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Navigating the Waves of Critical Care Echocardiography: Unveiling its Role, Advantages, and Pitfalls in the Cardiac Intensive Care Unit

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Abstract

Purpose of Review Critical Care Echocardiography (CCE) is now established as an important tool in the intensive care unit (ICU). This paper aims to examine the expanding role of cardiovascular ultrasound in the ICU, focusing on its applications, benefits, and challenges, while highlighting recent advancements shaping the future of critical care echocardiography.

Recent Findings Non-invasive echocardiographic measurement of hemodynamic parameters including stroke volume, cardiac output, left ventricular filling pressures, and pulmonary pressures have been well-validated against invasive measurements. Myocardial perfusion can also be evaluated using ultrasound enhancing agent techniques to further risk-stratify patients with chest pain.

Summary Echocardiography enables clinicians to visualize cardiac anatomy and physiology directly at the bedside, providing immediate feedback in rapidly changing clinical situations. Assessment of stroke volume, cardiac output, and left ventricular filling pressures can be readily measured at the bedside and correspond with clinical outcomes including mortality. Measurement of central venous pressure and pulmonary pressures may guide clinical decisions in fluid management and mechanical ventilation strategies. Lastly, myocardial perfusion imaging can supplement the 2D echocardiographic evaluation to further risk-stratify patients presenting with chest pain.

Keywords Echocardiography · Critical care · Noninvasive · Hemodynamics

Abbreviations

LVEF	Left ventricular ejection fraction
SCAI	Society for Cardiovascular Angiography & Interventions
PASP	Pulmonary Artery Systolic Pressure
PADP	Pulmonary Artery Diastolic Pressure
MPAP	Mean Pulmonary Artery Pressure
PVR	Pulmonary Vascular Resistance
TR	Tricuspid Valve Regurgitation
PR	Pulmonary Valve Regurgitation
RVOT	Right Ventricular Outflow Tract
VTI	Velocity-Time Integral
CW	Continuous Wave
RV	Right Ventricle
RA	Right Atrium
RAP	Right Atrial Pressure
RVSP	Right Ventricular Systolic Pressure
PG	Pressure Gradient
PW	Pulsed Wave
AT	Acceleration Time
LVOT	Left ventricular outflow tract

Introduction

Cardiovascular ultrasound has become an indispensable tool in the intensive care unit (ICU) due to its non-invasive nature, real-time imaging capabilities, and comprehensive diagnostic utility. As critically ill patients often present with complex hemodynamic instability, the ability to rapidly assess cardiac function, volume status, and structural abnormalities is crucial for guiding therapeutic interventions. Point-of-care ultrasound (POCUS) has served an important role in the cardiac intensive care unit affording the evaluation of cardiac function and structural abnormalities in patients with cardiogenic shock. Furthermore, POCUS is pivotal in diagnosing life-threatening conditions such as

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mechanical complications of myocardial infarction, cardiac tamponade, and infective endocarditis. Since cardiac function and pathologies vary with loading conditions, a thorough hemodynamic assessment is crucial. Ready access to rapid bedside POCUS enhances decision-making for fluid management, mechanical ventilation strategies, and titration and positioning of mechanical circulatory support devices. This paper aims to explore the expanding role of cardiovascular ultrasound in the ICU, focusing on its applications, benefits, and challenges, while highlighting recent advancements that are shaping the future of critical care echocardiography. This review will focus on cardiovascular and hemodynamic assessment by echocardiography and will not explore the important role of lung ultrasound in critical care.

Two-Dimensional Echocardiography

Early identification and treatment of the cause for cardiogenic shock is the cornerstone of initial management. The ability to perform imaging rapidly at the bedside can permit early insight into the cause of cardiovascular compromise in conjunction with the physical exam, laboratory studies, and electrocardiogram as part of the initial assessment. Left ventricular ejection fraction (LVEF) along with regional wall motion assessment, right ventricular systolic function, valvular assessment, and the presence of pericardial effusion are paramount to a rapid initial assessment of cardiac function.

