

# Emergency Echocardiography

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## KEYWORDS

- Emergency echocardiography • Cardiac ultrasound
- Emergency medicine • Point-of-care cardiac ultrasound
- Focused cardiac ultrasound

## CASE 1

Your next patient presents with a chief complaint of feeling “weak and dizzy.” She has had gradual worsening shortness of breath for 2 weeks, a slight cough that is worse at night, and mild leg swelling. She denies any chest pain or fever. Her blood pressure is 88/58 mm Hg; heart rate is 120 beats per minute with a regular rhythm; respiratory rate is 28 breaths per minute with an oxygen saturation of 98% on room air; and her temperature is 99.3°F (37.4°C). Her physical examination is remarkable for distant heart sounds, bilateral rales at the lung bases, and moderate neck vein distention. Her abdomen is nontender and her legs are slightly edematous bilaterally. While the nurse inserts an intravenous line and draws tubes of blood, you bring in the ultrasound machine for a closer look.

## SCOPE

The practice of emergency medicine has evolved to now include competence in emergency ultrasound as a basic training requirement of emergency medicine residency graduates. Over the past 10 years, advancements have been made in emergency ultrasound such that the use of ultrasound technology has now become integral to the daily practice of emergency medicine in the community as well as academic institutions. Emergency echocardiography (also referred to as “bedside cardiac ultrasound,” “goal-directed cardiac ultrasound,” “point-of-care cardiac ultrasound,” “focused cardiac ultrasound” [FOCUS], or “bedside echo”) is a well-established core application of emergency ultrasound. Emergency echocardiography that is

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performed and interpreted by emergency physicians is supported by the American College of Emergency Physicians (ACEP), the Society for Academic Emergency Medicine (SAEM), and the American Society of Echocardiography (ASE).<sup>1,2,3</sup> This article discusses the current indications, goals, and limitations of emergency transthoracic echocardiography. Training and quality assurance issues related to emergency echocardiography are not discussed in detail. For the majority of emergency physicians using emergency echocardiography, the main scope of practice is to evaluate for the presence or absence of a pericardial effusion, provide a visual estimation (“gestalt”) of overall heart function and, to some extent, identify right heart strain and evaluate the inferior vena cava. Emergency ultrasound education is a requirement in the emergency medicine residency curriculum. Out of an increased presence of emergency ultrasound fellowships and the spread of emergency ultrasound education and programs to community emergency medicine practices, there is an emerging cadre of recently graduated and practicing emergency medicine and critical care physicians with an increased interest, level of use, and expertise in emergency echocardiography.<sup>3,4,5</sup>

Most of the discussion is centered on the uses, benefits, and limitations of clinically indicated emergency echocardiography as consulted by, if not performed by, the emergency physician.

### **EMERGENCY ECHOCARDIOGRAPHY IS GOAL-DIRECTED**

Comprehensive echocardiography is traditionally performed by a certified echocardiographer in an echocardiography laboratory, and interpreted by a cardiologist. By contrast, emergency echocardiography attempts to answer a focused clinical question at the bedside in a time-sensitive manner. It is usually performed and interpreted by the treating physician. Emergency echocardiography is often indicated in the case of the patient who is overtly critically ill or in extremis. However, it can also help to rapidly unmask ominous underlying cardiac pathology in patients with otherwise deceptively mild symptoms. Information obtained can help to stratify cardiac function, identify structural abnormalities, or narrow the differential diagnosis of a patient presenting in shock. Goal-directed echocardiography, when immediately available to the treating physician, is a rapid, repeatable, and accurate test for diagnosing cardiac conditions critical to the care of the emergency department (ED) patient.

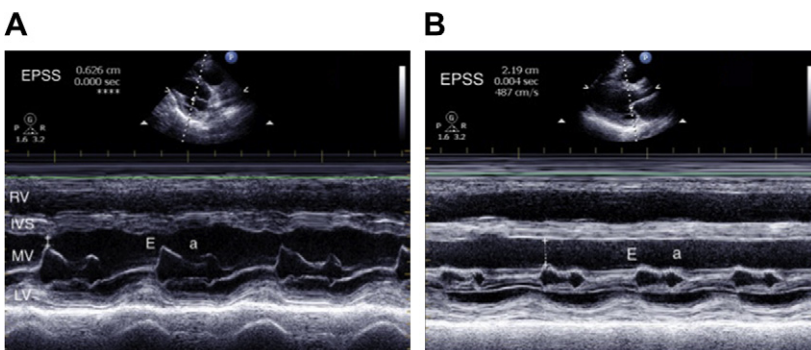
Echocardiography can be incorporated into the diagnostic and resuscitative phases of clinical care, as well as for safety in procedural guidance. The American College of Cardiology/American Heart Association Task Force on Practice Guidelines for the Clinical Application of Echocardiography includes common high-acuity situations seen daily in EDs. These indications include, but are not limited to, unexplained hypotension, dyspnea, signs suggesting the presence of elevated central venous pressure when cardiac causes are possible, suspected pericardial effusion, identification of tamponade physiology, and assessment of the size and function of the left ventricle when there is a suspicion of cardiomyopathy or cardiac failure.<sup>6,7</sup>

### **MODALITIES USED**

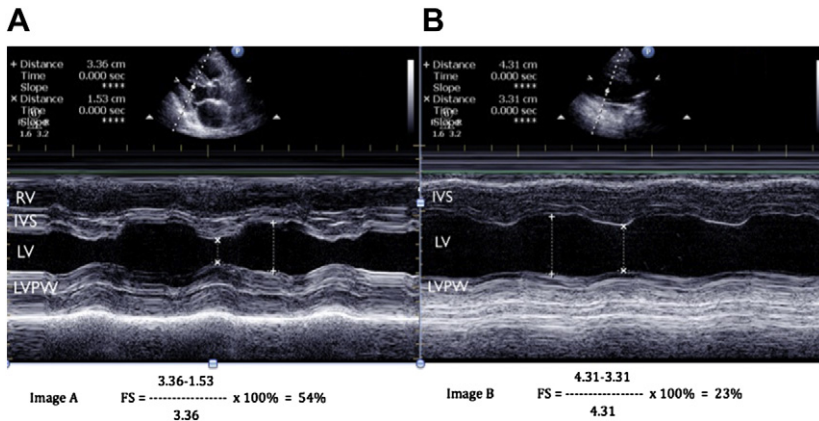
Multiple modalities are used in combination to evaluate different areas and different functions of the heart. The 3 most important that are performed by emergency physicians are B-mode, M-mode, and Doppler. Bedside comprehensive echocardiography is requested occasionally to complement and address suspicions raised by goal-directed/emergency echocardiography. Additional information obtained may include quantitative analyses of valve function, detailed calculations of ejection fraction (EF),

and tissue Doppler and diastolic function evaluations. More advanced modalities are beyond the scope of this article.

- *B-mode*. B-mode is also known as “brightness mode” or 2-dimensional (2D) ultrasound. Cardiac structures are shown in 2 dimensions and appear in shades of gray on the ultrasound monitor. It is ideal for real-time viewing of cardiac structures and their movements, and for the interpretation of cardiac function. Typically the real-time images are shown at a rate of 30 frames per second on the monitor. B-mode is the most common modality used in echocardiography, and is often used for the capture of still or video images.
- *M-mode*. This technology, also known as “motion mode,” provides the opportunity to view and trace the motion and dimensions of heart structures with time. The M line, a very thin and linear area of ultrasound evaluation, allows faster image processing than the 30° sector used in 2D imaging, and can be precisely positioned through structures of interest. The rate of image capture with M-mode is 1800 frames per second (much faster than the human eye), which can discern variations in valve position, wall thickness, or chamber size over time. See **Figs. 1** and **2** for examples of M-mode tracings.
- *Applied Doppler technology*. “Doppler,” as it is often described, actually includes several different subcategories including color Doppler, pulsed-wave Doppler, and continuous-wave Doppler. Each modality uses the same technology to measure heart parameters in a different way. The concept of Doppler is based on the ability of the ultrasound machine to measure the direction and magnitude of a frequency shift in cardiac muscle or blood flow relative to the transducer. This information is then translated into different colors or a graphic display of velocity magnitude on the ultrasound screen, depending on the subcategory being used. Color Doppler is most frequently used in emergency medicine to detect the presence of blood flow within a structure and to distinguish adjacent blood vessels (by showing different blood flow directions). Pulsed-wave and continuous-wave Doppler are used by emergency physicians with additional training, and their use is gradually increasing.



**Fig. 1.** E-point septal separation (EPSS). M-mode technology is used to trace the movement of the cardiac structures over time to more accurately estimate how closely the anterior leaflet of the mitral valve moves to the septum during left ventricular (LV) filling. The E wave (E) indicates the motion of the mitral valve toward the septum during LV filling. The A wave (a) indicates the motion of the mitral valve caused by atrial contraction. (A) An EPSS of 0.626 cm, indicating good systolic function. (B) The patient in this case. Her EPSS is calculated at 2.19 cm, indicating poor systolic function. IVS, interventricular septum; RV, right ventricle.



