Point-of-care echo in pulmonary embolism

Philips tutorial

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POC echo for the detection of pulmonary embolism

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1 Introduction

Pulmonary embolism (PE) is estimated to kill up to 300,000 people yearly in the United States.[1] While the diagnosis of PE may be straightforward in certain cases, the symptoms are often non-specific and PE remains a difficult diagnosis that may be lethal if missed.

Transthoracic echocardiography (TTE) may play a valuable role in the diagnosis of PE, particularly in those cases with hemodynamic instability. Once PE is diagnosed, TTE plays a key role in therapeutic considerations and prognosis.

TTE may be performed by a cardiologist when available. However, in the emergent presentation of PE, cardiology-performed TTE may not be available rapidly enough and, in many emergency departments, it is not available at all during certain hours. Point-of-care (POC) TTE performed by emergency medicine and critical care physicians to aid in the diagnosis and therapy of patients with severe PE may be lifesaving.

2 Clinical case

A 58-year-old woman with breast cancer who is currently being treated with chemotherapy (last dose one week ago) presents with acute onset of shortness of breath and associated bilateral anterior chest discomfort.

She arrives by ambulance. She is distressed, diaphoretic, and breathing rapidly. Her vital signs include a blood pressure of 95/50 mmHg, heart rate of 115, respiratory rate of 22, temperature of 100.2°F (37.9°C), and an oxygen saturation of 92% on 2 liters of oxygen. On exam she is mildly obese, her lungs are clear, and her heart sounds are only notable for tachycardia. Both lower extremities are mildly swollen. The EKG reveals sinus tachycardia but is otherwise unremarkable. The portable chest X-ray is pending.

What is your differential diagnosis?
What would you do?

3 PE and cardiac pathophysiology

The vast majority of PEs occur when a deep venous thrombosis (DVT), typically from the lower extremities, dislodges and travels through the right heart to the pulmonary artery. PEs occur in a spectrum of sizes that range from small (subsegmental) and unilateral emboli to massive and bilateral emboli (the so-called "saddle embolus" across both pulmonary arteries). Thrombi in the pulmonary artery can cause mechanical obstruction as well as vasospasm, both of which can increase pressure in the pulmonary artery, resulting in changes to the right side of the heart that may be apparent on echo.

PEs may be divided into three categories based on severity with implications for prognosis and therapy[2]:

- Small
- Submassive
- Massive

The diagnostic criteria, the initial and long-term prognosis, and therapy for each category are summarized in Table 1.

As a diagnostic test, echo is not sensitive for small PEs as these may occur without right ventricular (RV) strain. However, with larger PEs the echo findings will be abnormal.

In the absence of direct visualization of a thrombus, echo is not specific for PE, as RV changes may occur as a result of lung disease or primary pulmonary hypertension (COPD is a frequent cause of chronic RV strain). However, the finding of RV pathology in a symptomatic patient without known cardiopulmonary disease makes a significant PE more likely.

In a patient with diagnosed PE, transthoracic echo also plays a central role in categorizing the severity of PE with direct implications for prognosis and therapy.

In stable patients with PE, echo findings are used to separate small or "low-risk" PE (no RV strain present) from submassive PE (RV strain present). Submassive PEs have a much higher mortality (~10% in contrast to under 2% in small PEs), and a greater potential for long-term morbidity (chronic RV strain and decreased exercise tolerance), and therefore may merit more aggressive therapy (systemic or directed thrombolysis) particularly in patients at lower risk for hemorrhage (e.g., younger patients).

In contrast, massive PEs cause hemodynamic instability resulting in hypotension or an elevated shock index (ratio of heart rate to systolic blood pressure >1:1). Massive PEs are extremely lethal and merit aggressive therapy including thrombolysis or clot removal.

If a patient is hemodynamically unstable from a known PE, echo does not necessarily add value. However, the absence of RV findings in a patient with hemodynamic instability essentially excludes PE as a cause.

Table 1: Categorization of PE severity

Small ("low-risk") PE	Submassive PE	Massive PE
Diagnosis		
PE without hemodynamic instability or signs of RV strain.	Hemodynamically stable PE with signs of right ventricular strain (echo is gold standard, CT may be used). Surrogate markers may include elevated troponin, elevated BNP, hypoxia, or EKG changes.	PE with hemodynamic instability: shock or sustained hypotension (systolic blood pressure <90 mmHg for >15 minutes).
TTE		
No signs of RV strain identified.	Signs of RV strain are part of the definition of submassive PE. They may be more pronounced in massive PE and include: • RV dilatation • RV hypokinesis • McConnell's sign • Paradoxical septal motion • D-shaped septum • Tricuspid regurgitation • Elevated RVSP (right ventricular systolic pressure) • Abnormal TAPSE (tricuspid annular plane systolic excursion)	
Initial prognosis		
Mortality <2%, unlikely to cause long-term symptoms.	Mortality >10%.	25-50% mortality, mostly in the first hour.
Therapy		
Unfractionated or low molecular weight heparin. Outpatient treatment may be considered in selected patients.	Unfractionated or low molecular weight heparin. Systemic thrombolytics should be considered.	Heparin in addition to systemic thrombolysis, catheter-directed thrombolysis, mechanical thrombectomy, or embolectomy.
Long-term prognosis		
Unlikely to cause long-term symptoms.	May cause significant long-term functional impairment (impaired exercise tolerance).	Frequent long-term functional limitation.

4 Cardiac A and P review

The heart can be pictured best as a cone that is situated obliquely in the chest. A cone has a long axis (a line from the center of the circular base through the apex) and a short axis (the plane perpendicular to the long axis). The base of the heart contains the valves and atria and is oriented posteriorly and to the patient's right shoulder (scapula). The aortic valve is in the center of the base of the heart. The apex of the heart is oriented anteriorly and towards the patient's left hip (apex at the "point of maximal impulse", lateral and inferior to the nipple or breast). The muscular left ventricle forms the structure of the cone with the thinner-walled right ventricle wrapped around the left ventricle anteriorly.

In contrast to the left side of the heart, the right heart is normally a low-pressure, high-compliance system. Normal right heart pressures are ~25/10 mmHg compared to ~120/80 mmHg for the systemic circulation.

When pulmonary artery pressure rises and is transmitted to the right ventricle (RV) the high-compliance chamber will dilate resulting in RV enlargement, RV hypokinesis, and other signs of increased RV pressure.

Rarely a thrombus may be seen in the proximal inferior vena cava, right atrium, or right ventricle. This is a very important finding and will be discussed in detail in the section on pathology.

5 Image orientation

A review of ultrasound image orientation is especially important for this tutorial. While orientation is important for all ultrasound studies, it is particularly relevant when performing echo for PE, as it is crucial to accurately differentiate the right side of the heart from the left side of the heart. Relying on the typical appearance of the right ventricle (i.e., smaller and more thin-walled than the left ventricle) may be misleading in pathological states.

Review

- All ultrasound transducers have an orientation marker/indicator on one side (usually a bump, groove, dot, or ridge)
- The orientation marker on the transducer corresponds to the orientation marker displayed on the ultrasound screen
- The convention for “general” ultrasound exams is the orientation marker is on the left side of the ultrasound screen with the transducer orientation marker pointing to the patient’s right side or towards the head
- The convention for traditional “cardiology” ultrasound exams is the orientation marker is on the right side of the ultrasound screen with the transducer orientation marker pointing to the patient’s left side or towards the head

The “general” and “cardiology” orientations are internally consistent, with the marker on the screen corresponding to indicator direction, and for two of the major TTE windows (apical and subxiphoid), left-sided cardiac structures should be on the right side of the screen as it is viewed regardless of which convention is used **(Figure 3a** and **Figure 3b)**.