LVEF remains a powerful prognostic variable. In the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial, an LVEF < 28% was independently associated with higher 30-day and 1-year mortality in patients with cardiogenic shock due to myocardial infarction [1]. LVEF declines with advancing Society for Cardiovascular Angiography & Interventions (SCAI) shock stages in an unselected critically ill cardiac cohort and lower LVEF corresponds with higher mortality [2].

While these variables by two-dimensional assessment are useful, they provide an incomplete picture of cardiac function in the absence of hemodynamic information. Systolic function and valvular lesions are dynamic based on loading conditions; thus, a complete understanding of the hemodynamic profile is critical in the assessment to guide clinical decisions.

Doppler Hemodynamics

Estimation of intracardiac pressures and flow through Doppler techniques supplements the two-dimensional information by characterizing the patient's hemodynamic profile. These techniques produce reliable values that have been validated against direct invasive measurements and can be instrumental in both prognostication and directing therapies to optimize cardiac performance.

Stroke Volume and Cardiac Output

Left ventricular stroke volume is first measured by calculating the cross-sectional area of the left ventricular outflow tract (LVOT) using the equation for the area in a circle, $area = \pi r^2$. Rather than imprecisely measuring the radius, the diameter of the LVOT is directly measured at the insertion point of the aortic valve leaflets from the parasternal long axis view zoomed into the aortic valve (Fig. 1). Close attention to precise measurement is important as any errors will be amplified when used in calculation:

$$LVOT\ cross\ setional\ area = \pi \times \left(\frac{LVOT\ diamater}{2}\right)^2$$

To calculate the stroke volume, the LVOT cross sectional area is multiplied by the LVOT time-velocity integral (TVI) which represents the distance traveled by the blood ejected through the orifice. LVOT TVI is measured from a laminar

$$SV = (D_{LVOT}^2 \cdot \pi / 4) \cdot VTI_{LVOT}$$

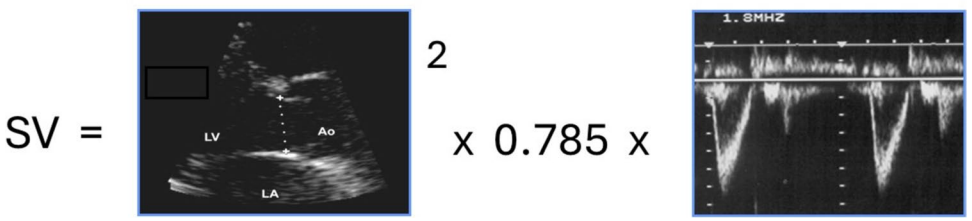

Fig. 1 Shown is the parasternal long-axis view of the left ventricle (LV) with measurement of the left ventricular outflow tract (LVOT) diameter just below the insertion of the aortic valve leaflets. Also shown is a pulsed-wave Doppler signal from an apical 5-chamber view demonstrating the LVOT velocity (note the modal signal with a bright rim and inner opaque appearance). Combining the two parameter in the equation shown allows for the calculation of stroke volume (SV). LA – left atrium, Ao – aorta, D – diameter, VTI – velocity time integral

pulsed wave Doppler signal placed within the LVOT in an apical long axis view, careful to avoid any areas of flow acceleration (Fig. 1).

$$Stroke\ Volume = LVOT\ cross\ setional\ area \times LVOT\ TVI$$

To calculate cardiac output and index, the stroke volume is multiplied by heart rate and divided by body surface area.

$$Cardiac\ output = stroke\ volume \times heart\ rate$$

$$Cardiac\ index = \frac{cardiac\ output}{body\ surface\ area}$$

Estimation of stroke volume and cardiac output by this method was first validated against invasive thermodilutional measurement in critically ill patients by a small study in 1984 [3]. Subsequent studies comparing echocardiographic estimations to thermodilutional measurements have had variable results, some demonstrating good correlation [4] while others suggesting discrepancies [5, 6]. The methods for echocardiographic measurement of cardiac output are widely variable in these studies, both in technique and experience in obtaining and interpreting these measurements. When cardiac output is calculating from the LVOT TVI as described here, and performed by an experienced sonographer there is excellent correlation with thermodilutional measurements [7].