**Fig. 2.** Fractional shortening. M-mode evaluation of the LV chamber in parasternal long-axis (PSLA) view. (A) Evidence of wall thickening and endocardial excursion consistent with good systolic function. (B) The patient in this case. Both endocardial excursion and myocardial wall thickening are reduced, and fractional shortening is calculated at 23%. A normal value for fractional shortening is between 30% and 45%. IVS, interventricular septum; LV, left ventricle; LVPW, left ventricular posterior wall; RV, right ventricle.

## WINDOWS USED

The main cardiac views obtained in transthoracic emergency echocardiography include the subcostal (subxiphoid), left parasternal (long and short axes), and apical windows. Subtle probe manipulations, such as angulation and rotation, can provide modified views of the heart chambers at different levels and perspectives. These different views may be used for identification of different wall segments, chambers, or valves, and may offer opportunities for Doppler interrogation and other physical or hemodynamic measurements. Each view may have technical challenges or feasibility issues in some patients. The indications and views listed in [Table 1](#) are discussed throughout this article.

## TECHNICAL LIMITATIONS

Common technical challenges to acquiring adequate images in transthoracic echocardiography may include obesity, abdominal distension, a muscular chest wall, rapid breathing, air interference (chronic obstructive lung disease/hyperinflation/mechanical ventilation), small intercostal windows, chest wall tenderness, the presence of thoracic devices or ongoing thoracic procedures, subcutaneous emphysema, and pneumopericardium. Each window has its own diagnostic limitations and advantages. For example, the parasternal long-axis (PSLA) view is used to evaluate the thoracic aorta, but is limited to its very proximal section (the aortic root) and a small portion of the descending aorta as seen in cross section of the same view. Additional views may be used, such as the suprasternal view of the aortic arch or a modified apical 2-chamber view, depending on the skill of the operator and clinical information sought.

## RETURN TO CASE 1

Your patient continues to be short of breath and tachypneic. Her blood pressure has decreased slightly, now at 79/44 mm Hg. Chest radiography is concerning for

<b>Clinical Indication</b>	<b>Recommended Cardiac Window</b>	<b>Comments</b>
Effusion detection	Subcostal PSLA	Often quickest and easiest to obtain Can differentiate pericardial from pleural effusion
RV evaluation	PSLA PSSA Apical 4-chamber	Evaluation for septal dyskinesis Look for "D sign" Comparison of chamber size
Global LV function <sup>a</sup>	PSLA	Ease of evaluation of mitral valve motion. Can also perform M-mode
Valve interrogation	Apical views	Allows for 0°–20° Doppler angle. Color or pulsed-wave modes
Volume assessment	Subcostal 4-chamber and IVC view (short or long)	Evaluation for respiratory variation of IVC and estimation of left ventricular end-diastolic area/volume
Shock/hypotension	Subcostal or PSLA	Use in combination with evaluation of aorta, pleura, and abdomen for free fluid to determine cause of shock
Proximal thoracic aortic disease	PSLA, modified apical 2, suprasternal view	Outflow >4 cm suggestive of proximal aortic disease (aneurysm) or intimal flap (dissection). Limited view of descending thoracic aorta. Suprasternal view may be used for evaluation of aortic arch <i>Note: TEE or CTA more sensitive and specific</i>
Cardiac arrest	Subcostal	Quick (<10 s) evaluation for presence of effusion, right ventricular strain, asystole

*Abbreviations:* CTA, computed tomographic angiography; IVC, inferior vena cava; LV, left ventricular; PSLA, parasternal long-axis view; PSSA, parasternal short-axis view; RV, right ventricular; TEE, transesophageal echocardiography.

<sup>a</sup> Detection of regional wall motion abnormalities requires multiple views.

pulmonary vascular congestion versus pneumonia. B-type natriuretic peptide is mildly elevated at 345 pg/mL. You suspect congestive heart failure, and turn to your bedside echocardiography skills for assistance.

### **Overall Cardiac Function**

#### **Overview**

Cardiac function can be evaluated by multiple different modalities in the intensive care unit and the ED. Invasively obtained hemodynamic profiles are better suited to the intensive care setting, are time consuming, and are no more accurate than echocardiographic data.<sup>8</sup> Echocardiography is more convenient and provides a visual estimate of global cardiac function at the bedside. Features used in the visual estimation of left ventricular (LV) function are the change in chamber size, motion of endocardial surfaces or valves, and thickening of the myocardium. The normal left ventricle of the heart squeezes with a complex twisting mechanism, making precise measurements difficult. Geometric approximations and assumptions are made regarding the

shape and motion of the heart to assess ventricular function, which can contribute to variability in the final calculations of EF, stroke volume, and cardiac output. There are several ways to evaluate cardiac function with ultrasound, including quantitative and semiquantitative measurements, and visual estimates of systolic function. Other methods used in comprehensive echocardiography may include regional systolic wall motion grading, the use of tissue Doppler, and a more complex use of pulsed-wave Doppler to determine the Tei index of myocardial performance. The use of each method depends on the clinical scenario and skill of the sonographer. Multiple windows and modalities can be used to evaluate global systolic function, but in emergency echocardiography this may not be feasible.

### **Quantitative methods (ejection fraction)**

LV systolic function is most frequently expressed as the EF. The EF is the percent volume change in ventricle size during systole, and is described by the equation  $EF = \text{stroke volume} / \text{end-diastolic volume}$ . Using a geometric model of a bullet shape, the endocardial surfaces of the left ventricle are traced from the base to the apex in both the apical 4-chamber view and the apical 2-chamber view to provide dimensions in 3 different planes. Diastolic volumetric measurements are subtracted from systolic measurements to determine the stroke volume. The stroke volume divided by the end-diastolic volume determines the EF. Many current ultrasound machines have packages available to assist with this calculation. For most clinical scenarios, though, an approximation is adequate and a formal calculation of EF is not necessary. In addition, it may be time consuming and beyond the scope of emergency medicine practice.

### **Semiquantitative methods**

Other methods that closely approximate visual estimates of systolic function include fractional shortening (FS) and E-point septal separation (EPSS). Measurements of the changes in ventricular size are made in two planes (fractional area shortening) or in one plane (FS). The advantage of the semiquantitative approach is that measurements are easier to obtain than for EF while limiting the inherent human error or bias present with pure visual estimates.

- **EPSS.** An important marker of cardiac function is movement of the anterior leaflet of the mitral valve toward the interventricular septum during LV filling. In a heart with normal function, this leaflet should almost contact the interventricular septum. EPSS merely seeks to quantify how close the leaflet comes to the septum using M-mode evaluation of the mitral valve in the PSLA view. The E point is the top of the first waveform seen during diastole. The distance between the E point and the septum wall is called the EPSS. See **Fig. 1** for an example. Measurements greater than 0.6 to 1 cm for EPSS generally indicate severe systolic dysfunction ( $EF < 30\%$ ). However, it should be noted that isolated or co-existing aortic insufficiency, dilated chambers, or mitral stenosis also increase the EPSS.<sup>9,10</sup> In one study, significant correlations between EPSS measurements and angiographic EF measurements were found except in cases with known mitral regurgitation and atrial fibrillation.<sup>11</sup>
- **Fractional shortening.** This method measures the fraction of a single LV diastolic dimension that is lost during systole. This fraction can be measured in various anatomic planes but with M-mode the parasternal views are most often used. Rather than using multiple different views and tracing the chambers as done with volumetric EF calculation, one simple view (typically the basal aspect of the LV diameter) and 2 measurements are all that are needed (see **Fig. 2**). The result of the comparison between systole and diastole dimensions is described

as a fraction or a percent change. A normal value for FS is approximately 30% to 45%. Fractional area shortening involves the loss in ventricular area (2 dimensions) between diastole and systole.

### **Visual estimates**

Fortunately, the visual estimation of LV systolic function agrees with semiquantitative and quantitative measurements, allowing for accurate real-time assessments. Global systolic function assessments by emergency medicine physicians with focused training have good agreement with interpretations of the same echocardiography studies done by cardiologists.<sup>12,13</sup> Studies in adults and children have also shown good correlation between bedside estimation of LV function and semiquantitative measurements, with good interrater (blinded) agreement in visual LV function estimation when evaluation guidelines are followed.<sup>11,14</sup>

Visual estimates of systolic function are typically described as normal, moderately depressed, or poor. Emergency physicians tend to be relatively accurate at identifying normal or severely depressed cardiac function. However, the determination of moderately depressed cardiac function can be more difficult and there is a wider variation in this category. The 3 parameters that are most closely examined are mitral valve motion, myocardial wall thickening, and endocardial excursion. Each parameter is described here. The clinical interpretation depends on the clinical scenario, and a visual estimate of systolic function is often sufficient for the decision at hand. Patients may have comorbid cardiac conditions or acute cardiac dysfunction that may affect their response to therapeutic interventions such as acute volume loading.