Confusion arises when:

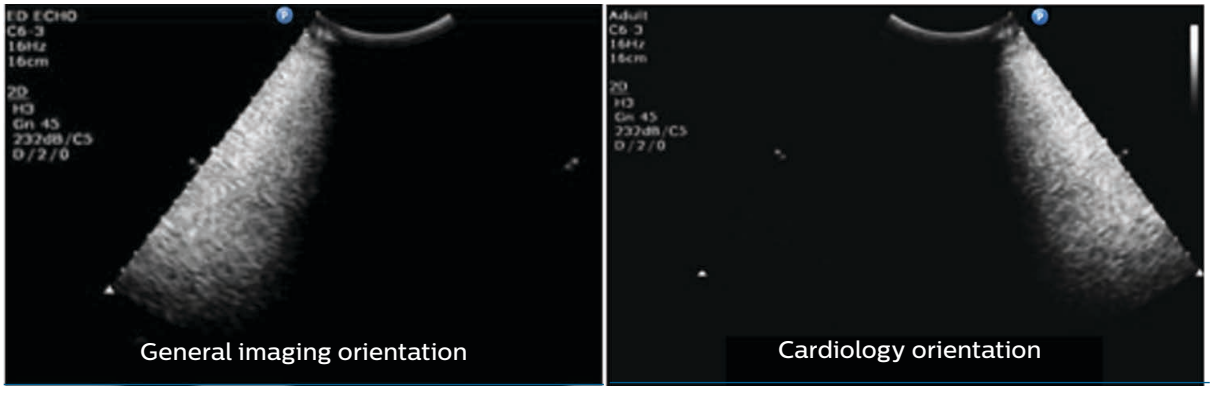
- A POC ultrasound exam integrates imaging of the heart with imaging of the abdomen or chest in a single exam or protocol (e.g., extended FAST exam, RUSH protocol, others)
- The transducer is oriented towards or away from the head (with “general” orientation, cephalad is screen left; with “cardiology” orientation, cephalad is screen right), hence image orientation for the parasternal views can be confusing

The physician performing POC echo needs to decide whether he/she will use the “general” indicator-to-screen orientation or the “cardiology” indicator-to-screen orientation. Therefore, the physician must always:

- Confirm the orientation marker on the ultrasound screen
- Confirm that the transducer orientation marker is directed correctly on the patient
- Understand how structures will be displayed on the screen[3]

A quick visual way to verify orientation is by placing a small amount of gel on the transducer on the side of the orientation marker **(Figure 1)**. The gel will correspond to the side of the ultrasound screen with the orientation marker. **Figure 2** demonstrates both the “general” orientation convention and the “cardiology” orientation convention.

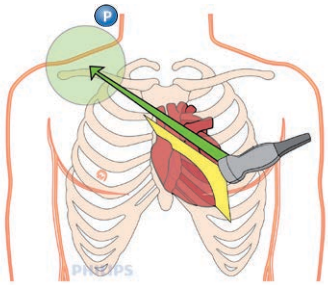
[Figure 1] Photo demonstrating small amount of gel on the side of the transducer corresponding to the orientation marker.

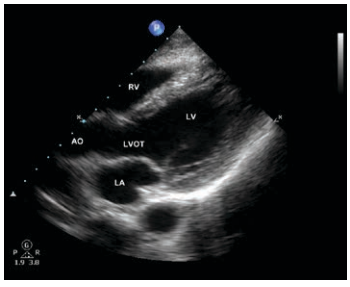


[Figure 2] Demonstrates the “general” and “cardiology” orientation conventions. For “general” the indicator/gel corresponds to left of screen as it is viewed. For “cardiology” the indicator/gel corresponds to right of screen as it is viewed.

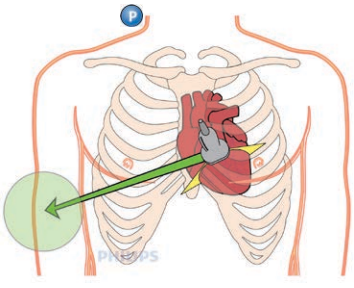
General convention

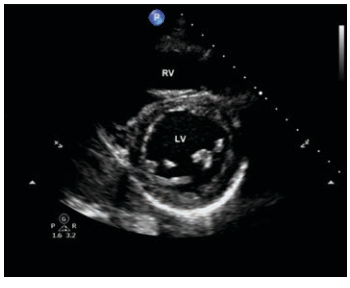
Parasternal long-axis



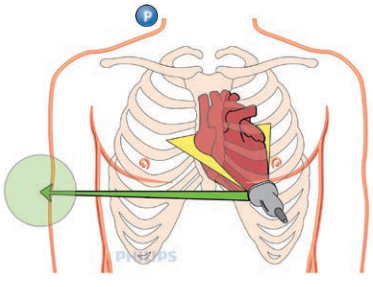


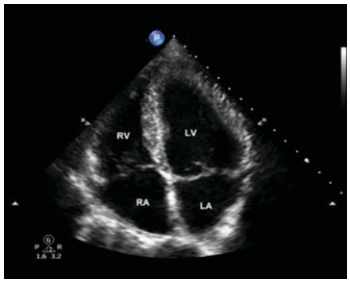
Parasternal short-axis



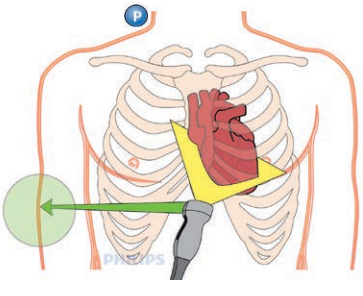


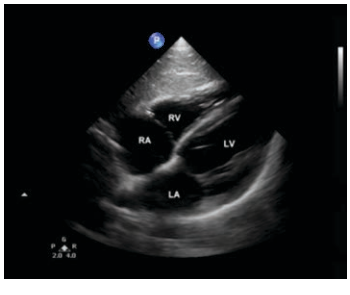
Apical four-chamber





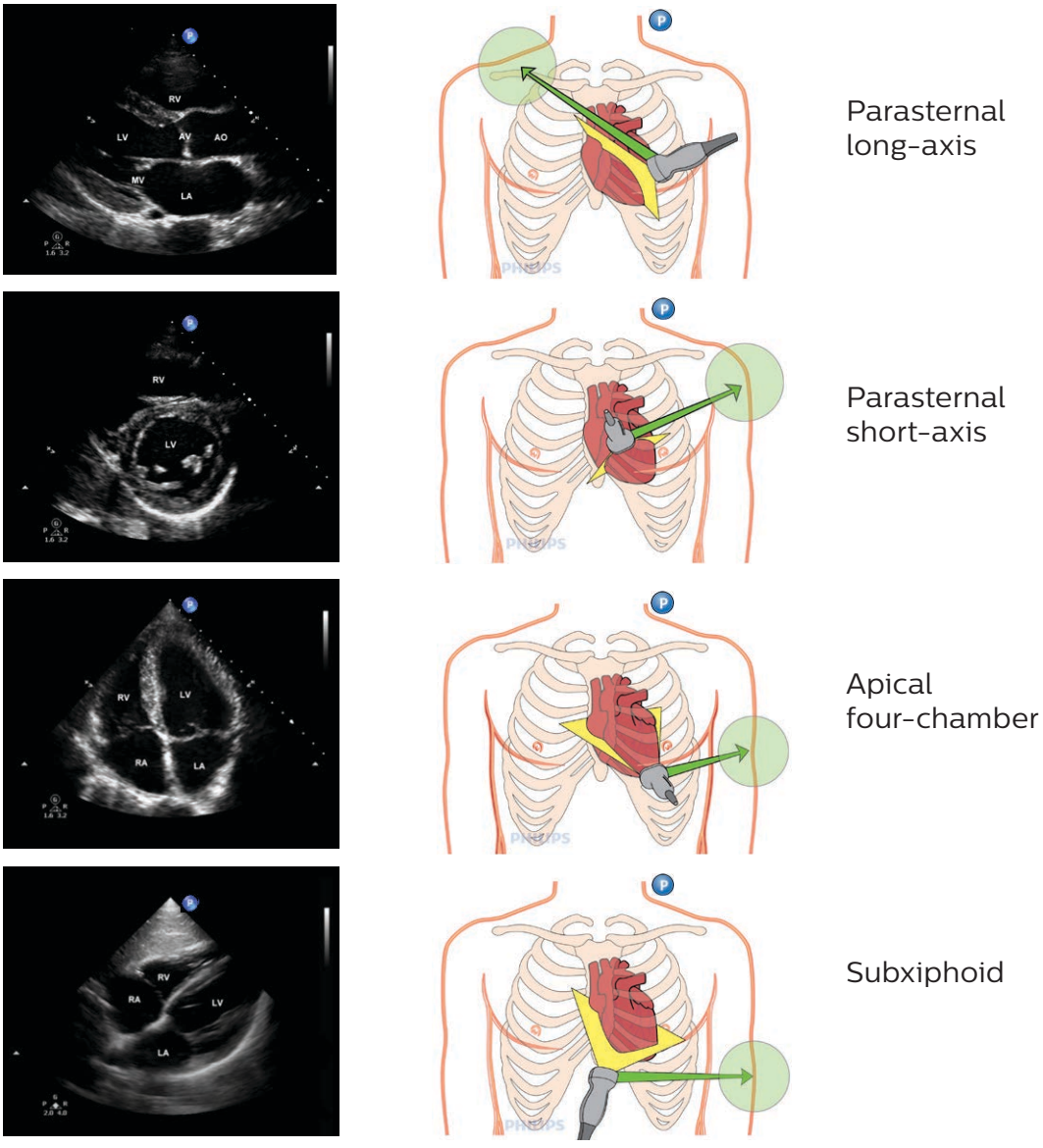
Subxiphoid





[Figure 3a] Illustrations demonstrating the direction of the transducer orientation marker and the corresponding images for cardiac images when using the “general” convention.

