Cardiac Point-Of-Care Ultrasound

An Emergency Medicine Review

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KEYWORDS

- Point-of-care ultrasound • POCUS • cardiac POCUS • Echocardiography
- Left ventricle • Systolic dysfunction • Diastolic dysfunction • Right ventricle
- Cardiac tamponade • Valvular dysfunction • Cardiac arrest

KEY POINTS

- Qualitative assessment is sufficient for differentiating normal and abnormal left ventricular (LV) ejection fraction.
- Patients with signs and symptoms of heart failure may have normal LV contractility, yet have poor relaxation and compliance suggestive of diastolic dysfuncion.
- The echocardiographic signs suggestive of cardiac tamponade are systolic right atrial collapse (sensitive), diastolic right ventricular (RV) collapse (specific), a plethoric and non-collapsible inferior vena cava (sensitive), and sonographic pulsus paradoxus.
- The most readily available parameters for assessing RV dysfunction are right ventricular dilation and septal dyskinesia (qualitative), as well as tricuspid annular plane systolic excursion (quantitative).
- B-mode can evaluate for valvular excursion, coaptation, and masses, whereas color and spectral Doppler can detect regurgitant jets and their velocities.
- Point-of-care ultrasound in cardiac arrest can evaluate for global cardiac function, activity versus standstill, cardiac tamponade, massive pulmonary embolism, and ventricular fibrillation.

Video content accompanies this article at http://www.emed.theclinics.com.

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INTRODUCTION

Cardiac point-of-care ultrasound (POCUS) performed at the bedside can make critical diagnoses and monitor the response to interventions in the emergency department (ED). As opposed to consultative echocardiography, cardiac POCUS serves to answer a specific clinical question. Over time, the indications and breadth of cardiac POCUS have developed to incorporate more advanced diagnoses.[1] In this review, the authors describe key aspects of cardiac POCUS alongside high-quality media.

DISCUSSION

Core Echocardiographic Views

The heart can be visualized in multiple planes (**Fig. 1**) (Video 1).

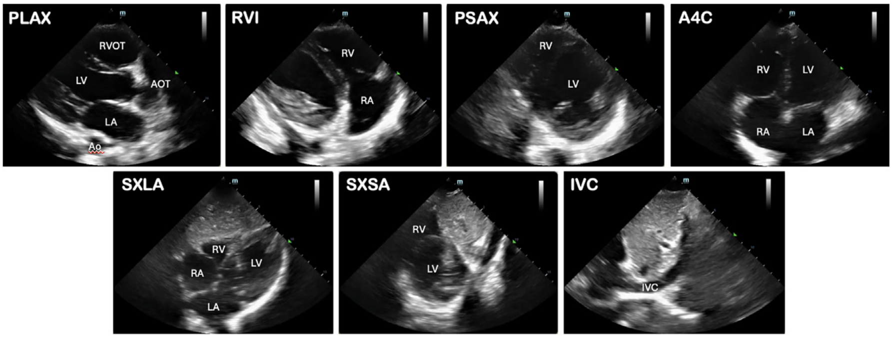

Fig. 1. Basic echocardiographic views. PLAX, parasternal long-axis; RVI, right ventricular inflow; PSAX, parasternal short-axis; A4C, apical 4-chamber; SXLA, subxiphoid long-axis; SXSA, subxiphoid short-axis; IVC, inferior vena cava.

Left Ventricular Systolic Dysfunction

Global qualitative assessment

Global left ventricular (LV) systolic function can be assessed qualitatively with strong accuracy, which improves with acquisition of multiple echocardiographic views.[2] The parameters to evaluate include: (1) how well the endocardial walls contract during systole and (2) how much those walls thicken (Video 2).

E-point septal separation

E-point septal separation (EPSS) is a quantitative measurement for differentiating patients with normal and low ejection fraction (EF). In diastole, blood travels from the left atrium (LA) to the LV through the mitral valve (MV), causing the anterior MV leaflet to open toward the interventricular septum. In early diastolic filling, the anterior leaflet reaches a point of maximal excursion (ie the E-point) and comes closest to the septum. In the parasternal long-axis (PLAX) view using M-mode, the distance between the anterior MV leaflet and septal wall is referred to as the EPSS (**Fig. 2**).

A normal, healthy anterior MV leaflet may directly contact the septum, producing an EPSS of 0 mm. In patients with decreased LV contractility, less blood flows through the MV during diastole to refill the LV, so the anterior leaflet does not fling open as widely. Moreover, there is a compensatory increase in LV cavity size that aims to maintain stroke volume. Together, these factors lead to an increase in EPSS.[3,4] One study demonstrated that an EPSS > 7 mm was 87% sensitive and

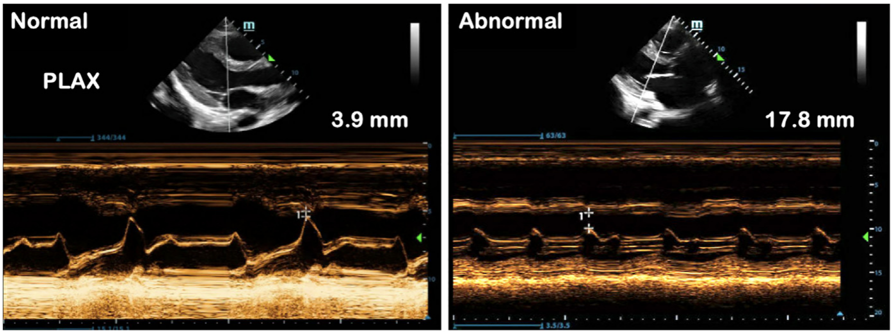

Fig. 2. E-point septal separation

75% specific for identifying an EF < 50%.[3] Another study demonstrated that an EPSS > 7 mm was 100% sensitive and 52% specific for an EF < 30%.[4] Of note, an elevated EPSS can occur despite a normal EF when opening of the anterior MV leaflet is restricted due to other etioolgies (eg mitral stenosis, aortic regurgitation [AR]).

Regional wall motion abnormalities

Echocardiography may be more sensitive for detecting cardiac ischemia than an electrocardiogram (ECG) and biomarkers (**Box 1**).[5] Therefore, assessing for echocardiographic regional wall motion abnormalities (RWMAs) should be considered in patients with clinical history and physical examination concerning for acute coronary occlusion, but with an equivocal or non-diagnostic ECG. These findings may prompt cardiology evaluation, suggest reperfusion, or provide prognostic information.

Box 1
Ischemic Cascade

Coronary hypoperfusion → diastolic dysfunction → regional wall motion abnormalities → systolic dysfunction → electrocardiogram changes → angina → biomarker elevation

RWMAs have been accurately identified by emergency physicians based on segmental wall thickening and endocardial motion using multiple echocardiographic views (Video 3).[5] These segments (eg anterior, posterior, inferior, and lateral) correspond to specific coronary arteries and their ECG distributions.

