



EDUCATIONAL ADVANCE

The “5Es” of Emergency Physician–performed Focused Cardiac Ultrasound: A Protocol for Rapid Identification of Effusion, Ejection, Equality, Exit, and Entrance

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Abstract

Emergency physician (EP)-performed focused cardiac ultrasound (EP FOCUS) has been increasingly recognized as a crucial tool to help clinicians diagnose and treat potentially life-threatening conditions. The existing literature demonstrates a variety of EP FOCUS applications and protocols; however, EP FOCUS is not taught, practiced, or interpreted consistently between institutions. Drawing on over 12 years of experience in a large-volume, high-acuity academic emergency department, we have developed a protocol for teaching and performing EP FOCUS known as “The 5Es,” where each E represents a specific assessment for immediately relevant clinical information. These include pericardial effusion, qualitative left ventricular ejection, ventricular equality, exit (aortic root diameter), and entrance (inferior vena cava diameter and respirophasic variation). Each of these assessments has been well described in the emergency medicine literature and is within the scope of EP-performed echocardiography. This approach provides a reliable and easily recalled framework for assessing, teaching, and communicating EP FOCUS findings that are essential in caring for the patient in the emergency setting.

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It has been recognized for more than 25 years that emergency physician (EP)-performed focused cardiac ultrasound (EP FOCUS) is an important skill for the care of patients with potentially life-threatening presentations.^{1,2} A recent review detailed 16 specific protocols that included cardiac ultrasound (US) as part of the point-of-care US assessment in medical shock.³ The term “focused cardiac ultrasound” has been addressed in some detail (FOCUS,⁴ FCU,⁵ and FoCUS⁶). However, this term is not specialty-specific, may include assessments that are not relevant in the acute/emergency setting, and has not included assessment of the thoracic aortic root (“exit”), which may be particularly applicable to acute and emergent presentations. We have found that the proximal thoracic aorta can be reli-

ably assessed, providing vital information about potential aortic pathology in patients presenting with acute symptoms.⁷ We thus propose the “5Es” to assess for the presence of a pericardial effusion, left ventricular ejection, ventricular equality, exit (aortic root diameter), and entrance (inferior vena cava [IVC] diameter and respirophasic variation). The 5Es protocol provides an easy-to-teach, evidence-based, and standardized approach to EP FOCUS for the rapid identification and management of time-sensitive pathologic conditions.

APPROACH TO EXAM

Typically EP FOCUS uses one or more of three windows and five views: a parasternal long-axis (PSLA), a

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parasternal short-axis (PSSA), an apical four-chamber (A4C), a subcostal long-axis (SCLA), and a subcostal four-chamber (SC4C) view. While pertinent findings are optimally confirmed in at least two views, it is understood that time constraints, patient acuity, patient mobility, and patient habitus may limit views.^{6,8} For images in this article, we will use an emergency medicine convention for cardiac imaging with the probe marker oriented to the patient's right, which keeps the anatomic right on the screen-left, as is the convention for other US imaging.⁹ This is in contrast with image and probe orientation utilized in traditional cardiology-performed US, but has been recognized as an accepted convention that we find to be conceptually easier, particularly when performing EP FOCUS as part of an integrated examination such as the focused assessment with sonography for trauma (FAST) or the rapid US for shock and hypotension.^{3,6}

As has been discussed previously in the emergency US literature, EP FOCUS is not intended to replace comprehensive echocardiography (echo) when more thorough cardiology evaluation is indicated. The clinical questions addressed by EP FOCUS tend to be limited and qualitative but it should be understood that EP FOCUS findings may fall on a spectrum that can make binary categorization challenging. The EP is encouraged to use professional judgment for the interpretation and integration of his or her findings into the diagnosis and care of the patient, as well as the need for specialist consultation.

EFFUSION

The first "E" in our protocol is an assessment for pericardial effusion. Of the echo components in our protocol, detection of pericardial effusion was the first to be clearly investigated and delineated in the literature and has been incorporated as part of the FAST for more than 20 years.^{1,2,10,11} Given the wide range of symptoms and the potential for hemodynamic collapse, timely and accurate detection of a pericardial effusion is essential for expediting diagnosis and management.¹² EP FOCUS can identify pericardial effusions at the bedside with a high degree of diagnostic accuracy.¹³ EP FOCUS has been shown to improve mortality in penetrating cardiac trauma.¹⁴ In a study of emergency department (ED) patients with unexplained dyspnea, more than 10%

were found to harbor pericardial effusions.¹⁵ Tamponade physiology is detectable earlier with US than with traditional exam findings, such as Beck's triad.^{13,16}

Pericardial effusions may be characterized as focal, circumferential, simple, or complex. While circumferential effusions are most common, it is important to obtain as many views as possible so as not to miss a focal effusion. This is of particular importance following any invasive cardiac procedure. Complex effusions (with internal echoes) may occur in the presence of pericardial hemorrhage or infectious effusions or in long-standing effusions with fibrous stranding. Of note, while uncommon in the developed world, tuberculosis is the most common cause of pericardial effusion in the developing world and often presents with complex effusions.¹⁷

Techniques for Assessing Effusion

The subcostal window (either SC4C or SCLA) is the most reliable view for detecting pericardial effusions because the most dependent portion of the pericardium is closest to the face of the probe. In this window, the liver can also help provide an acoustic window to the inferior pericardium. In the parasternal views (PSLA and PSSA), significant effusions should be visualized posterior to the left ventricle (LV) and not just anteriorly, as this will often be a fat pad and not an effusion (Figure 1). In the A4C view, small effusions may be visible lateral to the LV free wall, and moderate to large effusions may be seen tracking completely around the apex of the heart.

Normal patients have a trace amount physiologic pericardial fluid that may be seen with modern equipment and described as trivial or "not clinically significant." True effusions can be categorized as "small," "moderate," or "large." This categorization is often qualitative, although an effusion can be measured by assessing the largest pocket of fluid at end-diastole and measured orthogonally to the surface of the heart. By convention, small effusions are smaller than 1 cm, moderate effusions are 1 to 2 cm, and large effusions are >2 cm.¹⁸ Moderate to large effusions are more likely to have an effect on hemodynamics; however, even small effusions can also result in tamponade physiology.^{19,20} Prognosis in the setting of pericardial effusion is largely related to time course and etiology, and it is important to recognize that the hemodynamic sequelae of an effusion is much more important than its actual size.^{18,21}

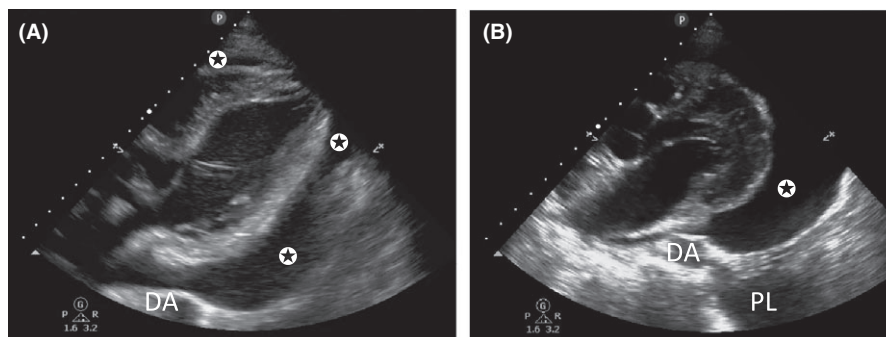


Figure 1. Effusion. (A) Pericardial effusion seen circumferentially (stars); (B) pericardial effusion (star) seen anterior to the descending aorta (DA) and a pleural effusion (PL) is seen posterior to aorta.

