

Resuscitative Cardiopulmonary Ultrasound and Transesophageal Echocardiography in the Emergency Department

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KEYWORDS

- Ultrasound • Echocardiography • Point-of-care ultrasound • Resuscitation • Shock
- Critical care • Transesophageal echocardiography

KEY POINTS

- The use of point-of-care ultrasound is the standard of care in the evaluation of patients with shock, hypotension, or acute hemodynamic decompensation in the emergency department.
- The scope of focused cardiopulmonary ultrasound has expanded to include some advanced techniques that tailor therapeutic interventions to a patient's underlying physiology.
- Qualitative and quantitative evaluation of left and right ventricular function, identification of pericardial effusion and tamponade, evaluation of preload and fluid responsiveness, and hemodynamic monitoring are key ultrasound applications during the resuscitation phase of critical care.

INTRODUCTION

The scope of focused cardiopulmonary ultrasound (FOCUS), resuscitative ultrasound, and transesophageal echocardiography (TEE) has evolved rapidly over the past 2 decades in critical care and emergency environments. The concept of “resuscitative ultrasound” is used to describe specific applications of FOCUS for diagnostic assessment, monitoring, therapy titration, and procedural guidance in critically ill patients. There are several distinctive qualities of resuscitative ultrasound, compared with comprehensive ultrasound or echocardiography:

1. Aims to answer specific clinical questions
2. Can be performed multiple times during the patient's clinical course

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3. Is a scalable diagnostic tool that can be tailored to fit both the patient's complexity (ie, number or sophistication of questions asked) and the experience of the clinician.

The increase of emergency ultrasound (EUS) and emergency medicine–critical care trained physicians has contributed to the expansion of EUS, beyond the traditional core applications.¹ This review provides a description of the fundamentals and main clinical applications of resuscitative ultrasound that can be helpful in the emergency department (ED) setting.

RESUSCITATIVE CARDIOPULMONARY ULTRASOUND

Resuscitative cardiopulmonary ultrasound can allow the emergency physician (EP) to characterize the predominant physiology of a patient with shock, establish the cause of acute hypoxemia, and assess the effect of therapeutic interventions, such as administration of vasoactive drugs or intravenous fluids.

Although a core set of 5 parasternal and subcostal views is often described in FOCUS, with some additional knowledge, practice, and views, the care provider can improve his or her clinical decision making. A description of the probe location, visualized anatomy, and clinical applications of 9 resuscitative cardiac ultrasound views is provided in [Table 1](#).

Evaluation of Left Ventricular Systolic Function

The evaluation of left ventricular (LV) systolic function and ejection fraction (LVEF), is the most common echocardiographic evaluation performed in the ED. Differentiating between a normal and depressed LVEF has a number of clinical implications, including the risk of cardiogenic shock, cardiac versus pulmonary cause of dyspnea, and the need for resuscitative fluids or vasoactive drugs. Quantitative methods, traditionally used in comprehensive echocardiography,² are technically challenging, time-consuming, and not suitable for use at the point of care.

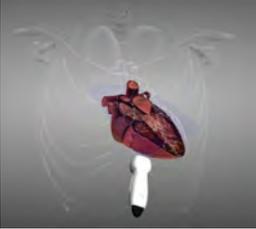
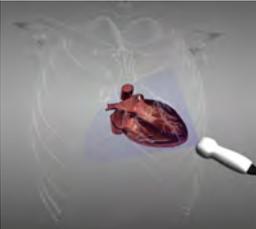
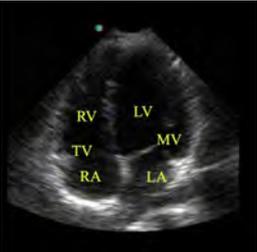
Simplified qualitative and semiquantitative methods to assess LV systolic function have been described and validated in the ED setting. These include visual estimation of LVEF³ and E-point septal separation (EPSS).⁴ Both of these methods have good correlation with LVEF using volumetric methods. When used by ED physicians in real time, these methods have shown to accurately categorize patients among normal, reduced, and severely reduced LVEF.^{3,5–7}

The visual assessment of LV systolic function can be performed using 3 elements: (1) systolic excursion of the endocardium toward the center of the LV (*endocardial excursion*), (2) systolic thickening of the myocardium (*myocardial thickening*), and (3) excursion of the anterior leaflet of the mitral valve (MV) toward the septum during early diastole (*EPSS*).

The first 2 elements are the foundation of the assessment of ventricular contractility. The third, EPSS, is a semiquantitative parameter that represents the closest distance between the anterior leaflet of the MV and the septum obtained by M-mode. Multiple cutoff values have been used to accurately identify patients with reduced LVEF.^{4,8,9} An EPSS >7 mm has been found to have 100% sensitivity for identifying patients with an LVEF less than 30%.⁴ The accuracy of visual estimation of LVEF appears to be more dependent on experience compared with EPSS ([Fig. 1](#)).

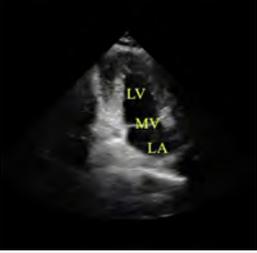
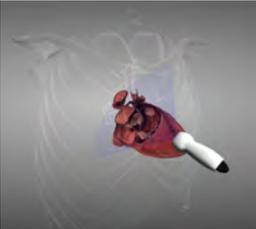
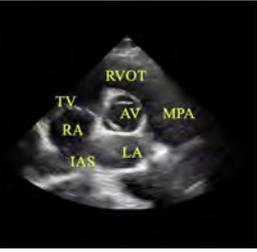
Clinicians must be aware of the limitations when estimating LV systolic function. Mitral stenosis and aortic regurgitation will both affect the distance between the MV and septum during diastole.^{8,10} If present, EPSS should not be used to estimate LVEF. LVEF estimation also may not be useful in patients with LV hypertrophy,

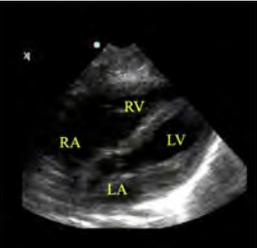
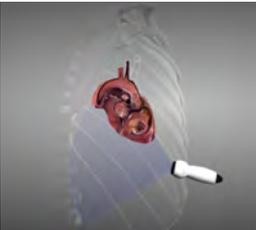
Table 1
Description of the probe location, visualized anatomy and clinical applications of 9 resuscitative cardiac ultrasound views

View	Probe Position	Ultrasound Anatomy	Clinical Applications
Parasternal long axis (PLAX)			LV systolic function E-point septal separation RV dilation Chamber dimensions Pericardial effusion
Parasternal short axis (PSAX)			LV systolic function RV dilation Septal kinetics Wall motion abnormalities Pericardial effusion
Apical 4 chamber (A4C)			LV systolic function Chamber dimensions RV function (TAPSE) Pericardial effusion Wall motion abnormalities MV/TV pathology Left atrial pressures

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Table 1
(continued)

View	Probe Position	Ultrasound Anatomy	Clinical Applications
Apical 5 chamber (A5C)			Quantitative LV function (LVOT-VTI)
Apical 2 chamber (A2C)			LV systolic function Wall motion abnormalities (inferior/anterior) MV pathology Mitral inflow velocities Left atrial pressures
Parasternal short of aortic valve (AV SAX)			Chamber dimensions Clot in transit IAS defects AV pathology Procedural guidance

Suprasternal notch of the aorta (SSNA)			Type A Aortic dissection
Subxiphoid (SSX)			LV systolic function Chamber dimensions Pericardial effusion
Inferior vena cava (IVC)			IVC diameter/respirophasic changes

Abbreviations: IAS, Inter atrial septum; LV, left ventricular; LVOT, left ventricular outflow tract; MV, mitral valve; RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion; TV, tricuspid valve; VTI, velocity-time integral.