Cardiology convention

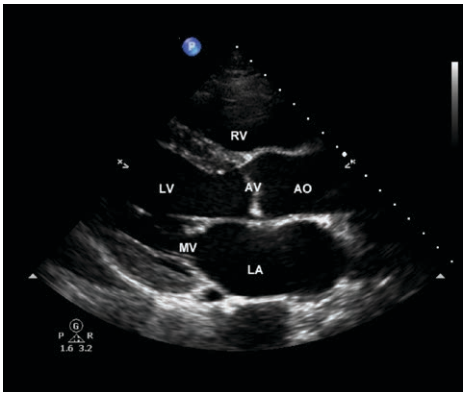


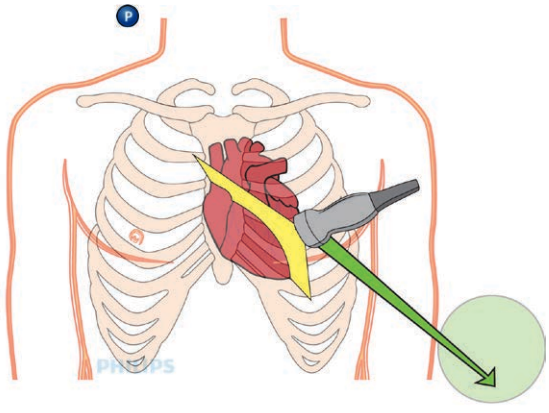
[Figure 3b] **Illustrations demonstrating the direction of the transducer orientation marker and the corresponding images for cardiac images when using the “cardiology” convention.**

In order to minimize confusion about indicator-to-screen orientation, our emergency department uses the “general” orientation for all ultrasound exams, including cardiac exams **(Figure 3a)**.

We created a preset optimized for cardiac imaging (“ED ECHO”) with the indicator-to-screen orientation consistent with “general” ultrasound exams (orientation marker to left of screen).

Using our convention for the parasternal long-axis view, the transducer orientation marker is directed towards the patient’s right shoulder, hence the apex of the heart is on the right side of the screen as it is viewed (consistent with the other “general” imaging views).





If a view that appears like the “cardiology” convention for the parasternal long-axis view is desired using a “general” indicator-to-screen convention, the orientation marker on the transducer can be turned so it is directed towards the patient’s left hip (the “fourth and long” direction: i.e., indicator to the four o’clock position).

Note: For the parasternal long-axis view using the cardiology convention (apex of the heart, a left-sided structure, to the left of the screen as it is viewed), the screen orientation marker is to the right of the screen and the transducer orientation marker is directed to patient’s right shoulder (Figure 3b).

NOTE OF CAUTION: If the right ventricle is enlarged and hypertrophied it could be mistakenly identified as the left ventricle in the apical four-chamber view.

This may occur:

- ***IF** the "general" screen orientation is used but the clinician uses the "cardiology" transducer orientation (the right ventricle would be displayed on the right side of the screen and not the left).*
- ***IF** the "cardiology" screen orientation is used but the clinician uses the "general" transducer orientation (again, the right ventricle would be displayed on the right side of the screen and not the left).*

This error is not likely in the parasternal and subxiphoid views because the right ventricle will always be anterior to the left ventricle regardless of indicator-to-screen or transducer orientation.

In summary, as long as the cardiac structures on the image are correctly identified, the convention that is used is not important. However, particular care should be taken with orientation of the apical four-chamber image. While the right ventricle typically appears smaller and more thin-walled than the left ventricle, this may not be a reliable finding in a pathological state, and consequently the orientation should be verified.

Unless otherwise noted, the images in this tutorial will use the “general” imaging convention (orientation marker on the left of the screen and transducer orientation marker to the patient’s right). In this convention, right-sided structures will always be on left of screen. Remember that the parasternal long-axis images will appear flipped to those accustomed to a “cardiology” convention.

6 Sonographic views and anatomy

Equipment considerations

Point-of-care transthoracic echo for the evaluation of right-sided pathology is best done using equipment from manufacturers with an emphasis on cardiac applications. A small-footprint, low-frequency, phased-array transducer should be used. Tissue harmonic imaging will enhance 2D visualization. Continuous wave (CW) spectral Doppler is essential for measuring right-sided pressures.

Approach

It is recommended to approach the patient from a consistent side, allowing the same hand to hold and manipulate the transducer each time. This will help develop confidence in obtaining images and maintaining orientation. This is particularly important for the clinician who is also a novice sonologist.

We recommend that the patient be approached from the patient's right side for all examinations when possible. This is in contrast to the approach used by cardiologists who typically approach the patient from the patient's left side and use their left hand to scan (which is why their orientation conventions are different).

Sonographic views and anatomy

The typical point-of-care TTE examination includes views from three ultrasound windows. The parasternal and apical windows are most applicable for determining the presence of RV strain.

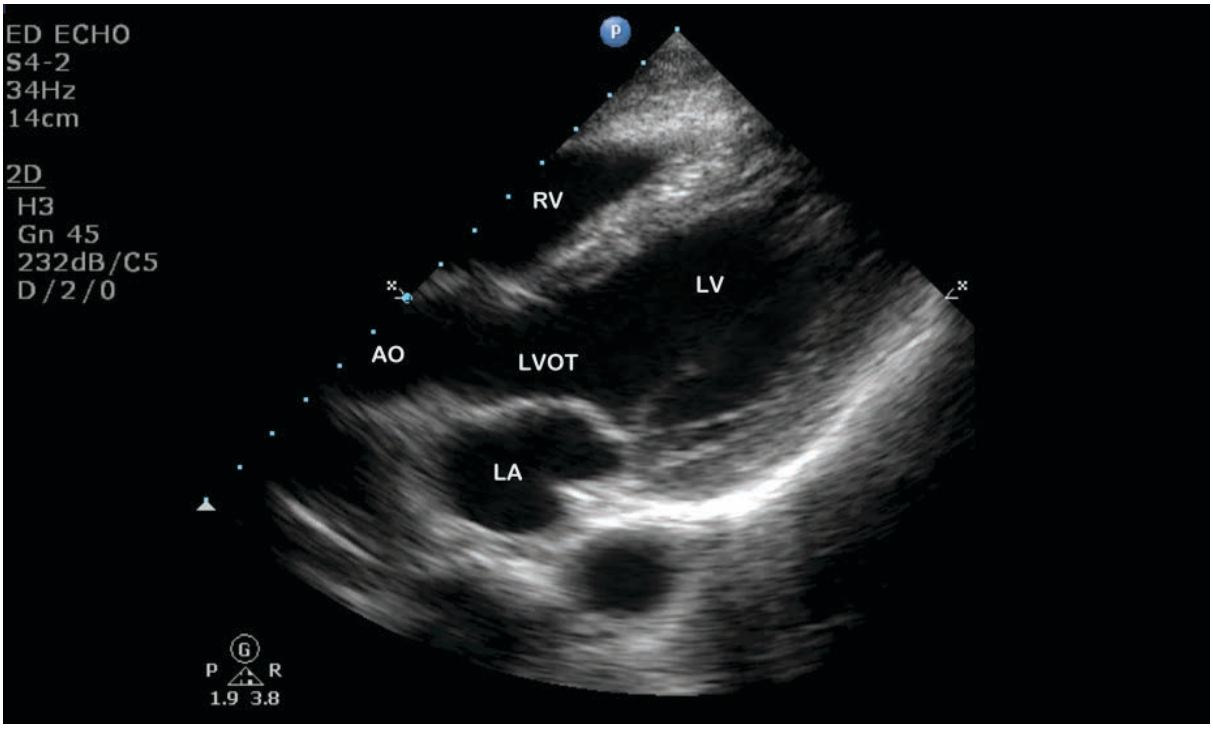
TTE Windows	TTE Views
Parasternal	Parasternal long-axis (PSLA) Parasternal short-axis (PSSA)
Apical	Apical four-chamber (A4C)
Subxiphoid	Subxiphoid four-chamber (SX) Subxiphoid IVC/hepatic veins

The parasternal window is the most reliably available and is often the first performed. However, the parasternal window may not be available in patients with chronic obstructive pulmonary disease (COPD) or those on a ventilator.

It is unusual that all three windows will be perfectly obtainable on any single patient. However, in most patients at least one window that can help define RV anatomy is obtainable. The clinician should attempt to obtain views from all three windows when possible.

Parasternal long-axis (PSLA) view

The parasternal long-axis (PSLA) view is obtained by placing the transducer perpendicular to the chest and lateral to the sternum at the 3rd, 4th, or 5th interspace with the ultrasound plane directed from the patient's right shoulder (base of heart) to the left hip (apex). The PSLA view may sometimes be improved by having the patient turn onto their left side, but this is not always necessary.



[Figure 4] PSLA view. Right ventricle (RV), left ventricle (LV), left atrium (LA), aorta (AO), and left ventricular outflow tract (LVOT).