Left Ventricular Diastolic Dysfunction

Physiology and pathophysiology of diastolic filling

Normal LV filling during diastole occurs when the myocardium relaxes easily and accepts blood compliantly. The first phase of diastole (ie early filling) comprises 70-80% of LV filling, whereas the second phase (ie the atrial kick) comprises 20-30%. With diastolic dysfunction, the myocardium becomes stiff and non-compliant over time. This leads to elevations in LV and left atrial pressures (LAP), as well as LA dilatation. Upon opening of the MV in diastole, elevated LAP pushes blood into the LV. Abnormal diastolic function appears commonly even when systolic function is normal and is associated with increased mortality.[6]

Table 1
Echocardiographic assessment of diastolic dysfunction[8]

	Echocardiographic Assessment of Diastolic Function	
Parameter	1. Velocity of blood moving from left atrium to left ventricle	2. Myocardial tissue velocity
Modality	Pulsed wave Doppler	Tissue Doppler imaging
Sampling Gate Location	In between tips of open mitral valve leaflets	At septal or lateral mitral annulus
Doppler Waveform (Fig. 3A, B)	Above baseline: 1. E-wave (early filling) 2. A-wave (atrial kick)	Below baseline: 1. e′ 2. a′

Source Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2016;17(12):1321-60.

Echocardiographic assessment and interpretation

Cardiac POCUS is 89-93% sensitive and 53-80% specific for diastolic dysfunction (**Table 1**).[7] Diastolic function can be categorized through a systematic approach (**Table 2**) (**Fig. 4**).

Pericardial Effusion and Tamponade

Definition and pathophysiology

Cardiac tamponade occurs when the intrapericardial pressure from an effusion exceeds the chambers' intracardiac pressure, thus preventing one or more chambers from filling with blood.[9,10] The severity of tamponade exists along a spectrum, so cardiac tamponade is best diagnosed through a combination of the clinical examination and cardiac POCUS. The development of tamponade physiology is dependent on the rate of pericardial fluid accumulation and the pericardium's compliance (**Fig. 5**).[9,10] Nevertheless, larger effusions are more likely to cause tamponade physiology.[9,10]

Pericardial effusions are classified according to several variables (**Tables 3** and **4**).[9] False positives include a pericardial fat pad, left-sided pleural effusion, and ascites (**Figs. 6** A-C and) (Video 4A-C).[9,10]

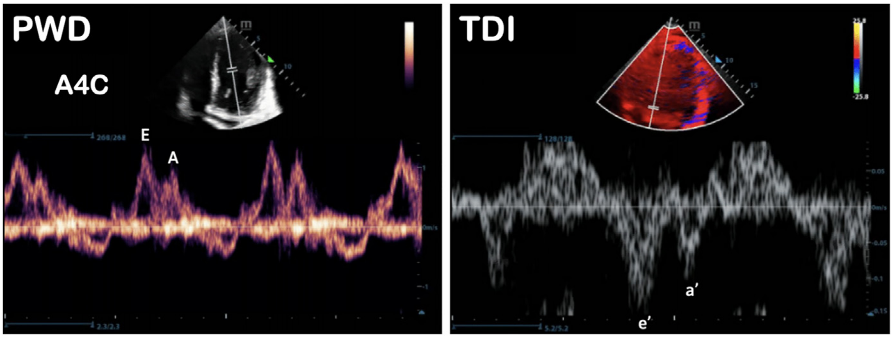

Fig. 3. Normal diastolic function.

Table 2
Diastolic function classification

Diastolic Function Classification	Pathophysiology
Normal	• Most blood flows into the LV during early filling. • A small percent of blood is pushed into the LV by the atrial kick.
Grade 1 - Mild Dysfunction (Impaired Relaxation)	• $\Delta P_{LA\text{-}LV}$ is lower, so early diastolic filling decreases. The atrial kick pushes the remainder of blood into the LV. • Impaired relaxation corresponds to a lower tissue velocity, but the LV remains compliant. • The atrial kick overcomes the ΔP, resulting in normal LAP.
Grade 2 - Moderate Dysfunction (Pseudonormal)	• The LV is now non-compliant, resulting in an increase in LAP and reestablishment of $\Delta P_{LA\text{-}LV}$. • When the MV opens early in diastole, blood is actively pushed from LA into LV.
Grade 3 - Severe Dysfunction (Restrictive)	• LAP is significantly elevated. • When the MV opens early in diastole, blood is pushed rapidly into the LV. • Diastasis is reached quickly, so there is little contribution from the atrial kick.

Abbreviations: LV, left ventricle; LAP, left atrial pressure; MV, mitral valve; LA, left atrium.

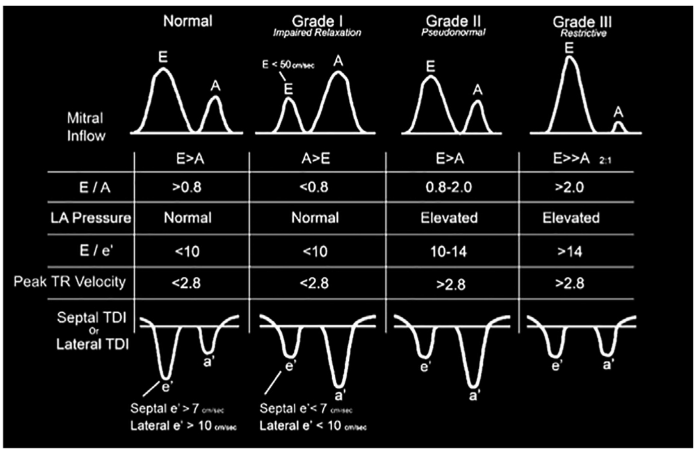

Fig. 4. Categorization of diastolic function

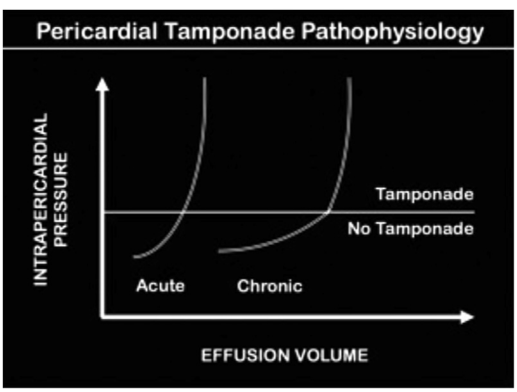

Fig. 5. Pericardial tamponade physiology. (*Adapted with permission from* Alerhand, S., R.J. Adrian, B. Long, and J. Avila, Pericardial tamponade: A comprehensive emergency medicine and echocardiography review. Am J Emerg Med, 2022. 58: p. 159–174.)

Table 3
Classification model for pericardial effusions

Hemodynamic impact	Size	Distribution	Onset	Composition
Hemodynamically stable Hemodynamically unstable	Trivial Small Moderate Large	Localized adjacent to specific chamber(s) Circumferential	Acute Subacute Chronic	Blood Transudate Exudate Air Gas

Adapted with permission from Alerhand, S., R.J. Adrian, B. Long, and J. Avila, Pericardial tamponade: A comprehensive emergency medicine and echocardiography review. Am J Emerg Med, 2022. 58: p. 159-174.

Table 4
Quantitative and qualitative size categorization of pericardial effusions

Size	Estimated Volume	Appearance	End-Diastolic Diameter Between Visceral and Parietal Pericardium
Trivial	< 50 mL	Only in systole	0 cm
Small	50–100 mL	Systole and diastole	< 1 cm
Moderate	100–500 mL	Circumferential	1–2 cm
Large	> 500 mL	Qualitatively large	> 2 cm

Adapted with permission from Alerhand, S., R.J. Adrian, B. Long, and J. Avila, Pericardial tamponade: A comprehensive emergency medicine and echocardiography review. Am J Emerg Med, 2022. 58: p. 159-174.