Identification of an effusion should prompt the practitioner to look for signs of tamponade physiology. As pressures inside the pericardium elevate, US will show a progression of findings beginning with collapse of the right atrium (RA), collapse of the right ventricle (RV), and finally LV collapse.²⁰ One of the most easily obtained and sensitive signs of tamponade is the presence of a noncollapsible, plethoric IVC (the fifth “E”), indicating impaired filling from extrinsic compression of the heart.²² Collapse of the RA in ventricular systole or the RV in diastole indicates tamponade physiology, but tachycardia may make it difficult to differentiate normal systolic ventricular collapse from pathologic diastolic ventricular collapse. To confirm RV diastolic collapse, M-mode can be used in either the PSLA or SX4C views to demonstrate RV free wall motion as it relates to the anterior leaflet of the mitral valve (Figure 2). If the RV free wall is collapsed when the mitral (anterior) valve is open, it indicates diastolic collapse of the RV.

Tamponade physiology can also be demonstrated on echo by exaggerated respiratory variation of ventricular in-flow velocities (the echo equivalent of pulsus paradoxus).²³ In an A4C view, a pulsed wave spectral Doppler gate is placed at the tips of the mitral valve during diastole. A change of more than 25% in the early filling signal (“E wave”) indicates impaired filling (Figure 2). While these techniques can help determine the presence of tamponade physiology, they are not completely sensitive or specific and should be used in conjunction with clinical judgment.

Pearls and Pitfalls for Effusion

A common pitfall, particularly among novice practitioners of EP FOCUS, is confusing epicardial or pericardial fatty tissue for an effusion.²⁴ Fatty tissue can be characterized by its heterogeneous echo-texture, its coordinated movement in conjunction with the myocardium, and its failure to track around the heart, especially at the apex and posteriorly. A false-positive diagnosis may occur when a hypoechoic space is seen only anterior to the heart on the PSLA view. In the parasternal views, pathologic effusions are typically visible posteriorly, in the most dependent portion of the pericardium (Figure 1). Most clinically significant effusions will not obliterate during diastole and can be traced with US around the apex of the heart and/or posteriorly. Exceptions are loculated or focal effusions and therefore multiple views are recommended.

It should be noted that there are other causes of RA and RV diastolic collapse, including severe hypovolemia and large pleural effusions. Pleural effusions may be misinterpreted as pericardial effusions, particularly in the PSLA window where left-sided pleural fluid lies adjacent to the LV. They can be differentiated by their relationship to the descending aorta. Pericardial effusions will track *between* the descending aorta and the LV free wall, while pleural effusions will track *posterior and lateral* to the descending aorta (Figure 1).

Due to the nonspecific clinical presentations of pericardial effusion and tamponade, we recommend having a low threshold for employing US, particularly when a

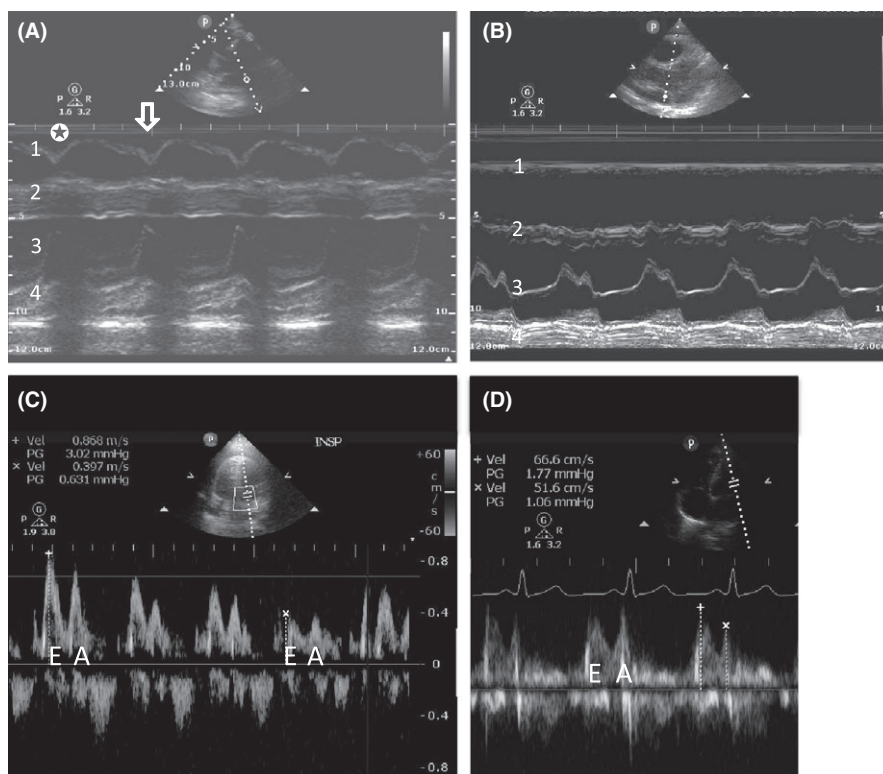


Figure 2. Pericardial effusion and tamponade. (A) M-mode used to show RV collapse during ventricular filling (*arrow*) when mitral valve is open (*star* = pericardial effusion, 1 = RV free wall, 2 = interventricular septum, 3 = mitral valve, 4 = LV free wall). (B) M-mode tracing provided of normal heart for comparison. (C) Mitral inflow velocity measured with pulse wave Doppler with more than 25% decrease in E-wave. (D) Normal mitral inflow velocity measured with pulse-wave Doppler with no change in E-wave.

patient presents with unexplained dyspnea, tachycardia, hypotension, near-syncope, or cardiomegaly on chest radiograph. While visualization of the pericardium is essential in the initial evaluation of penetrating thoraco-abdominal trauma, pericardial effusion as a result of blunt trauma is rare, and such patients are unlikely to survive to ED presentation.²³ The identification of a pericardial effusion in blunt trauma should raise the suspicion of either a false-positive or an incidental pericardial effusion and should not necessarily indicate the need for acute intervention unless there is severe hemodynamic compromise without another source.