[Video 1]

Video of the parasternal long-axis view. Note the screen orientation marker is set for "general." Right ventricle (RV), aorta (AO), aortic valve (AV), left ventricle (LV), left atrium (LA), and mitral valve (MV).

The PSLA view includes the right ventricle anteriorly; the left atrium, mitral valve, and left ventricle posteriorly; and the aortic valve, aortic root, and left ventricular outflow tract in the center of the heart **(Figure 4)**. The aortic arch curves out of view, but the descending aorta can be seen in cross-section posterior to the heart and anterior to the thoracic vertebral bodies. The structures seen in the PSLA view are similar to those seen in the subxiphoid or apical four-chamber views, except that the right atrium is typically not seen, while the aortic valve and left ventricular outflow tract are well visualized (more cephalad structures) **(Video 1)**.

The PSLA view can help define the size of the right ventricle relative to the left ventricle, a ratio that should be about 0.6:1. However, when comparing the chambers in the PSLA, it is important that the ultrasound plane not be angled through the right ventricle in a way that overemphasizes RV size relative to the LV. Fanning the ultrasound plane through the PSLA view to maximize LV size (which occurs when the plane of the ultrasound is orthogonal to the long axis of the LV) will ensure that the RV size is not overemphasized.

While angling the transducer may overemphasize RV size, it can be effective in visualizing the tricuspid valve (TV) when looking for regurgitation and measuring RV pressures. This “right ventricular view” is obtained by a very slight rotation and angling of the ultrasound plane slightly rightward and inferiorly as shown in **Video 2a**, and bringing the RV and tricuspid valve into view as shown in **Video 2b**. Color flow and spectral Doppler may then be applied.

[Video 2a]

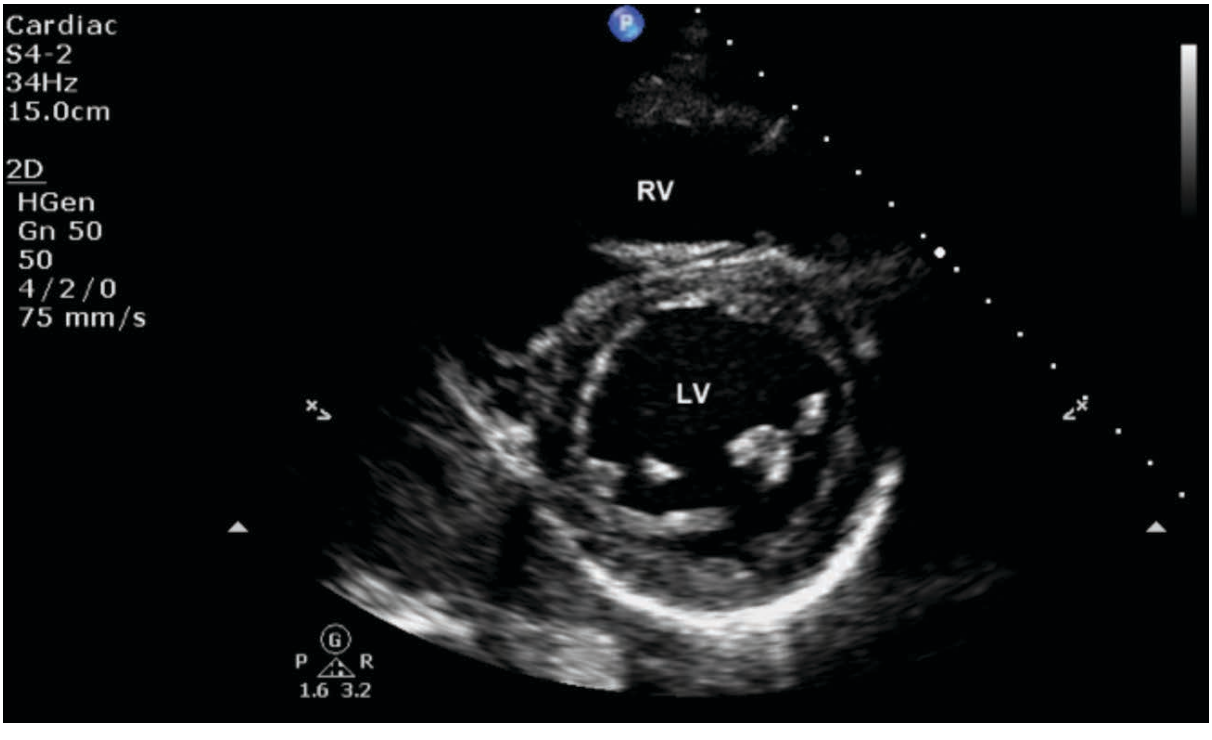
This video demonstrates how to obtain a parasternal right ventricular view. From a standard PSLA, slightly angle the transducer face inferiorly and to the right by rotating and tilting a very small amount.

[Video 2b]

This video demonstrates the transition from a standard PSLA view to a right ventricular parasternal view. As the transducer is rotated and tilted slightly, the LV is minimized while the RV and TV come into view. This also demonstrates the pitfall of how transducer angle can overemphasize the RV vs the LV.

Parasternal short-axis (PSSA) view

The parasternal short-axis (PSSA) view may be very helpful in defining RV geometry. It is obtained by rotating the transducer 90° counterclockwise (general convention) or 90° clockwise (cardiology convention) from the PSLA view so that the ultrasound plane is perpendicular to the long axis of the heart. The plane should then be fanned through the long axis so that the LV is visualized as a "donut" at the papillary muscle or mitral valve level. The RV will be anterior to the LV. A normal RV is thin-walled and crescent-shaped around the muscular circle of the LV **(Figure 5)**.



[Figure 5] **Parasternal short-axis view using the "general" orientation. Right ventricle (RV), left ventricle (LV).**

Apical four-chamber (A4C) view

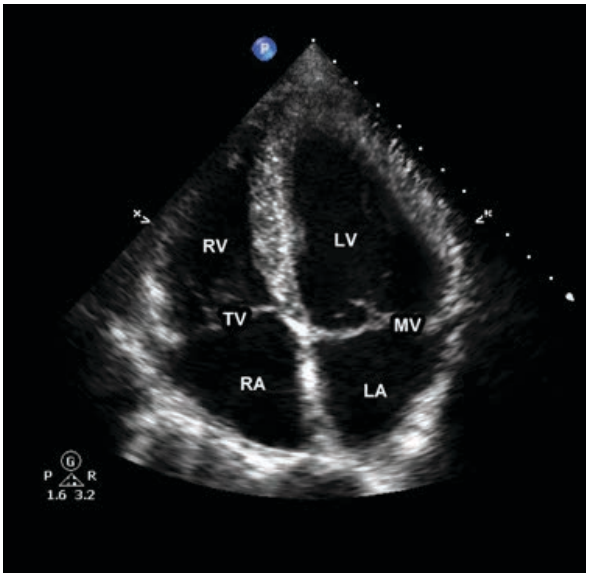
The apical four-chamber (A4C) view is often the most difficult to obtain due to overlying breast or fatty tissue and/or due to problems positioning the patient. It is, however, the most useful view for side-by-side comparison of RV-to-LV geometry and RV pressures as it allows interrogation of tricuspid valve flow that is in line with the direction of blood flow (which is optimal for Doppler).

The A4C view is obtained by placing the transducer at the point of maximal impulse (PMI) where the apex of the heart is closest to the chest wall. Typically, this is at about the fourth intercostal space, inferior and lateral to the nipple in men and underneath the breast in women. The transducer orientation marker should be directed towards the patient's right (general indicator-to-transducer orientation), or slightly towards the ceiling in a supine patient when the transducer is on the lateral part of the chest. The plane of the transducer will need to be angled slightly toward the patient's right shoulder in line with the long axis of the heart.

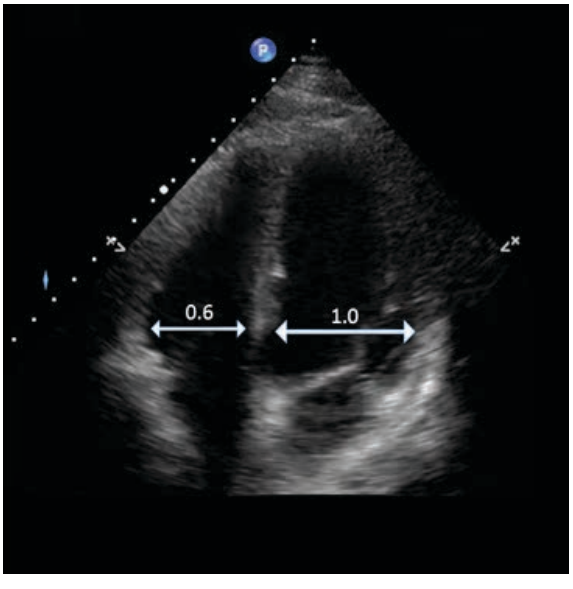
If possible, have the patient roll onto their left side and place their left forearm under their head. This positioning will often dramatically improve the ability to visualize the cardiac structures in this view.