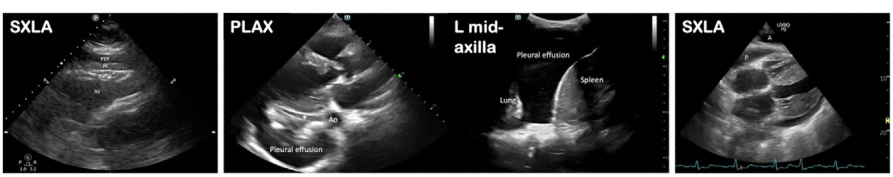

Fig. 6. False positives for a pericardial effusion: (*A*) pericardial fat pad (*B*) left-sided pleural effusion (*C*) ascites.

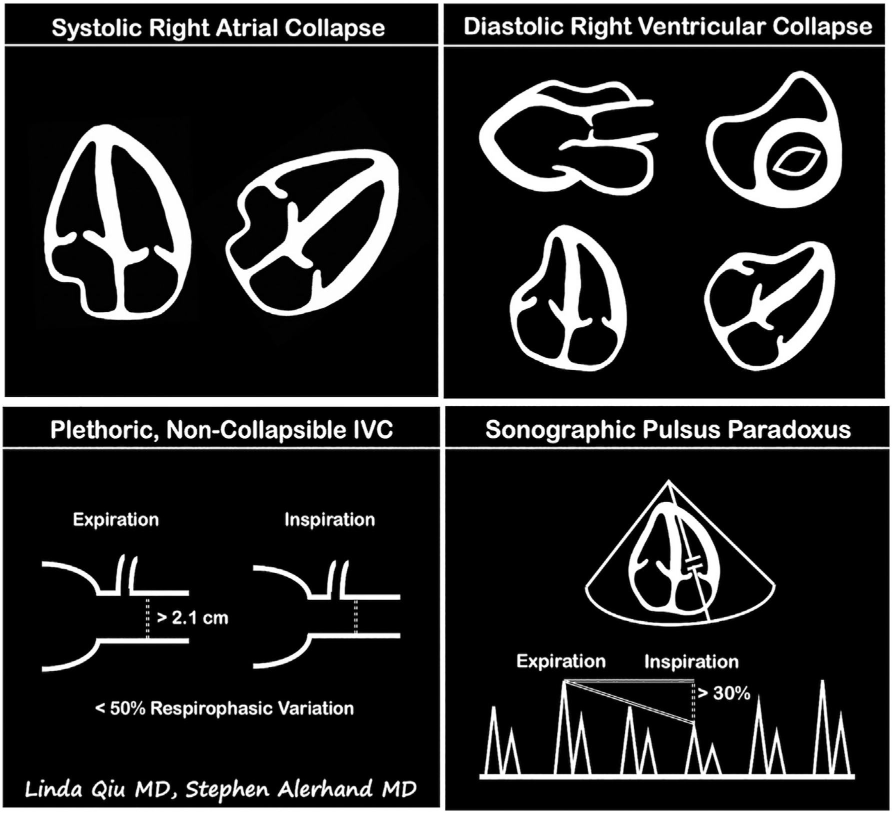

Fig. 7. Infographic of echocardiographic signs suggestive of cardiac tamponade. (*Adapted with permission from* Alerhand, S., R.J. Adrian, B. Long, and J. Avila, Pericardial tamponade: A comprehensive emergency medicine and echocardiography review. Am J Emerg Med, 2022. 58: p. 159–174.)

Echocardiographic findings suggestive of tamponade (Fig. 7)

Systolic right atrial collapse. Systolic right atrial (RA) collapse occurs when intrapericardial pressure exceeds RA intracardiac pressure (**Figs. 7 and 8** [top left]) (Video 5A).[9,10] The RA is thin-walled and carries the lowest pressure of any chamber. Therefore, its collapse in systole is the earliest sign of tamponade.[9,10] It is 50% sensitive for tamponade early in the disease but 100% sensitive with progression.[9,10] Its specificity is 33-100% and increases when the duration of collapse is greater than 1/3 of the cardiac cycle.[9,10]

Assessment for systolic RA collapse is best visualized in the apical 4-chamber (A4C) and subxiphoid long-axis (SXLA) views. With use of the ultrasound machine's cine function, clips can be frozen and cycled through time to confirm that RA collapse is in fact occurring during systole (ie when the atrioventricular valves are closed).

Diastolic right ventricular collapse. Diastolic right ventricular (RV) collapse occurs when intrapericardial pressure exceeds RV intracardiac pressure (**Figs. 7** and **8**, top right) (Video 5A).[9,10] The longer the duration of the chamber's wall collapse during diastole, the more advanced the tamponade physiology has progressed.[9,10] This sign is 48-60% sensitive and 75-90% specific for tamponade.[9,10]

Assessment of diastolic RV collapse is best visualized in the PLAX, parasternal short-axis (PSAX), A4C, SXLA, and subxiphoid short-axis (SXSA) views. In addition

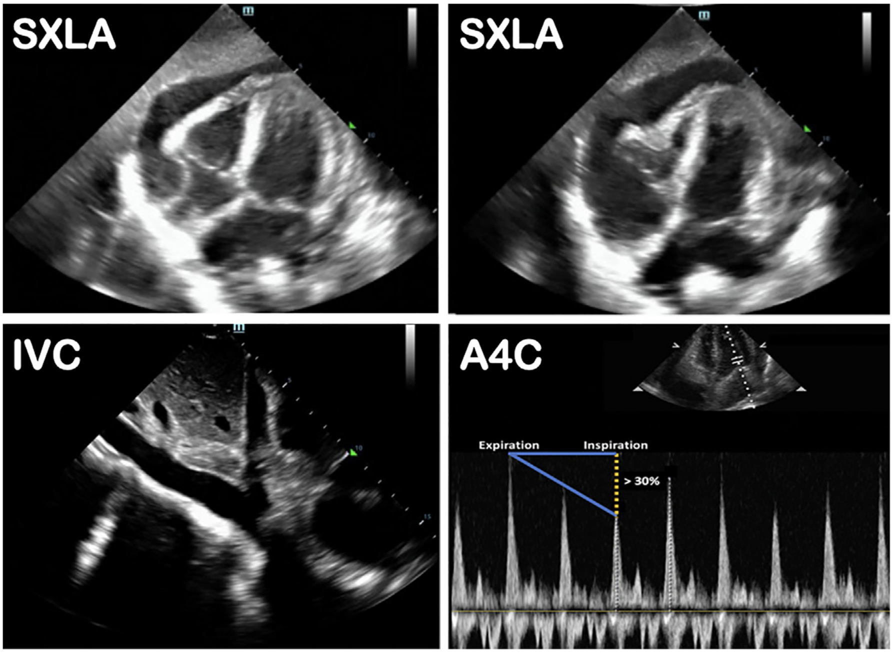

Fig. 8. Echocardiographic signs suggestive of cardiac tamponade. Top left: systolic RA collapse; top right: diastolic RV collapse; bottom left: plethoric, non-collapsible IVC; bottom right: sonographic pulsus paradoxus.

to use of the cine function, the higher temporal resolution of M-mode can be utilized by directing the beam through the RV outflow tract's (RVOT) free wall and the tip of the anterior MV leaflet (**Fig. 9**).