- **Mitral valve motion.** The PSLA view is most often used for this indication. During diastolic filling, the anterior leaflet of the mitral valve moves upwards toward the interventricular septum. In a patient with normal LV function, the mitral valve should nearly contact the septum. On the other hand, patients with severely depressed LV function may have only a slight flicker of the mitral valve. Patients with moderately depressed function fall somewhere in between.
- **Myocardial wall thickening.** In a heart with normal cardiac function, the ventricular walls thicken significantly during systole and relax during diastole, as shown in [Video 1](#). Patients with moderate and severely depressed dysfunction will show a decrease in this parameter. A visual gestalt is needed to estimate this parameter after viewing numerous echocardiograms of patients with varying degrees of cardiac function.
- **Endocardial excursion.** During systole the myocardium contracts, bringing the endocardial borders of the heart closer together as blood is ejected through the LV outflow tract. In a normal heart, the endocardial borders should come relatively close together, although no exact measurement is defined. A patient with moderately depressed or severely depressed cardiac function will have an echocardiogram that shows limited movement of the endocardial borders toward each other during systole. The ability to estimate this parameter is gained over time after viewing many echocardiograms of hearts with varying degrees of LV function.

A visual estimation scale of LV function that factored in the aforementioned parameters was shown to correlate well with bedside measurements of LV function and provided moderate interrater agreement.<sup>11</sup> See [Videos 1–4](#) for examples.

One of the most valuable features of bedside global cardiac assessments is in ruling out severe systolic dysfunction. Cardiogenic shock carries a high morbidity and mortality, and early detection is crucial.<sup>15</sup> For example, the finding of severe systolic

dysfunction in a patient with dyspnea and rales may be an indication for aggressive inotropic support. In another patient with hypotension of uncertain etiology, discovering normal to hyperdynamic systolic function may make it unlikely that the patient will require inotropic support or suffer from fluid toxicity in response to acute volume loading. Such a patient may instead tolerate and respond favorably to acute volume loading. Other causes of cardiogenic shock aside from severe systolic dysfunction include tamponade, severe valvular dysfunction, and ventricular septal wall rupture.

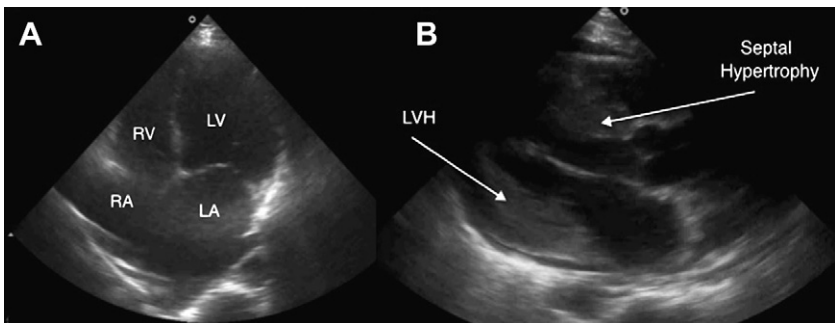
### Case 1 Conclusion

A bedside echocardiogram shows evidence of severely depressed cardiac function by visual gestalt and an EPSS is calculated at 1.4 cm. The patient is admitted for diuresis and improves symptomatically. A comprehensive echocardiogram obtained the next day shows an EF of 15% to 20%.

### Special Circumstances

While emergency physicians can accurately discriminate between depressed and normal LV systolic function by visual estimation, there are several additional circumstances that should be noted.

- **Cardiomyopathies.** During evaluation for systolic function, evidence of cardiomyopathies can often be found. The two most common are shown here. Dilated cardiomyopathy appears as thin-walled, dilated chambers with poor systolic function. Hypertrophic cardiomyopathy appears as thick ventricle walls (>15 mm) with impaired relaxation (diastolic dysfunction), and usually intact systolic function unless it is at the end stage. A detailed discussion of the cardiomyopathies is beyond the scope of this article, but is mentioned in the event it is seen, so that the sonographer can become familiar with their appearance and arrange appropriate subspecialty consultation and follow-up if found. See [Fig. 3](#), and [Videos 5](#) and [6](#) for examples of dilated and hypertrophic cardiomyopathy.
- **Diastolic dysfunction.** it is important to remember that diastolic function is usually evaluated by comprehensive echocardiography, and a detailed discussion is beyond the scope of this article. Diastolic dysfunction can be evaluated at the bedside using M-mode of the mitral valve leaflet movement or Doppler



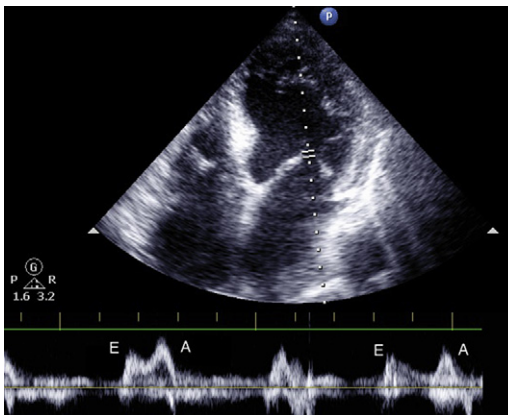
**Fig. 3.** Cardiomyopathies. Compare and contrast the thin-walled, dilated chambers of a patient with dilated cardiomyopathy (A; apical 4-chamber view) with the thick-walled chambers of the patient with hypertrophic cardiomyopathy (B; PSLA view). See [Videos 5](#) and [6](#). LA, left atrium; LV, left ventricle; LVH, left ventricular hypertrophy; RA, right atrium; RV, right ventricle.

interrogation of valvular flow. Normal filling of the left ventricle (diastole) begins with, and is predominantly achieved by, isovolemic relaxation of the left ventricle. The rest of the LV filling is supplemented by atrial contraction. The anterior leaflet opening (using M-mode) or blood flow velocity (using pulsed-wave Doppler) during isovolemic relaxation phase of diastole is depicted as the E wave. The second and smaller A wave represents movement/blood flow due to the left atrial contraction phase of diastole. A reduction or reversal of the normal E/A wave ratio indicates impaired relaxation of the left ventricle. It should be noted that up to a third of cases of heart failure is caused by diastolic dysfunction, and that a visual estimation of normal systolic function does not rule out the possible presence of diastolic dysfunction or heart failure. See [Fig. 4](#) and [Video 7](#) for an example of pulsed-wave Doppler imaging in a patient with diastolic dysfunction.

- **Hyperdynamic heart.** Hyperdynamic heart refers to the near obliteration of the ventricular cavity during systole, and may represent an elevated EF but not necessarily an increased stroke volume. This condition does not necessarily refer to the rate of contractions but rather to its vigor. Hyperdynamic heart activity can be found in hyperthyroidism or adrenergic surge states (after epinephrine/dopamine administration or certain drug overdoses). It may also be a compensatory response to poor oxygen-carrying abilities such as in low-volume states or anemia. A hyperdynamic heart effectively rules out systolic dysfunction as the cause of a patient's symptoms. A well-filled hyperdynamic heart can be found in distributive shock states such as sepsis or anaphylaxis. A barely filled hyperdynamic heart suggests hypovolemia or hemorrhage, and a search for hemorrhage or vascular emergency should be pursued based on the clinical context. See [Video 7](#) for an example of a hyperdynamic heart in the PSLA view.

## CASE 2

Your next patient has persistent hiccups for 4 days. He has a history of chronic renal failure and has received all of his hemodialysis treatments as scheduled this week. He endorses generalized fatigue but denies chest pain, vomiting, or abnormal colored



**Fig. 4.** Diastolic dysfunction. A pulsed-wave Doppler apical 4-chamber view in a patient with impaired cardiac relaxation and evidence of diastolic dysfunction. Note the reversal of the E/A wave ratio, indicating that there is a greater contribution to LV filling from atrial contraction due to the increased resistance to passive flow into the LV during diastole. Refer also to [Fig. 13](#) for diastolic dysfunction.

stool. On examination, he has stable vital signs and an unremarkable cardiovascular examination with the exception of mild jugular venous distension. He has clear lungs bilaterally and a nontender abdomen. You order a chest radiograph and note mild pulmonary vascular congestion, no peridiaphragmatic lesions, and cardiomegaly that is unchanged compared with his previous chest radiographs. His electrocardiogram (ECG) shows low-voltage QRS complexes but no PR or ST segment abnormalities. Due to suspicion of pericardial effusion, you proceed with bedside cardiac ultrasound and it confirms the diagnosis. Next, you carefully look at his heart chambers.

### ***Pericardial Effusion Detection***

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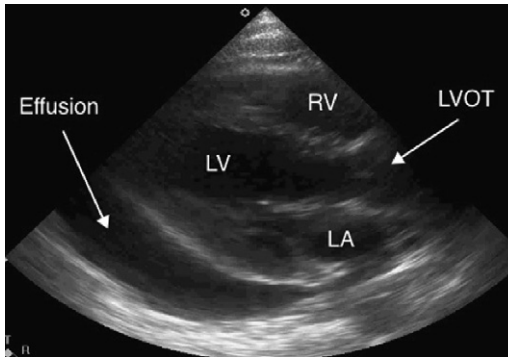
#### ***Overview***

The normal pericardial space usually contains a small volume (5–10 mL) of pericardial fluid, which functions as a lubricant between the visceral and parietal pericardium. The pericardial space of the normal adult heart can hold up to 50 mL of pericardial fluid before clinically significant elevations in intrapericardial pressure occur. Small accumulations may be barely discernible without the use of multiple windows.