When correctly obtained, the A4C view should include the LV, RV, LA, RA, and mitral and tricuspid valves. The apex of the heart will be at the top of the screen with the interventricular septum running vertically between the RV and the LV (the RV will be on the left of the screen). The valves and atria are farther from the transducer and are seen lower on the screen. A common error is to place the transducer too medially, which will cause the interventricular septum to be angled on the screen. If necessary, move the transducer laterally so the septum is completely vertical from the apex to the base **(Figure 6a)**.

The A4C view allows a visual comparison of the RV-to-LV diameter, which can be measured in diastole across the tips of the valves **(Figure 6b)**. A normal RV:LV ratio is approximately 0.6:1. An RV diameter equal to or greater than the LV diameter is specific for RV dilatation.



[Figure 6a]
Apical four-chamber view. Right ventricle (RV), tricuspid valve (TV), right atrium (RA), left ventricle (LV), mitral valve (MV), left atrium (LA).



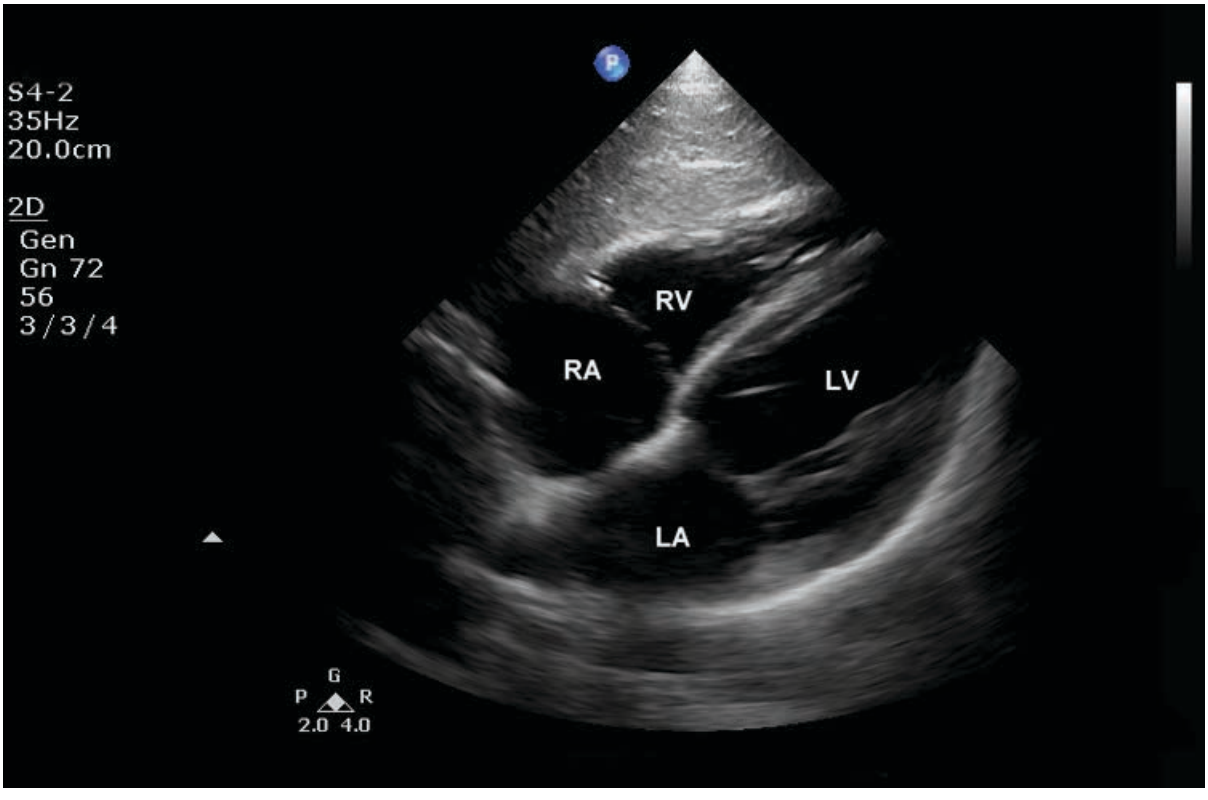
[Figure 6b]
Normal apical four-chamber image comparing RV:LV ratio.

Subxiphoid (SX) view

The subxiphoid (SX) view is best obtained by placing the right hand over the transducer (thumb will be on the transducer orientation marker pointing to the patient's right). Place the transducer under the xiphoid process and, using sufficient pressure, direct the ultrasound beam towards the heart by dropping the tail of the transducer so that it is nearly parallel to the patient's thorax.

The SX view uses the liver as a window. The right atrium, tricuspid valve, and right ventricle (adjacent to the liver) are seen more anteriorly. The left atrium, mitral valve, and left ventricle are seen more posterior **(Figure 7)**.

The subxiphoid window also allows the clinician to follow the hepatic veins into the right atrium. Scanning through the proximal inferior vena cava and right atrium into the RV may occasionally reveal thrombus, an uncommon finding but one that directly impacts management if present.



[Figure 7] Subxiphoid view. Right ventricle (RV), right atrium (RA), left ventricle (LV), left atrium (LA).

The subxiphoid view may also help define RV geometry, although it may overemphasize RV size relative to the LV size if the plane of the ultrasound beam cuts through the wide part of the RV when the plane is angled up from under the costal margin.

Due to the potential for overestimating RV size and being at a poor angle for Doppler interrogation of the tricuspid valve, the subxiphoid view is usually of the lowest utility for RV evaluation.

7 Doppler for calculation of RVSP

Doppler can be used to determine the pressure gradient between the RV and RA by interrogating the tricuspid valve and estimating the RV systolic pressure (RVSP).

Formula for the calculation of right ventricular systolic pressure (RVSP)[4]

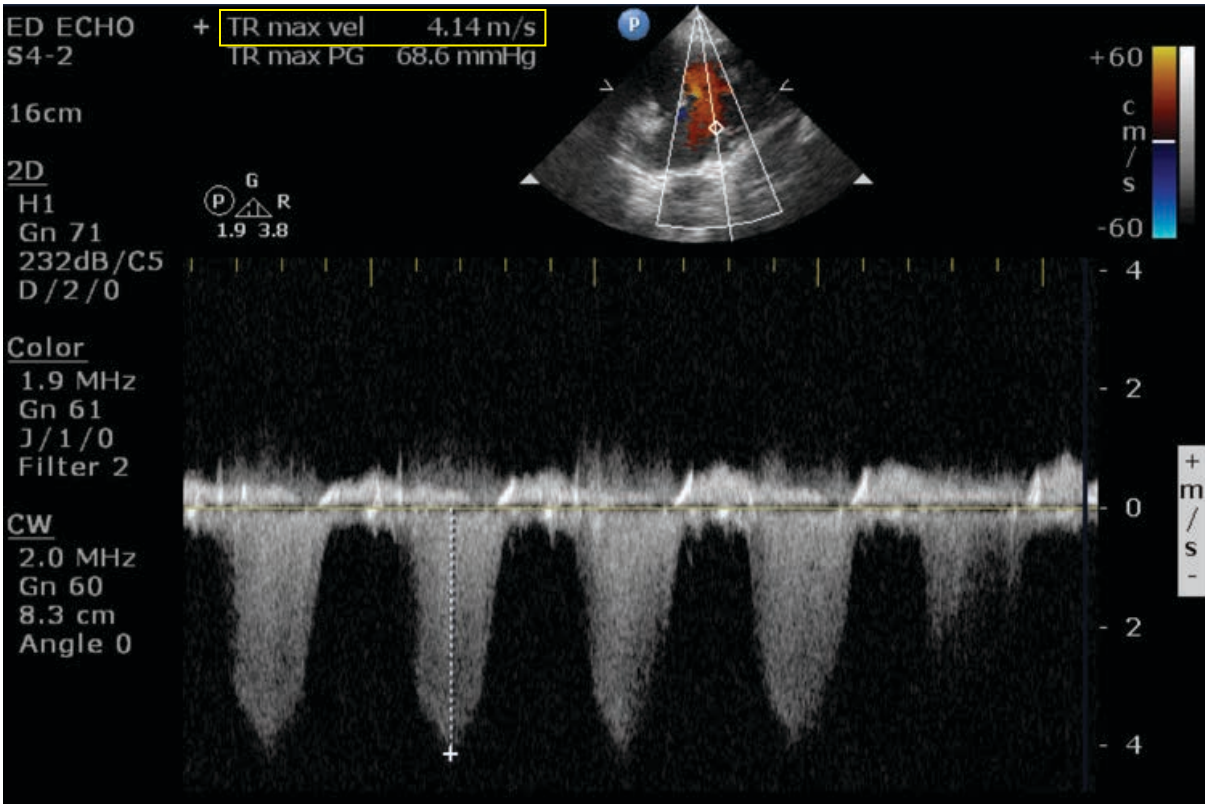
$$RVSP = P_{RV\text{-}RA} + P_{RA}$$

P = Pressure
RV = Right ventricle
RA = Right atrium

Color flow Doppler (CFD) should first be applied across the tricuspid valve to determine if tricuspid regurgitation (TR) is present. Sensitivity to flow in both color and spectral Doppler may be maximized by reducing the scale (also known as pulse repetition frequency or PRF) or by increasing the Doppler gain. Sensitivity must be balanced against noise and aliasing.