Plethoric inferior vena cava with minimal respirophasic collapsibility. When increased intrapericardial pressure collapses the RA, incoming preload to that chamber is diminished, thereby producing a dilated, plethoric inferior vena cava (IVC) proximally (bottom left) (**Figs. 7** and **8**, bottom left) (Video 5b).[9] This finding is 95-97% sensitive for tamponade.[9,10] Assessment of IVC diameter and respirophasic collapsibility is best visualized in the subxiphoid area in long-axis, approximately 3 cm from the IVC-RA junction. For the purposes of this specific assessment, a qualitative determination of plethoric versus thin and collapsible is sufficient.[9,10]

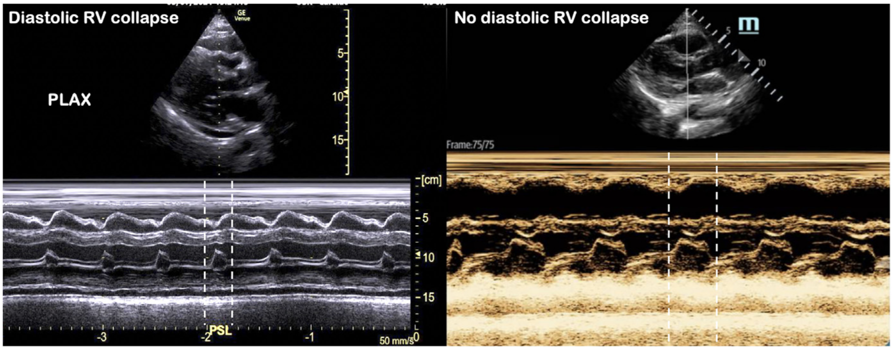

Fig. 9. M-mode evaluating for diastolic right ventricular collapse.

Table 5
Sonographic pulsus paradoxus

Evaluation of Sonographic Pulsus Paradoxus		
Parameter being evaluated	MV inflow velocity	TV inflow velocity
Pulsed wave Doppler sampling gate location	In between the open MV leaflet tips	In between the open TV leaflet tips
Change in waveform amplitude with inspiration	$\geq$ 30% decrease	$\geq$ 60% increase

Abbreviations: MV, mitral valve; TV, tricuspid valve.

Sonographic pulsus paradoxus. Sonographic pulsus paradoxus in the A4C view serves as a surrogate for its physical examination correlate (ie decrease in inspiratory systolic blood pressure by > 10 mm Hg) (**Table 5**) (**Figs. 7** and **8**, bottom right).[9,10] In the presence of a pericardial effusion, this finding carries a positive likelihood ratio of 3.3 and negative likelihood ratio of 0.03 for tamponade.[9,10]

Right Ventricular Dysfunction

Value of recognizing acute and chronic right ventricular dysfunction

Cardiac POCUS can evaluate for acute RV dysfunction such as with pulmonary embolism (PE) (**Fig. 10**).[11] It can also help differentiate between acute and chronic etiologies such as pulmonary hypertension (PH) (**Fig. 11**).[12]

Increased right ventricular:left ventricular size ratio

In patients with RV dysfunction, the RV:LV size ratio increases due to elevated pulmonary vascular resistance and RV afterload (**Table 6**) (**Fig. 12**) (Video 6A-C). This increase in pressure causes the normally thin, compliant RV free wall to dilate outward.

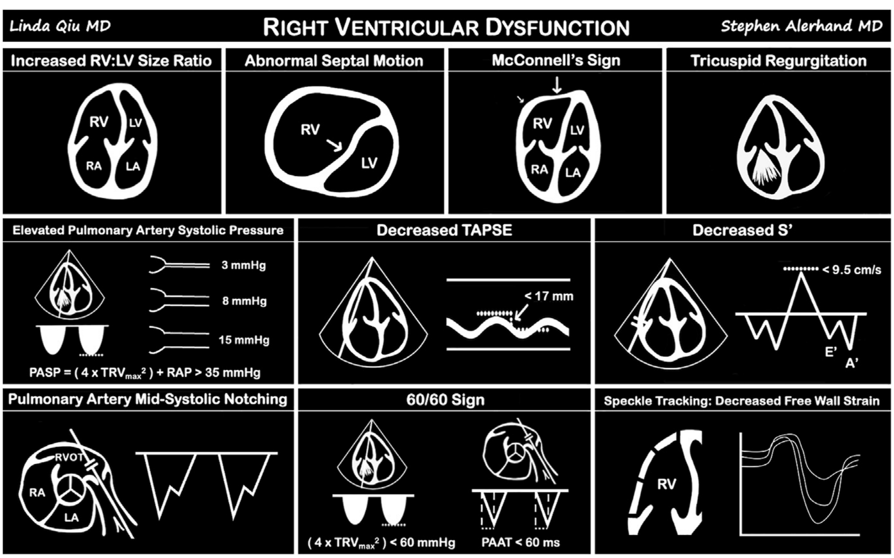

Fig. 10. Infographic of echocardiographic signs suggestive of acute right ventricular dysfunction. (*Adapted with permission from* Alerhand, S., T. Sundaram, and M. Gottlieb, What are the echocardiographic findings of acute right ventricular strain that suggest pulmonary embolism? Anaesth Crit Care Pain Med, 2021. 40(2): p. 100852.)

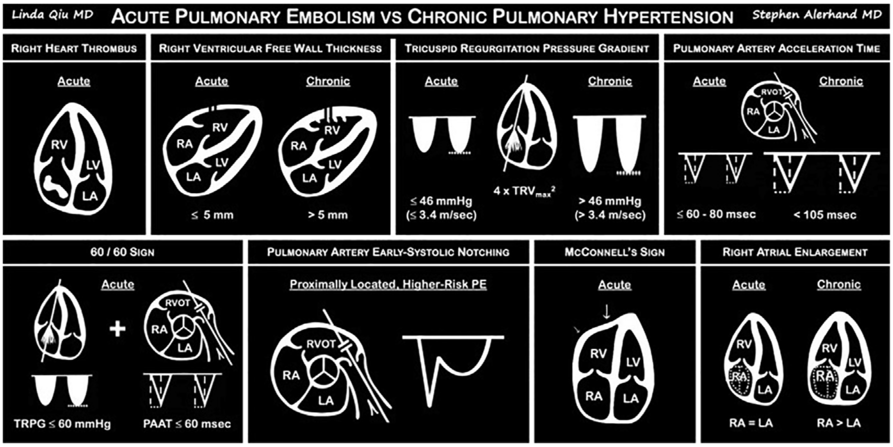

Fig. 11. Infographic of echocardiographic signs distinguishing acute pulmonary embolism and chronic pulmonary hypertension. (*Adapted with permission from* Alerhand, S. and R.J. Adrian, What echocardiographic findings differentiate acute pulmonary embolism and chronic pulmonary hypertension? Am J Emerg Med, 2023. 72: p. 72–84.)

Table 6
Right ventricular size assessment

Size Ratio of Right Ventricle: Left Ventricle[14]			
Normal	Mild Enlargement	Moderate Enlargement	Severe Enlargement
0.67:1	0.67:1–1:1	1:1–1.5:1	1.5:1

Source: Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr. 2010;23(7):685-713; quiz 86-8.

