79%).

Use of POCUS to determine ultrasonographic JVP was evaluated in 6 studies of 592 patients,[19-24] including 4 studies of 472 patients in both inpatient and outpatient settings, 1 of 76 hospitalized patients, and 1 of 44 intensive care unit patients. These studies included 9 different techniques for determining JVP by ultrasonography, and none of the techniques were evaluated in more than 1 study. However, the results obtained from these techniques were similar, indicating that there are multiple acceptable methods to obtain relevant clinical data from this examination. An ultrasonographic JVP that was not elevated (<8 cm) by POCUS was useful to identify patients less likely to have volume overload (LR, 0.26 [95% CI, 0.20-0.33]; sensitivity, 81%); individuals with increased ultrasonographic JVP (>8 cm) were more likely to have volume overload (LR, 2.8 [95% CI, 2.2-3.5]; specificity, 71%).

### Laboratory Findings

Six studies involving 3494 patients examined the use of BNP to determine the presence of volume overload in patients presenting with dyspnea.[10,43,53-56] Three studies evaluated 2595 patients in the emergency department, 2 assessed 578 patients in the hospital, and 1 study evaluated 321 patients in an urgent care setting. In 5 studies, the upper limit of normal was defined as BNP levels of 100 ng/mL or higher; the sixth study used a threshold of BNP levels of 94 ng/mL or higher. Across these studies, intravascular volume overload was more likely among individuals with elevated BNP concentration (LR, 6.9 [95% CI, 2.4-20.4]; specificity, 87%) and much less likely among individuals with normal-range BNP levels (LR, 0.14 [95% CI, 0.08-0.24]; sensitivity, 87%).

Six studies of 1103 patients evaluated NT-proBNP,[47,57-61] but each used a variety of values for the upper limit of normal to define volume overload (range, 283-817 pg/mL for single-point cutoff; 450-1800 pg/mL for age-related cutoff). Lack of consistency in the threshold prevented creation of a summary threshold.

## Discussion

There are 4 commonly used methods to evaluate volume overload: physical examination (jugular venous distension, lower extremity edema, and presence of crackles on lung auscultation), POCUS (lung ultrasonographic assessment of B-lines, ultrasonographic JVP, and inferior vena cava collapsibility), biochemical markers (BNP and NT-proBNP), and chest radiographic studies. Physical examination, while associated with specificity ranging from 80% to 95%, has lower sensitivity for detecting volume overload and less discriminating LRs, suggesting that these tests alone do not reliably exclude volume overload.

With POCUS evaluation for volume overload, including pulmonary B-lines, ultrasonographic JVP, and inferior vena cava collapsibility index, B-lines showed the highest sensitivity for identifying volume overload (93%) and the lowest LR (0.09), indicating that lung ultrasonography has a high degree of reliability in excluding volume overload. Summative statistics of the accuracy of ultrasonographic JVP were unable to be performed because none of the included studies used the same POCUS technique to evaluate central venous pressure. However, compared with physical examination of JVP, it had a marked increase in sensitivity, likely due to better visualization of the internal jugular vein. Less heterogeneity was observed across studies examining the inferior vena cava collapsibility index, suggesting that this examination is more consistently performed in a standardized manner than pulmonary B-lines on POCUS or ultrasonographic JVP.

BNP has high sensitivity, which makes it useful for identifying patients at lower risk of having volume overload. Similarly, intravascular congestion on chest radiography displayed high specificity and a positive LR for identifying patients at higher risk of having volume overload. In contrast to the physical examination and POCUS, results from BNP and chest radiography are not obtained immediately, although these tests are readily available and generally reported rapidly (within an hour to several days, depending on the clinical setting).

POCUS is currently included as a core competency for training in emergency medicine, general surgery, anesthesiology, and critical care.[62] Although the Society of Hospital Medicine advocates inclusion of POCUS into routine clinical care, there is no US national standard or certification to establish competency.[63] Both physical examination and POCUS techniques require education and experience to generate accurate results.