Some degree of TR may be present even in normal patients. CFD may help determine the presence and maximal intensity of regurgitant flow. In order to quantify velocity of flow and estimate the pressure gradient, spectral Doppler must be used – specifically, continuous wave (CW) Doppler as CW Doppler allows measurement of higher velocities.

If TR is present on CFD, the maximal intensity should be noted and the CW cursor placed across this area. CW Doppler will plot velocity of flow on the y-axis. TR is typically a parabolic arc below the baseline, as regurgitant flow is away from the transducer **(Figure 8)**. Scale (PRF) and baseline should be adjusted to optimize the display of the TR jet.



[Figure 8] CW Doppler demonstrating TR. Note the peak velocity of this TR jet is 4.14 m/s.

NOTE: When performing Doppler interrogation, ensure that the ultrasound beam is aligned with the direction of flow; any angle will create an underestimation of flow. Although TR is typically best obtained in the A4C view, a parasternal RV view may be obtained in some patients (angle correction of the Doppler signal may be needed).

Several cardiac cycles should be measured to ensure that the maximal TR velocity is captured. Peak velocity should then be measured.
The velocity gradient between the RV and RA can be calculated using the modified Bernoulli equation **$\Delta P = 4 \times V^2$** where **ΔP** is the pressure gradient and **V** is the maximum velocity in meters per second.

Velocity			Gradient
1m/s :	4×1^2	=	4 mmHg
2m/s :	4×2^2	=	16 mmHg
3m/s :	4×3^2	=	36 mmHg
4m/s :	4×4^2	=	64 mmHg

Estimating right atrial pressure

In order to accurately measure RVSP, the pressure gradient between the RV and RA must be added to the estimated right atrial pressure (RAP), typically ~10 mmHg, although it may range from near zero to ~20 mmHg. RAP may be estimated by interrogating the proximal inferior vena cava (IVC) for size and collapsibility. An IVC diameter greater than 2-2.5 cm and/or the absence of collapsibility indicates an elevated RAP, while a flat and/or completely collapsible IVC indicates a low RAP.

Normal RVSP is ~25 mmHg, corresponding to a TR velocity of ~2 m/s. A TR velocity of 2.7 m/s or greater is often used as a cutoff for elevated RV systolic pressure. While an acute submassive or a massive PE will elevate RVSP, an acute PE alone should not generate extremely high RVSPs, as these require RV hypertrophy from a chronic process in order to generate very high pressures. A TR velocity of 4 m/s or greater suggests chronic RV strain and hypertrophy.

8 Diagnosis of acute PE using POC echo

Signs of PE that may be evident on TTE include:

- Changes in RV geometry and/or contractility
- Elevated RV systolic pressure as measured by Doppler
- Direct visualization of thrombus in the proximal IVC or RA (rare)

Changes in RV geometry and RVSP may be acute or chronic. Certain characteristics on TTE may help differentiate acute from chronic RV strain.

Echo findings in acute RV strain may include:

- Right ventricular dilatation
- Right ventricular hypokinesis
- McConnell's sign (RV hypokinesis with apical sparing)
- Paradoxical septal motion
- D-shaped septum
- Tricuspid regurgitation
- Elevated RV systolic pressure
- Abnormal TAPSE

Some combinations of the above findings will often be evident. Indications of strain may also be present with more chronic conditions and are not necessarily specific for PE (except for McConnell's sign). Clues for differentiating chronic from acute RV strain are discussed below. McConnell's sign (described on the next page) has been shown to be fairly specific for acute RV strain (almost invariably from PE).

As discussed previously, not all PEs are large enough to cause RV strain, so the absence of these signs should not be used to exclude PE.

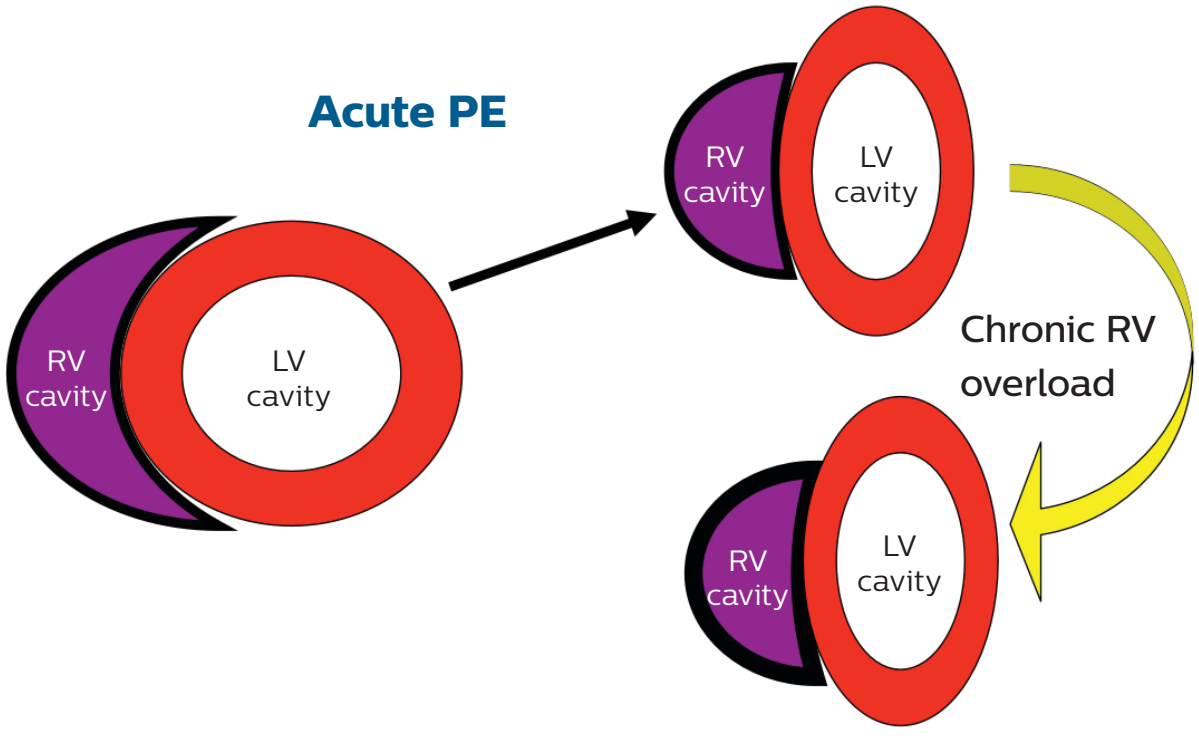
RV dilatation

RV dilatation is typically the most readily apparent sign of RV strain. As described earlier, the normal RV is a low-pressure, high-compliance system wrapped in a crescent around the LV. When pulmonary artery pressures rise from a significant PE, the RV will dilate **(Figure 9)** and become hypokinetic.

RV dilatation and hypokinesis

RV dilatation and hypokinesis may be seen qualitatively in any view, but are typically best appreciated in the PSLA, PSSA, and A4C views **(Video 3). Video 3** contrasts normal RV size and function with RV dilatation and hypokinesis in all three views.

When the RV enlarges, the PSSA view will show the characteristic "D-shaped" septum that occurs as the RV pushes into the LV, creating the flat part of the "D" that is normally circular.



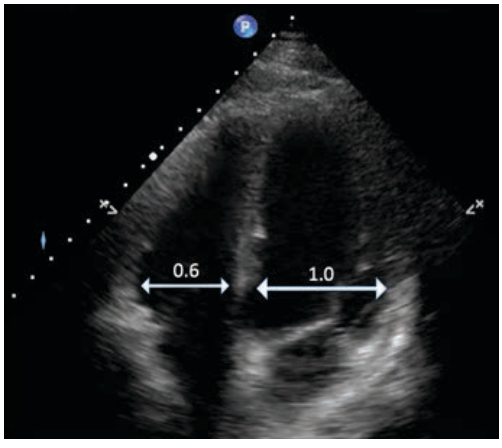
[Figure 9] In acute PE there is an increase in pulmonary artery pressure due to the obstruction by the thrombus. The RV dilates (pressing into the LV) and becomes hypokinetic. In patients with chronic RV pressure overload the RV wall hypertrophies over time, allowing higher pressures to develop.