EJECTION

The second “E” in our protocol is assessment of LV ejection fraction (LVEF). This E can help differentiate causes of hypotension, chest pain, and dyspnea and may aid in expediting condition-specific therapies.^{3,25,26}

There are multiple quantitative modalities for LVEF calculation; however, qualitative estimates of ejection fraction by EP FOCUS correlate well with both quantitative measurements and subjective estimates by cardiologists.^{25,27} Qualitative assessment of global ejection is typically categorized as “hyperdynamic” (LVEF > 65%), “normal” (LVEF 50% to 65%), “moderately depressed” (LVEF 30% to 50%), or “severely depressed” (LVEF < 30%).^{25,28} Additionally, in cardiac arrest patients, one can recognize “no coordinated myocardial activity” as a likely indicator of futility of further resuscitation.²⁹ Hyperdynamic states are typically associated with decreased afterload, and are most commonly found in patients with sepsis or severe hypovolemia. A severely depressed ejection fraction, particularly when paired with a plethoric IVC (and/or B-lines on thoracic images), indicates systolic heart failure.

Techniques for Assessing Ejection Fraction

For a visual determination of LVEF, the PSLA view is an excellent initial window. The PSLA includes the septum, apex, and posterior LV wall. Additionally, the PSLA view provides good visualization of the anterior leaflet of the mitral valve, allowing for assessment of E-point septal separation (EPSS). Movement of the anterior leaflet of the mitral valve so that it nearly touches the septum in diastole correlates with good LV filling and thus a good ejection fraction.³⁰ EPSS may be assessed by visual estimation or measured quantitatively using B-mode or M-mode to measure the smallest distance from the anterior mitral valve tip to the septal wall during diastole (Figure 3). EPSS measurements greater than 7 mm have been shown to correlate with severely depressed LVEF.³¹ Rotating from the PSLA to a PSSA view at the level of the papillary muscles shows the entire circumference of the muscular portion of the LV.³² This is an optimal window to observe the concentric squeeze of the LV by estimating the degree of internal chamber collapse in systole versus diastole. This view is also useful to detect focal wall motion abnormalities. In a PSSA view proceeding in a clockwise direction, the septal, anterior, lateral, posterior, and inferior wall segments of the mid-portion of the LV are visible (mnemonic “SALPI”).

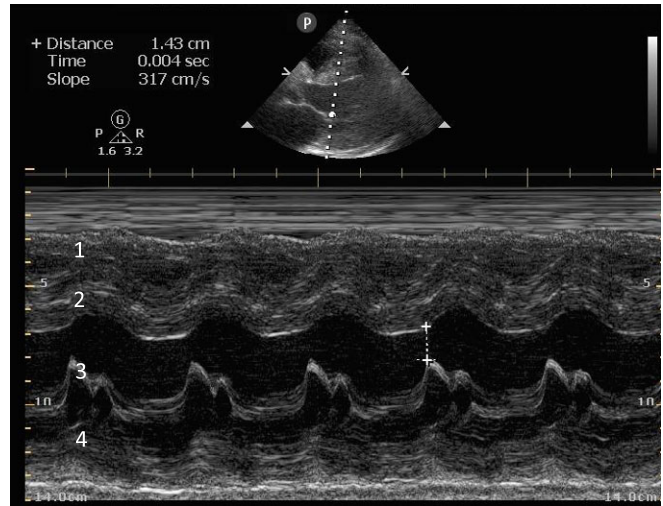


Figure 3. Ejection fraction. Depiction of E-point septal separation: M-mode is used to trace the movement of the anterior mitral valve in relation to the ventricular septum. Measurements of greater than 7 mm are suggestive of depressed systolic ejection. Above the measurement is 14.3 mm, which is abnormal (1 = right ventricle free wall, 2 = interventricular septum, 3 = mitral valve, 4 = left ventricle free wall).

The A4C window also provides a good view of the global function of the myocardium, but should include an apical two-chamber view to evaluate all parts of the myocardium. The subxiphoid views may also provide information about global myocardial function, but it may be difficult to see all portions of the LV.

Pearls and Pitfalls of Ejection Fraction

While EPs are very good at distinguishing normal function from severe dysfunction, moderate LV dysfunction can be more difficult to reliably discern.²⁵ It is important to optimize the LV view at the widest diameter of the LV chamber with clearly visible endocardial borders. Being oblique to the LV chamber may underestimate its size and overestimate its emptying (Figure 4). EPSS is a good surrogate measure of ejection fraction, but can be confounded by septal hypertrophy, mitral valve stenosis, or mismeasurements. Two-dimensional images (especially in the A4C view) can be limited by poor lateral resolution and decreased visualization of endocardial borders.³¹

While the basic EP FOCUS assessment is for estimation of global ejection fraction, focal wall motion abnormalities can affect overall function and represent a pitfall if not appropriately recognized or characterized. Incomplete visualization of all portions of the LV can result in missed focal wall motion abnormalities. When present, these typically indicate myocardial dysfunction or scarring from ischemia, which may be acute or chronic. Myocardial contraction should be observed for several heartbeats, particularly if there are premature ventricular contractions or underlying arrhythmias, such as atrial fibrillation.

Even with a preserved ejection fraction, heart failure still remains a possible cause of dyspnea. Approximately half of all heart failure cases may have some component of impaired relaxation leading to diastolic dysfunction,

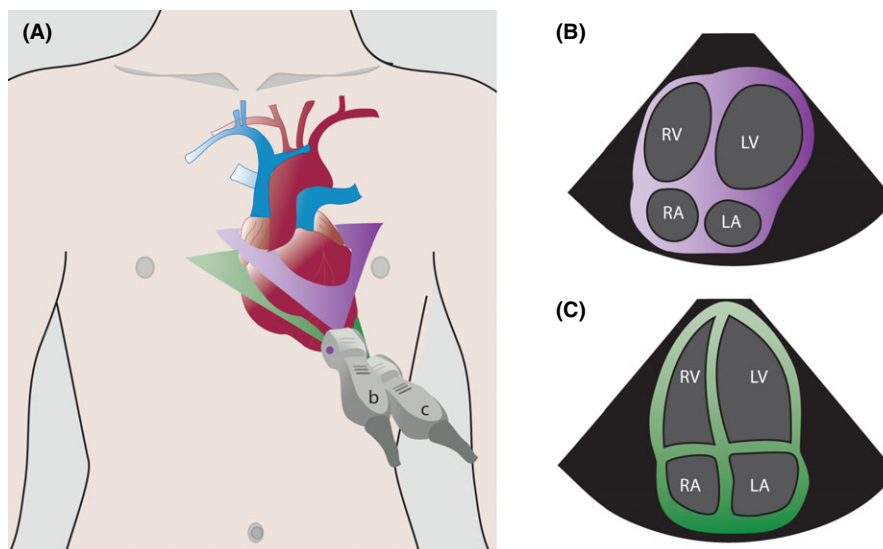


Figure 4. Foreshortening. Ejection: (A) Incorrect and correct angle for image acquisition. (B) Foreshortening of heart. (C) Ideal image for apical view without foreshortening. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

also known as heart failure with preserved ejection fraction.³²

EQUALITY

The third “E” in our protocol is for equality, referring to the relative size of the RV to the LV. In healthy patients, the RV is a low-pressure, thin-walled, high-compliance chamber that is wrapped anteriorly around the muscular, cone-shaped LV. The normal RV systolic pressure is approximately 25 mm Hg with an RV:LV diameter ratio of less than 0.6:1. When pressure in the pulmonary artery rises, the RV will dilate (Figure 5). While not perfectly sensitive, using “equality” (i.e., a 1:1 ratio) as the cutoff ensures specificity for detecting true RV strain by EP FOCUS.^{33,34}