- ***Clinical clues.*** The spectrum of symptoms and signs is variable and can include dyspnea, chest pain, and/or tachycardia. However, there are other vague symptoms such as nausea and singultus that may indicate the presence of an underlying effusion. The emergency physician must therefore be vigilant about comorbid conditions that are typically associated with pericardial effusion accumulations. A high index of suspicion will lower the threshold for the evaluation and detection of pericardial effusion.
- ***Sonographic appearance.*** Pericardial effusions are usually echolucent (black) and contrast sharply with the echogenicity of fibrous pericardium and myocardium. Some pericardial accumulations are more challenging to discern, as they may have a heterogeneous gray appearance if composed of clotting blood or other debris. Benign epicardial fat is usually located anteriorly, and can be mistaken for a small pericardial effusion.
- ***Sonographic views.*** In a trauma setting, the subxiphoid view is commonly used and is often the easiest view to obtain. However, the PSLA view offers the advantage in more accurately differentiating pericardial effusion from pleural effusion, and can help to further evaluate the size of the effusion and its hemodynamic effects. The apical 4-chamber view may be used as well, especially when considering ultrasound-guided pericardiocentesis. See **Figs. 5** and **6**, and **Videos 8** and **9** for parasternal views of a moderate-sized pericardial effusion.
- ***Size.*** A pericardial effusion is usually quantified as small, moderate, or large. Effusion volume size can be estimated as being small if the distance between the epicardium to pericardium is less than 0.5 cm; moderate if 0.5 to 2 cm, and large if greater than 2 cm. By obtaining multiple views of the heart, one can increase confidence in ruling out a small or loculated effusion. Small effusions are usually confined to the posterior space. However, moderate to large effusions tend to envelope the heart and have a greater width. These effusions may be confirmed using a single cardiac window. Larger effusions increase the likelihood of acute tamponade physiology and need for emergent pericardial drainage. **Fig. 7A** and **Video 10** show pericardial effusions of 3 different sizes from a subxiphoid view.

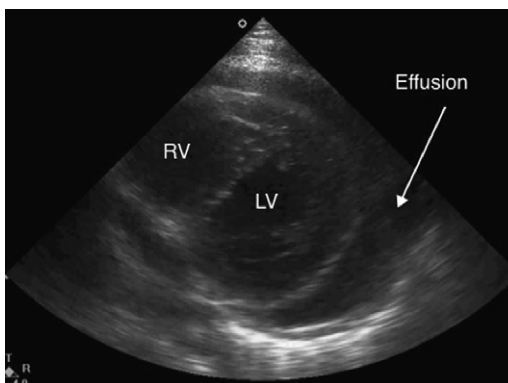
#### ***Etiology***

There are multiple causes of pericardial fluid accumulations, which can roughly be divided into traumatic and nontraumatic mechanisms.

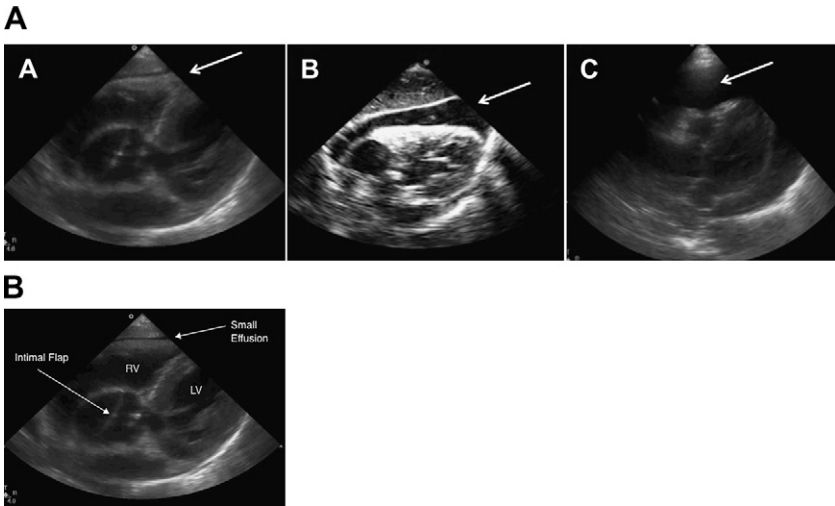


**Fig. 5.** Pericardial effusion (PSLA view), showing moderate to large pericardial effusion without evidence of cardiac tamponade. This particular patient had a history of end-stage renal disease. Despite the large size, no significant hemodynamic effects are seen, due to its chronicity. See [Video 8](#). LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract; RV, right ventricle.

- **Trauma.** Cardiac ultrasound is part of the Focused Assessment with Sonography in Trauma (FAST). Examination of cardiac windows is especially important in cases of penetrating injuries near the heart or in truncal injuries where the trajectory of a bullet is uncertain. With blunt trauma, in addition to pericardial effusion detection, it is also advised to examine for decreased cardiac activity caused by myocardial contusion.<sup>1</sup> In cases of blunt trauma where the presence of a pericardial effusion may be an incidental but preexisting finding, an expeditious query of previous records or comparison of previous imaging may be required. In these cases, a pericardial effusion is considered a false positive if it is erroneously considered a consequence of acute traumatic injury. Conversely, penetrating injuries to the pericardium and heart can lead to spillage of blood and negligible pericardial accumulations. In such a case, the nonvisualization of pericardial fluid during FAST should be reported as such and not be considered a definitive



**Fig. 6.** Pericardial effusion. This parasternal short-axis (PSSA) view shows a moderate to large pericardial effusion. Note that there is no evidence of right ventricular collapse or developing cardiac tamponade. See [Video 9](#). LV, left ventricle; RV, right ventricle.



**Fig. 7.** (A) Pericardial effusion size comparison (arrows). (A) A small anterior pericardial effusion. (B) An example of a moderate-sized effusion. (C) Presence of a large effusion. All views are subcostal. Note that a small anterior pericardial effusion is sometimes confused with an epicardial fat pad. (B) Aortic dissection with small anterior effusion. This subcostal view is an enlargement of image A in the previous figure. It shows a small anterior effusion from an ascending aortic dissection. The intimal flap of the dissection is seen here as well. Although the effusion is small, the mechanism suggests potential for rapid accumulation and development of cardiac tamponade. See [Video 10](#).

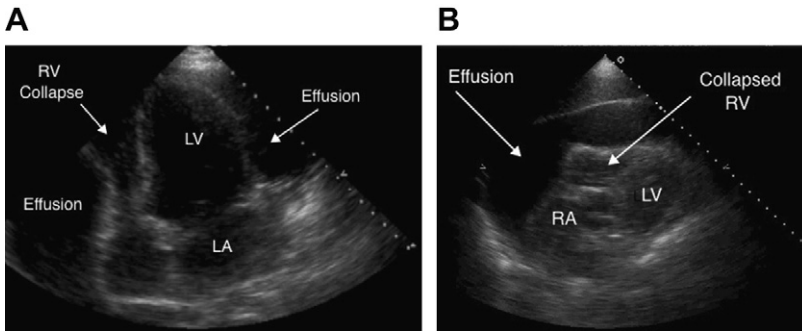
“ruling out” of cardiac injury. In the penetrating trauma setting, smaller fluid collections are more likely to provoke tamponade physiology and hemodynamic instability. A hypovolemic patient will have lower right heart pressures and, thus, cardiac tamponade physiology can occur with much lower pericardial pressures resulting from smaller effusions.

- **Nontrauma.** Many possible causes are appropriate to consider in the absence of trauma and may include inflammatory, infectious, malignant, or autoimmune conditions as well as aortic dissection. Pericardial effusions are also commonly seen in patients with renal failure, lupus, cancer, acquired immunodeficiency syndrome, and pregnancy. Transudative effusions tend to appear similar to traumatic effusions (echolucent or black), whereas exudative or malignant effusions can vary widely in their appearance.

### **Tamponade physiology**

The presence of a pericardial fluid accumulation is considered a necessary but not sufficient condition for the clinical diagnosis of tamponade. For a critically ill patient with a pericardial effusion, it is essential to be aware of the clinical factors affecting the development of cardiac tamponade.

