




AKADÉMIAI KIADÓ

# Echocardiography in the intensive care unit: An essential tool for diagnosis, monitoring and guiding clinical decision-making

## IMAGING

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## REVIEW ARTICLE



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### ABSTRACT

In the last years, new trends on patient diagnosis for admission in cardiac intensive care unit (CICU) have been observed, shifting from acute myocardial infarction or acute heart failure to non-cardiac diseases such as sepsis, acute respiratory failure or acute kidney injury. Moreover, thanks to the advances in scientific knowledge and higher availability, there has been increasing use of positive pressure mechanical ventilation which has its implications on the heart. Therefore, there is a growing need for Cardiac intensivists to quickly, noninvasively and repeatedly evaluate various hemodynamic conditions and the response to therapy.

Transthoracic critical care echocardiography (CCE) currently represents an essential tool in CICU, as it is used to evaluate biventricular function and complications following acute coronary syndromes, identify the mechanisms of circulatory failure, acute valvular pathologies, tailoring and titrating intravenous treatment or mechanical circulatory support. This could be completed with trans-esophageal echocardiography (TOE), advanced echocardiography and lung ultrasound to provide a thorough evaluation and monitoring of CICU patients. However, CCE could sometimes be challenging as the acquisition of good-quality images is limited by mechanical ventilation, suboptimal patient position or recent surgery with drains on the chest. Moreover, there are some technical caveats that one should bear in mind while performing CCE in order to optimize its use and avoid misleading findings. The aim of this review is to highlight the key role of CCE, providing an updated overview of its main applications and possible pitfalls in order to facilitate its use in CICU for clinical decision-making.

### HIGHLIGHTS

- Echocardiography is an essential bedside tool for the management of critically ill patients.
- Integrated Echocardiography and Lung Ultrasound are very useful aids for the diagnosis, monitoring and guiding clinical decision-making in the CICU and should be an extension of bedside physical examination.
- CCE requires understanding of the complex pathophysiology of the haemodynamics of critical illness.
- Cardiac intensivists should be aware of the technical challenges and pitfalls of CCE.

### KEYWORDS

echocardiography, intensive care, diagnosis, monitoring, critical care

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## Introduction

As cardiac critical care is developing as a discipline on its own accord, crossing the boundaries of cardiology and critical care medicine, cardiac intensivists are facing multiple challenges when trying to acquire/maintain the necessary skills and competencies [1].

Critical care echocardiography (CCE) is one of the tools they use when dealing with diagnostic complexities and dilemmas: increasing severity of disease, labile hemodynamics, high levels of organ support and iatrogenic complications. CCE seems to be ideally designed for such a setting: it is safe, non-invasive, portable, rapid and repeatable. The major limitation to CCE is the operator her/himself: scanning a critically ill patient and being able to interpret the images with confidence is a demanding task that among other complex skills requires modesty and acceptance of one's own limitations.

Evaluation of ventricular function following an acute coronary syndrome or in the case of hypotension of unknown or suspected cardiac etiology [2], identifying the mechanisms of circulatory failure [3], tailoring and titrating pharmacologic or mechanical circulatory support [4], are among the most frequent indications to perform CCE. When integrated with lung ultrasound, this becomes a powerful bedside tool to allow optimization and weaning of these therapies [5]. Despite the difficulties with acquisition of good-quality images in critical care due to mechanical ventilation, suboptimal patient positioning or recent surgery with drains and dressings on the chest, recent evidence has demonstrated the great feasibility of transthoracic echocardiography (TTE) for basic evaluation even by less-experienced operators. Moreover, performing focused echocardiography in patients who are in the prone position has been increasingly utilized during the COVID-19 pandemic and found feasible for qualitative assessment of RV and LV function even in obese patients and those on high PEEP [10].