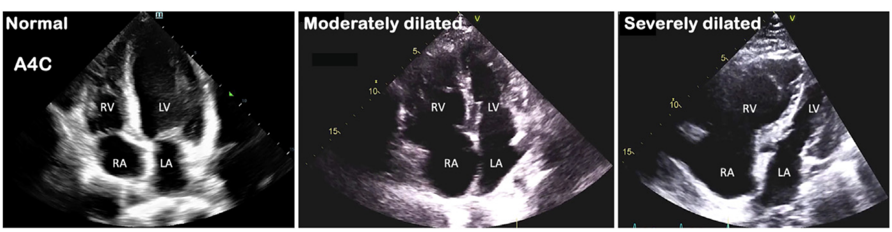

Fig. 12. Right ventricular size

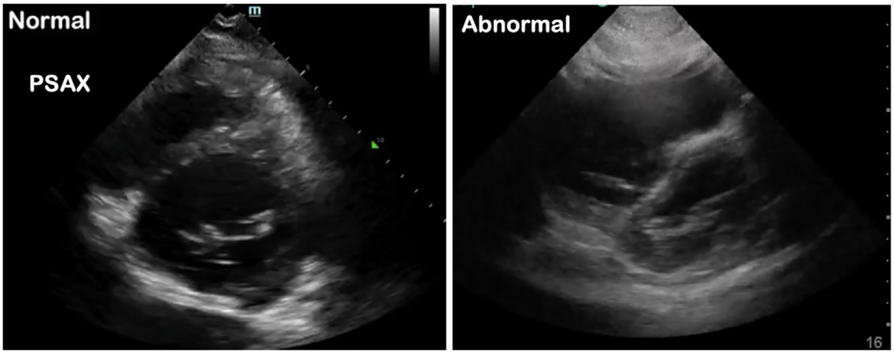

Fig. 13. Interventricular septal kinetics.

A meta-analysis of physician-performed echocardiograms found that the RV:LV ratio was 46% sensitive and 91% specific for PE.[13] This assessment is best visualized in the A4C or SXLA views.

Abnormal septal motion

In patients with normal physiology, the interventricular septum moves toward the RV in systole due to higher LV filling pressures. In patients with RV dysfunction, elevated right-sided pressures cause the septum to flatten or bow into the LV, demonstrating abnormal septal motion or septal dyskinesia (**Fig. 13**) (Video 7). In a meta-analysis of physician-performed echocardiograms, septal dyskinesia was 28% sensitive and 96% specific for PE.[13] Sensitivity increases in patients who are tachycardic or hypotensive.[15] Assessment for abnormal septal motion is best visualized in the PSAX and SXSA views at the level of the papillary muscles (ie the “D-shaped septum”), but can also be seen in the A4C and SXLA views.

Decreased tricuspid annular plane systolic excursion

Tricuspid annular plane systolic excursion (TAPSE) represents a quantitative estimation of RV longitudinal systolic function that correlates well with advanced imaging modalities and 30-day PE-related mortality.[16] TAPSE measures the vertical displacement of the lateral tricuspid annulus during the cardiac cycle using M-mode in the A4C view. Guidelines posit that a TAPSE < 17 mm (normal average 24 mm) suggests RV systolic dysfunction (**Fig. 14**).[17]

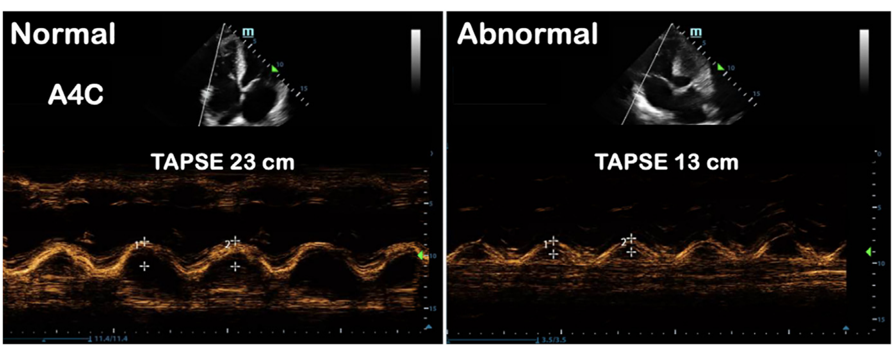

Fig. 14. Tricuspid annular plane systolic excursion.

Box 2
Estmimation of tricuspid regurgitation pressure gradient (TRPG)[14]

1. Color Doppler: Visualize a tricuspid regurgitation jet (Video 8).
2. Continuous wave Doppler: Obtain the maximal jet velocity (TRV_{max}) (Fig. 15).
3. Input TRV_{max} into the simplified Bernoulli equation ($TRPG = \Delta P_{RV\text{-}RA} = 4 \times TRV_{max}^2$), or allow automatic calculation by ultrasound machine software.[14]

Abbreviations: RV, right ventricle; RA, right atrium; TRV_{max}, maximal tricuspid regurgitation jet velocity.

Source: Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr. 2010;23(7):685-713; quiz 86-8.

Increased tricuspid regurgitation pressure gradient and pulmonary artery systolic pressure

The Doppler-derived tricuspid regurgitation pressure gradient (TRPG) non-invasively estimates the difference in pressure between the RV and RA ($\Delta P_{RV\text{-}RA}$) (Box 2). In patients with RV dysfunction, increased pulmonary artery (PA) pressure leads to RV dilation, outward bulging of the anterior tricuspid valve (TV) leaflet, decreased coaptation of the TV leaflets, and resultant functional (ie, secondary) tricuspid regurgitation (TR) (Table 7).[11]

Assessment of TRPG can be performed in any view in which the continuous wave Doppler (CWD) beam and direction of TR flow can be oriented roughly parallel: right ventricular inflow (RVI), PSAX at aortic valve (AV) level, A4C, SXLA, and SXSA at AV level. The angle between CWD beam and TR flow should ideally be 0° but is recommended to lie within 20°, as larger errors become compounded in the simplified Bernoulli equation.[12] Of note, the PA systolic pressure can be estimated by adding the estimated RA pressure (derived from IVC diameter and respirophasic variation[11]) to the TRPG. A thin, collapsible IVC corresponds to 3 mmHg. A dilated, non-collapsible IVC corresponds to 15 mmHg. An IVC that does not meet either category corresponds to 8 mmHg.

Decreased pulmonary artery acceleration time

PA acceleration time (PAAT) refers to the duration from onset of PA flow to its peak velocity on the PA systolic ejection waveform (Fig. 16) (Table 8). Assessment of PAAT is performed

Table 7
Tricuspid regurgitation pressure gradient ranges

Physiology	Maximal Tricuspid Regurgitation Velocity (TRV_{max}) (m/s)	Tricuspid Regurgitation Pressure Gradient (TRPG) (mmHg)
Normal[12]	2.0–2.1	16.0–18.3
Elevated RV systolic and pulmonary arterial pressures[14]	> 2.8–2.9	> 31.4–33.6
Acute RV dysfunction[12]	≤ 3.4	≤ 46
Chronic RV dysfunction[12]	> 3.4	> 46

Abbreviation: RV, right ventricle.
Source: 12.Alerhand S, Adrian RJ. What echocardiographic findings differentiate acute pulmonary embolism and chronic pulmonary hypertension? Am J Emerg Med. 2023;72:72-84.14. Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr. 2010;23(7):685-713; quiz 86-8.