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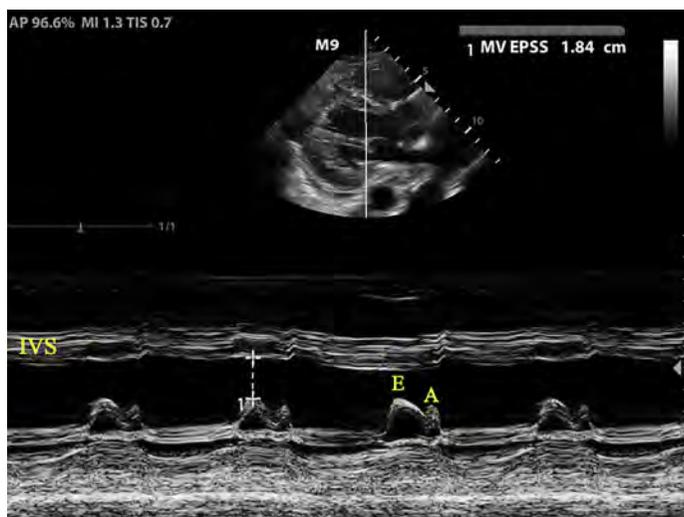


Fig. 1. Evaluation of LV systolic function with EPSS. Parasternal long view with M-mode through the anterior leaflet of the MV. In this example, EPSS is 1.8 cm, which suggests severe systolic dysfunction. IVS, intraventricular septum; E, early diastole; A, late diastole.

hypovolemia, and right ventricular (RV) dysfunction. Stroke volume (SV) assessment is likely more useful in these patients.

Qualitative Evaluation of Right Ventricular Function

The importance of RV function has been increasingly recognized over the past decade. Identification of acute RV failure is essential during a resuscitation. Identifying acute RV dysfunction at the point of care can significantly alter patient management, and is an independent predictor of mortality in patients with acute pulmonary embolism (PE), acute respiratory distress syndrome (ARDS), and acute myocardial infarction.^{11–13} A summary of echocardiographic findings of RV dysfunction is listed in [Table 2](#).

Table 2 Summary of basic and advanced applications of resuscitative cardiopulmonary ultrasound	
Basic Resuscitative Cardiopulmonary Ultrasound	Advanced Resuscitative Cardiopulmonary Ultrasound
Qualitative LV function	Quantitative LV function: SV and CO estimation
Qualitative RV function	Quantitative RV function
Identification of pericardial effusion	Evaluation of tamponade physiology
Evaluation of IVC diameter/respirophasic changes	Resuscitative TEE
Identification of pneumothorax	Evaluation fluid tolerance (IVC, SVC, LA pressures)
Identification of pulmonary edema	Hemodynamic monitoring
Identification of pleural effusion	

Abbreviations: CO, cardiac output; IVC, inferior vena cava; LA, left atrial; LV, left ventricular; RV, right ventricular; SV, stroke volume; SVC, superior vena cava; TEE, transesophageal echocardiography; US, ultrasound.

RV function can be evaluated with qualitative or quantitative assessments. RV dilation and septal kinetics are the main qualitative screening tools that can be easily and reliably performed by ED physicians.^{14–16} Weekes and colleagues¹⁴ prospectively demonstrated 100% sensitivity and 99% specificity for identifying RV dysfunction in normotensive patients with PE, and that FOCUS had better diagnostic accuracy identifying RV dysfunction than troponin or brain natriuretic peptide alone.

Qualitative assessment of the RV relies on understudying its key anatomic features. The RV has a characteristic crescent shape, normally is approximately two-thirds the size of the LV, and has a thinner myocardial free wall. Dilation of the RV with an RV/LV ratio greater than 0.6 and deviation of the septum toward the LV (leading the LV to adopt a D-shape) indicates right ventricular dysfunction.² An RV free wall thickness <5 mm, can further help distinguish acute pathology, as patients with chronically elevated RV pressures develop RV hypertrophy.

Regional hypokinesia or akinesia of the RV free wall with preserved function at the apex, known as the McConnell sign, has a high specificity but low sensitivity for acute PE or acute RV infarct.^{17,18} The McConnell sign also may be found in patients with acute chest syndrome.¹⁹ **Table 3** summarizes the main echocardiographic findings of RV dysfunction.

The main challenge of qualitative RV is obtaining adequate in-plane images that accurately reflect the RV size (the RV is normally located under the sternum). Clinicians should be careful with underestimating RV size, particularly in the apical 4 and subxiphoid chamber views.

Evaluation of Pericardial Effusion

Identification of pericardial effusion is one of the original elements of the FOCUS examination,¹ and several studies have shown that ED physicians can accurately and reliably detect pericardial effusions compared with comprehensive echocardiography.²⁰

The pericardial space is normally minimal, with only 10 mL of fluid that normally does not separate the pericardium from the myocardial wall. As the volume increases, pericardial effusion can be seen as an anechoic (black) stripe posterior to the LV in parasternal views, whereas in the subxiphoid view it can be seen between the pericardium and RV free wall (**Fig. 2**). The amount of fluid should be measured during diastole, and can be classified as a small, moderate, or large effusion.²¹

Clinicians must be familiar with pericardial fluid mimics. Epicardial fat pads can mimic a small anterior effusion. Small, nonoculated effusions will follow gravity and will accumulate posteriorly. Larger, simple effusions will eventually become circumferential. Epicardial fat is often hypoechoic (shade of gray) as opposed to simple

Table 3
Summary of some of the main echocardiographic findings seeing in presence of RV dysfunction

Echocardiography Findings of RV Dysfunction	
RV dilation	RV/LV diameter ratio >0.6
Septal flattening	D-shape of LV due to increased RV pressure
Moderate/Severe TR	Dilation of tricuspid annulus due to increased RV volume
McConnell sign	Akinesia of the RV free wall with preservation of the apex
TAPSE <17 mm	Longitudinal excursion of the lateral tricuspid annulus

Abbreviations: LV, left ventricle; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.

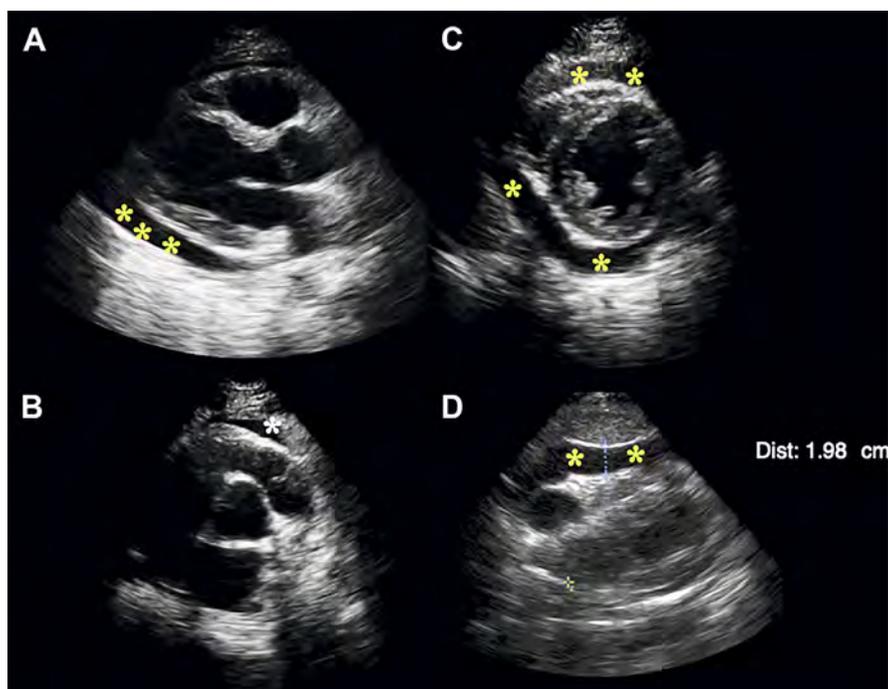


Fig. 2. Visualization of pericardial effusion (*asterisk*) in different views. (A) Parasternal long view. (B) Parasternal short aortic valve. (C) Parasternal short midpapillary. (D) Subxiphoid view with a measured pericardial effusion of 1.98 cm (moderate) between the pericardium and RV free wall.

effusions that are anechoic (black). Pleural effusions can mimic pericardial effusions, but track posterior to the descending thoracic aorta (DTA), whereas pericardial effusion will be anterior to the DTA (**Fig. 3**).