Right ventricle dilatation may be acute, chronic, or acute-on-chronic. However, in patients presenting with undifferentiated chest pain, shortness of breath, hypotension, or syncope, the presence of any RV dilatation

should raise the diagnostic suspicion of an acute pulmonary embolism (PE). PEs can range in severity from small subsegmental disease with minimal morbidity and mortality, to massive with resultant RV failure, shock, and death. PEs are typically defined as “massive” when sustained hypotension ensues. The remaining PE categories are stratified into “submassive,” when there are signs of RV strain on echo, versus “low-risk” (also labeled “small” or “minor”), when there is no RV strain.³⁵ EP FOCUS can help stratify these patients, with RV hypokinesis being an independent predictor of mortality.³⁶ The presence of RV strain in suspected massive PE or diagnosed submassive PE may signal the need for more aggressive therapy such as thrombolysis or thrombectomy.^{35,37–39}

Techniques for Assessing Equality

When the A4C view is properly obtained, with all four chambers visible and divided by a vertically oriented interventricular septum, ventricular size can be accurately

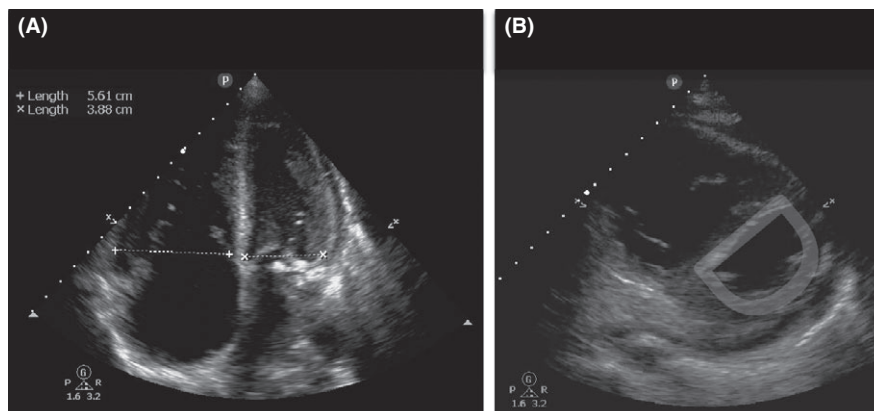


Figure 5. Right ventricle (RV) enlargement. (A) RV enlargement with RV:LV ratio of >1:1. (B) Enlargement of RV cavity with bowing into LV cavity creating “D”-shaped LV. LV = left ventricle.

compared either qualitatively or quantitatively (Figure 5). If measured, ventricular size should be obtained between endocardial borders at the tips of the valves in diastole. However, the A4C may be technically challenging to obtain correctly. The PSLA window may show a prominent and hypokinetic anterior chamber when RV strain is present. However, tilting of the plane in the PSLA (known as the “tricuspid tilt”) may cut across the RV obliquely causing overemphasis of the RV relative to the LV and should be used with caution. On the other hand, the PSSA at the level of the papillary muscles can often provide an excellent and reliable estimation of RV:LV ratio. In the PSSA view, the greatest chamber diameters for both the RV and the LV are usually visible side by side at the level of the papillary muscles. When RV pressure rises the septum will be pushed toward the LV. The PSSA is thus the preferred view to demonstrate this septal flattening, resulting in the characteristic “D-shaped” LV (Figure 5).⁴⁰ The subxiphoid view may also show RV enlargement, but should be used with caution as the RV may be overemphasized if the plane of the US cuts through it obliquely, and RV size should be confirmed in other planes.

In addition to enlargement, EP FOCUS may show RV hypokinesis or be used to measure elevated RV pressure. While the LV tends to contract circumferentially and perpendicular to the long axis of the heart, the RV tends to move longitudinally, from base to apex. The A4C is the best view to demonstrate the movement of the tricuspid annulus during RV systole, allowing assessment and measurement of tricuspid annular plane systolic excursion (TAPSE, Figure 6). A TAPSE of 18 mm or greater is typically considered normal.⁴¹ TAPSE is a technique for assessing RV function that is well described in the cardiology literature, but to our knowledge only described once in the EP literature.^{42–45} In our experience it is easily measured when an adequate A4C view is present and has been described as reproducible and perhaps better than RV size as a predictor of PE severity.^{41,43,45}

Right ventricle systolic pressure may be estimated quantitatively when tricuspid regurgitation is present. The peak velocity of the tricuspid regurgitant jet should be measured using continuous wave spectral Doppler with the Doppler signal in line with the jet (typically in an A4C view). The pressure difference between the RV and RA can then be estimated using the modified Bernoulli equation, with $\Delta P = 4 \times V^2$. A velocity greater

than 2.7 m/sec typically indicates elevated RV systolic pressure (Figure 6), with velocities of 4 m/sec or greater indicating chronic RV pressure overload.

Pearls and Pitfalls of Equality

One of the primary pitfalls of RV assessment is overestimation of RV to LV ratio (false-positive) based on the US plane cutting through the RV in an oblique plane that makes the RV look relatively larger than the LV. This can be an issue in the PSLA, SX4C, or A4C views. For apical views, be sure to slide the probe sufficiently lateral on the chest wall so that the probe lies over the point of maximum intensity and true apex. Flattening the plane to transect through the base of the heart avoids foreshortened chambers and misinterpretation of their sizes (Figure 4). For the PSLA view it is important to fan through the long axis of the heart to make sure the LV is maximized relative to the RV.

An understanding of probe marker orientation conventions and relative probe placement on the patient is essential because if reversed, the normally larger LV may be mistaken for an abnormally enlarged RV (or an enlarged RV may be mistaken for a normal LV), especially in the A4C view.⁹

When imaged correctly by an EP with appropriate experience in echo, the presence of an RV:LV ratio of 1:1 or greater is highly specific for RV strain, as determined by consultant-performed echo.^{33,34} However, because using a 1:1 ratio for a dilated RV sets a higher threshold (specificity) for pathology, sensitivity is sacrificed.³⁵ Additionally, it is important to keep in mind that PEs (albeit “small”) may occur without any signs of right heart strain at all.