When aortic regurgitation is present, stroke volume quantified with the method should be interpreted carefully. The added left ventricular preload from the aortic regurgitation will increase the volume ejected during systole resulting in an elevated measured stroke volume. The meticulous interpreter would then calculate the effective forward stroke volume by subtracting the aortic regurgitant volume from the measured stroke volume.

These values also correlate with clinical outcomes in cardiogenic shock. Low indexed stroke volume and cardiac output correlate with higher SCAI shock stages and in-hospital mortality [2]. Specifically, a low indexed stroke volume (< 35 mL/m2) corresponds to 2-fold higher odds for in hospital mortality at each shock stage [2].

Left Ventricular Filling Pressures

The importance of left ventricular filling pressure in critically ill patients remains under-appreciated. Elevated LV end diastolic pressure corresponds to higher mortality in patients with acute myocardial infarction [8]. LV end diastolic pressure can be estimated echocardiographically by the ratio of early mitral inflow velocity to early mitral annular tissue velocity (E/e'). In an apical four-chamber view, E can be measured as the peak velocity in early diastole on a pulsed-wave Doppler signal placed at the tips of the mitral valve leaflets. Whereas E' is measured as the peak velocity in early diastole on Tissue Doppler Imaging (TDI) of the medial or lateral mitral annulus.

Noninvasive E/E' values have been shown to correspond to invasively measured LV end diastolic pressures by simultaneous measurement in the cardiac catheterization lab. With high fidelity, E/E' < 8 corresponds to normal LV filling pressure and E/E' > 15 corresponds to high LV filling pressure [9]. E/E' values between 8 and 15 had variable LV end diastolic pressures and thus represents an indeterminate range.

Mitral valve E' should probably not be utilized when mitral valve surgery has been performed, in the presense of significant mitral annular calcification, or when LBBB is present as annular motion will be altered. Similarly, high flow states such as mitral regurgitation or anemia can cause erroneously high E, in which case E/E' must be interpreted with caution.

Clinically, E/E' ratio is important prognostically, even in critically ill patients. Elevated E/E' independently predicts mortality after acute myocardial infarction and out of hospital cardiac arrest more strongly than LVEF [10, 11]. This is similarly true in an unselected critically ill cardiac cohort [11].

Central Venous Pressure

Echocardiography is a pivotal non-invasive tool in the estimation of right atrial pressure (RAP). The assessment of the inferior vena cava (IVC) via echocardiography is one of the most commonly used methods [12]. The size and collapsibility of the IVC during respiration provide valuable insights into RAP. A dilated IVC with reduced respiratory variation is indicative of elevated RAP, whereas a small IVC with significant respiratory variation suggests normal or low RAP. The ASE Guidelines suggest that an IVC diameter greater than 2.1 cm that collapses less than 50% with a sniff correlates with elevated RAP, typically greater than 10 mmHg [13].

In addition, respiratory variation in vena cava diameter measured by ultrasound (distensibility index > 15%) predicts fluid responsiveness in critically ill patients [14], but this technique works better in patients on controlled mechanical ventilation than on patients who are breathing spontaneously [15].

In addition to IVC assessment, the size and function of the right atrium (RA) are critical parameters evaluated through echocardiography. An enlarged RA can be a sign of chronic pressure overload and elevated RAP. The ASE recommends using RA area measurements to estimate RAP, with an RA area greater than 18 cm^2 being suggestive of elevated pressures [16].

Doppler assessment of the hepatic veins also can assist with the assessment of RAP as summarized

Table 1 Estimated right atrial pressures based on ratio of systolic and diastolic forward flow seen on hepatic vein Doppler

Hepatic vein doppler flow	Estimated right atrial pressure (mmHg)
Systolic > Diastolic	5
Systolic = Diastolic	10
Systolic < Diastolic	15
Absent Systolic Flow	20

in Table 1 [17]. The ratio of the systolic to diastolic forward flow peak velocity or TVI correlates with RAP. A systolic > diastolic predominant pattern is associated with a RAP of 5 mmHg. An equal systolic to diastolic ratio suggests a RAP of 10 mmHg. A diastolic > systolic predominant pattern suggests a RAP of 15 mmHg. Finally, absence of any systolic forward hepatic vein velocity (all forward flow in diastole) correlates with a RAP of 20 mmHg.