Evaluation Preload Using the Inferior Vena Cava

The evaluation of the inferior vena cava (IVC) represents a noninvasive parameter of preload with several clinically relevant applications. It has been widely used in

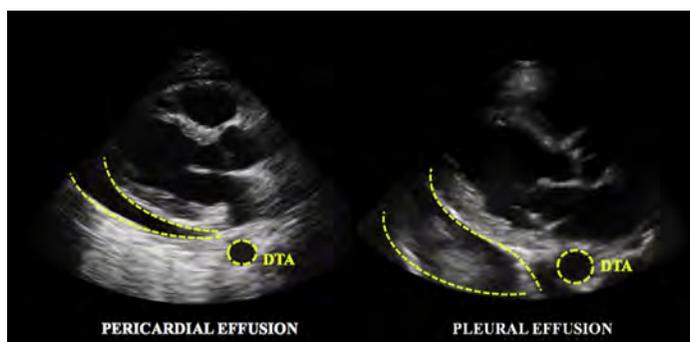


Fig. 3. Echocardiographic appearance of pericardial effusion versus pleural effusion in parasternal long axis view. Pericardial effusion follows a plane that is anterior to the DTA, whereas pleural effusion is posterior.

emergency medicine and critical care point-of-care ultrasonography because it is often easy to visualize, but has significant limitations.

IVC diameter and variation provide a noninvasive estimate of central venous and right atrial pressures. The IVC is best visualized through the subcostal window in short or long axis. In long axis view, the IVC is visualized entering the right atrium (RA). Using M-mode, the maximum and minimum diameters are measured and the percentage of collapse estimated. Diameter measurements should be made just distal to the hepatic vein, approximately 2 to 3 cm distal to the RA (Fig. 4).

The use of IVC variation to predict volume responsiveness has been extensively studied in both spontaneously breathing (SB) and mechanically ventilated patients.^{22–24} Despite widespread use of IVC ultrasound to guide volume resuscitation, the heterogeneity of populations and measurement techniques has made the interpretation of the evidence challenging.

The use of IVC diameter and collapsibility to predict volume responsiveness has many limitations Table 4 Via and colleagues²⁵ provided a comprehensive review of the multiple physiologic mechanisms that affect the reliability of using IVC a measure of preload.

Recent systematic reviews and meta-analyses, including studies conducted in the intensive care unit (ICU) and ED over the past 2 decades, suggest that IVC diameter evaluation as a predictor of fluid responsiveness is most reliable in the following^{23,24}:

1. Non-SB patients, receiving mechanical ventilation
2. Prescribed tidal volume (TV) ≥ 8 mL/kg and positive end-expiratory pressure (PEEP) ≤ 5 cm H₂O
3. In sinus rhythm, without evidence of RV dysfunction

In SB patients, IVC should be used with caution and in conjunction with other parameters, such as LV systolic function and lung ultrasound profile. Recently, Corl and colleagues²⁶ conducted the largest ICU study assessing the predictive value of IVC in SB patients and found that an IVC collapsibility cutoff $\geq 25\%$ identified fluid responsive patients with sensitivity and specificity of 87% and 81%, respectively, with a positive likelihood ratio of 4.5. As in prior studies, the lack of respiratory effort standardization during IVC measurements likely explains the misclassification of patients as fluid responsive (16%), and limits the generalizability of these findings.

For the EP, the clinical scenario in which IVC evaluation can be most helpful is in managing the hypotensive patient whose IVC is plethoric, without tamponade, and

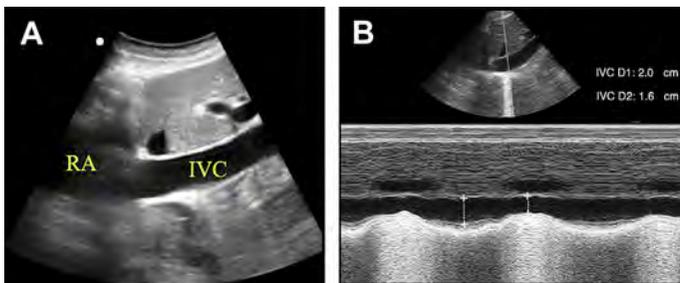


Fig. 4. (A) Long axis view of the IVC. (B) M-mode image of the IVC depicting maximum (IVC D1) and minimum (IVC D2) diameters in an SB patient. Inspiration lowers the intrathoracic pressure, which augments the venous return. This method allows determination of the IVC collapsibility index.

Table 4
Conditions affecting the reliability of IVC assessment as a predictor of fluid responsiveness in spontaneously breathing (SB) and mechanically ventilated (MV) patients

False Positives	False Negatives	Type of Ventilation
Significant inspiratory effort leading to exaggerated intrathoracic pressures	Weak inspiratory effort leading to small intrathoracic pressures	SB
Patients with COPD/asthma: forced expiration (ie, "abdominal breathing") leading to expiratory collapse of IVC	Patients with COPD/asthma: lung hyperinflation and auto-PEEP leading to reduced venous return	SB
Off-plane imagine during longitudinal measurement in M-mode (false collapse of IVC)	Chronic RV dysfunction: increased RA pressures and chronic dilation of IVC	SB or MV
Extrinsic compression of the IVC by masses	Increased abdominal pressure: IVC collapsibility reduced by external pressure over IVC	SB or MV
	High levels of PEEP: increased RA pressures with reduced venous return	MV
	Small tidal volume (<8 mL/kg): smaller variations in IVC in response to lung-heart interactions	MV

Abbreviations: COPD, chronic obstructive pulmonary disease; IVC, inferior vena cava; PEEP, positive end-expiratory pressure; RA, right atrium; RV, right ventricle.