Because there is currently more evidence supporting use of POCUS to evaluate volume overload than physical examination,[64] POCUS education could help physicians determine volume overload more accurately. Additionally, POCUS has the advantage of generating preservable images, allowing quantitative evaluation and external verification of examination findings that may be useful to track clinical status over time. Moreover, POCUS may provide more accurate information in patients with elevated body mass index because volume overload is often more difficult to assess with obesity.

## Limitations

This review has several limitations. First, most included studies used expert clinician opinion to determine if volume overload was present, which may be a less accurate reference standard than using right heart catheterization to confirm elevated right atrial pressure. Second, patient acuity varied substantially from studies in outpatient clinics to studies evaluating critically ill patients in intensive care units, and the accuracy of volume measurement techniques may differ among these patient populations. Third, exclusion criteria varied among studies. Fourth, techniques for performing physical examination maneuvers and, particularly, POCUS evaluation varied among studies. Fifth, studies including BNP data did not adjust BNP thresholds for age, obesity, or the presence of end-stage kidney disease. Sixth, no included studies evaluated the combination of physical examination, POCUS findings, laboratory, or radiographic results for volume overload.

# Scenario Resolution

The pretest probability of volume overload, based on the data from the 2 level 1–quality studies of dyspnea, was between 40% and 50%. Given the limited collapsibility of the patient's inferior vena cava (LR, 3.9) and bilateral B-lines on POCUS (LR, 4.0), the probability of volume overload was between 72% and 80%. The patient's diuresis was intensified. Repeat POCUS performed 4 days after presentation showed resolution of pulmonary B-lines (LR, 0.09), resolution of elevated upright JVP by POCUS (LR, 0.26), and decreased inferior vena cava diameter to 1.9 cm with 70% collapsibility (LR, 0.22), suggesting the probability of volume overload had decreased to between 5.6% and 21%. The patient's creatinine levels improved to 1.4 mg/dL and the patient was discharged from the hospital.

# Conclusions

A BNP level of 100 ng/mL or higher and the presence of vascular congestion on chest radiography may be the most useful tests to identify patients with volume overload. Absence of pulmonary B-lines using POCUS or BNP levels lower than 100 ng/mL may be the most useful findings to exclude volume overload.

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REFERENCES

1. Miller WL. Fluid volume overload and congestion in heart failure: time to reconsider pathophysiology and how volume is assessed. *Circ Heart Fail*. 2016;9(8):e002922. doi:10.1161/CIRCHEARTFAILURE.115.002922

2. Frank Peacock W, Soto KM. Current technique of fluid status assessment. *Congest Heart Fail*. 2010;16(suppl 1):S45-S51. doi:10.1111/j.1751-7133.2010.00166.x

3. Chen HH, Schrier RW. Pathophysiology of volume overload in acute heart failure syndromes. *Am J Med*. 2006;119(12)(suppl 1):S11-S16. doi:10.1016/j.amjmed.2006.09.012

4. Schrier RW. Water and sodium retention in edematous disorders: role of vasopressin and aldosterone. *Am J Med*. 2006;119(7)(suppl 1):S47-S53. doi:10.1016/j.amjmed.2006.05.007

5. Trayes KP, Studdiford JS, Pickle S, Tully AS. Edema: diagnosis and management. *Am Fam Physician*. 2013;88(2):102-110.

6. Vyshedskiy A, Alhashem RM, Paciej R, et al. Mechanism of inspiratory and expiratory crackles. *Chest*. 2009;135(1):156-164. doi:10.1378/chest.07-1562

7. Ii S, Wada S. Direct numerical simulation of expiratory crackles: relationship between airway closure dynamics and acoustic fluctuations. *J Biomech*. 2017;50:234-239. doi:10.1016/j.jbiomech.2016.11.033

8. Knudsen CW, Omland T, Clopton P, et al. Diagnostic value of B-type natriuretic peptide and chest radiographic findings in patients with acute dyspnea. *Am J Med*. 2004;116(6):363-368. doi:10.1016/j.amjmed.2003.10.028