TDI is an advanced echocardiographic technique that assesses the movement of the tricuspid annulus. The velocity of the tricuspid annular motion can provide insights into right ventricular (RV) function and indirectly reflect RAP. Reduced tricuspid annular systolic velocity (S') is associated with elevated RAP and RV dysfunction [18]. This technique adds another layer of diagnostic accuracy in evaluating right heart pressures.

In clinical practice, the integration of ultrasound findings with the patient's clinical presentation and other diagnostic tests is essential for a comprehensive evaluation of RAP. Echocardiography provides a non-invasive, readily available, and effective means of estimating RAP, which is crucial in the diagnosis and management of various cardiovascular conditions, including heart failure, pulmonary hypertension, and congenital heart diseases.

Pulmonary Pressures

Despite some limitations, a complete estimation of pulmonary artery pressures can be obtained using routine Doppler assessment. The simplified Bernoulli's equation is utilized to determine the pressure gradient between two chambers based on the peak velocity of flow between them.

$$Pressure\ Gradient = 4\ (peak\ velocity)^2$$

Using peak velocities during different times in the cardiac cycle, this Benoulli equation can be used to estimate pulmonary artery systolic pressure (PASP), pulmonary artery diastolic pressure (PADP), mean pulmonary artery pressure (MPAP), and pulmonary vascular resistance (PVR) utilizing Doppler signals from tricuspid valve regurgitation (TR), pulmonary valve regurgitation (PR), and right ventricular outflow tract (RVOT) velocity-time integral (VTI).

First, the PASP can be estimated utilizing the simplified Bernoulli equation to estimate the pressure difference between RV and PA in mid-systole. The peak Tricuspid Regurgitation (TR) velocity is measured utilizing continuous wave (CW) Doppler. Multiple views should be interrogated to obtain the best Doppler alignment to obtain the highest velocity. The velocity of the TR jet, when combined with the right atrial pressure, can be used to calculate the right ventricular systolic pressure (RVSP) [13]. In the absence of pulmonary valve stenosis or prosthesis, the RVSP is equal to the PASP [19].

$$PASP = 4\ (TR\ velocity)^2 + RAP$$

The upper limit of normal for a peak TR velocity is 2.8 m/s. Assuming a normal RAP of 3–5 mmHg, this will result in a PASP of 36 mmHg [20–22]. This method has been validated in numerous studies and is widely used in clinical practice [23].

There are limitations to this estimation. As described above, estimation of RAP can be challenging in some circumstances especially in patients requiring positive pressure ventilation or those with severe TR. Additionally, not all patients will have a strong TR signal. If an incomplete TR signal is utilized, this will lead to underestimation of the PASP.

Next, the PADP can be estimated utilizing the simplified Bernoulli equation to estimate the pressure difference between RV and PA at end-diastole. A CW Doppler tracing through the pulmonic valve can be used to measure the peak velocity of pulmonary regurgitation at the end of diastole and then added to the RV end diastolic pressure to calculate the PADP. The RV end diastolic pressure is equal to the estimated RAP as described above [24–26].

$$PADP = 4\ (End\ PR\ Velocity)^2 + RAP$$

Limitations of this assessment are similar to those for estimating the PASP. The end PR velocity can be more challenging to obtain due to limited visualization of the pulmonary valve. Also, in the setting of severe PR rapid equalization of pressure between the PA and RV will lead to underestimation of the PADP.