Adapted from 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):1164–7. <https://doi.org/10.1007/s00134-016-4357-9>; with permission.

minimal respiratory collapse. In this case, the patient is unlikely to benefit from intravenous fluids and vasoactive therapy should be considered instead. In light of the available evidence, additional methods to establish the patient's preload and fluid tolerance should be used.²⁷

Focused Lung Ultrasound

Combined with FOCUS, lung ultrasound (LUS) can provide critical diagnostic information to narrow the differential diagnosis and guide management in critically ill patients with respiratory distress.^{28,29} Lung ultrasound can identify pulmonary edema, pleural effusion, and pneumothorax with greater diagnostic accuracy compared with clinical examination and chest radiography.^{30–32} Lung ultrasound relies on the presence or absence of specific ultrasound artifacts. An in-depth review of these artifacts (and the LUS technique) is beyond the scope of this article, but is well described in Dietrich and colleagues³³ and Bianco and colleagues.³⁴

The first step of focused LUS is the evaluation of pleural sliding by visualizing the pleural line moving during respirations with a high-frequency (ie, linear) probe in the anterior chest. The absence of lung sliding suggests the possibility of pneumothorax, but is not the only possible diagnosis. M-mode in the same location can distinguish between a normal pattern or nonventilated lung parenchyma known as the stratosphere sign (Fig. 5). The diagnosis of pneumothorax can be made with reported specificity of 100% if there is no lung sliding, no stratosphere sign, or the presence of a lung point.³⁵

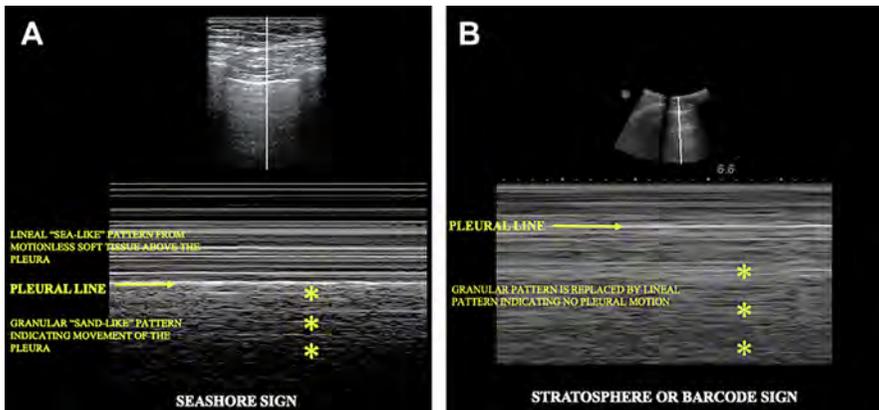


Fig. 5. Evaluation pleural sliding for the assessment of pneumothorax using LUS. (A) The interface between the lineal “sealike” pattern of the motionless tissues above the pleural line, with the granular “sandlike” pattern of the subpleural space indicating normal sliding forms the “seashore sign.” (asterisk) (B) A continuous, smooth pattern is seen in presence of lung that is not being ventilated. This is known as the “stratosphere sign” (asterisk) and is suggestive of pneumothorax.

The second part of focused LUS is the evaluation of lung parenchyma using a low-frequency (ie, curvilinear or phased array) probe set at a depth of 18 cm. A 6-window protocol (3 in each hemithorax) can provide similar diagnostic accuracy to a traditional 8-view examination.^{30,36} Patients can be classified into 4 main ultrasonographic lung patterns, each suggesting different diagnostic entities: A-line pattern, B-line pattern, effusion, or consolidation.

A-lines are horizontal linear artifacts, representing reverberation (mirror effect) of the pleural line, with normal lung. The presence of A-lines in a patient with dyspnea should orient to noninterstitial pathology (ie, consider chronic obstructive pulmonary disease [COPD], PE, or acute coronary syndrome).

B-lines are vertical lines resulting from increased interstitial tissue density. The presence of more than 3 B-lines per lung zone suggests the presence interstitial fluid, which can suggest pulmonary edema (diffuse and bilateral), ARDS (bilateral with areas of sparing), or focal interstitial syndrome (unilateral, often with patchy appearance) such as pneumonia (Fig. 6).^{37,38}

ADVANCED FOCUSED CARDIAC ULTRASOUND TECHNIQUES

Although the basic applications of FOCUS are sufficient in most cases, the management of complex critically ill patients often requires additional information to understand the patient’s cardiovascular physiology and hemodynamics.

Examples of these scenarios include cases in which LVEF does not accurately represent the SV, when quantitative assessment of the cardiac output is needed to titrate vasoactive drugs, suspected acute RV failure, and in the evaluation of tamponade. By understanding additional principles, clinicians can extend their scope of resuscitative ultrasound and better guide management in these clinical situations.

Principles of Doppler

Doppler allows a measurement of frequency shift in moving elements, which in the case of echocardiography enables the detection of blood flow velocities. Pulse

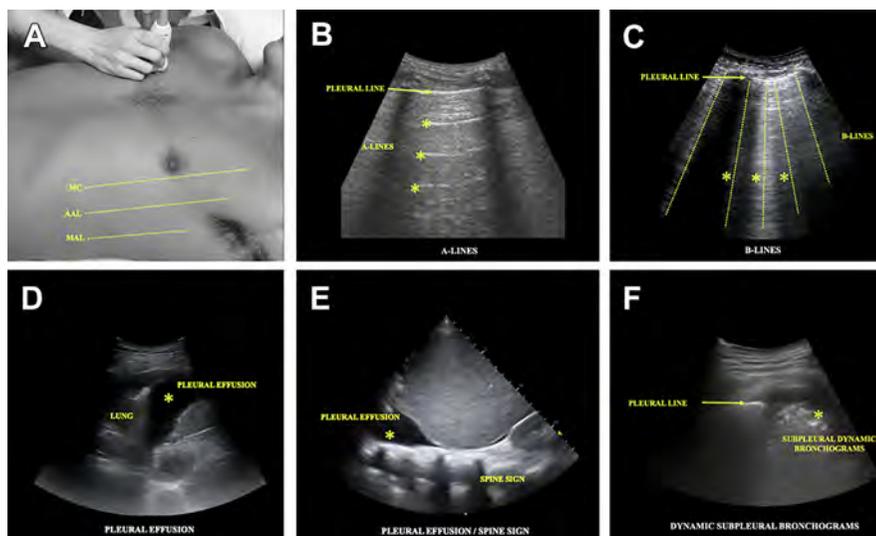


Fig. 6. Main LUS findings. (A) Six zone scanning protocol for LUS. The left and right sides of chest wall are divided following 3 lines: mid-clavicular (MC), anterior axillary line (AAL) and mid-axillary line (MAL). (B) A-line pattern, indicating normal parenchyma (*asterisk*). (C) B-line pattern (*asterisk*) indicating increased extravascular lung water and interstitial lung syndrome. (D) Left-sided pleural effusion (*asterisk*). (E) Right-sided pleural effusion (*asterisk*). The thoracic spine vertebral bodies are seen due to enhanced transmission of ultrasound by pleural effusion.⁵⁹ (F) Subpleural bronchograms (*asterisk*) indicating lung consolidation from pneumonia. ([A] Adapted from [A] Pivetta E, Goffi A, Lupia E, et al. Lung ultrasound-implemented diagnosis of acute decompensated heart failure in the ED: A SIMEU multicenter study. *Chest* 2015;148(1):202–10; with permission.)

Wave (PW) Doppler uses pulsed wave ultrasound signals to determine the depth of a measurement. Continuous Wave Doppler uses continuous signals that are simultaneously emitted and received, yielding measurement of velocities throughout the entire ultrasound beam.

Doppler velocity is reliable only if the ultrasound beam is less than 15° from the parallel position of blood flow. As this angle increases, velocity will be progressively underestimated, reaching no detected signal (no flow) when the beam is perpendicular to flow. This concept has important clinical implications, as obtaining acceptable alignment between ultrasound signal and blood flow is often difficult and represents an important source of error.

Quantitative Evaluation of Systolic Left Ventricular Function

EPs can incorporate PW Doppler to perform quantitative assessment of LV function, including estimation of SV and cardiac output (CO). SV is estimated by calculating the product of the area patient's LV outflow tract velocity-time integral (LVOT-VTI) and aortic valve area (AVA).

From the parasternal long axis (PSL) view, the LVOT diameter is measured during early systole from intima to intima to calculate the AVA. To measure the LVOT-VTI, the PW Doppler gate should be placed proximal to the aortic valve leaflets in an apical 5-chamber window. The image is then captured to trace the contour of the Doppler velocity profile to obtain the VTI (**Fig. 7**). This measurement should be averaged over 3 consecutive waveforms to account for beat-to-beat SV variation due to respiration.