Nevertheless, to obtain a reliable assessment of hemodynamics, the role of expert operators remains fundamental for performing advanced critical care echocardiography for complex critically ill patients [6]. Handheld devices can be used for a focused initial approach and the findings have shown good agreement with formal TTE examinations when performed by experienced operators [7]. The coronavirus disease-2019 (COVID-19) pandemic has given us several paradigms of how versatile and adaptive as a tool CCE can be, even in the extreme setting of intensive care unit (ICU) overflow [8, 9].

This review aims to highlight the important role of CCE in the clinical decision-making and also the unique challenges and 'secrets' that it bears.

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## Echocardiography as a support to clinical diagnosis

### Pericardial effusion and tamponade

This is a potentially life-threatening complication which could develop after cardiac surgery, acute myocardial infarction

(AMI), acute or chronic heart failure (HF), pericarditis, rheumatological disease and after catheter interventional procedures. The subcostal view is usually the best window to provide a quick assessment of pericardial effusion. This view is also used to guide pericardiocentesis if needed [11]. Although cardiac tamponade is a clinical diagnosis, echocardiography is the method of choice to identify a pericardial effusion and assess its hemodynamic consequences; its pathophysiology depends primarily on the speed of accumulation of fluid, rather than on the absolute volume of fluids [12]. Table 1 summarizes the key echocardiographic findings in patients with cardiac tamponade [13].

### Dynamic left ventricular outflow tract obstruction

This pattern of obstructive shock could be impossible to diagnose without the use of echocardiography in which several findings could be seen: Small LV cavity, basal septal hypertrophy, elongated anterior mitral leaflet and the presence of systolic anterior motion (SAM) of the anterior mitral leaflet creating a high systolic velocity across the LV outflow tract (LVOT). Dynamic obstruction to forward flow should also be assessed at the mid-LV cavity which could also demonstrate the presence of systolic turbulence at any point in the LV cavity, especially with small, hyperdynamic LV. The typical echocardiographic finding is the presence of an increased peak systolic velocity which is more pronounced at the late systole with the evidence of a 'Dagger-shaped' CW Doppler waveform.

The echocardiographic diagnosis of dynamic LVOT obstruction or LV mid-cavitary obstruction often leads to drastic changes in the clinical management as it entails stopping or reducing inotropes, increasing afterload by vasopressors, optimizing preload by administering intravenous fluids and also reducing heart rate with beta-blockers or pacing optimization. Mitral regurgitation may also develop as a result of the loss or distortion of systolic mitral leaflet coaptation, leading to an eccentric regurgitation jet in the direction of the left pulmonary veins.

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## Hemodynamic monitoring by echocardiography

Echocardiography is the most reliable bedside method to assess cardiac function repeatedly, assisting clinicians not only in characterizing hemodynamic disorders, but also in helping to guide and monitor the response to advanced therapeutic options (intravenous fluids, inotropes and ultrafiltration) [15].

In patients with signs of hemodynamic instability, an echocardiographic examination is required for an immediate differential diagnosis to guide therapy [8]; this could be initially limited to a focused evaluation following specific protocols, which have proved to enhance decision-making in the acute settings [14,16].

### Assessment of LV systolic function and cardiac output

There are currently no validated referenced values for LV Ejection Fraction (EF) in the critically ill patients. Moreover,