Table 8
Pulmonary artery acceleration time[12]

Pulmonary Circulation Physiology	Pulsed Wave Doppler Waveform	Pulnonary Artery Acceleration Time (msec)
Normal	Smooth, dome-shaped	> 105; average 138.7
Low compliance, high resistance (↑ probability of pulmonary hypertension)	Triangular-shaped	< 105
Pulmonary hypertension (sufficient time to develop compliance)	Triangular-shaped	81–104
Pulmonary embolism (less time to develop compliance)	Steeper, narrower triangle	$\leq$ 60–80

Source: 12.Alerhand S, Adrian RJ. What echocardiographic findings differentiate acute pulmonary embolism and chronic pulmonary hypertension? Am J Emerg Med. 2023;72:72-84.

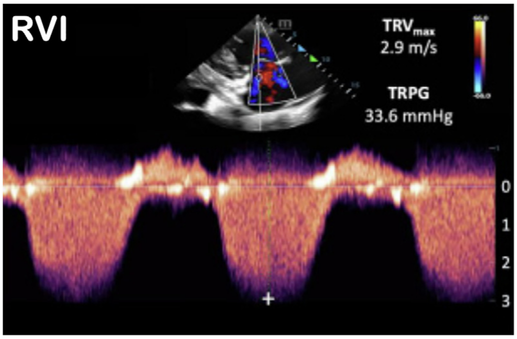

Fig. 15. Obtaining the tricuspid regurgitation pressure gradient using continuous wave Doppler.

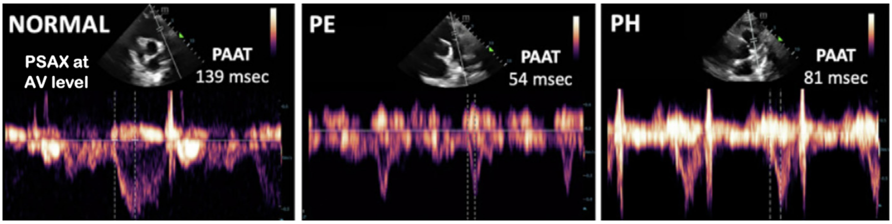

Fig. 16. Pulmonary artery acceleration time.

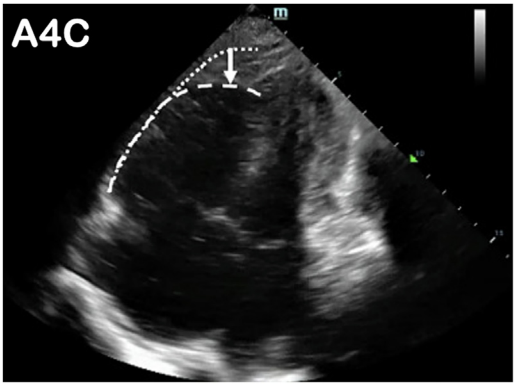

Fig. 17. McConnell's sign.

in the PSAX or SXSA views at the AV level, ensuring visualization of the RV outflow tract (RVOT), PA, and pulmonic valve (PV). The PWD sampling gate should be placed 0.5-1 cm proximal to the PV, in approximate alignment with the direction of blood flow.[12]

60/60 sign

The 60/60 sign is the combination of TRPG $\leq$ 60 mm Hg (**Fig. 15**) and PAAT $\leq$ 60 msec (**Fig. 16**B).[11,12] Both findings on their own (and together) suggest acute RV dysfunction. The 60/60 sign is 13-71% sensitive and 69-98% specific for PE.[12] It is more sensitive for proximally-located, higher-risk PEs than for lower-risk PEs.[12]

McConnell's sign

McConnell's sign refers to regional RV free wall hypokinesis with intact apical contraction, as visualized in the A4C view (**Fig. 17**) (Video 9).[11,12] An systematic review and meta-analysis (SRMA) reported McConnell's sign as 22% sensitive and 97% specific for PE.[13] Evidence shows higher sensitivity in proximally-located, higher-risk PEs.[12]

Valvular Dysfunction

Evaluation of the cardiac valves can be performed systematically (**Table 9**).

Color Doppler assessment

Color jet area. Color Doppler allows visual estimation of a regurgitant jet (**Fig. 18**) (Video 8). Of note, an eccentric jet (ie directed toward the atrium's medial or lateral wall) demonstrates a smaller jet area than a centrally-directed jet of equal or lower severity. This occurs due to loss of the jet's kinetic energy as it gets absorbed by the atrial wall.

Color jet width relative to left ventricular outflow tract. Color jet width refers to the ratio of the AR jet width and LVOT diameter (**Fig. 19**) (Video 10), best obtained in PLAX just proximal to the AV.

Table 9
Approach to valvular assessment

B-mode	Color Doppler
Leaflet excursion (ie how well the leaflets open)	Color jet area
Leaflet coaptation (ie how well the leaflets close together)	Color jet width relative to left ventricular outflow tract (aortic regurgitation)
Masses or vegetations	Vena contracta width

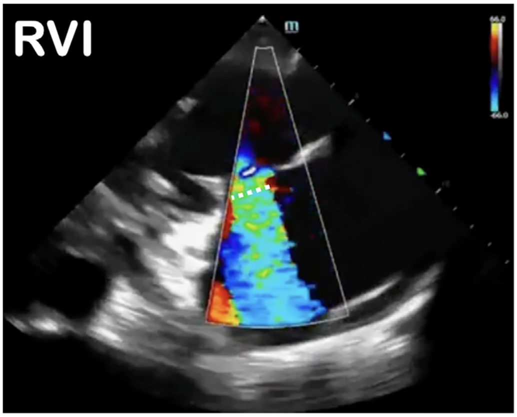

Fig. 18. Color jet area and vena contracta (denoted by *dotted line*) of tricuspid regurgitation.

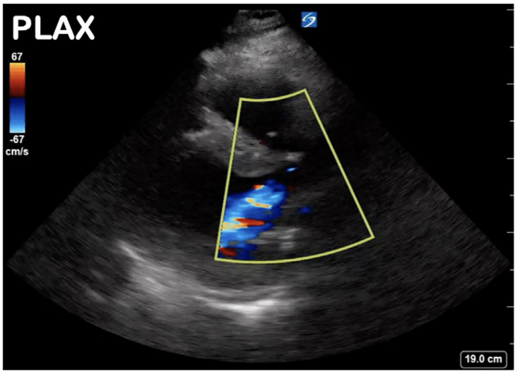

Fig. 19. Color jet width of aortic regurgitation.

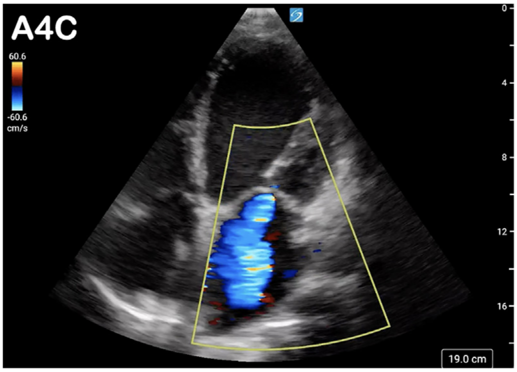

Fig. 20. Mitral regurgitation.