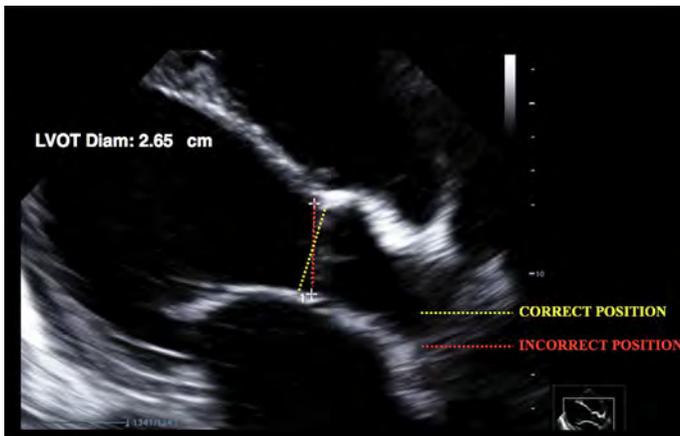


Fig. 7. Measurement of the LVOT diameter for estimation of SV. In parasternal long view, the LVOT should be measured placing caliper perpendicular to the direction of flow (yellow dotted line). Note that measurement is being made with image zoomed to increase accuracy.

There are a few important sources of error the clinician should be aware of when estimating SV using VTI. First, because the LVOT diameter is squared in the AVA calculation, small errors in the measurement will lead to significant error in the final product. Zooming in to the LVOT allows for the most accurate measurement possible (Fig. 8). The caliper

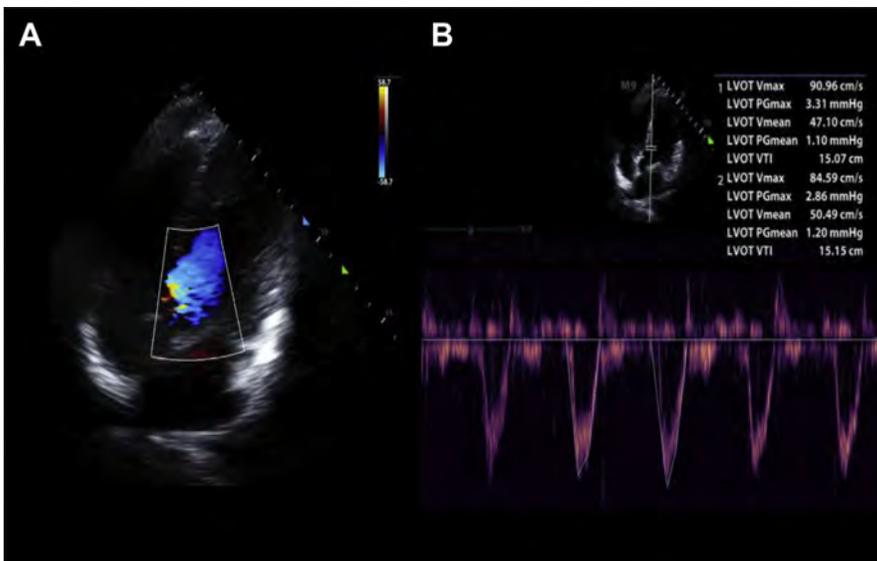


Fig. 8. Measurement of the VTI using PW Doppler for SV estimation. (A) After developing a 5-chamber view, CF Doppler is used to visualize the center of laminar flow into the LVOT. Blue denotes flow away from the probe. (B) The sample volume of PW Doppler is positioned at the LVOT aiming for an angle of insonation of less than 15 degrees to avoid underestimation of velocity. At least two or three envelopes are traced to improve accuracy, and the average calculated VTI is used to estimate the SV. Once the LVOT diameter and VTI are known with these methods, the SV can be estimated using the formula: $SV = \pi [LVOT\ diameter/2]^2 \times [LVOT-VTI]$. CF, color flow.

should be positioned perpendicular to the direction of blood flow and not following a vertical line. Second, adequate positioning of the PW sample volume, approximately 5 mm proximal to the aortic valve, is key for reliable measurements. If the sample volume is too close to the valve, velocities will be overestimated due to higher flow through the valve, while measuring too far from the valve will underestimate the velocities.

Quantitative Evaluation of Right Ventricular Function

Unlike assessment of the LV, the qualitative evaluation of RV systolic function is much less reliable.³⁹ Chamber dimensions and septal deviation may be sufficient to make the diagnosis of RV dysfunction in many cases, but a quantitative approach can improve the accuracy and reliability of the RV function. There are several quantitative methods used to assess RV function, including fractional area of change, myocardial performance index, tricuspid annular velocity, and tricuspid annular plane systolic excursion (TAPSE). Although there is no universal agreement, TAPSE has been the most studied parameter in ED and ICU settings.⁴⁰

TAPSE can be measured using M-mode in an apical 4-chamber view to measure the maximum longitudinal excursion of the lateral tricuspid annulus, between the end of systole and the end of diastole. A TAPSE ≥ 17 mm has been shown to have good correlation with normal RV function^{2,41,42} Abnormal TAPSE has been established as a marker of poor clinical outcome in several pathologies, including ARDS, PE, and septic cardiomyopathy.⁴⁰

One important ED application is the risk stratification of patients with suspected or diagnosed PE. A recent prospective study of TAPSE in patients with suspected PE found that a TAPSE less than 15.2 mm was able to identify ED patients with clinically significant acute PE (specificity of 100% but a low sensitivity of 53%).⁴³ A similar study showed that a TAPSE ≤ 17 mm had 90% sensitivity for centrally located PE and that when combined with lower extremity ultrasound for deep vein thrombosis, sensitivity was 100%.⁴⁴

Although quick and relatively easy to perform, TAPSE has some limitations due to its single dimensional measurement. First, it measures only longitudinal excursion, which is one component of RV function. Some patients with frank RV dysfunction, due to pulmonary hypertension, for example, may have a preserved TAPSE. Second, it is angle dependent. If the beam is not parallel to the RV free wall, the longitudinal excursion will be underestimated.

Evaluation of Pericardial Tamponade Physiology

EPs can assess for echocardiographic signs of tamponade physiology in the patient with a pericardial effusion. The hallmark of pericardial tamponade is impaired diastolic filling of right-sided chambers, leading to a decrease of LV SV and hemodynamic compromise.²¹

The first step is evaluation of IVC diameter and respiratory variation. Due to the increased intrapericardial pressures during tamponade, the RA cannot receive venous return, causing distension of the IVC throughout the respiratory cycle. A plethoric IVC, with no or minimal respiratory variation, is highly sensitive for tamponade physiology (>95%). In the absence of this finding, the presence of tamponade physiology is extremely unlikely.^{21,45}

The second step is evaluation of RV diastolic collapse. When intrapericardial pressure exceeds the intracardiac filling pressure, the RV free wall collapses. This can be measured using M-mode in PSL view, aligning the beam through the RV free wall and the anterior leaflet of the MV. Diastolic collapse of the RV has a specificity between 75% and 90% and a relatively low sensitivity of 48% to 60%.²¹

Last, using PW Doppler in A4C view, pathologic inflow velocity variation through the mitral or tricuspid valves can be evaluated. Respiratory variation of the

peak inflow velocities greater than 30% at the MV and greater than 60% at the TV suggest increased ventricular interdependence and is highly specific for tamponade.^{21,46}

Integrating Resuscitative Ultrasound for Hemodynamic Monitoring

Hemodynamic monitoring has an important role in the management of critically ill patients guiding therapeutic decisions and providing endpoints of resuscitation. There has been an increasing use of ultrasound for hemodynamic monitoring in recent years, likely because it meets many of the properties of an “ideal” hemodynamic monitor.^{47,48}

A number of echocardiographic parameters can be used to tailor interventions to meet an individual patient’s physiologic needs. For instance, serial VTI measurements with transthoracic echocardiogram (TTE) or TEE to estimate SV and CO can monitor fluid responsiveness and response to vasoactive therapy. Systemic vascular resistance (SVR) can be estimated using the formula: $SVR = 80 \times (\text{MAP} - \text{RA pressure [mm Hg]}) / \text{CO (L/min)}$. These quantitative data can be helpful when differentiating a patient’s macro circulatory shock phenotype and choosing a therapeutic intervention.