Next, the MPAP can be estimated utilizing the simplified Bernoulli's to estimate the pressure difference between RV and PA in early-systole. A CW Doppler tracing through the pulmonic valve can be used to measure the peak velocity of pulmonary regurgitation at the beginning of systole and then added to the RV end diastolic pressure, or RAP.

$$MPAP = 4\,(Peak\ PR\ Velocity)^2 + RAP$$

Limitations to this technique are difficulty acquiring an adequate CW Doppler tracing that shows the complete PR signal in early systole. In patients with pulmonary hypertension a peak PR velocity can generally be obtained, however this measurement can be challenging in other patients [27]. When this cannot be achieved, MPAP can alternatively be calculated from PASP and PADP, understanding that this relies on the accuracy of the input variables.

$$MPAP = \frac{1}{3}PASP + \frac{2}{3}PADP$$

When these values are unable to be obtained, the pressure gradient between RV and PA can be estimated by measuring the TVI of the TR signal on CW Doppler and added to the RAP to calculate MPAP [28].

$$MPAP = TR\ mean\ pressure\ gradient + RAP$$

The final and most challenging method to estimate the MPAP can be obtained using a pulsed wave (PW) Doppler signal of the RVOT. With the marker placed just proximal to the pulmonary valve the signal is obtained. With the PW Doppler signal the acceleration time (AT) is measured from the onset of flow through the pulmonary valve to the peak velocity. A value of > 130 ms is normal, while a short AT (< 100ms) is highly suggestive of pulmonary hypertension [29, 30].

$$MPAP = 90 - (0.62\ x\ RVOT\ Acceleration\ Time)$$

While a PW Doppler signal of the RVOT can be obtained in most cases appropriately measuring the AT can be challenging which is the main limitation of this technique.

Estimating pulmonary vascular resistance (PVR) becomes clinically relevant since elevated pulmonary pressure may represent underlying pulmonary vascular disease or increased pulmonary flow. To estimate a PVR, the RVOT VTI is measured from a PW Doppler signal through the RVOT and peak TR velocity acquired from CW Doppler signal through the tricuspid valve [30].

$$PVR = 10\,x\left(\frac{TR\ Velocity}{RVOT\ TVI}\right) + 0.16$$

This will provide the PVR in Wood units (multiply by 80 to convert to dynes*cm/s2). A normal PVR is < 1.5 Wood units (120 dynes*cm/s2) and significant pulmonary hypertension is defined as a PVR > 3 Wood units (240 dynes*cm/s2).

Myocardial Perfusion Assessment

Contrast agents may be used to aid in the application of ultrasound [31]. The fundamental property of ultrasound enhancing agents (UEA) is microbubbles containing gas that differs in acoustic impedance from blood. Agitated saline has been used for decades to opacify vascular structures [32]. However, given the size of the microbubbles produced prohibits flow through pulmonary capillaries, contemporary use is reserved for assessment of intracardiac shunts [33]. Contrast agents consisting of smaller microspheres with a shell of either albumin (Optison®, GE Healthcare, Chalfont St. Giles, UK) or phospholipids (Definity®, BMS, Billerica, Massachusetts; Sonovue®, Bracco, Milan, Italy) containing high-molecular-weight gases allow left-heart and arterial opacification by easily traversing the pulmonary capillaries [34]. The gases in the contrast agents are biologically inert while the shells are metabolized similar to natural albumin and lipids [35, 36]. They are also generally safe with very rare severe reactions, including anaphylaxis, with few contraindications [31]. Before preparing the contrast agent of choice, ultrasound machine settings should be optimized for image acquisition. Typical ultrasound output with harmonic imaging is not high enough energy to inflict harmful bioeffects. However, resonant oscillations can disrupt the contrast agent shell, causing microbubble destruction and impair image quality. Output power should be reduced to achieve a mechanical index of < 0.3 ("low") for the machine to process the harmonic tissue signals without destroying microbubbles, improving signal-to-noise ratio and, for instance, the endocardial-blood interface [31]. The mechanical index can be lowered even further (0.05–0.2, "very low") to attenuate tissue signals and amplify contrast signals, which can be advantageous in assessment of contrast in the myocardial microcirculation. Brief "flashes" of high mechanical index impulses (0.8–1.0) can destroy the contrast microbubbles in the myocardium followed by replenishment in the next diastolic period, thereby assessing myocardial perfusion [37, 38].