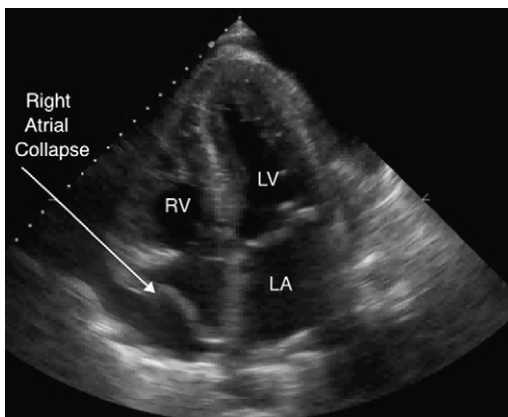
- **Identification.** Tamponade physiology is most commonly identified by decreased right ventricular (RV) size with early diastolic collapse and late expansion (specific not sensitive), as seen in [Fig. 8A](#) and B, and [Videos 11](#) and [12](#). This condition is a result of an elevation in intrapericardial pressure limiting RV free wall expansion and thus compromising LV filling and cardiac output. Other features suggestive of tamponade physiology include right atrial collapse at early diastole, as seen in [Fig. 9](#) and [Video 13](#) (most sensitive feature). This finding becomes more specific



**Fig. 8.** (A) Apical 4-chamber pericardial tamponade. This apical 4-chamber view shows a moderate-sized pericardial effusion with collapse of the right ventricle (RV), indicating high intrapericardial pressures and subsequent cardiac tamponade. See [Video 11](#). (B) Subcostal view of pericardial tamponade. A large pericardial effusion is seen with compression of all heart chambers, including the RV. See [Video 12](#). LA, left atrium; LV, left ventricle; RA, right atrium.

when right atrial diastolic collapse persists.<sup>16</sup> Of note, right atrial collapse may also be found in the presence of a pleural effusion or hypovolemia. Finally, tamponade physiology should be suspected when there is also minimal to absent respiratory diameter variation of the inferior vena cava (IVC). Technical limitations to tamponade detection include the presence of tachycardia, tachypnea, or challenging windows due to body habitus, making it difficult to confidently distinguish diastolic and systolic phases. If the sonographer is uncertain, M-mode technology may be used to evaluate for subtle posterior movement of the RV free wall during diastole in the PSLA view.

- **Rate of accumulation.** The pericardium is mostly fibrous and has a limited degree of elasticity. Gradual increases in pericardial fluid volume and intrapericardial



**Fig. 9.** Pericardial effusion with right atrial collapse. This apical 4-chamber view shows a moderate-sized effusion with evidence of increasing intrapericardial pressures causing collapse of the right atrium. The right ventricle (RV) size is normal because the right ventricular diastolic pressure is greater than the intrapericardial pressure. See [Video 13](#). LA, left atrium; LV, left ventricle.

pressure can be tolerated until the relatively low pressure of the thin right heart walls is exceeded. A pericardial effusion can accumulate insidiously or suddenly, depending on the clinical context. As a general rule, rapidly accumulating effusions tend to have more notable physiologic effects. For example, the chronic effusion of a patient with renal failure may not affect hemodynamic parameters until it is quite large. By contrast, a small traumatic pericardial bloody effusion may quickly lead to hemodynamic collapse in an otherwise healthy patient, due to its rapid rate of accumulation.

- *Clinical decision making.* In the unstable patient (eg, cardiac arrest, chest pain, or shock), when a significant pericardial effusion is detected then clinical tamponade should be considered the likely cause of the patient's instability. If tamponade physiology is still uncertain or suspected and the patient is quasi-stable, emergency comprehensive echocardiography and cardiology consultation are suggested. Comprehensive studies usually perform contemporaneous electrocardiographic monitoring to definitively determine systolic and diastolic phases. Another sign of developing tamponade is paradoxical septal wall motion. Detection of tamponade may also require the use of Doppler interrogation to detect increased tricuspid flow velocity or decreased mitral forward flow velocity with inspiration. Emergent pericardiocentesis (nontrauma setting) or pericardial window placement (preferred) is the definitive treatment of tamponade.

### **Pericardiocentesis**

If pericardiocentesis is indicated in the ED, real-time ultrasound guidance is suggested to improve success and reduce complication rates.<sup>17,18</sup> Direct needle guidance and entry into the pericardial space, a reduction of pericardial fluid volume, and improved right chamber expansion can be seen sonographically. The most common methods are a direct in-line approach with the subxiphoid view or an apical approach with sonographic evaluation of the subxiphoid view simultaneously.

### **Case 2 Conclusion**

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This patient is found to have a moderate-sized pericardial effusion without evidence of tamponade physiology. His blood pressure remains stable and the patient is admitted for further workup of the source of his effusion. Consideration is also made for placement of a pigtail catheter for drainage of the effusion.

### **Pitfalls**

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There are several tips to remember when evaluating a patient for a pericardial effusion or signs of tamponade.

- *Chronic pulmonary hypertension.* It must be noted that in conditions causing chronically elevated right-sided chamber pressure, there may not be right atrial or RV collapse until the intrapericardial pressure is very high. Such conditions may show a pericardial effusion with a distended right atrium and ventricle, RV hypertrophy, and plethora of the IVC. Consider the patient's previous history when making this determination.
- Right-sided heart chamber pressures are more easily exceeded (leading to collapse) in conditions of volume depletion.
- *Differentiation from pleural effusion.* To the untrained eye, a left pleural effusion can be mistaken for a pericardial effusion. The pericardial space surrounds most of the heart except at the base and stops near the atrioventricular sulcus and anterior to the adjacent descending aorta. Unclotted fluid in the pericardial

space is contained within the pericardial space, and does not appear posterior to the descending aorta or atrioventricular sulcus on the parasternal view (as seen in [Fig. 10](#) and [Video 14](#)). Fluid seen posterior to the descending aorta is considered pleural fluid in the left thorax. Pleural fluid can therefore be confused with a pericardial fluid (false positive) if the descending aorta is not used as a reference point.

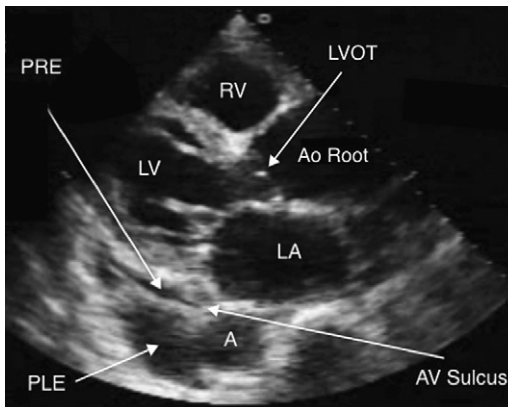
### CASE 3

A 56-year-old man experiences sudden pleuritic chest pain and shortness of breath. He is mildly hypoxic at 92% but his chest radiograph shows clear lungs and no cardiomegaly. His ECG shows tachycardia but no classic signs of ischemia or infarction. Emergency echocardiogram shows no pericardial effusion, normal LV systolic function, and something else...

#### *Right Ventricular Size and Function*

##### **Anatomy**

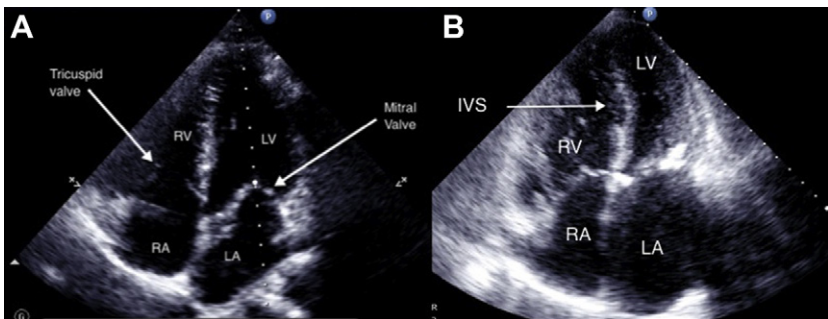
The right ventricle of the normal adult heart has a basal width that is less than 60% that of the left ventricle. The normal left ventricle is more muscular and its apex is bluntly curved, occupying two-thirds of the heart's apex. By contrast, the right ventricle's apex tapers sharply when viewed with the apical 4-window. The RV free wall is thin and creates a relatively low-pressure chamber. The interventricular septal wall is therefore contoured (bowed) toward the right ventricle. The normal right ventricle has a crescentic (semilunar) appearance on the parasternal short-axis (PSSA) view and is significantly smaller than the left ventricle.



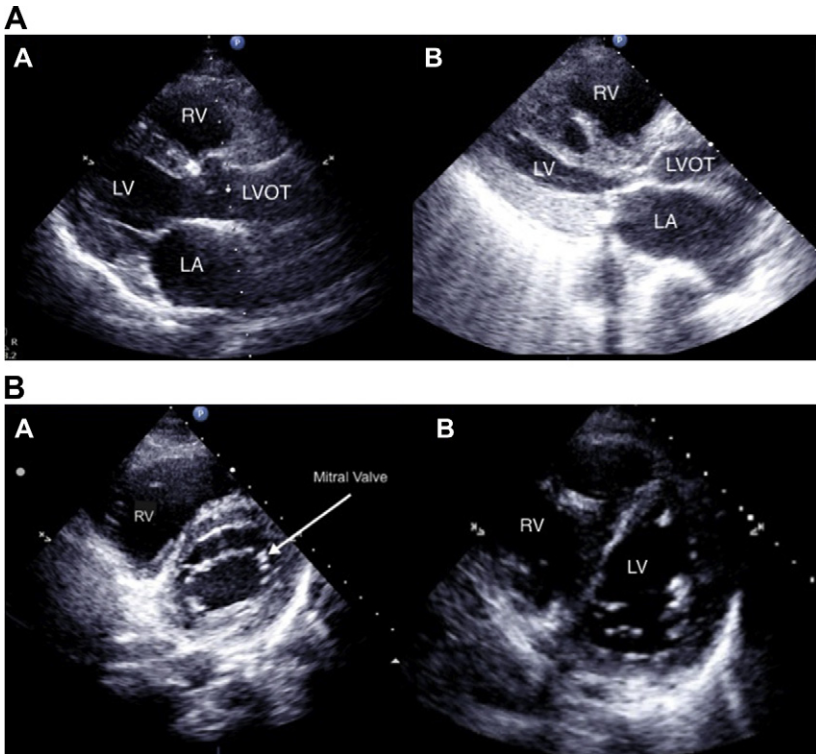
**Fig. 10.** Pericardial and pleural effusion. This PSLA view shows both a left pleural and a pericardial effusion. The descending aorta (A) is a key anatomic landmark. The pericardium attaches to the heart at the atrioventricular (AV) sulcus, limiting the expansion of the pericardial effusion (PRE) and thus helps to differentiate it from a pleural effusion (PLE), which is also seen here. Pericardial fluid accumulations do not extend posterior to the descending aorta (A). A left pleural effusion extends behind the descending aorta and thus is distinguished from the pericardial effusion. Note also that the aortic (Ao) root is dilated (>4 cm), which is suggestive of an ascending thoracic aortic aneurysm. The left atrium (LA) is enlarged as well. See [Video 14](#). LV, left ventricle; LVOT, left ventricular outflow tract; RV, right ventricle.