TEE has the unique advantage that allows visualization of the superior vena cava (SVC), which can be used to predict fluid responsiveness, avoiding confounders such as abnormal intra-abdominal pressure. In mechanically ventilated patients, SVC collapsibility greater than 36% has been found to predict fluid responsiveness with sensitivity and specificity of 90% and 100%, respectively.⁴⁹

Last, although the assessment of diastolic function has been traditionally beyond the scope of FOCUS, mitral inflow velocities and Tissue Doppler Imaging of the mitral annulus using TTE or TEE can provide valuable information during resuscitation.⁵⁰ A ratio $E/A \geq 2$ in patients with decreased LV function (ejection fraction <40%) or $E/e' \geq 14$ with normal LV systolic function, has been shown to predict elevated left atrial pressure (LAP), indicating severe LV diastolic dysfunction. Although the exact values of LAP do not correlate with preload sensitivity,⁵¹ this binary assessment (ie, normal vs elevated) can help to identify patients at high risk for developing hydrostatic pulmonary edema from fluid therapy, and differentiate cardiogenic pulmonary edema from ARDS.

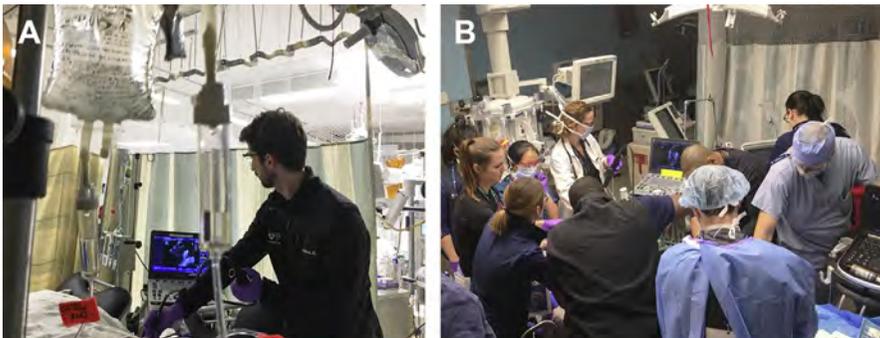
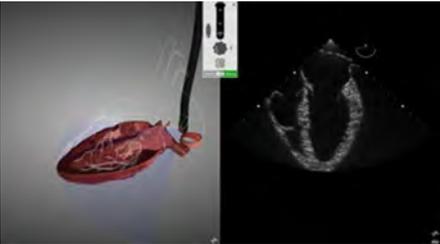
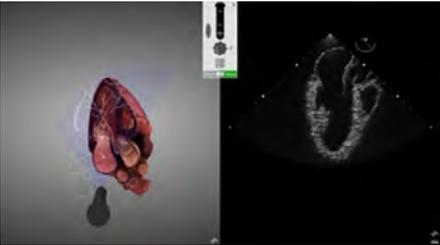
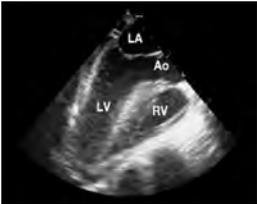


Fig. 9. Common applications of resuscitative TEE in the ED. (A) Physician performing assessment of undifferentiated shock in a mechanically ventilated patient. (B) Performance of resuscitative TEE for the guidance of cannula placement, with ongoing cardiopulmonary resuscitation during a case of ECPR. ECPR, extracorporeal cardiopulmonary resuscitation; ED, emergency department.

Table 5
TEE probe graphics courtesy of Heart Works by Intelligent Ultrasound

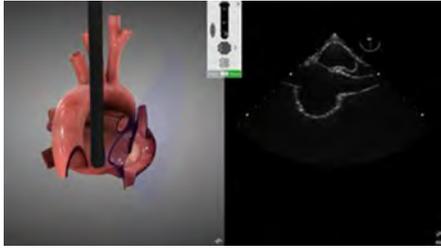
View	Probe Position	TEE Anatomy	Clinical Application
ME 4C			Pathology pericardium LV/RV size and function RWMA Valvular pathology
ME LAX			Quality of CPR (AMC) LV function Pathology MV Pathology AV

LV function
RWMA
Pathology pericardium



TG SAX Pap

Procedure guidance
Venous guidewire ECMO
Volume responsiveness



ME Bicaval

Abbreviations: AMC, area of maximal compression; AV, aortic valve; CPR, cardiopulmonary resuscitation; ECMO, extracorporeal membrane oxygenation; LV, left ventricle; ME Bicaval, midesophageal bicaval; ME LAX, midesophageal long axis view; ME TG SAX Pap, midesophageal transgastric short axis papillary view; ME4C, midesophageal 4 chamber; MV, mitral valve; RV, right ventricle; RWMA, regional wall motion abnormality; TEE, transesophageal echocardiography.

3D Graphics Courtesy of Heartworks, Intelligent Ultrasound, Ltd, Abingdon, United Kingdom.

RESUSCITATIVE TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Over the past 2 decades, the practice of TEE has expanded from its traditional indications (ie, patients undergoing cardiac surgery, suspected endocarditis, or cardioversion in atrial fibrillation), to assist the hemodynamic evaluation of patients with acute decompensation, shock, and cardiac arrest.^{52–56} Following landmark publications demonstrating the feasibility of EP-performed TEE, a number of institutions in the United States have implemented ED-based TEE programs (Fig. 9). In 2017, the American College of Emergency Physicians published a Policy Statement to provide guidelines for the use of TEE in cardiac arrest.⁵⁷

EP and intensivist-performed TEE examinations are feasible, safe, and clinically impactful in the management of critically ill patients.⁵⁶ The 4 core views in resuscitative TEE are midesophageal (ME) 4 chamber, ME long axis, transgastric short axis at the level of papillary muscles, and ME bicaval view (Table 5). Fig. 10 provides an algorithm integrating the 4 core resuscitative TEE views.