The physical properties of these contrast agents improve the assessment of left ventricular systolic function and regional wall motion, particularly in critically-ill patients with difficult imaging windows [39–42]. There is also an association of use of ultrasound contrast agents and lower mortality, perhaps due to management changes [43]. Contrast use offers additional opportunities at improving diagnosis and clinical decision making, building upon stress echocardiography where regional wall motion (RWM) assessment is improved and myocardial perfusion (MP) can be quantified [11, 44, 45]. MP and reduced subendocardial thickening may represent coronary stenosis and at-risk myocardium that may be missed with transmural wall motion assessment alone. [31, 46, 47]

MP assessment adds incremental diagnostic value in the assessment for myocardial ischemia in the acute setting, including the Emergency Department and the cardiac ICU, particularly when the electrocardiogram and troponin

levels are non-diagnostic [48–50]. A finding of combined abnormal RWM and MP have been shown to be superior to abnormal RWM alone in predicting nonfatal myocardial infarction or cardiac death at 48 hours after Emergency Department presentation. Abnormal myocardial perfusion in combination with impaired RWM correlate with a high rate of adverse cardiac events (64–74%) at follow-up [49]. In contrast, normal perfusion and RWM suggest normal myocardial blood flow and contractile function and these patients have a low rate of adverse events. In the acute setting, if contrast echocardiography suggests that a cardiac ischemic contribution to symptoms or illness is unlikely, then shifting diagnostic and therapeutic intervention to other causes may be necessary [49].

Lastly, use of mechanical circulatory support (MCS) is common in the CICU, which can also predispose to difficult imaging windows. While there is a paucity of data in the use of contrast agents in MCS, they are generally safe to use in patients with a durable left ventricular assist device [51] and in those on venoarterial extracorporeal membrane oxygenation [52], although precautions must be taken for air detection devices that may affect circuit function [53].

Advantages and Pitfalls

Non-invasive echocardiographic evaluation of a critically ill patient offers many advantages over more invasive assessments. Firstly, transthoracic echocardiography is readily accessible and allows more rapid acquisition of measurements. This facilitates more rapid assessment to support clinical decisions in deteriorating patients and permits repeated assessments in a cohort with rapidly changing clinical status. Furthermore, choosing to forego invasive procedures also repeals the risk for procedural complications. Performance of the echocardiographic assessment at the bedside also limits the significant resource utilization needed to transport patients to other areas of the hospital. Similarly, non-invasive echocardiography can offer cost savings in these patients with high resource utilization. CCE can also be used to guide procedures like pericardiocentesis and placement of intra-aortic balloon pumps as well as identifying the effects of mechanical ventilation on cardiac function.

However, there are important considerations that may limit the utility of echocardiographic assessment. The measurements are dependent on the image quality in the available echocardiographic windows. Critically ill patients may have interference from cardiac devices such as mechanical circulatory support devices, fewer accessible quality windows, or interference from hemodynamic factors such as LVOT obstruction. There may be greater demands on provider time for initial and serial assessments compared to invasive measurements from an indwelling pulmonary artery catheter. Lastly, measurements are prone to both acquisition and interpretation errors based on user experience.

Conclusions

CCE is a vital diagnostic and monitoring tool used in the ICU to assess and manage patients with acute cardiovascular conditions. It assists in the rapid diagnosis of various cardiac conditions such as myocardial infarction, heart failure, pericardial effusion, and valvular heart diseases. It provides real-time imaging of the heart, allowing for immediate assessment of cardiac function and structure. CCE is used to evaluate hemodynamic status, including cardiac output, preload, afterload, and contractility. This is crucial for managing patients with shock, sepsis, or other critical conditions. CCE assists in guiding therapeutic interventions such as fluid management and vasopressor/inotrope administration. One of the major advantages of CCE is that it is non-invasive and can be performed at the bedside, making it ideal for critically ill patients who cannot be easily transported. Importantly, proper training and expertise are required to perform and interpret CCE accurately. CCE can also provide prognostic information, helping to predict outcomes and guide long-term management strategies for critically ill patients. In summary, CCE is an essential tool in the ICU for diagnosing cardiac conditions, monitoring hemodynamics, guiding interventions, and providing prognostic information. Its non-invasive nature and ability to be performed at the bedside make it invaluable in the management of critically ill patients.

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Findings support utilization of ultrasound myocardial perfusion assessment in risk-stratification of patients presenting with chest pain and no ST elevation on ECG.

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Compliance with Ethical Standards

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