[Video 3]

Video demonstrating normal RV size and RV dilatation in PSLA, PSSA, and A4C views. Note on the abnormal PSSA view there is paradoxical septal motion and a characteristic D-shape as the RV pushed into the LV. On the abnormal A4C view note the RV and LV appear to be roughly equal size.

The normal ratio of the maximal diameter of the RV:LV is ~0.6:1 **(Figure 6b)**. For point-of-care ultrasound, an RV:LV ratio of 1:1 or greater provides a cutoff that may be qualitatively estimated with good specificity for true RV strain.

Measurement of RV:LV diameters (if performed) is optimally done in an A4C view, across the tips of the tricuspid/mitral valves in diastole.



With careful interrogation, qualitative estimation of RV:LV ratio may be obtained using a PSLA view, taking care not to overestimate RV size relative to LV size. Overestimation of RV size can occur if a “more RV” PSLA view **(Video 2)** is used without attempting to compare RV size to a maximized LV cavity diameter.

McConnell's sign

While RV hypokinesis may be global, acute pulmonary embolism often causes the midportion of the RV to become hypokinetic while the apical portion of the RV continues to contract well.

Dilatation and akinesis of the mid-portion of the RV with "apical sparing" is known as McConnell's sign after the author who first described this sign in 1996.[5] In the initial series, this pattern was 96% specific for pulmonary embolism, and a follow-up study showed resolution of this pattern with thrombolytic therapy for PE.[6]

Examples of McConnell's sign in acute PE are shown in **Video 4**.

[Video 4]

Video demonstrating three examples of McConnell's sign. Note that while most of the RV is dilated and hypokinetic, the apex is moving vigorously. This "apical sparing" is the key finding in McConnell's sign, which is specific for acute pulmonary embolism.

Paradoxical septal motion and D-shaped septum

RV strain may also affect the interventricular septum. When RV pressure is elevated, it pushes on the septum, impairing and delaying left ventricular filling. This may be apparent as "paradoxical septal motion" **(Video 5)** in which the septum does not relax and fill normally.

When viewed in a short axis, RV pressure will cause the septum to flatten, creating the D-shape of the LV with the septum being the flat part of the D **(Video 5)**.

[Video 5]

Two examples of RV dilatation with paradoxical septal motion. Normally the septum should contract and expand from the LV center with LV systole and diastole. This expansion is delayed by RV pressure overload.

Tricuspid regurgitation

Elevations in RV pressure from acute PE will cause tricuspid regurgitation (TR), which can be identified using color flow Doppler (CFD) and quantified using spectral Doppler.

Visualization and quantification of TR is best done in the A4C view, or can be obtained using a parasternal RV view **(Video 6)**.

If CFD is applied to the hepatic veins in a subxiphoid view in a patient with significant TR, reversal of flow in the hepatic veins may be seen **(Video 7)**.

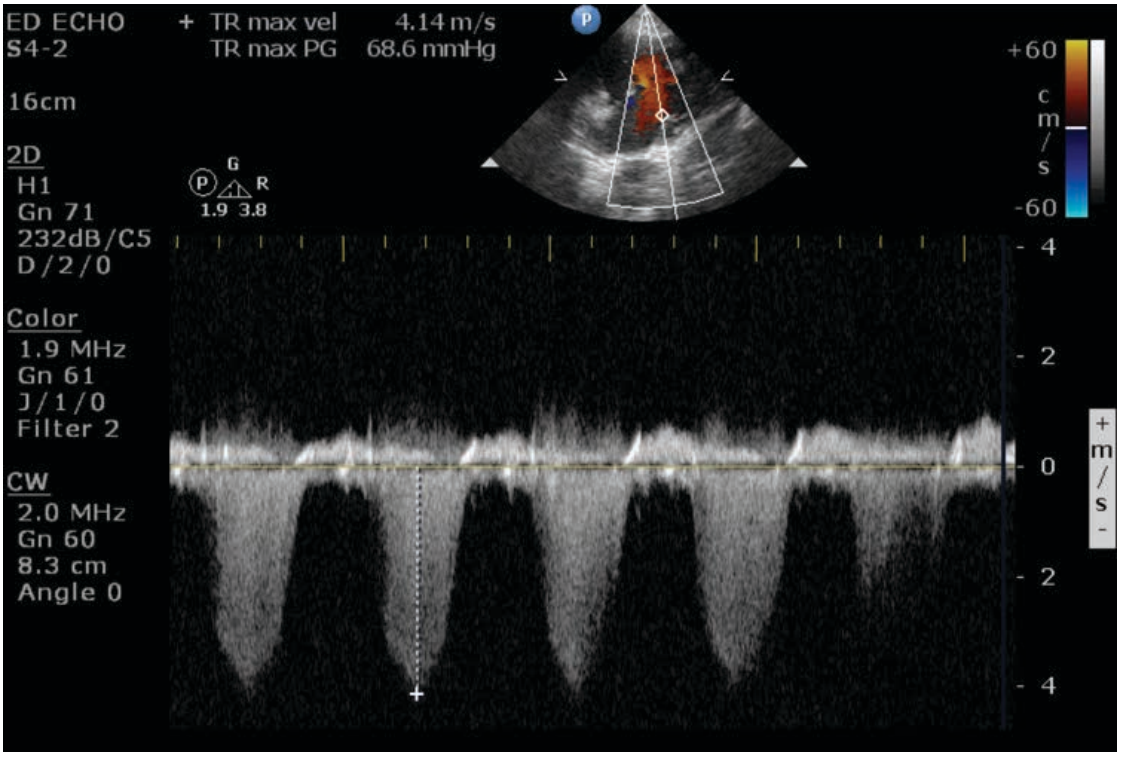
[Video 6]

Four examples of TR and marked RV dilatation.
Example 1 – Prominent TR on CFD in modified apical view. Note the right atrial enlargement.
Example 2 – RA enlargement and substantial TR jet using CFD.
Example 3 – Large TR jet on CFD using a parasternal RV view.
Example 4 – Modified apical view demonstrating RV dilatation and a small TR jet.

[Video 7]

Video demonstrating color flow Doppler of the hepatic veins in a patient with acute, severe PE. The regurgitant jet across the tricuspid is being transmitted through the right atrium (RA) into the hepatic circulation. This can cause liver congestion and right upper quadrant pain in the patient.

In order to quantitatively estimate RV systolic pressure, the maximal velocity of the TR jet should be measured. Because velocities may be quite high, continuous wave (CW) spectral Doppler should be used.



[Figure 10] **CW Doppler across the TV. The TR is measured at 4.14 m/s, corresponding to an RV-to-RA pressure differential of 68.6 mmHg (using the modified Bernoulli equation ΔP = 4 x V2). This high pressure (>60 mmHg) is indicative of chronic RV strain.**

Elevated RV systolic pressure

The ultrasound system can automatically calculate this pressure difference based on the maximal velocity. Normal RV systolic pressure (RVSP) is about 25 mmHg. While estimation of RVSP from a maximal TR jet also requires an RA pressure estimation, most sources use a TR velocity of 2.7 m/s or greater as a cutoff to indicate elevated RVSP (this correlates to a pressure gradient of 29 mmHg).

As described below, a TR jet velocity of 4 m/s or greater (pressure gradient of 64 mmHg or greater) is unlikely to occur in acute PE.

[Video 8]

Video of continuous wave (CW) Doppler across the tricuspid valve with the characteristic parabolic TR jet below the baseline.

Chronic RV strain

As mentioned before, RV strain may not always be acute. Over time, with persistent increased pressure the RV will hypertrophy, allowing higher pressures to develop. Findings on echo that indicate chronic rather than acute RV strain include:

- RV dilatation/hypokinesis without McConnell's sign
- RV hypertrophy (RV free wall >5 mm or RV septum >7 mm)
- Extremely high RV pressures (RVSP >60 mmHg)

Video 9a demonstrates an A4C view with a markedly dilated RV. However, the RV wall is circumferentially thickened, indicating a chronic process.

Video 9b demonstrates a subxiphoid view with a dilated RV with marked RV thickening (more than 1 cm), indicating chronic RV strain.

While acute RV pressure overload will cause elevated RVSP and TR, the acutely strained RV cannot typically generate extremely high pressures, as the RV does not have the muscle mass. With chronic RV strain and RV hypertrophy, RV pressures can go higher. As mentioned earlier, maximal TR velocities of 4 m/s or more indicate chronic RV strain.

[Video 9a]

A4C view with a markedly dilated RV. Note the RV wall is circumferentially thickened, indicating a chronic process. The thickness of the RV appears to be ~1 cm or more (using the calibration markers on the side of the screen as a guide). Also note that the entire RV, including the apex, does not contract well.