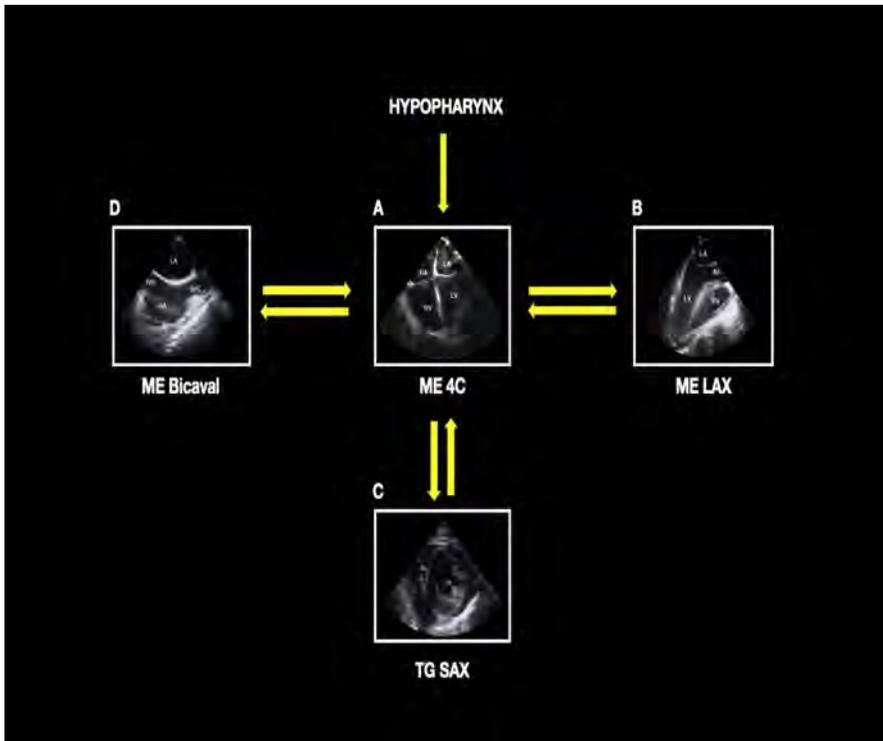


Fig. 10. Algorithm integrating 4 resuscitative TEE views. (A) ME4C view. ME4C represents the “home base” of resuscitative TEE and is the starting point for all examinations. This view is obtained at the mid-esophageal level (probe insertion 35–40 cm from the incisors), omniplane at 0° with sector depth at around 12–14 cm depending on the heart size. (B) ME LAX. From the ME 4C view, the image is centered on the LV and then the omniplane should be rotated to 130–140°. (C) TG SAX. From the ME 4C view, the probe is advanced out of the esophagus and into the stomach at around 40–45 cm from the incisors, with the sector depth at around 10–12 cm. As the probe exists the esophagus, gentle ante-flexion is applied to establish contact with the gastric wall. (D) ME Bicaval. From the ME 4C, the image is centered over the RA and then the omniplane rotated to 90°, with the sector depth at around 8–10 cm. Image courtesy of the Resuscitative TEE Project (www.resuscitativetee.com).

The primary indication for use of resuscitative TEE in the ED is during cardiac arrest.^{54,58} However, TEE can be clinically influential in several other clinical scenarios:

1. Evaluation of patients in shock who have inadequate transthoracic windows.⁵⁶
2. Assessment of fluid responsiveness in mechanically ventilated patients using SVC variation.⁴⁹
3. The guidance of extracorporeal membrane oxygenation cannulation during initiation of extracorporeal circulation.⁵⁷

SUMMARY

Resuscitative cardiopulmonary ultrasound is a powerful tool in the assessment of critically ill patients, and continues to rapidly grow in emergency medicine. The basic elements include the qualitative evaluation of LV and RV function, the identification of pericardial effusion, and the evaluation of preload. Advanced applications such as identification of tamponade physiology, quantitative function of the RV and LV, and hemodynamic monitoring can be incorporated with relatively simple measurements. Resuscitative cardiopulmonary ultrasound and TEE represent a dynamic and scalable field in acute care imaging. EPs can and should adapt the use of these tools to their individual needs, considering skill level, clinical environment, and wider institutional practices.

REFERENCES

1. 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(12):1225–30.
2. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging* 2015;16(3):233–71.
3. Moore CL, Rose GA, Tayal VS, et al. Determination of left ventricular function by emergency physician echocardiography of hypotensive patients. *Acad Emerg Med* 2002;9(3):186–93.
4. Mckaigney CJ, Krantz MJ, La Rocque CL, et al. E-point septal separation: a bedside tool for emergency physician assessment of left ventricular ejection fraction. *Am J Emerg Med* 2014;32(6):493–7.
5. Randazzo MR, Snoey ER, Levitt MA, et al. Accuracy of emergency physician assessment of left ventricular ejection fraction and central venous pressure using echocardiography. *Acad Emerg Med* 2003;10(9):973–7.
6. Shahgaldi K, Gudmundsson P, Manouras A, et al. Visually estimated ejection fraction by two dimensional and triplane echocardiography is closely correlated with quantitative ejection fraction by real-time three dimensional echocardiography. *Cardiovasc Ultrasound* 2009;7(1):1–7.
7. Secko MA, Lazar JM, Saliccioli LA, et al. Can junior emergency physicians use E-point septal separation to accurately estimate left ventricular function in acutely dyspneic patients? *Acad Emerg Med* 2011;18(11):1223–6.
8. Massie BM, Schiller NB, Ratshin RA, et al. Mitral-septal separation: new echocardiographic index of left ventricular function. *Am J Cardiol* 1977;39(7):1008–16.

9. Silverstein JR, Laffely NH, Rifkin RD. Quantitative estimation of left ventricular ejection fraction from mitral valve E-point to septal separation and comparison to magnetic resonance imaging. *Am J Cardiol* 2006;97(1):137–40.
10. Ahmadpour H, Shah AA, Allen JW, et al. Mitral E point septal separation: a reliable index of left ventricular performance in coronary artery disease. *Am Heart J* 1983; 106(1 PART 1):21–8.
11. Sanchez O, Trinquart L, Colombet I, et al. Prognostic value of right ventricular dysfunction in patients with haemodynamically stable pulmonary embolism: a systematic review. *Eur Heart J* 2008;29(12):1569–77.
12. Mekontso Dessap A, Boissier F, Charron C, et al. Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact. *Intensive Care Med* 2016;42(5):862–70.
13. Engström AE, Vis MM, Bouma BJ, et al. Right ventricular dysfunction is an independent predictor for mortality in ST-elevation myocardial infarction patients presenting with cardiogenic shock on admission. *Eur J Heart Fail* 2010;12(3):276–82.
14. Weekes AJ, Johnson AK, Troha D, et al. Prognostic value of right ventricular dysfunction markers for serious adverse events in acute normotensive pulmonary embolism. *J Emerg Med* 2017;52(2):137–50.
15. Gaspari R, Weekes A, Adhikari S, et al. A retrospective study of pulseless electrical activity, bedside ultrasound identifies interventions during resuscitation associated with improved survival to hospital admission. A REASON Study. *Resuscitation* 2017;120:103–7.
16. Rutz MA, Clary JM, Kline JA, et al. Emergency physicians are able to detect right ventricular dilation with good agreement compared to cardiology. *Acad Emerg Med* 2017;24(7):867–74.
17. McConnell MV, Solomon SD, Rayan ME, et al. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol* 1996;78(4):469–73.
18. Mediratta A, Addetia K, Medvedofsky D, et al. Echocardiographic diagnosis of acute pulmonary embolism in patients with McConnell's sign. *Echocardiography* 2016;33(5):696–702.
19. McCutcheon JB, Schaffer P, Lyon M, et al. The McConnell sign is seen in patients with acute chest syndrome. *J Ultrasound Med* 2018. <https://doi.org/10.1002/jum.14585>.
20. Mandavia DP, Hoffner RJ, Mahaney K, et al. Bedside echocardiography by emergency physicians. *Ann Emerg Med* 2001;38(4 SUPPL):377–82.
21. Klein AL, Abbara S, Agler DA, et al. American Society of Echocardiography clinical recommendations for multimodality cardiovascular imaging of patients with pericardial disease: endorsed by the Society for Cardiovascular Magnetic Resonance and Society of Cardiovascular Computed Tomography. *J Am Soc Echocardiogr* 2013;26(9):965–1012.e15.
22. Zhang Z, Xu X, Ye S, et al. Ultrasonographic measurement of the respiratory variation in the inferior vena cava diameter is predictive of fluid responsiveness in critically ill patients: systematic review and meta-analysis. *Ultrasound Med Biol* 2014;40(5):845–53.
23. Long E, Oakley E, Duke T, et al, Paediatric Research in Emergency Departments International Collaborative (PREDICT). Does respiratory variation in inferior vena cava diameter predict fluid responsiveness: a systematic review and meta-analysis. *Shock* 2017;47(5):550–9.