9. Yamanoğlu A, Çelebi Yamanoğlu NG, Parlak İ, et al. The role of inferior vena cava diameter in the differential diagnosis of dyspneic patients; best sonographic measurement method? *Am J Emerg Med*. 2015;33(3):396-401. doi:10.1016/j.ajem.2014.12.032

10. Morrison LK, Harrison A, Krishnaswamy P, Kazanegra R, Clopton P, Maisel A. Utility of a rapid B-natriuretic peptide assay in differentiating congestive heart failure from lung disease in patients presenting with dyspnea. *J Am Coll Cardiol*. 2002;39(2):202-209. doi:10.1016/S0735-1097(01)01744-2

11. Wang CS, FitzGerald JM, Schulzer M, Mak E, Ayas NT. Does this dyspneic patient in the emergency department have congestive heart failure? *JAMA*. 2005;294(15):1944-1956. doi:10.1001/jama.294.15.1944

**12**. Kearney D, Reisinger N, Lohani S. Integrative volume status assessment. *POCUS J*. 2022;7 (Kidney):65-77. doi:10.24908/pocus.v7iKidney.15023

**13**. Damy T, Kallvikbacka-Bennett A, Zhang J, et al. Does the physical examination still have a role in patients with suspected heart failure? *Eur J Heart Fail*. 2011;13(12):1340-1348. doi:10.1093/eurjhf/hfr128

**14**. Assaad S, Kratzert WB, Shelley B, Friedman MB, Perrino A Jr. Assessment of pulmonary edema: principles and practice. *J Cardiothorac Vasc Anesth*. 2018;32(2):901-914. doi:10.1053/j.jvca.2017.08.028

**15**. Suneja M, Szot JF, LeBllond RF, Brown DD. The chest: chest wall, pulmonary, and cardiovascular systems; the breasts. In: *DeGowin's Diagnostic Examination*. 11th ed. McGraw Hill; 2020. Accessed January 25, 2025. https://accessmedicine.mhmedical.com/content.aspx?aid=1188917506

**16**. Cook DJ, Simel DL. Does this patient have abnormal central venous pressure? *JAMA*. 1996;275 (8):630-634. doi:10.1001/jama.1996.03530320054034

**17**. Volpicelli G, Elbarbary M, Blaivas M, et al; International Liaison Committee on Lung Ultrasound (ILC-LUS) for International Consensus Conference on Lung Ultrasound (ICC-LUS). International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med*. 2012;38(4):577-591. doi:10.1007/s00134-012-2513-4

**18**. Picano E, Pellikka PA. Ultrasound of extravascular lung water: a new standard for pulmonary congestion. *Eur Heart J*. 2016;37(27): 2097-2104. doi:10.1093/eurheartj/ehw164

**19**. Albaeni A, Sharma M, Ahmad M, Khalife WI. Accurate estimation of right-filling pressure using handheld ultrasound score in patients with heart failure. *Am J Med*. 2022;135(5):634-640. doi:10.1016/j.amjmed.2021.11.020

**20**. Siva B, Hunt A, Boudville N. The sensitivity and specificity of ultrasound estimation of central venous pressure using the internal jugular vein. *J Crit Care*. 2012;27(3):315.e7-315.e11. doi:10.1016/j.jcrc.2011.09.008

**21**. Vaidya GN, Ghafghazi S. Correlation of internal jugular and subclavian vein diameter variation on bedside ultrasound with invasive right heart catheterization. *Indian Heart J*. 2021;73(2):231-235. doi:10.1016/j.ihj.2021.01.024

**22**. Vaidya GN, Kolodziej A, Stoner B, et al. Bedside ultrasound of the internal jugular vein to assess fluid status and right ventricular function: The POCUS-JVD study. *Am J Emerg Med*. 2023;70:151-156. doi:10.1016/j.ajem.2023.05.042

**23**. Wang L, Harrison J, Dranow E, Aliyev N, Khor L. Accuracy of ultrasound jugular venous pressure height in predicting central venous congestion. *Ann Intern Med*. 2022;175(3):344-351. doi:10.7326/M21-2781