### Echocardiographic signs

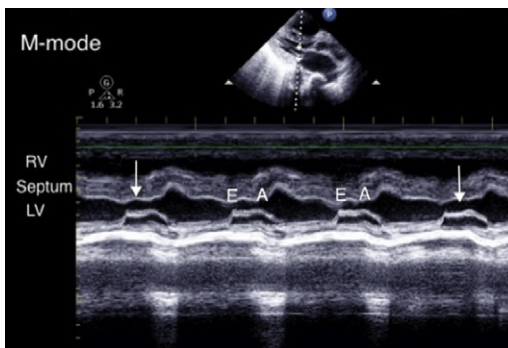
- RV dilation.** As previously mentioned, the right ventricle is typically smaller (<60%) than the left ventricle under normal conditions. However, with increases in right-sided pressure or volume, the right ventricle may enlarge to equal or exceed the size of the left ventricle. The causes of RV dilation (acute cor pulmonale) are multiple and may include pulmonary embolus (PE), primary pulmonary hypertension, chronic obstructive pulmonary obstruction, elevated intrathoracic pressure from mechanical ventilation, RV infarction, certain cardiomyopathies, and other conditions. See **Fig. 11**, **Fig. 12A** and **B**, and **Videos 15–20** for examples of RV dilation.
- Paradoxical septal motion.** In a normal heart, the higher pressures of the left ventricle move the septal wall bowed toward the right ventricle throughout a normal cardiac cycle. With RV strain, RV enlargement due to volume or pressure increases can compete with or may even exceed the LV pressures. Significant RV pressure may straighten the septal wall or even cause it to bow toward the left ventricle, as seen in **Fig. 13**, an M-mode tracing in the PSLA view. This abnormal septal motion is also called septal dyskinesia. The resulting decrease in LV filling may lead to significant reductions in cardiac output.<sup>19</sup>
- Decreased IVC respiratory variation.** Elevated right-sided pressure or volume will reduce the variability of the IVC during a normal respiratory cycle and give the appearance of physiologic fluid overload; this is discussed in more detail in a later section. However, it should be noted that the presence of decreased respiratory variation as a result of pulmonary hypertension should not be automatically interpreted as fluid overload. Fluid administration may still be an important intervention in certain cases, including acute PE.
- Other findings.** One study determined 100% hospital mortality for pulmonary embolism when shock signs were accompanied by echocardiographic findings of diastolic LV impairment (E/A wave <1), RV hypokinesis, RV/LV ratio >1, and end-diastolic RV diameter >3 cm.<sup>20</sup> Another study determined that in normotensive patients with PE and RV systolic overload, tricuspid velocity and troponin level were better at risk-stratifying patients than RV to LV diameter ratio.<sup>21</sup> The bottom line is that for patients in extremis with acute RV enlargement signs, massive pulmonary embolism should be considered. When the patient is not in



**Fig. 11.** Right ventricular strain (4-chamber). (A) A normal apical 4-chamber view. (B) Apical 4-chamber view of a patient with elevated right heart pressures. Notice the enlargement of the right ventricle (RV) and bowing of the septum toward the left ventricle (LV) in (B). See **Videos 15** and **16**. LA, left atrium; IVS, interventricular septum; RA, right atrium.



**Fig. 12.** (A) Right ventricular strain (PSLA). (A) A normal PSLA view. (B) A dilated right ventricle (RV) compressing the left ventricle (LV), due to high right-sided pressures. A dilated left atrium (LA) is also seen. Notice how the interventricular septum has shifted toward the LV, compromising filling. See [Videos 17](#) and [18](#). LVOT, left ventricular outflow tract. (B) Right ventricular strain (PSSA). (A) A normal PSSA view, showing a circular left ventricle (LV) at the level of the mitral valve. (B) A dilated right ventricle (RV) causing shift of the septal wall, and a D-shaped LV caused by the elevated right heart pressure. See [Videos 19](#) and [20](#).



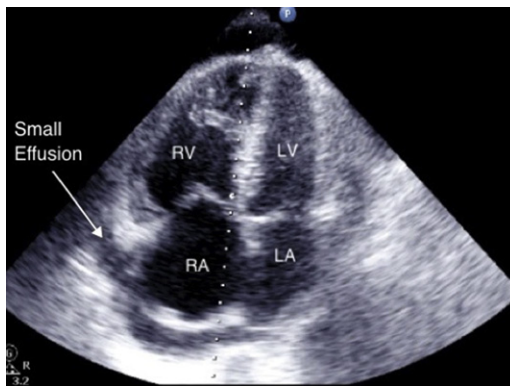
**Fig. 13.** RV strain (M-mode). This parasternal long-axis M-mode snapshot shows evidence of elevated right heart pressure and septal dyskinesia. The E and A waves represent the diastolic phases of passive LV filling and atrial contraction, respectively. In this case, the E and A wave are of similar size, consistent with diastolic dysfunction. Note that in diastole the septum shifts toward the LV (arrows), due to the abnormally elevated right heart pressures, as indicated by the arrows.

extremis, more advanced emergency echocardiography parameters and biomarkers can be used for risk stratification of suspected or diagnosed pulmonary embolism.

- *Acute versus chronic.* Right ventricular hypertrophy, defined as an RV free wall thickness greater than 5 mm, usually suggests chronic RV pressure elevation. This condition occurs because chronic RV pressure increases lead to RV enlargement with RV hypertrophy, and may include right atrial dilation as well. By contrast, an acute increase in RV wall tension may compromise perfusion, leading to ischemia-related reductions in RV systolic function without an increase in RV free wall thickness. See [Fig. 14](#) and [Video 21](#) for an example.

### Diagnostic utility

- *PE.* Emergency transthoracic echocardiography rarely makes a definitive diagnosis of massive pulmonary embolism or a thrombus in transit.<sup>22,23</sup> Unlike transesophageal echocardiography, which can detect thrombi in the central pulmonary arterial tree, transthoracic echocardiography merely searches for signs of pressure buildup from a massive or submassive pulmonary embolism lodged in the proximal or lobar pulmonary arteries.<sup>24</sup> A large PE is needed to achieve this effect, and occlusion of more than 30% of the pulmonary arterial tree usually can lead to RV dilation and evidence of poor RV function.<sup>25</sup>
- *Echocardiography.* Echocardiography should not be used to reduce the suspicion or stop the search for a clinically significant PE, as it is not considered a good screening test for this diagnosis.<sup>26</sup> However, echocardiography combined with venous ultrasound showed improved sensitivity when severe dyspnea or high pretest suspicion existed.<sup>27</sup> Computed tomographic angiography (CTA) of the thorax is still the preferred imaging study to definitively make or exclude the diagnosis of pulmonary embolism.
- *Undifferentiated shock and hypotension.* In patients with undifferentiated shock and hypotension, emergency echocardiography searches for previously



**Fig. 14.** Right ventricle (RV) hypertrophy. This apical 4-chamber view shows prominent dilation of the right atrium (RA) and RV compared with the left heart chambers. The enlarged RV now occupies most of the cardiac apex. The RV free wall is thickened, suggesting chronic pulmonary hypertension. A small pericardial effusion is seen lateral to the RA and RV. Given the chronically elevated right heart pressures, tamponade is less likely to occur, as it will require higher intrapericardial pressures to collapse this RV. See [Video 21](#). LA, left atrium; LV, left ventricle.

discussed pericardial effusion, global heart function, and then for RV enlargement. If RV dilation is discovered, the next step is to determine whether it is acute or chronic. Chronic RV dysfunction and enlargement are suggested if there is associated left-sided dilation or dysfunction, RV hypertrophy, or right atrial dilation. Both RV pressure and volume overload conditions can lead to enlargement of the right ventricle with straightening or bowing of the interventricular septum into the left ventricle. In the cardiac arrest scenario, a massive PE is definitely a possibility, and emergency echocardiography screening may be the only diagnostic imaging option available.<sup>28</sup> RV strain can explain associated IVC plethora, and this makes volume assessment by using echocardiography challenging. The findings of RV dilation and ischemia-induced RV dysfunction are important risk-stratification criteria in patients with confirmed acute submassive PE. Its presence portends increased short-term mortality and long-term morbidity.<sup>26,29–34</sup> Primary RV infarction also may show up as RV hypokinesis, but is commonly associated with LV inferior wall segment motion abnormalities.