Right ventricle dilation may be a result of acute, chronic, or acute-on-chronic RV pressure elevation. While differentiation between these entities can be challenging, there are a few clues that may be available using both two-dimensional imaging and Doppler. With acute right heart dilation the RV wall remains thin, but over time the RV will hypertrophy. If the RV free wall myocardium measures over 5 mm, this is indicative of chronic strain (Figure 7). Furthermore, while both acute and chronic conditions produce measurable right heart pressure elevations, the acutely strained, thin-walled RV will not be able to generate extremely high pressures. A pressure gradient of more than 60 mm Hg (tricuspid regurgitation jet of about 4 m/sec or more) indicates chronic RV pressure elevation. McConnell’s sign, the

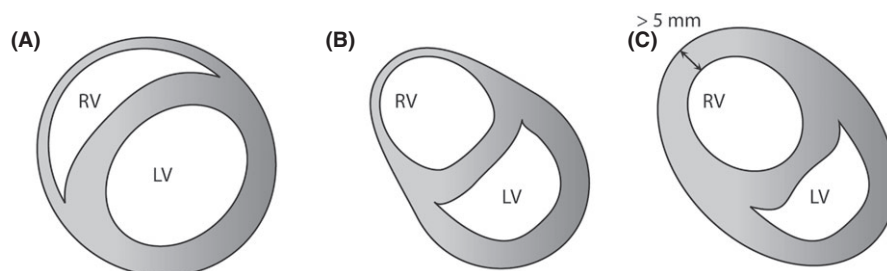


Figure 6. Equality of the ventricle. (A) Normal ventricular ratio and muscular thickness; (B) enlargement of RV cavity with bowing into LV cavity creating “D” shaped LV with thin RV wall, suggestive of acute RV strain; (C) enlargement of RV cavity with bowing into LV cavity creating “D”-shaped LV with thick RV wall (>5 mm) suggestive of chronic RV strain. LV = left ventricle; RV = right ventricle.

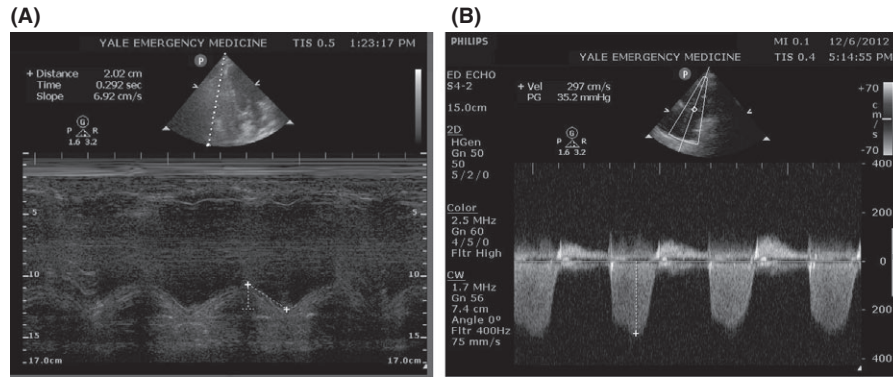


Figure 7. Right ventricle (RV) function. Other measures of RV function: (A) Normal tricuspid annular plane systolic excursion (TAPSE), >16 mm; (B) Elevated RV systolic pressure (>2.7 m/sec), measured using continuous wave Doppler peak velocity of the tricuspid regurgitant jet. Pressure differential is 35.2 mm Hg ($4 \times \text{velocity}^2$).

presence of RV dilation and hypokinesis of the mid and basal portions of the RV with “apical sparing” (as opposed to global RV dysfunction), has been reported to be specific for acute RV strain, although it may not differentiate well between acute strain from PE versus acute strain from RV infarction.^{37,46–48}

EXIT

The fourth “E” in our protocol is for exit from the heart, or the assessment of the aortic root for thoracic aortic aneurysm, and thoracic aortic dissection (TAD). Aneurysmal disease of the thoracic aorta predisposes to aortic dissection.⁴⁹ TAD is a time-dependent and potentially deadly disease process that can present silently or masquerade as a variety of clinical presentations. TAD results from a tear in the intima of the aorta that can propagate in either an anterograde or a retrograde fashion. Retrograde dissections can produce a number of US findings such as an intimal flap, aortic valve insufficiency, retrograde aortic flow, or rupture into the pericardium with pericardial effusion and tamponade. While contrast enhanced computed tomography (CT) remains the test of choice for diagnosing thoracic aortic disease, transthoracic echo has been shown to be accurate for detection of aortic root dilation and may be performed rapidly with a high degree of specificity by the EP at the bedside in a patient presenting with acute chest pain and/or hemodynamic instability.^{7,50}

Techniques for Assessing Exit

The proximal aortic root is best assessed using a PSLA window, and EP measurement of the aorta in this view correlates well with measurements on CT angiogram (CTA).⁷ The aortic root should be measured from leading edge to leading edge (outside wall to inside wall) at the widest visible point during diastole, which is typically across the sinuses of Valsalva (Figure 8). A thoracic aortic root of over 4.5 cm is typically considered aneurysmal. However, measurement on CTA may be slightly lower than measurements on echo, probably due to the angle of measurement.^{7,49} For EP FOCUS, we recommend that measurements of <4 cm be considered normal, 4.0 to 4.5 cm borderline, and >4.5 cm aneurysmal.

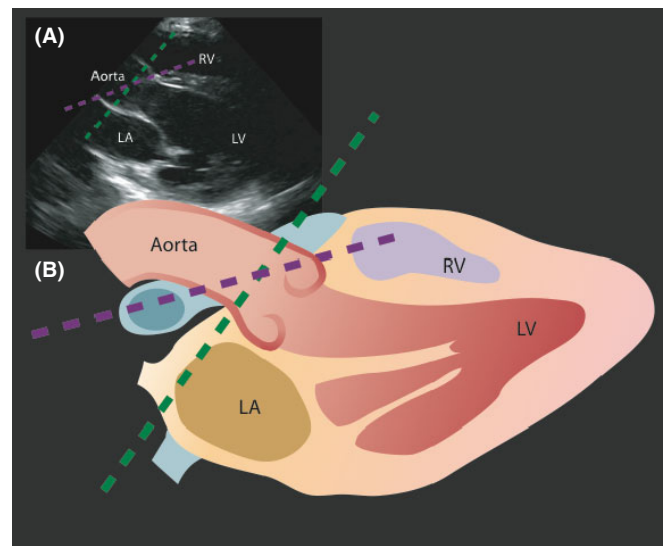


Figure 8. The exit. Evaluation of proximal aorta: (A) Image obtained showing proper (long-dashed line) and incorrect (short-dashed line) measurement of proximal aorta. Measurement obtained at widest point; (B) cartoon of correct measurement with anatomy labeled. LA = left atrium; LV = left ventricle; RV = right ventricle.

With respect to aortic dissection, dynamic images of the ascending aorta may detect an intimal flap seen as a hyperechoic linear structure within the aortic lumen that moves with each heartbeat. While the sensitivity of TTE for intimal flap is low, its visualization carries a high specificity.⁵⁰ Furthermore, a retrograde flap may cause aortic insufficiency and/or bleeding into the pericardium. The presence of aortic root dilation and/or intimal flap with a pericardial effusion should prompt immediate consultation to cardiothoracic surgery, with CTA if the patient will tolerate it.

Pearls and Pitfalls of Exit

A common error occurs when practitioners place their measurement cursors parallel to the edge of the US footprint rather than perpendicular to the long axis of the vessel. This results in oblique measurements that can overestimate the true aortic diameter (Figure 8).