24. Si X, Xu H, Liu Z, et al. Does respiratory variation in inferior vena cava diameter predict fluid responsiveness in mechanically ventilated patients? A systematic review and meta-analysis. *Anesth Analg* 2018;127(5):1157–64.
25. 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):1164–7.
26. Corl KA, George NR, Romanoff J, et al. Inferior vena cava collapsibility detects fluid responsiveness among spontaneously breathing critically-ill patients. *J Crit Care* 2017;41:130–7.
27. Lee CWC, Kory PD, Arntfield RT. Development of a fluid resuscitation protocol using inferior vena cava and lung ultrasound. *J Crit Care* 2016;31(1):96–100.
28. Zanobetti M, Scorpiniti M, Gigli C, et al. Point-of-care ultrasonography for evaluation of acute dyspnea in the ED. *Chest* 2017;151(6):1295–301.
29. Buhumaid RE, St-Cyr Bourque J, Shokoohi H, et al. Integrating point-of-care ultrasound in the ED evaluation of patients presenting with chest pain and shortness of breath. *Am J Emerg Med* 2018. <https://doi.org/10.1016/j.ajem.2018.10.059>.
30. Pivetta E, Goffi A, Lupia E, et al. Lung ultrasound-implemented diagnosis of acute decompensated heart failure in the ED: A SIMEU multicenter study. *Chest* 2015;148(1):202–10.
31. Wooten WM, Shaffer LET, Hamilton LA. Bedside ultrasound versus chest radiography for detection of pulmonary edema. *J Ultrasound Med* 2018;1–7. <https://doi.org/10.1002/jum.14781>.
32. Patel CJ, Bhatt HB, Parikh SN, et al. Bedside lung ultrasound in emergency protocol as a diagnostic tool in patients of acute respiratory distress presenting to emergency department. *J Emerg Trauma Shock* 2018;11(2):125–9.
33. Dietrich CF, Mathis G, Blaivas M, et al. Lung artefacts and their use. *Med Ultrason* 2016;18(4):488–99.
34. Bianco F, Bucciarelli V, Ricci F, et al. Lung ultrasonography: a practical guide for cardiologists. *J Cardiovasc Med* 2017;18(7):501–9.
35. Lichtenstein D, Mezière G, Biderman P, et al. The “lung point”: an ultrasound sign specific to pneumothorax. *Intensive Care Med* 2000;26(10):1434–40.
36. Lichtenstein DA, Mezière GA, Lagoueyte JF, et al. A-lines and B-lines: lung ultrasound as a bedside tool for predicting pulmonary artery occlusion pressure in the critically ill. *Chest* 2009;136(4):1014–20.
37. Bataille B, Riu B, Ferre F, et al. Integrated use of bedside lung ultrasound and echocardiography in acute respiratory failure: a prospective observational study in ICU. *Chest* 2014;146(6):1586–93.
38. Mojoli F, Bouhemad B, Mongodi S, et al. Lung ultrasound for critically ill patients. *Am J Respir Crit Care Med* 2018. <https://doi.org/10.1164/rccm.201802-0236CI>.
39. Puchalski MD, Williams RV, Askovich B, et al. Assessment of right ventricular size and function: echo versus magnetic resonance imaging. *Congenit Heart Dis* 2007;2(1):27–31.
40. Huang SJ, Nalos M, Smith L, et al. The use of echocardiographic indices in defining and assessing right ventricular systolic function in critical care research. *Intensive Care Med* 2018;44(6):868–83.
41. Park JH, Kim JH, Lee JH, et al. Evaluation of right ventricular systolic function by the analysis of tricuspid annular motion in patients with acute pulmonary embolism. *J Cardiovasc Ultrasound* 2012;20(4):181–8.
42. Miller D, Farah MG, Liner A, et al. The relation between quantitative right ventricular ejection fraction and indices of tricuspid annular motion and myocardial performance. *J Am Soc Echocardiogr* 2004;17(5):443–7.

43. Lahham S, Fox JC, Thompson M, et al. Tricuspid annular plane of systolic excursion to prognosticate acute pulmonary symptomatic embolism (TAPSE/PAPSE study). *J Ultrasound Med* 2018. <https://doi.org/10.1002/jum.14753>.
44. Dwyer KH, Rempell JS, Stone MB. Diagnosing centrally located pulmonary embolisms in the emergency department using point-of-care ultrasound. *Am J Emerg Med* 2018;36(7):1145–50.
45. Vakamudi S, Ho N, Cremer PC. Pericardial effusions: causes, diagnosis, and management. *Prog Cardiovasc Dis* 2017;59(4):380–8.
46. Appleton CP, Hatle LK, Popp RL. Cardiac tamponade and pericardial effusion: respiratory variation in transvalvular flow velocities studied by Doppler echocardiography. *J Am Coll Cardiol* 1988;11(5):1020–30.
47. Vincent J, Rhodes A, Perel A, et al. Clinical review: update on hemodynamic monitoring—a consensus of 16. *Crit Care* 2011. <https://doi.org/10.1186/cc10291>.
48. Porter TR, Shillcutt SK, Adams MS, et al. Guidelines for the use of echocardiography as a monitor for therapeutic intervention in adults: a report from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2015;28(1):40–56.
49. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med* 2004;30(9):1734–9.
50. Combes A, Arnoult F, Trouillet JL. Tissue Doppler imaging estimation of pulmonary artery occlusion pressure in ICU patients. *Intensive Care Med* 2004;30(1):75–81.
51. Osman D, Ridel C, Ray P, et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. *Crit Care Med* 2007;35(1):64–8.
52. Memtsoudis SG, Rosenberger P, Loffler M, et al. The usefulness of transesophageal echocardiography during intraoperative cardiac arrest in noncardiac surgery. *Anesth Analg* 2006;102(6):1653–7.
53. Shillcutt SK, Markin NW, Montzingo CR, et al. Use of rapid "rescue" perioperative echocardiography to improve outcomes after hemodynamic instability in noncardiac surgical patients. *J Cardiothorac Vasc Anesth* 2012;26(3):362–70.
54. Teran F, Dean AJ, Centeno C, et al. Evaluation of out-of-hospital cardiac arrest using transesophageal echocardiography in the emergency department. *Resuscitation* 2019;137:140–7.
55. Mayo PH, Narasimhan M, Koenig S. Critical care transesophageal echocardiography. *Chest* 2015;148(5):1323–32.
56. Arntfield R, Pace J, Hewak M, et al. Focused transesophageal echocardiography by emergency physicians is feasible and clinically influential: observational results from a novel ultrasound program. *J Emerg Med* 2016;50(2):286–94.
57. Fair J, Tonna J, Ockerse P, et al. Emergency physician-performed transesophageal echocardiography for extracorporeal life support vascular cannula placement. *Am J Emerg Med* 2016;34(8):1637–9.
58. Van Der Wouw PA, Koster RW, Delemarre BJ, et al. Diagnostic accuracy of transesophageal echocardiography during cardiopulmonary resuscitation. *J Am Coll Cardiol* 1997;30:780–3.
59. Dickman E, Terentiev V, Likourezos A, et al. Extension of the thoracic spine sign: a new sonographic marker of pleural effusion. *J Ultrasound Med* 2015;34(9):1555–61.