- RV dysfunction can be found in as many as 40% of hemodynamically stable cases of confirmed PE. Other studies report up to 20% of confirmed PE cases have normal echocardiographic findings. Overall the finding of RV enlargement in an unstable patient should strongly place submassive PE on the differential diagnosis and consideration for definitive diagnosis.<sup>21,35</sup>

### **Case 3 Conclusion**

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The patient is found to have evidence of RV dilation on both the apical 4-chamber and PSSA views during evaluation by bedside echo. CTA of the chest is obtained, identifying a PE in the right pulmonary artery. The patient continues to decompensate with declining respiratory status and worsening hypotension. Thrombolytic agents are given based on the patient's computed tomography findings and evidence of RV strain. The patient is transferred to the medical intensive care unit for further care.

### **CASE 4**

A 47-year-old man collapses in the airport while transferring flights. The prehospital team appropriately initiates Advanced Cardiopulmonary Life Support (ACLS) protocol and after 10 minutes he arrives in your ED undergoing active cardiopulmonary resuscitation (CPR) by medics. He is morbidly obese. You notice an atrioventricular fistula and his left arm as well as some bilateral lower extremity edema. Pulses are very difficult to palpate because of the patient's body habitus, and a manual blood pressure is unobtainable. The new attending to your staff wants to use the ultrasound machine to look at his heart quickly.

### **Cardiac Arrest**

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#### **Overview**

Bedside cardiac ultrasound can be used to determine the presence and degree of cardiac activity in situations whereby identification of a palpable pulse is uncertain. Though not explicitly stated in ACLS guidelines, emergency physicians have realized that the usefulness of goal-directed emergency echocardiography is exemplified in the pulseless electrical activity (PEA) and asystole cardiac arrest situation. During pulse checks, a brief viewing of the heart can identify cardiac activity, screen for pericardial effusions and tamponade, look for significant right heart strain, or evaluate for severe volume depletion. The cardiac ultrasound views must be promptly started and skillfully obtained during the 5-second "pulse check." It should supplement and not

interrupt the course of the resuscitation. The subcostal view is typically easiest to obtain during insufflation in a mechanically ventilated patient. Serial heart evaluations can be performed during the course of the resuscitation.<sup>36–40</sup> See [Video 22](#) for an example of a subcostal view to evaluate for the presence of cardiac function.

### **Advantages**

- *Lack of interruptions with CPR.* There is minimal interruption on resuscitative efforts in exchange for the critical and time-sensitive information that is provided to the treating physicians. A quick look of several seconds on the subxiphoid cardiac window may give more information than attempting to palpate a pulse in a patient with a difficult body habitus.<sup>39</sup> Serial evaluations will show the return of spontaneous circulation, or if the heart activity deteriorates or is refractory to resuscitative efforts.
- *Identification of life-threatening pathology.* Recent ACLS guidelines list several potentially reversible causes that must be considered in the patient presenting in PEA.<sup>41,42</sup> Emergency echocardiography can quickly identify several of the causes of severe shock, including pericardial effusion/tamponade, evidence of RV strain (PE), and signs of cardiac underfilling or severe hypovolemia. The time-sensitive ruling-in and ruling-out of severe cardiac conditions can guide therapeutic interventions, improve resuscitation efforts, and may provide more informed decision making on each critically ill patient.<sup>3,43–45</sup>
- *Improvement in resuscitative efforts.* Once cardiac activity is identified, echo can be used to guide resuscitative efforts. Serial examinations can identify progression in hemodynamic status or contractility following the initiation of intravenous fluid resuscitation or adrenergic agents.<sup>46</sup>
- *Patient safety.* The days of the blind pericardiocentesis attempts during an undifferentiated PEA resuscitation are long gone. Furthermore, ultrasound can be used to guide the procedure if one is detected. It may also assist in determining the presence of transcutaneous or transvenous pacer capture in the right clinical scenario.

### **Survival**

When cardiac ultrasound shows no ventricular activity and there is asystole on the cardiac monitor after full ACLS resuscitative attempts by paramedics, survival is rare.<sup>39,47,48</sup> Other signs of failure of resuscitation include the development of coagulating blood and isolated atrial and valve movement without organized ventricular activity.<sup>40</sup> These small, isolated valve movements are not the same as organized cardiac contractions, and the resuscitation should not be continued based on these findings alone. It must also be noted that reversible and transient myocardial stunning is a phenomenon that exists and may result from acute coronary hypoperfusion, noncardiac causes such as intracranial hemorrhage, acute drug toxicity, and others.<sup>49–51</sup> Finding persistent asystole during serial ultrasound evaluations (while full resuscitative efforts continue) helps to better rule out peri-arrest transient myocardial stunning and confirms an unsuccessful resuscitation.<sup>52,53</sup>

### **Pediatric considerations**

The primary reasons for pediatric arrests are usually not cardiac but are due to respiratory complications and insufficiency.<sup>54</sup> The detection of pulses during pulse checks can be very challenging in the pediatric peri-arrest time. Emergency echocardiography evaluates pump function as part of the circulation assessment.<sup>55</sup>

### **Case 4 Conclusion**

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Subcostal cardiac views are obtained rapidly during pulse checks, and indicate that despite the absence of definite palpable pulses, the patient does have evidence of cardiac activity on your bedside echo. A stat basic metabolic panel is ordered and shows the presence of hyperkalemia at 7.3 mEq/L. The patient is treated with calcium, bicarbonate, insulin, and glucose, with improvement of his hemodynamic status. Nephrology is consulted for emergent dialysis. The patient is discharged 5 days later with no evidence of neurologic dysfunction.

### **CASE 5**

A 45-year-old man with cardiac disease and diabetes mellitus presents with weakness, hypotension, and tachycardia. He appears emaciated. His serum glucose, anion gap, and acetone levels are all elevated. Bedside echocardiography is notable for a hyperdynamic left ventricle and an IVC diameter of 2 cm with minimal respiratory size variation. Based on the patient's history and echocardiographic findings, what is his volume status?

### **Volume Assessment**

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#### **Overview**

The volume status of patients presenting to the ED with critical illness or injury is often unclear. Traditionally, physicians have relied on their physical examination findings to help make this determination. However, physical signs are not always accurate in the detection of hypovolemia.<sup>56,57</sup> Other traditional methods have proved unreliable as well. A recent meta-analysis has shown that invasively obtained hemodynamic information such as central venous pressure (CVP) has a poor correlation with actual volume status.<sup>50</sup> The potential consequences of inaccurate judgment of volume status are not minor. Acute volume loading can provoke fluid overload in patients with an unsuspected primary cardiogenic etiology. Suboptimal volume resuscitation can prolong end-organ hypoperfusion, dysfunction, or failure. Bedside cardiac ultrasound aims to noninvasively evaluate pump filling as well as filling of the IVC, the main conduit of venous blood return to the heart.

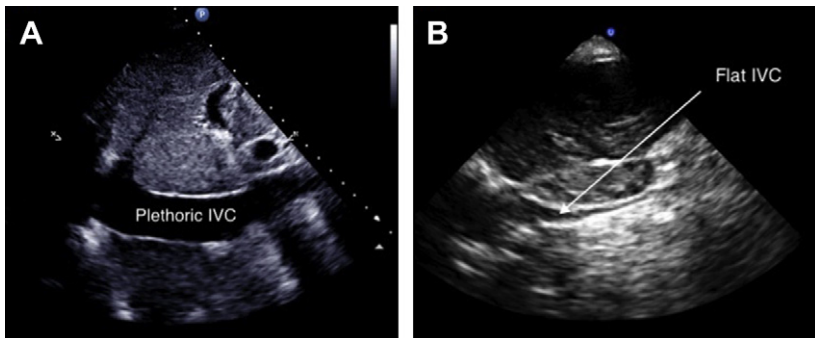
#### **Physiology**

There has been much research on the use of the IVC in estimating intravascular volume. The IVC is a distensible and collapsible vein returning venous blood to the right atrium of the heart. The two most often described sonographic parameters in determining volume status are IVC size and respiratory variation. Both features are influenced by pressure and volume changes within the heart. These changes may be related to myocardial compliance, valve function, intrathoracic pressure, pericardial pressure, or intra-abdominal pressure, and all are factors that influence IVC respiratory dynamics.

- **Pump filling.** Pump filling represents LV preload, and can be appreciated and quantified as LV end-diastolic area or LV end-diastolic volume. It can also be visually estimated during emergency echocardiography. Hypervolemia is often associated with increased LV end-diastolic dimensions (dilated left ventricle) and depressed systolic function. One cardiac ultrasound feature that is highly suggestive of hypovolemia includes PSSA views showing touching ("kissing") papillary muscles during systole. Another feature is very low to absent end-diastolic LV volumes when using the parasternal long, subcostal or apical 4-chamber views. Classic severe hypovolemia signs can also include near to

complete emptying of the heart, hypercontractility, and rapid contractions. Keep in mind that some patients may have preexisting systolic dysfunction or may be unable to mount a compensatory rate increase in response to low LV preload if on certain medications, including  $\beta$ -blockers.