While the “exit” of EP FOCUS reliably assesses the proximal aortic root, thoracic aortic dilatation or an intimal flap may occur distal to the aortic root. Assessment of other parts of the thoracic aorta may be enhanced by using a suprasternal notch window and by visualizing portions of the descending aorta, often seen in the far field on the PSLA view.

Mirroring and reverberation artifacts can mimic intimal flaps.⁵¹ To avoid errors in interpretation of intimal flaps, the examiner should interrogate the aorta from multiple angles and transducer locations, look for flap motion independent of surrounding structures, ensure that the structure is confined to the lumen, and use Doppler to demonstrate differential flow on either side of the flap.

Thoracic aortic dilatation is strongly associated with dissection; however, there can be aneurysm without dissection and dissection without aneurysm.⁴⁹ The presence of thoracic aortic dilatation should prompt greater suspicion of thoracic aortic disease, but its absence should not rule it out. If there is suspicion of disease, CTA remains the diagnostic test of choice, particularly if the patient is hemodynamically stable. Early EP FOCUS may prompt and expedite appropriate diagnostic testing and consultation when needed.⁵⁰

ENTRANCE

The fifth and final “E” in our protocol is for entrance to the heart or assessment of the IVC. The usefulness and reliability of IVC assessment and its correlation to central venous pressure, fluid status, and fluid responsiveness has been studied extensively over the past several years. While controversy exists over the correlation of IVC measurements to other quantitative measures of RA pressure, the qualitative assessment of the IVC may be clinically helpful, particularly if it is plethoric or non-collapsible or notably flat.⁵² IVC plethora is highly sensitive for tamponade as well as congestive heart failure.^{22,53} By contrast, a flat or highly collapsible IVC correlates well with acute blood loss and hypovolemia (Figure 9).^{54,55} While the majority of cardiac views should be performed first to provide context for IVC findings, early assessment of the IVC can help to quickly differentiate causes of shock, chest pain, and dyspnea.³

Techniques for Assessing Entrance

While the first 4Es can often be obtained from a parasternal view, assessment of the IVC requires a subcostal

or subxiphoid approach. The SXLA provides a longitudinal view of the IVC and has been shown to have the best inter-rater reliability.⁵⁶ The SX4C can also show the IVC in short axis, although craniocaudal movement of the IVC during respiration should be considered.⁵⁷

The IVC should be assessed in terms of overall size and collapsibility. Both size and collapsibility can also be measured quantitatively, although the significance of exact numbers is questionable. IVC diameter is typically measured at its largest diameter (end expiration) at about 2 cm distal to the junction of the IVC and RA or 1 cm distal to the where the hepatic veins join the IVC. An IVC diameter of ≥ 2 cm, especially with minimal or no collapsibility, is considered plethoric and correlates with increased RA pressure. An IVC of <1 cm, particularly with complete collapse, is considered flat and indicates low preload and potential fluid responsiveness.^{58,59} An IVC diameter between 1 and 2 cm is typically normal, but estimation of preload can be improved by including the degree of collapsibility, also known as the “caval index,” which ranges from 0 to 1. The percent collapsibility (caval index $\times 100$) is calculated as the difference between the inspiratory and expiratory diameters divided by the inspiratory diameter. A high caval index (high percent collapse) indicates lower preload.

Pearls and Pitfalls of Entrance

When in a long-axis plane, inadvertently sliding off the center of the vessel produces a cylinder tangent effect, which may underestimate the size of the IVC and overestimate its collapse. Obtaining short-axis and long-axis views of the IVC may help avoid this. The IVC also moves both anterolaterally and craniocaudally with inspiration, and this translocation should be taken into account during visualization or measurements.⁵⁷

Another pitfall is mistaking the descending aorta for the IVC when scanning in a long-axis plane. The IVC may appear to pulsate or vary with respiration; the aorta has a thicker wall and is positioned to the patient’s left. In a transverse or axial plane, the IVC should be seen on the left of the screen, to the patient’s right. Following the IVC proximally through the liver parenchyma should reveal the hepatic veins joining the IVC before it enters the RA, while the aorta will travel behind the heart. Therefore, an IVC in long axis will “curve up” on the left side of the screen making a “happy whale sign” whereas the aorta in long axis will “curve down” on the left side of the screen as seen in Figure 9.

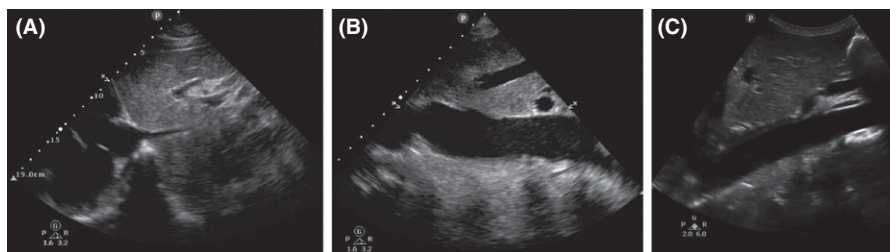


Figure 9. The entrance. Depiction of IVC: (A) flat IVC; (B) plethoric IVC; (C) aorta. The aorta may appear similar to the IVC. The sonographer can look for the IVC joining the RA or hepatic veins draining into the IVC to help distinguish between the structures. Additionally, the sonographer can identify the celiac trunk or superior mesenteric artery arising from the aorta to aid in identification. IVC = inferior vena cava.

The diameter of the IVC should be used in conjunction with the collapsibility to improve accuracy of preload estimation. For example, an IVC of 1.5 cm with a high caval index may be considered indicative of lower preload, while an IVC of 1.8 cm with a low caval index may indicate higher preload. In these latter situations, repeating IVC assessment after an intervention such as fluid administration may provide more valuable information.

CONCLUSIONS

The intent of this article is to codify elements of the cardiac US exam that we have found to be most relevant to patients presenting with acute or emergent complaints (Table 1). A recent international consensus statement defined FOCUS as being goal-directed, problem-oriented, limited in scope, simplified, time-sensitive and repeatable, qualitative and semiquantitative, performed at the point of care, and usually performed by clinicians.⁶ The 5Es described in this article meet all of these criteria. However, the international statement addressed the use of FOCUS in “all clinical settings” and included the assessment of chronic cardiac disease, as well as

gross valvular abnormalities and large intracardiac masses, without assessment of the thoracic aorta.

In our experience the 5Es encompass the cardiac US findings most applicable in patients who present emergently with hypotension, dyspnea, syncope, penetrating thoracic trauma, chest pain, or other acute complaints where diagnosis may be aided by visualization of the heart. While gross valvular abnormalities and intracardiac masses are important if they are seen, they are less common and less acute and tend to be less immediately deadly than acute thoracic aortic disease, which accounts for more than twice as many deaths as abdominal aortic aneurysm rupture.⁵¹

The 5Es are not meant to provide an absolute boundary for EP FOCUS, which will likely continue to evolve, but are intended to provide a framework for the acquisition and interpretation of the most relevant and applicable components of echocardiography in the emergent setting. We hope that adoption and subsequent application of the 5Es in EDs will help to standardize and effectively teach the echo findings that may allow EPs to save lives and expedite the care of patients with potentially life-threatening illness.