[Video 9b]

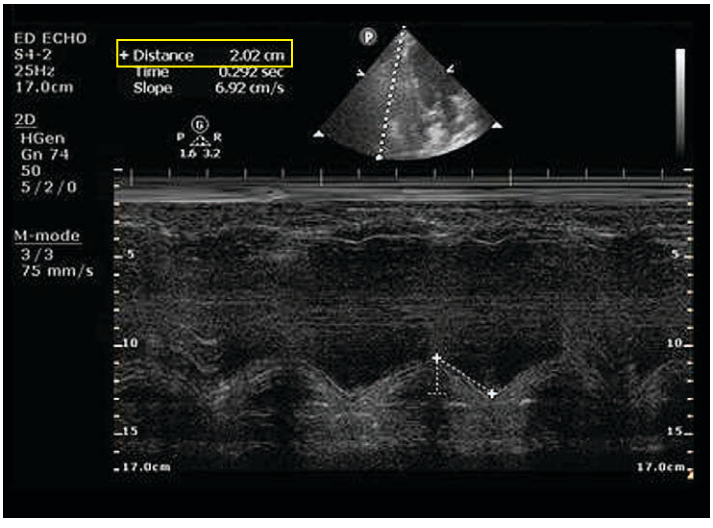
Subxiphoid view of the heart demonstrating substantial thickness of the right ventricular free wall, adjacent to the liver, consistent with RV hypertrophy from chronic RV pressure overload. If measured, an RV free wall diameter of over 5 mm is considered abnormal.

TAPSE

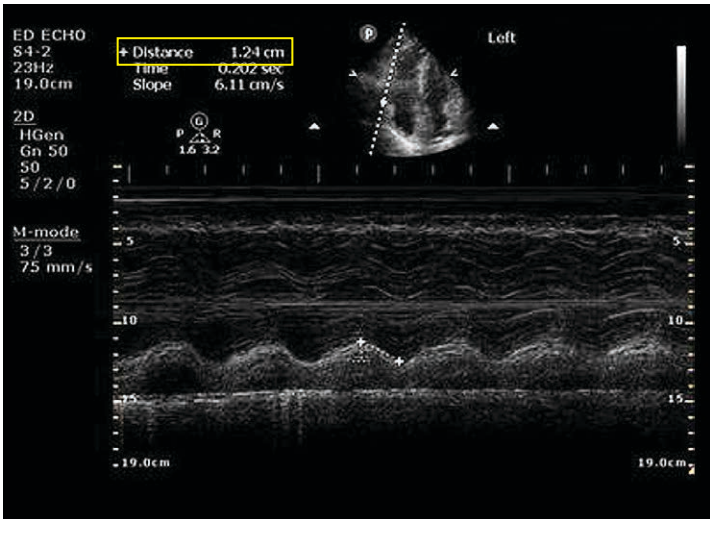
Another way to determine the degree of right ventricular dysfunction is using a measure called “tricuspid annular plane systolic excursion” or TAPSE.

While the left ventricular walls typically contract radially towards the long axis of the heart, the right ventricle moves longitudinally from the base to the apex of the heart. When the RV is strained or dysfunctional this motion (TAPSE) decreases. TAPSE has been shown to be reproducible, to have good correlation with more invasive measures of RV dysfunction, and to be an independent predictor of mortality.[8-11]

TAPSE should be obtained in an apical four-chamber view. In this view, the lateral annulus of the tricuspid valve is seen in the bottom left corner of the screen and should move from the bottom of the screen (base of the heart) towards the top of the screen (apex of the heart). By placing the M-mode cursor on the lateral part of the tricuspid annulus, the amount of tricuspid excursion can be measured **(Figures 11a** and **11b)**. Typically 17 mm or more of motion is considered normal.



[Figure 11a]
Mode tracing demonstrating a normal TAPSE, measured at 20 mm.



[Figure 11b]
M-mode tracing demonstrating an abnormal TAPSE, measured at 12 mm, in a patient diagnosed with an acute pulmonary embolism and right heart strain on echo.

[Video 10]

This video demonstrates an apical four-chamber view with normal TAPSE. Note the vigorous motion of the bright lateral annulus of the tricuspid valve at the bottom left of the image.

Direct visualization of thrombus and ancillary DVT studies

While uncommon, a thrombus may occasionally be directly visualized in the inferior vena cava (IVC), right atrium, or right ventricle **(Video 11)**. While thrombi have a characteristic appearance, other masses, such as an atrial myxoma, may have a similar appearance.

A visualized thrombus in a patient with PE is an indication for mechanical removal and/or thrombolysis.

While not a focus of this tutorial, evaluation for DVT (deep vein thrombosis) may play a role in the evaluation for PE. The presence of a DVT in a patient with symptoms of PE and/or signs of RV strain is essentially pathognomonic for pulmonary embolus from DVT. However, while nearly all PEs are thought to result from deep venous thrombi, only about 50% of patients with PE will have DVT detectable using ultrasound on the lower extremities.

[Video 11]

This video demonstrates two examples of a mobile thrombus. Example 1 – A mobile thrombus is identified in a sagittal view of the inferior vena cava (IVC) as it feeds into the right atrium. Example 2 – A thrombus is identified in a medial apical/PSLA view. Note the dilated RV with the thrombus near the RV apex.

9 Clinical case resolution

In the clinical case of the woman with cancer presenting with shortness of breath and hypotension, PE is clearly a top consideration in the differential diagnosis. However, other diagnoses may present similarly: pneumonia, sepsis, myocardial infarction, cardiomyopathy, pericardial effusion, etc. In this case, a real case that presented to our ED at 1:40 AM, bedside echo was used.

The image we obtained is shown in **Video 12**. The video demonstrates massive RV dilatation impinging on a left ventricle that is small and is having trouble filling due to RV pressure. Based on this finding, in a patient that was deemed too unstable for CT scan, intravenous thrombolysis was used within minutes of her arrival.

Video 13 shows her heart about one hour later, after fibrinolysis. While the RV is still dilated, the size has decreased substantially. The patient survived to ultimately undergo thrombectomy and was later discharged from the hospital.

[Video 12]

This somewhat limited A4C view shows a massively dilated right ventricle and a small, minimally filled left ventricle (LV). Note the time (1:40 AM).

[Video 13]

Approximately one hour later (2:44 AM) in the same patient, after systemic thrombolysis. The RV is still dilated, but the ratio to the LV has decreased and the LV is now filling and allowing for systemic circulation.

10 Summary

Integration of bedside ultrasound into patient care

The presence or absence of signs of PE on TTE may be integrated into the diagnostic evaluation of patients with suspected PE, and into the prognostic and therapeutic management of patients with PE.

Perhaps the most dramatic impact of bedside echo is in the hemodynamically unstable patient who may have PE. The history and physical examination alone of a patient with hypotension, with or without other symptoms (such as dyspnea, chest pain, or tachycardia), may initially yield a broad differential aside from PE. For example, the differential may include acute coronary syndrome, sepsis, exacerbation of COPD, aortic dissection, pericardial tamponade, or others.

If, however, PE is the cause of hemodynamic instability, RV strain is invariably present. The absence of any signs of RV strain in an unstable patient should cause the clinician to think of other etiologies. If a patient with suspected PE is in extremis and RV strain is present, TTE may provide sufficient evidence for aggressive intervention, including thrombolysis or thrombectomy, particularly when a patient is too unstable for a CT angiogram.

In patients with PE that are stable, RV strain may not be present. However, in a symptomatic patient – particularly one without known pulmonary disease – the presence of signs of acute RV strain should lead the clinician to consider PE as a much more likely diagnosis. In these cases, another diagnostic test is typically performed (often a CT angiogram) to confirm the presence of PE prior to aggressive intervention.

Additionally, in patients without contraindications, anticoagulation may be initiated if suspicion of PE is high prior to definitive diagnosis.

Once PE is diagnosed, presence of RV strain indicates an increased risk of mortality and long-term morbidity. The American Heart Association recommends the use of systemic fibrinolysis (100 mg of alteplase given over two hours) in addition to heparin for patients with PE and evidence of moderate-to-severe RV strain (hypokinesis or RVSP >40 mmHg) that do not have a contraindication to thrombolysis.[2]

Whichever therapy is chosen, the presence of RV strain indicates a more severe PE, which may merit closer in-hospital observation, perhaps including ICU-level care. Conversely, the absence of RV strain may identify a group of lower-risk patients (perhaps the absence of RV strain may be used to identify these patients) that could be safely treated without hospitalization, although outpatient treatment criteria are still being evaluated.[7]

Conclusion

While echo is only one tool of many in the evaluation of pulmonary embolism, it can play a key role in the diagnosis, prognosis, and therapy of PE. Knowledge of the basic findings of right ventricular strain that may be identified using bedside transthoracic echo may help guide clinicians in the diagnosis and management of PE. In certain cases, appropriate management based on findings from bedside TTE may be lifesaving.

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