Table 10
Acute mitral regurgitation categorization[18]

Mitral Regurgitation	B-Mode	Color Jet Area of Left Atrium (%)	Vena Contracta Width (cm)
Mild	Normal leaflets Normal left atrial and ventricular size	< 20	< 0.3
Moderate	-	20–40	0.3–0.7
Severe	Enlarged left atrium Flail leaflet Papillary muscle rupture	> 40 or eccentric	> 0.7
Common causes	Endocarditis Papillary muscle rupture Ischemic papillary muscle dysfunction Chordae tendon rupture		

Source: Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr. 2003;16(7):777-802.

Table 11
Acute aortic regurgitation categorization[18]

Aortic Regurgitation	B-Mode	Color Jet Width/ LVOT Width (%)	Vena Contracta Width (cm)
Mild	Normal leaflets Normal left ventricular size	< 25	< 0.3
Moderate	Normal or abnormal leaflets Normal or dilated left ventricle	25–65	0.3–0.6
Severe	Flail leaflet or coaptation defect Dilated left ventricle	> 65	> 0.6
Common causes	Endocarditis Aortic dissection Traumatic valve rupture		

Source: Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr. 2003;16(7):777-802.

Table 12
Tricuspid regurgitation categorization[18]

Tricuspid Regurgitation	B-Mode	Color Jet Area of Right Atrium (%)	Vena Contracta Width (cm)
Mild	Normal leaflets Normal right atrial and ventricular size	Thin, small, central	< 0.3
Moderate	-	-	0.3–0.69
Severe	Flail leaflet or coaptation defect Dilated right atrium and ventricle, inferior vena cava dilation	> 50	$\geq$ 0.7
Common causes	Endocarditis Acute pulmonary embolism Pulmonary hypertension		

Source: Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr. 2003;16(7):777-802.

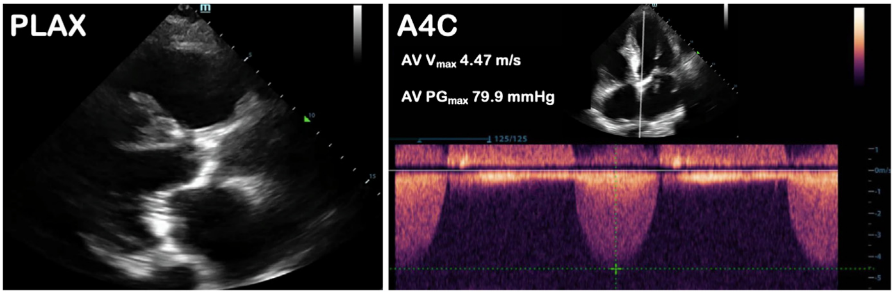

Fig. 21. Aortic stenosis: (*A*) B-mode (*B*) Continuous wave Doppler.

Vena contracta. The vena contracta is the narrowest diameter of the regurgitant jet, taken just proximal to the regurgitant orifice (**Fig. 18**) (Video 8). It correlates with the size of the effective regurgitant orifice and severity of regurgitation.[18]

Valvular regurgitation

Mitral (**Fig. 20**) (Video 11), aortic (see **Fig. 19**) (Video 10), and tricuspid regurgitation (**Fig. 18**) (Video 8) can be categorized by severity (**Tables 10–12**).

Valvular Stenosis

The assessment for aortic stenosis (**Fig. 21**) (Video 12) can be approached systematically (**Table 13**) (**Box 3**) (**Table 14**).[19]

Endocarditis

A valvular vegetation appears as an irregular, lobulated, or amorphous shape with high-frequency oscillating movement (**Fig. 22**A) (Video 13B).[12] It attaches to the proximal, lower-pressure side of the valve (ie in the path of the associated high-velocity regurgitant jet) or to implanted prosthetic material—though it oscillates independently of these structures. It must be differentiated from a right heart thrombus that floats freely within the chambers.

Cardiac Arrest

Transthoracic echocardiography can identify reversible causes of cardiac arrest and guide pathology-specific management.[20] When the etiology of the arrest is identified, there is a significant survival benefit.[21] Nevertheless, despite the benefits of POCUS, its use during pulse checks must not cause delay in the resumption of high-quality chest compressions (**Box 4**).[20]

Table 13
Echocardiographic evaluation of aortic stenosis

B-mode (Fig. 21A) (Video 10)	Spectral Doppler
Hyperechoic, calcified valve	Continuous wave Doppler: Peak systolic velocity across the aortic valve (V_{max}) (Fig. 21B)
Reduced leaflet excursion	Continuous wave Doppler: Mean $\Delta P_{aorta\text{-}LV}$ during systole, determined by simplified Bernoulli equation ($\Delta P = 4V_{max}^2$) (Fig. 21B)
Small valve orifice	Aortic valve area based on the continuity equation

Box 3
Determination of aortic valve area

Continuity equation: flow passing through the LVOT is equal to flow through the AV.

$CSA_{LVOT} = \pi\ [(LVOT_{diameter})/2]^2$

$Area_{AV} = (Area_{LVOT} \times VTI_{LVOT})/VTI_{AV}$

–$LVOT_{diameter}$ is obtained from the parasternal long-axis view.

–VTI_{LVOT} is obtained using pulsed wave Doppler in the apical 5-chamber view.

–VTI_{AV} is obtained using continuous wave Doppler in the apical 5-chamber view.

Abbreviations: AV, aortic valve; CSA, cross-sectional area; LVOT, left ventricular outflow tract; AVA, aortic valve area; VTI, velocity time integral.

Table 14
Aortic stenosis categorization

Aortic Stenosis	Aortic Valve Area (cm^2)	Mean Pressure Gradient Between Aorta and Left Ventricle (mmHg)	Peak Systolic Velocity Across Aortic Valve (m/s)
Normal	-	-	< 2.5
Mild	> 1.5	< 20	2.6–2.9
Moderate	1.0–1.5	20–40	3–4
Severe	< 1.0	> 40	> 4
Common Causes	Congenitally abnormal valve (e.g. bicuspid valve) Inflammation Age-related calcification		

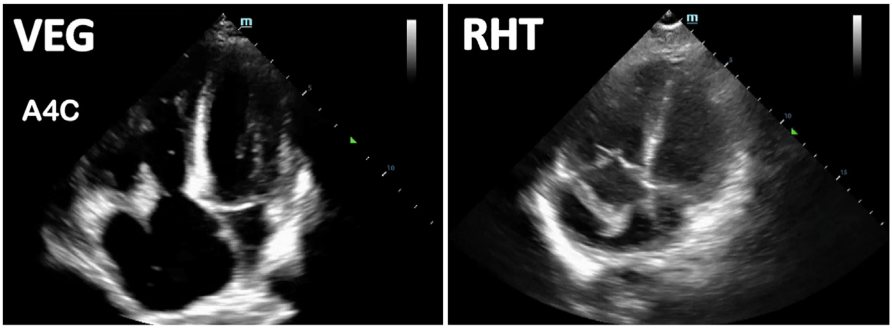

Fig. 22. Endocarditis vegetation versus right heart thrombus.