Table 1
Explanation of the 5Es

5 Es	Categorization of Findings	Additional Findings
1. Effusion	None (trace) Small (<1 cm) Moderate (1–2 cm) Large (>2 cm)	<ul style="list-style-type: none"> • Tamponade physiology <ul style="list-style-type: none"> ◦ RV free wall collapse in diastole (may use M-mode) ◦ RA collapse in ventricular systole ◦ 25% respirophasic variation in peak velocity using spectral Doppler ◦ Plethoric IVC
2. Ejection	Hyperdynamic (EF > 65%) Normal (EF 50%–65%) Moderately depressed (EF 30%–50%) Severely depressed (EF < 30%) No significant myocardial activity	<ul style="list-style-type: none"> • E-point septal separation < 7 mm is normal • LV diameter > 6 cm in diastole is abnormal • Wall motion abnormalities using “SALPI” mnemonic (septal, anterior, lateral, posterior, inferior) on short-axis view
3. Equality	Normal (RV:LV < 1) Enlarged (RV:LV > 1)	<ul style="list-style-type: none"> • Other signs of RV strain <ul style="list-style-type: none"> ◦ Septal bowing (D-shaped septum) ◦ McConnell’s sign ◦ Elevated RV pressure estimated using continuous wave Doppler with peak TR ◦ Tricuspid annular plane systolic excursion (TAPSE) < 16 mm • Beware chronic RV strain (COPD, pulmonary hypertension) <ul style="list-style-type: none"> ◦ RV free-wall thickness > 5 mm ◦ TR jet very elevated (>4 m/sec) ◦ Absent McConnell’s sign
4. Exit	Normal root (<4 cm) Borderline root (4–4.5 cm) TAA (>4.5 cm)	<ul style="list-style-type: none"> • Dissection flap • Aortic regurgitation using color Doppler • Pericardial effusion
5. Entrance	Flat IVC (<1 cm, > 75% collapse) Normal collapse Full IVC (>2 cm, < 25% collapse)	<ul style="list-style-type: none"> • Can use M-mode to calculate caval index: $\frac{\text{max IVC diameter} - \text{min IVC diameter}}{\text{max IVC diameter}}$ • Measurements within 1–2 cm of hepatic vein

COPD = chronic obstructive pulmonary disease; EF = ejection fraction; IVC = inferior vena cava; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; TAA = thoracic aortic aneurysm; TR = tricuspid regurgitation.

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References

1. Plummer D. Principles of emergency ultrasound and echocardiography. *Ann Emerg Med* 1989;18:1291–7.
2. Hauser AM. The emerging role of echocardiography in the emergency department. *Ann Emerg Med* 1989;18:1298–303.
3. Seif D, Perera P, Mailhot T, Riley D, Mandavia D. Bedside ultrasound in resuscitation and the rapid ultrasound in shock protocol. *Crit Care Res Pract* 2012;2012:503254.
4. Labovitz AJ, Noble VE, Bierig M, et al. Focused cardiac ultrasound in the emergent setting: a consensus statement of the American Society of Echocardiography and American College of Emergency Physicians. *J Am Soc Echocardiogr* 2010;23:1225–30.
5. Spencer KT, Kimura BJ, Korcarz CE, Pellikka PA, Rahko PS, Siegel RJ. Focused cardiac ultrasound: recommendations from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2013;26:567–81.
6. Via G, Hussain A, Wells M, et al. International evidence-based recommendations for focused cardiac ultrasound. *J Am Soc Echocardiogr* 2014;27(683):e1–33.
7. Taylor RA, Oliva I, Van Tonder R, Eleftheriades J, Dziura J, Moore CL. Point-of-care focused cardiac ultrasound for the assessment of thoracic aortic dimensions, dilation, and aneurysmal disease. *Acad Emerg Med* 2012;19:244–7.
8. Chisholm CB, Dodge WR, Balise RR, Williams SR, Gharabaghian L, Beraud AS. Focused cardiac ultrasound training: how much is enough? *J Emerg Med* 2013;1–5.
9. Moore CL. Current issues with emergency cardiac ultrasound probe and image conventions. *Acad Emerg Med* 2008;15:278–84.
10. Rozycki GS, Ochsner MG, Jaffin JH, Champion HR. Prospective evaluation of surgeons' use of ultrasound in the evaluation of trauma patients. *J Trauma* 1993;34:516–27.
11. Moss AJ, Bruhn F. The echocardiogram. An ultrasound technique for the detection of pericardial effusion. *N Engl J Med* 1966;274:380.
12. Spodick DH. Acute cardiac tamponade. *N Engl J Med* 2003;349:684–90.
13. Mandavia DP, Hoffner RJ, Mahaney K, Henderson SO. Bedside echocardiography by emergency physicians. *Ann Emerg Med* 2001;38:377–82.
14. Plummer D, Brunette D, Asinger R, Ruiz E. Emergency department echocardiography improves outcome in penetrating cardiac injury. *Ann Emerg Med* 1992;21:709–12.
15. Blaivas M. Incidence of pericardial effusion in patients presenting to the emergency department with unexplained dyspnea. *Acad Emerg Med* 2001;8:1143–6.
16. Jacob S, Sebastian JC, Cherian PK, Abraham A, John SK. Pericardial effusion impending tamponade: a look beyond Beck's triad. *Am J Emerg Med* 2009;27:216–9.
17. Mayosi BM, Burgess LJ, Doubell AF. Tuberculous pericarditis. *Circulation* 2005;112:3608–16.
18. Imazio M, Mayosi BM, Brucato A, Adler Y. Pericardial effusion triage. *Int J Cardiol* 2010;145:403–4.
19. Saito Y, Donohue A, Attai S, et al. The syndrome of cardiac tamponade with "small" pericardial effusion. *Echocardiography* 2008;25:321–7.
20. Nagdev A, Stone M. Point-of-care ultrasound evaluation of pericardial effusions: does this patient have cardiac tamponade? *Resuscitation* 2011;82:671–3.
21. Sagristà-Sauleda J, Mercé AS, Soler-Soler J. Diagnosis and management of pericardial effusion. *World J Cardiol* 2011;3:135–43.
22. Himelman RB, Kircher B, Rockey DC, Schiller NB. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiography sign of cardiac tamponade. *J Am Coll Cardiol* 1988;12:1470–7.
23. Burstow DJ, Oh JK, Bailey KR, Seward JB, Tajik AJ. Cardiac tamponade: characteristic Doppler observations. *Mayo Clin Proc* 1989;64:312–24.
24. Press GM, Miller S. Utility of the cardiac component of FAST in blunt trauma. *J Emerg Med* 2013;44:9–16.
25. Moore CL, Rose GA, Tayal VS, Sullivan DM, Arrowswood JA, Kline JA. Determination of left ventricular function by emergency physician echocardiography of hypotensive patients. *Acad Emerg Med* 2002;9:186–93.
26. Jones AE, Tayal VS, Sullivan DM, Kline JA. Randomized, controlled trial of immediate versus delayed goal-directed ultrasound to identify the cause of nontraumatic hypotension in emergency department patients. *Crit Care Med* 2004;32:1703–8.
27. Gudmundsson P, Rydberg E, Winter R, Willenheimer R. Visually estimated left ventricular ejection fraction by echocardiography is closely correlated with formal quantitative methods. *Int J Cardiol* 2005;101:209–12.
28. Randazzo MR. Accuracy of emergency physician assessment of left ventricular ejection fraction and central venous pressure using echocardiography. *Acad Emerg Med* 2003;10:973–7.
29. Blaivas M, Fox JC. Outcome in cardiac arrest patients found to have cardiac standstill on the bedside emergency department echocardiogram. *Acad Emerg Med* 2001;8:616–21.
30. Weekes A, Reddy A, Lewis M, Norton H. E-Point Septal Separation Compared to Fractional Shortening Measurements of Systolic Function in Emergency Department Patients Prospective Randomized Study. *J Ultrasound Med* 2012;1891–7.
31. McKaigney CJ, Krantz MJ, La Rocque CL, Hurst ND, Buchanan MS, Kendall JL. E-point septal separation: a bedside tool for emergency physician assessment of left ventricular ejection fraction. *Am J Emerg Med* 2014;32:493–7.
32. Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the cardiac imaging committee of the council on clinical cardiology of the American Heart Association. *Circulation* 2002;105:539–42.