**24**. Simon MA, Schnatz RG, Romeo JD, Pacella JJ. Bedside ultrasound assessment of jugular venous compliance as a potential point-of-care method to predict acute decompensated heart failure 30-day readmission. *J Am Heart Assoc*. 2018;7(15):e008184. doi:10.1161/JAHA.117.008184

**25**. Xing CY, Liu YL, Zhao ML, et al. New method for noninvasive quantification of central venous pressure by ultrasound. *Circ Cardiovasc Imaging*. 2015;8(5):e003085. doi:10.1161/CIRCIMAGING.114.003085

**26**. Jang T, Aubin C, Kaji AH. The imprecision of sonographic assessment of jugular venous distension among novice operators. *J Med Ultrasound*. 2013;21(3):143-145. doi:10.1016/j.jmu.2013.07.001

**27**. Taniguchi T, Ohtani T, Nakatani S, et al. Impact of body size on inferior vena cava parameters for estimating right atrial pressure: a need for standardization? *J Am Soc Echocardiogr*. 2015;28 (12):1420-1427. doi:10.1016/j.echo.2015.07.008

**28**. Via G, Tavazzi G, Price S. Ten situations where inferior vena cava ultrasound may fail to accurately predict fluid responsiveness: a physiologically based point of view. *Intensive Care Med*. 2016;42 (7):1164-1167. doi:10.1007/s00134-016-4357-9

**29**. Simel DL, Rennie D. *The Rational Clinical Examination: Evidence-Based Clinical Diagnosis*. McGraw Hill; 2009.

**30**. Mueller T, Gegenhuber A, Poelz W, Haltmayer M. Diagnostic accuracy of B type natriuretic peptide and amino terminal proBNP in the emergency diagnosis of heart failure. *Heart*. 2005;91(5):606-612. doi:10.1136/hrt.2004.037762

**31**. Sterne JAC, Gavaghan D, Egger M. Publication and related bias in meta-analysis: power of statistical tests and prevalence in the literature. *J Clin Epidemiol*. 2000;53(11):1119-1129. doi:10.1016/S0895-4356(00)00242-0

**32**. Dao Q, Krishnaswamy P, Kazanegra R, et al. Utility of B-type natriuretic peptide in the diagnosis of congestive heart failure in an urgent-care setting. *J Am Coll Cardiol*. 2001;37(2):379-385. doi:10.1016/S0735-1097(00)01156-6

**33**. Januzzi JL Jr, Camargo CA, Anwaruddin S, et al. The N-terminal Pro-BNP investigation of dyspnea in the emergency department (PRIDE) study. *Am J Cardiol*. 2005;95(8):948-954. doi:10.1016/j.amjcard.2004.12.032

**34**. Mueller C, Frana B, Rodriguez D, Laule-Kilian K, Perruchoud AP. Emergency diagnosis of congestive heart failure: impact of signs and symptoms. *Can J Cardiol*. 2005;21(11):921-924.

**35**. Knudsen CW, Riis JS, Finsen AV, et al. Diagnostic value of a rapid test for B-type natriuretic peptide in patients presenting with acute dyspnoe: effect of age and gender. *Eur J Heart Fail*. 2004;6(1):55-62. doi:10.1016/j.ejheart.2003.10.006

**36**. Logeart D, Saudubray C, Beyne P, et al. Comparative value of Doppler echocardiography and B-type natriuretic peptide assay in the etiologic diagnosis of acute dyspnea. *J Am Coll Cardiol*. 2002; 40(10):1794-1800. doi:10.1016/S0735-1097(02)02482-8

**37**. Bayés-Genís A, Santaló-Bel M, Zapico-Muñiz E, et al. N-terminal probrain natriuretic peptide (NT-proBNP) in the emergency diagnosis and in-hospital monitoring of patients with dyspnoea and ventricular dysfunction. *Eur J Heart Fail*. 2004; 6(3):301-308. doi:10.1016/j.ejheart.2003.12.013