Box 4
Pearls for minimizing pauses in chest compressions[20]

Most experienced sonographer performs the scan

Scan performed by non-team leader

Perform non-cardiac evaluations (eg lung, airway, deep vein thrombosis) during ongoing chest compressions

Pre-scan prior to pausing compressions to identify the optimal cardiac window

Record the clip during the pause, then analyze it once compressions have resumed

Use a nearby towel to wipe off ultrasound gel immediately

A designated person counts down from 10 seconds

Source: Gottlieb M, Alerhand S. Managing Cardiac Arrest Using Ultrasound. Ann Emerg Med. 2023;81(5):532-42.

Cardiac tamponade

In a large prospective ED study, a pericardial effusion was found in 4% of patients with nontraumatic cardiac arrest.[22] Those who underwent pericardiocentesis had a higher survival-to-discharge rate than those who did not (15% vs 1%, respectively).[22] In cases of traumatic arrest, approximately 10% occurred due to cardiac tamponade.[23] Emergency physicians evaluating pulseless electrical activity (PEA) and cardiac arrest patients have identified pericardial effusions and cardiac tamponade with strong positive predictive value.[24] However, the echocardiographic signs of tamponade (described earlier) are less reliable in cardiac arrest due to the absence of normal forward flow and cardiovascular pressures (Video 14). Therefore, a pericardial effusion found in a patient in cardiac arrest should be drained when there is heightened suspicion that this effusion is causing tamponade.[20]

Massive pulmonary embolism

Data suggests that PE may be the primary etiology of cardiac arrest in approximately 2-13% of cases.[20] However, echocardiographic signs that normally suggest PE are less reliable in cardiac arrest patients who do not have the normal intracardiac pressures and pressure gradients. For example, right-sided dilation also can occur due to lack of forward blood flow.[20] Moreover, new thrombus formation can occur within 6 minutes of arrest due to the low-flow state (**Fig. 22**B) (Video 13A).[20] A non-cardiac adjunct in early arrest that could raise suspicion for PE as the cause of cardiac arrest is the diagnosis of a deep vein thrombosis.[20]

Ventricular fibrillation

Data suggests that ventricular fibrillation (VF) occurs in approximately 17% of out-of-hospital cardiac arrests (OHCA) and 13% of in-hospital cardiac arrests (IHCA).[25] Fine VF appears less pronounced on the cardiac monitor, but can be visualized on the ultrasound screen as rhythmic twitching movements of the myocardium (Video 15). Given the improved outcomes of shockable versus non-shockable rhythms, even for fine VF, the differentiation between shockable VF and asystole is critical.

Aortic dissection

A meta-analysis of OHCA found a pooled incidence of acute aortic dissection of 4% with 100% mortality (**Box 5**).[26]

Box 5
Echocardiographic findings suggesting aortic dissection[27]

Intimal flap (direct)

Aortic root enlargement (indirect) (**Fig. 23**) (Video 16)

Aortic regurgitation (indirect) (see **Fig. 19**) (Video 10)

Hemorrhagic pericardial effusion (indirect) (**Fig. 24**) (Video 17)

Source: Sutarjono B, Ahmed AJ, Ivanova A, et al. Diagnostic accuracy of transthoracic echocardiography for the identification of proximal aortic dissection: a systematic review and meta-analysis. Sci Rep. 2023;13(1):5886.

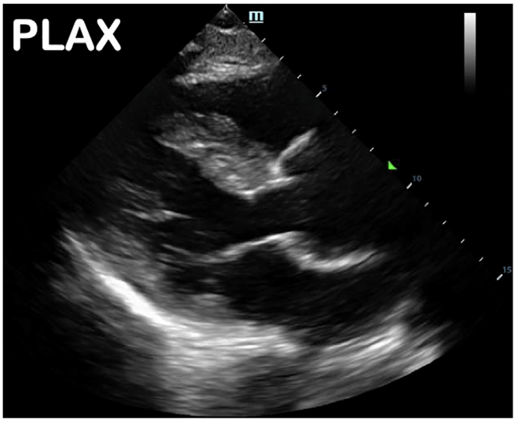

Fig. 23. Dilated aortic root in proximal aortic dissection.

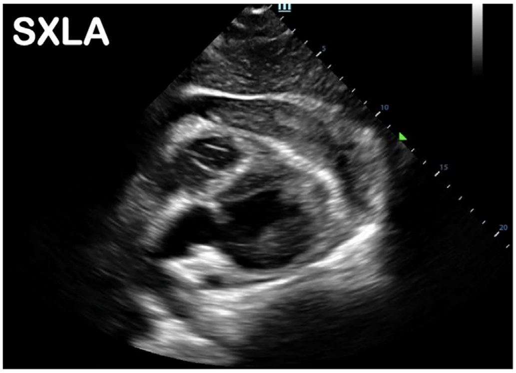

Fig. 24. Traumatic hemopericardium.

Box 6
Advantages of transesophageal echocardiography versus transthoracic echocardiography[20]

Shorter duration of pulse checks

Continuous cardiac visualization

Higher-quality images

Procedural guidance

Optimal localization of chest compressions

Overcomes situation-related factors (eg compression devices and hands, defibrillator pads)

Overcomes patient factors (eg obesity, gastric insufflation from positive-pressure ventilation)

Source: Gottlieb M, Alerhand S. Managing Cardiac Arrest Using Ultrasound. Ann Emerg Med. 2023;81(5):532-42.

Prognostication

Studies have demonstrated that cardiac activity found by POCUS is associated with improved prognosis in cardiac arrest, including for patients previously thought to be in PEA or asystole.[20] Expert sonographers have demonstrated substantial interrater reliability in identifying cardiac standstill, but less-experienced users have shown more variability.[28,29] Experts have thus deemed organized cardiac activity "as movement of the myocardium with change in size of the ventricular cavity and synchronized movement of the ventricular wall."[30]

Transesophageal echocardiography

Placement of the transesophageal echocardiography (TEE) probe directly into the esophagus behind the heart provides several advantages over TTE (**Box 6**). Therefore, TEE can better evaluate for organized cardiac contractility (**Fig. 25**) (Video 18), assess LV systolic function, identify shockable rhythms, diagnose reversible causes of arrest, evaluate chest compression depth and location (Video 19), and monitor the response to these interventions.[20] An systematic review (SR) consisting of both OHCA and IHCA found that TEE identified reversible causes in 41% of patients.[31] From large data sets

Fig. 25. Transesophageal echocardiography in cardiac arrest (MELAX = mid-esophageal long-axis).

of intraoperative TEE, and smaller samples in cardiac arrest, the complication rate has been reported as only 0-1.2%.[20] Experts recommend that only a small number of views are needed in cardiac arrest.[32]

SUMMARY

A fundamental understanding of the key aspects of cardiac POCUS is essential to managing sick patients in the ED. Each individual application can be broken down for a systematic approach at the bedside.

CLINICS CARE POINTS

- The more echocardiographic views that are acquired, the more accurate the qualitative LV global assessment will be.
- LV hypertrophy and a dilated LA can also suggest diastolic dysfunction.
- What might appear as a small pericardial effusion (or no effusion) in one view may appear larger in another, and vice versa.
- A combination of echocardiographic parameters is more reflective of right-heart pathophysiology than a single parameter in isolation.
- Left-sided valvular lesions can cause hemodynamic instability and death in the acute setting.
- Improper use of POCUS can inadvertently cause delays in resumption of chest compressions during cardiac arrest.

DISCLOSURE

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SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.emc.2024.05.004.

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