- *IVC size.* In previous studies, several attempts were made to correlate CVP with IVC diameter and respiratory variation. However, these IVC size measurements alone as an indicator of fluid status have fallen out of favor, due to their unreliability. One of the first studies to support this finding showed a significant overlap in IVC diameter between normal subjects and those with cardiac disease, indicating that size alone is not a reliable indicator of fluid status.<sup>58</sup> The same study also found that patients with isolated right heart disease had distinctly lower respiratory variations in IVC diameter when compared with their healthy counterparts.<sup>58</sup> Therefore, variation in IVC size was thought to hold some promise as an estimation of right-sided filling pressures. Note that right-sided filling pressures are affected by several factors, including pulmonary hypertension, myocardial compliance, valve function, presence of pericardial effusion and/or PE, and general hydration status of the patient. Each assessment must be taken within the clinical context of the patient, and assumptions should not be made about the hydration status based on isolated IVC measurements. See [Fig. 15](#), and [Videos 23](#) and [24](#) for a comparison between a plethoric and collapsible IVC.
- *IVC respiratory variation.* Usually there is some degree of variation in IVC diameter during each breathing cycle. However, the nature of the variation differs depending on the mode of ventilation. During spontaneous breathing, inhalation decreases intrathoracic pressure, causing the return of venous blood to the right atrium and a brief decrease in IVC diameter; this is also called IVC collapsibility. On the other hand, patients being mechanically ventilated show the opposite effects. Positive pressure ventilation increases intrathoracic pressure, causing less inflow of blood to the lungs, thus increasing the IVC diameter; this is also known as IVC distensibility. Some studies use IVC distensibility in monitoring acute volume-loading adjustments in mechanically ventilated patients.<sup>59,60</sup> The absolute value of the change of IVC size is more important than the direction of change. Usually with volume depletion, the change in size of the IVC is more dramatic than in normovolemic or hypervolemic states.



**Fig. 15.** Inferior vena cava (IVC) evaluation. (A) A subcostal longitudinal view of a dilated, plethoric IVC. By contrast, (B) shows an IVC that is nearly fully collapsed. This appearance often indicates hypovolemia, but clinical correlation is needed, as discussed in the text. See [Videos 23](#) and [24](#).

The measurement of IVC collapsibility is referred to as the caval index, often expressed as a percentage. The caval index is calculated by the equation:

$$\text{Caval Index} = (\text{IVC}_{\text{exp}} - \text{IVC}_{\text{insp}}) / \text{IVC}_{\text{exp}} \times 100\%$$

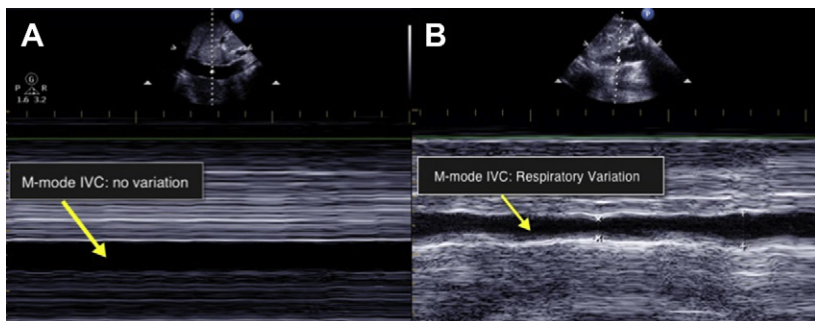
A high caval index in general indicates a higher probability of hypovolemia, although the cutoff values vary depending on which study is cited. Several other studies have shown good interrater agreement in the calculated caval index measurements when compared with visual estimates of the same parameters.<sup>11,12,61</sup> **Fig. 16** shows a comparison between a high and low caval indices by M-mode evaluation.

### **Clinical utility**

In a study of acute volume loading of hypotensive ED patients without signs of congestive heart failure, no fluid toxicity was provoked even in patients with low caval index before volume load. IVC diameters increased with volume loading as well. Baseline high caval indices acutely decreased (filled up) whereas low caval indices at baseline remained low.<sup>11</sup> When IVC plethora is encountered, a search for signs of right and left heart strain or effusion with tamponade is strongly suggested. IVC size and caval index are influenced by pressure elevation and may not be due to a state of volume overload unless other clinical signs (eg, pulmonary vascular congestion) are present. When there is an elevated caval index (increased IVC collapsibility), low pressures and low volume states are more likely.

### **Limitations of sonographic evaluation of the IVC for determination of fluid status**

- **Correlation with CVP unreliable.** Previous studies have attempted to show correlations of IVC size and dynamics with central venous pressure measurements.<sup>58,62–64</sup> Acute fluid loading and fluid removal studies have demonstrated temporally associated acute changes in IVC size and dynamics, but these have been performed in a myriad of subjects (healthy, mechanically ventilated, cardiac diseases, sepsis, and right heart strain).<sup>59,60,65,66</sup> It must be emphasized that in clinical studies IVC dynamics and size have been mainly correlated with right heart pressure measurements rather than volume states.<sup>58,67,68</sup> A recent study by Brennan and colleagues<sup>69</sup> reassessed the previously used model that categorized CVP into 5-mm Hg ranges based on IVC findings, and found it to



**Fig. 16.** Inferior vena cava (IVC) in M-mode (*arrows*). Both images are of the proximal IVC (subcostal long-axis view) using M-mode during a respiratory cycle. In (A), no respiratory variation is seen, which suggests an elevated right heart pressure (one of the causes can be fluid overload). In (B), moderate variation of IVC diameter is seen, likely within the normal range. See text for further discussion on IVC dynamics.

be only 43% accurate when prospectively applied to its own study group. Based on this reappraisal, the investigators offered a simpler approach: an elevated right heart pressure was suggested by an IVC diameter greater than 2.0 cm and a caval index less than 40%.

- *Right heart pressure and the caval index.* Studies have shown an overlap in baseline IVC diameters of subjects with no cardiac disease and those with left and right heart disease. However, the caval index was notably lower in those with right-sided heart disease.<sup>58</sup> This finding suggests that a key influence in caval index is right-sided pressure. Right-sided pressure increases may be elevated in many forms of heart disease, including congestive heart failure (considered a fluid overload states), and pericardial and several pulmonary conditions.<sup>70</sup> IVC plethora itself is not pathognomonic of a single cardiopulmonary disease state or volume status.
- *Evaluation of preload status.* Ultrasound of the IVC does not reflect LV preload status. Volume status assessments with echocardiography should also include an evaluation of LV end-diastolic dimensions.
- *Mechanical ventilation.* In patients undergoing positive pressure ventilation, the IVC will collapse with expiration, rather than inspiration as seen with spontaneously breathing patients. Although the pattern is opposite, the principle of examination for variation with respirations is still the same and can be interpreted in a similar fashion.
- *Pediatrics.* In pediatric emergency medicine studies, IVC indices had moderately good sensitivities but low specificities for gastroenteritis-related dehydration.<sup>57,71</sup> The wide range of sizes in the pediatric age spectrum and patient compliance issues can sometimes make technical measurements of IVC indices difficult. Often the IVC is compared with the aorta for a size reference, and IVC/aorta ratios are commonly measured in children for this reason.<sup>57,71</sup> Note that incidental findings of congenital heart abnormalities are often encountered during emergency echo of children for other indications. Congenital heart abnormality detection is beyond the scope of emergency echocardiography. If the clinician is suspicious of a congenital anomaly, comprehensive pediatric echocardiography is needed for detailed analysis.

### **Case 5 Conclusion**

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Based on clinical factors and vital signs alone, this patient appears to be volume depleted. He received volume resuscitation in the ED with clinical improvement. Comprehensive echocardiography was performed on stabilization and, sure enough, this patient was found to have evidence of pulmonary hypertension. The elevated right-sided pressures were likely responsible for his lack of IVC variation despite his dehydrated state.

The bottom line is that, in general, significant IVC diameter respiratory variation usually indicates a hypovolemic state especially when combined with a hyperdynamic and poorly filled heart. However, the presence of a plethoric IVC should not rule out a low volume state, due to the factors previously mentioned. Other causes of elevated right heart pressure should be considered when an IVC diameter greater than 2 cm with less than 40% collapsibility is discovered.<sup>69</sup>

### **SUMMARY**

The responsible and proficient use of emergency echocardiography requires training, knowledge of ultrasound physics, consistent practice in adequate image acquisition,

and judicious interpretation. The ACEP *Emergency Ultrasound Guidelines* provides details of minimum training requirements and suggestions for practice-based pathways to training and credentialing in emergency echocardiography. Emergency echocardiography has its limitations and challenges in the emergency setting, but, when used properly, is frequently instrumental and invaluable in improving patient care in the ED.

## SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at [doi:10.1016/j.emc.2011.08.002](https://doi.org/10.1016/j.emc.2011.08.002).

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