**38**. Özkan B, Ünlüer EE, Akyol PY, et al. Stethoscope versus point-of-care ultrasound in the differential diagnosis of dyspnea: a randomized trial. *Eur J Emerg Med*. 2015;22(6):440-443. doi:10.1097/MEJ.0000000000000258

**39**. Inui G, Tomita K, Fukuki M, et al. Clinical characteristics for distinguishing between acute cardiogenic pulmonary edema and community-acquired pneumonia in elderly patients: a prospective observational study. *Monaldi Arch Chest Dis*. 2023;94(2):1. doi:10.4081/monaldi.2023.2633

**40**. Patel H, Skok C, DeMarco A. Peripheral edema: evaluation and management in primary care. *Am Fam Physician*. 2022;106(5):557-564.

**41**. Öhman J, Harjola VP, Karjalainen P, Lassus J. Rapid cardiothoracic ultrasound protocol for diagnosis of acute heart failure in the emergency department. *Eur J Emerg Med*. 2019;26(2):112-117. doi:10.1097/MEJ.0000000000000499

**42**. Perrone T, Maggi A, Sgarlata C, et al. Lung ultrasound in internal medicine: a bedside help to increase accuracy in the diagnosis of dyspnea. *Eur J Intern Med*. 2017;46:61-65. doi:10.1016/j.ejim.2017.07.034

**43**. Plichart M, Orvoën G, Jourdain P, et al. brain natriuretic peptide usefulness in very elderly dyspnoeic patients: the BED study. *Eur J Heart Fail*. 2017;19(4):540-548. doi:10.1002/ejhf.699

**44**. Prosen G, Klemen P, Štrnad M, Grmec S. Combination of lung ultrasound (a comet-tail sign) and N-terminal pro-brain natriuretic peptide in differentiating acute heart failure from chronic obstructive pulmonary disease and asthma as cause of acute dyspnea in prehospital emergency setting. *Crit Care*. 2011;15(2):R114. doi:10.1186/cc10140

**45**. Cibinel GA, Casoli G, Elia F, et al. Diagnostic accuracy and reproducibility of pleural and lung ultrasound in discriminating cardiogenic causes of acute dyspnea in the emergency department. *Intern Emerg Med*. 2012;7(1):65-70. doi:10.1007/s11739-011-0709-1

**46**. Russell FM, Ehrman RR, Cosby K, et al. Diagnosing acute heart failure in patients with undifferentiated dyspnea: a lung and cardiac ultrasound (LuCUS) protocol. *Acad Emerg Med*. 2015;22(2):182-191. doi:10.1111/acem.12570

**47**. Liteplo AS, Marill KA, Villen T, et al. Emergency thoracic ultrasound in the differentiation of the etiology of shortness of breath (ETUDES): sonographic B-lines and N-terminal pro-brain-type natriuretic peptide in diagnosing congestive heart failure. *Acad Emerg Med*. 2009;16(3):201-210. doi:10.1111/j.1553-2712.2008.00347.x

**48**. Anderson KL, Jenq KY, Fields JM, Panebianco NL, Dean AJ. Diagnosing heart failure among acutely dyspneic patients with cardiac, inferior vena cava, and lung ultrasonography. *Am J Emerg Med*. 2013;31(8):1208-1214. doi:10.1016/j.ajem.2013.05.007

**49**. Kajimoto K, Madeen K, Nakayama T, Tsudo H, Kuroda T, Abe T. Rapid evaluation by lung-cardiac-inferior vena cava (LCI) integrated ultrasound for differentiating heart failure from pulmonary disease as the cause of acute dyspnea in the emergency setting. *Cardiovasc Ultrasound*. 2012;10(1):49. doi:10.1186/1476-7120-10-49

**50**. Sekiguchi H, Schenck LA, Horie R, et al. Critical care ultrasonography differentiates ARDS, pulmonary edema, and other causes in the early course of acute hypoxemic respiratory failure. *Chest*. 2015;148(4):912-918. doi:10.1378/chest.15-0341