33. Dresden S, Mitchell P, Rahimi L, et al. Right ventricular dilatation on bedside echocardiography performed by emergency physicians aids in the diagnosis of pulmonary embolism. *Ann Emerg Med* 2014;63:16–24.
34. Taylor RA, Moore CL. Accuracy of emergency physician-performed limited echocardiography for right ventricular strain. *Am J Emerg Med* 2014;32:371–4.
35. Jaff MR, McMurtry MS, Archer SL, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. *Circulation* 2011;123:1788–830.
36. Taylor RA, Davis J, Liu R, Gupta V, Dziura J, Moore CL. Point-of-care focused cardiac ultrasound for prediction of pulmonary embolism adverse outcomes. *J Emerg Med* 2013;45:392–9.
37. Bomann JS, Moore C. Emergency department echocardiogram of right ventricle thrombus and McConnell's sign in a patient with dyspnea [abstract]. *Acad Emerg Med* 2009;16:474.
38. Borloz MP, Frohna WJ, Phillips CA, Antonis MS. Emergency department focused bedside echocardiography in massive pulmonary embolism. *J Emerg Med* 2011;41:658–60.
39. Madan A, Schwartz C. Echocardiographic visualization of acute pulmonary embolus and thrombolysis in the ED. *Am J Emerg Med* 2004;22:294–300.
40. Matthews JC, McLaughlin V. Acute right ventricular failure in the setting of acute pulmonary embolism or chronic pulmonary hypertension: a detailed review of the pathophysiology, diagnosis, and management. *Curr Cardiol Rev* 2008;4:49–59.
41. Holley AB, Cheatham JG, Jackson JL, Moores LK, Villines TC. Novel quantitative echocardiographic parameters in acute PE. *J Thromb Thrombolysis* 2009;28:506–12.
42. Kopecna D, Briongos S, Castillo H, et al. Interobserver reliability of echocardiography for prognostication of normotensive patients with pulmonary embolism. *Cardiovasc Ultrasound* 2014;12:29.
43. Lobo JL, Holley A, Tapson V, et al. Prognostic significance of tricuspid annular displacement in normotensive patients with acute symptomatic pulmonary embolism. *J Thromb Haemost* 2014;12:1020–7.
44. Zanobetti M, Converti C, Conti A, et al. Prognostic value of emergency physician performed echocardiography in patients with acute pulmonary embolism. *West J Emerg Med* 2013;14:509–17.
45. Pruszczyk P, Goliszek S, Lichodziejewska B, et al. Prognostic value of echocardiography in normotensive patients with acute pulmonary embolism. *JACC Cardiovasc Imaging* 2014;7:553–60.
46. Casazza F, Bongarzone A, Capozzi A, Agostoni O. Regional right ventricular dysfunction in acute pulmonary embolism and right ventricular infarction. *Eur J Echocardiogr* 2005;6:11–4.
47. Haller EP, Nestler DM, Campbell RL, Bellamkonda VR. Point-of-care ultrasound findings of acute pulmonary embolism: McConnell sign in emergency medicine. *J Emerg Med* 2014;47:e19–21.
48. McConnell MV, Come PC, Solomon SD, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol* 1996;78:469–73.
49. Elefteriades JA, Farkas EA. Thoracic aortic aneurysm clinically pertinent controversies and uncertainties. *J Am Coll Cardiol* 2010;55:841–57.
50. Nazerian P, Vanni S, Castelli M, et al. Diagnostic performance of emergency transthoracic focus cardiac ultrasound in suspected acute type A aortic dissection. *Intern Emerg Med* 2014;9:665–70.
51. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2010;55:e27–129.
52. Brennan JM, Blair JE, Goonewardena S, et al. Reappraisal of the use of inferior vena cava for estimating right atrial pressure. *J Am Soc Echocardiogr* 2007;20:857–61.
53. Blehar DJ, Dickman E, Gaspari R. Identification of congestive heart failure via respiratory variation of inferior vena cava diameter. *Am J Emerg Med* 2009;27:71–5.
54. Lyon M, Blaivas M, Brannam L. Sonographic measurement of the inferior vena cava as a marker of blood loss. *Am J Emerg Med* 2005;23:45–50.
55. Sefidbakht S, Assadsangabi R, Abbasi HR, Nabavizadeh A. Sonographic measurement of the inferior vena cava as a predictor of shock in trauma patients. *Emerg Radiol* 2007;14:181–5.
56. Saul T, Lewiss RE, Langsfeld A, Radeos MS, Del Rios M. Inter-rater reliability of sonographic measurements of the inferior vena cava. *J Emerg Med* 2012;42:600–5.
57. Blehar DJ, Resop D, Chin B, Dayno M, Gaspari R. Inferior vena cava displacement during respirophasic ultrasound imaging. *Crit Ultrasound J* 2012;4:18.
58. Weekes AJ, Tassone HM, Babcock A, et al. Comparison of serial qualitative and quantitative assessments of caval index and left ventricular systolic function during early fluid resuscitation of hypotensive emergency department patients. *Acad Emerg Med* 2011;18:912–21.
59. Nagdev AD, Merchant RC, Tirado-Gonzalez A, Sisson CA, Murphy MC. Emergency department bedside ultrasonographic measurement of the caval index for noninvasive determination of low central venous pressure. *Ann Emerg Med* 2010;55:290–5.

Supporting Information

The following supporting information is available in the online version of this paper:

Video Clip S1. First E: effusion.

Video Clip S2. Second E: ejection fraction.

Video Clip S3. Third E: equality.

Video Clip S4. Fourth E: exit.

Video Clip S5. Fifth E: entrance.