**51**. Dehbozorgi A, Eslami Nejad S, Mousavi-Roknabadi RS, Sharifi M, Tafakori A, Jalli R. Lung and cardiac ultrasound (LuCUS) protocol in diagnosing acute heart failure in patients with acute dyspnea. *Am J Emerg Med*. 2019;37(11):2055-2060. doi:10.1016/j.ajem.2019.02.040

**52**. Miller JB, Sen A, Strote SR, et al. Inferior vena cava assessment in the bedside diagnosis of acute heart failure. *Am J Emerg Med*. 2012;30(5):778-783. doi:10.1016/j.ajem.2011.04.008

**53**. Nakata K, Komukai K, Yoshii Y, et al. The optimal cut-off value of plasma BNP to differentiate heart failure in the emergency department in Japanese patients with dyspnea. *Intern Med*. 2015; 54(23):2975-2980. doi:10.2169/internalmedicine.54.4786

**54**. Zhao SQ, Hu YM, Li Q, et al. The clinical value of rapid assay for plasma B-type natriuretic peptide in differentiating congestive heart failure from pulmonary causes of dyspnoea. *Int J Clin Pract*. 2008;62(2):214-220. doi:10.1111/j.1742-1241.2007.01591.x

**55**. Maisel AS, Krishnaswamy P, Nowak RM, et al; Breathing Not Properly Multinational Study Investigators. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *N Engl J Med*. 2002;347(3):161-167. doi:10.1056/NEJMoa020233

**56**. Kevin Rogers R, Stehlik J, Stoddard GJ, et al. Adjusting for clinical covariates improves the ability of B-type natriuretic peptide to distinguish cardiac from non-cardiac dyspnoea: a sub-study of HEARD-IT. *Eur J Heart Fail*. 2009;11(11):1043-1049. doi:10.1093/eurjhf/hfp127

**57**. Sanz MP, Borque L, Rus A, Vicente B, Ramírez Y, Lasa L. Comparison of BNP and NT-proBNP assays in the approach to the emergency diagnosis of acute dyspnea. *J Clin Lab Anal*. 2006;20(6):227-232. doi:10.1002/jcla.20146

**58**. Su Q, Liu H, Zhang X, et al. Diagnostic values of NT-proBNP in acute dyspnea among elderly patients. *Int J Clin Exp Pathol*. 2015;8(10):13471-13476.

**59**. Li G, Daniels CE, Kojicic M, et al. The accuracy of natriuretic peptides (brain natriuretic peptide and N-terminal pro-brain natriuretic) in the differentiation between transfusion-related acute lung injury and transfusion-related circulatory overload in the critically ill. *Transfusion*. 2009;49(1):13-20. doi:10.1111/j.1537-2995.2008.01941.x

**60**. Steinhart B, Thorpe KE, Bayoumi AM, Moe G, Januzzi JL Jr, Mazer CD. Improving the diagnosis of acute heart failure using a validated prediction model. *J Am Coll Cardiol*. 2009;54(16):1515-1521. doi:10.1016/j.jacc.2009.05.065

**61**. Robaei D, Koe L, Bais R, Gould I, Stewart T, Tofler GH. Effect of NT-proBNP testing on diagnostic certainty in patients admitted to the emergency department with possible heart failure. *Ann Clin Biochem*. 2011;48(Pt 3):212-217. doi:10.1258/acb.2010.010137

**62**. Díaz-Gómez JL, Mayo PH, Koenig SJ. Point-of-care ultrasonography. *N Engl J Med*. 2021; 385(17):1593-1602. doi:10.1056/NEJMra1916062

**63**. Soni NJ, Schnobrich D, Mathews BK, et al. Point-of-care ultrasound for hospitalists: a position statement of the Society of Hospital Medicine. *J Hosp Med*. 2019;14:E1-E6. doi:10.12788/jhm.3079

**64**. Maw AM, Huebschmann AG, Mould-Millman NK, Dempsey AF, Soni NJ. Point-of-care ultrasound and modernization of the bedside assessment. *J Grad Med Educ*. 2020;12(6):661-665. doi:10.4300/JGME-D-20-00216.1