Table 1. Parameters and pitfalls of Echocardiography in CICU

	Parameters	Pitfalls
LV systolic function/cardiac output	LVOT VTI/SV MAPSE TDI S'	<p><b>LVOT VTI:</b></p> <ul style="list-style-type: none"> <li>• Avoid plain foreshortening when measuring the LVOT diameter <ul style="list-style-type: none"> <li>• Avoid SV under-estimation: <ul style="list-style-type: none"> <li>-Optimize PWD settings to measure LVOT VTI</li> <li>-Put the PWD beam parallel to LVOT flow</li> </ul> </li> </ul> </li> <li>• Shock -&gt; check for dynamic LVOT obstruction (+/- MV SAM) [especially in case of hypovolemia, LV hypertrophy and hyperdynamic LV (e.g. vasoplegia)]. <ul style="list-style-type: none"> <li>• Not applicable in aortic stenosis or subaortic obstruction <ul style="list-style-type: none"> <li>• AF: average of 5 beats</li> </ul> </li> <li>• AR: SV overestimation (the regurgitant diastolic flow is not considered)</li> </ul> </li> </ul> <p><b>MAPSE</b></p> <ul style="list-style-type: none"> <li>• Monodimensional and regional parameter (only analyzes mitral annular LV portion)</li> </ul> <p><b>TDI S'</b></p> <ul style="list-style-type: none"> <li>• Lower spatial resolution</li> </ul>
Fluid responsiveness (static)	LVEDA IVC diameter	<p><b>LVEDA</b></p> <ul style="list-style-type: none"> <li>• Hypovolemia: Small LV cavity with diastolic dysfunction and elevated filling pressures -&gt; good response to fluids, but low threshold to overload with a large volume fluid challenge <i>or</i> exaggerate response with increase in filling pressures with a small volume of fluid challenge <ul style="list-style-type: none"> <li>• Vasoplegia: often preserved LVEDA</li> <li>• Hypovolemia reduced LVEDA</li> </ul> </li> <li>• Not valid in patients with RV dysfunction</li> </ul> <p><b>IVC diameter</b></p> <ul style="list-style-type: none"> <li>• High sensitivity, low specificity for hemodynamic overload</li> </ul>
Fluid responsiveness (dynamic)	IVC distensibility in spontaneous breath SVC collapsibility in ventilated patients LVOT VTI respiratory variability Aortic velocity respiratory variability	<ul style="list-style-type: none"> <li>• Other causes of <b>dilated IVC</b>: RV failure, cardiac tamponade, pulmonary embolism, TR, pulmonary hypertension. <b>IVC Collapse</b> -&gt; increased abdominal pressure</li> <li>• <b>Passive leg raise</b>: Avoid patient discomfort (e.g. <i>abdominal compartment syndrome, pelvic fractures</i>) and maintain his privacy</li> <li>• <b>LVOT VTI</b>: Severely hypovolaemic patients may not have an increased LVOT VTI due to lack of blood in the lower extremities <ul style="list-style-type: none"> <li>• RV dysfunction may cause false volume respiratory changes</li> </ul> </li> </ul> <p><b>Aortic velocity variability:</b></p> <ul style="list-style-type: none"> <li>• Can be exaggerated by PEEP or cardiac translational motion <ul style="list-style-type: none"> <li>• Not applicable in patients with an open chest</li> </ul> </li> <li>• Non-sinus rhythm: multiple measurements of SV must be averaged for a reliable assessment</li> </ul>

(continued)



**Table 1. Continued**

	Parameters	Pitfalls
Pulmonary artery pressures	RA, RV size PASP, PADP, MPAP PVR IVS shape and movement IVC diameter and collapsibility	<p><b>Inaccuracies in estimation of PASP:</b></p> <ul style="list-style-type: none"> <li>• TR underestimated in compensated RV (for high ventricular pressure)</li> <li>• Severe (free flow) TR -&gt; equalization of right atrial and ventricular pressures <ul style="list-style-type: none"> <li>• Pulmonary stenosis</li> <li>• Severe RV systolic dysfunction.</li> </ul> </li> <li>• Misalignment of CW Doppler signal: e.g, eccentric jets. <ul style="list-style-type: none"> <li>• Suboptimal CWD envelope <ul style="list-style-type: none"> <li>• Can vary with BMI.</li> </ul> </li> </ul> </li> </ul> <p>Tip:</p> <ul style="list-style-type: none"> <li>• I.V agitated saline can be used to improve the TR envelope</li> </ul> <p>Echocardiographic measurement of PVR is not fully validated to initiate or monitor treatment of pulmonary hypertension. This method should not replace invasive measurement by right heart catheterization.</p>
Right ventricular function	RA, RV size Visual assessment of RV free wall and longitudinal wall motion RV FAC TAPSE TDI S' MPI	
Right heart preload and RAP	Evidence of left heart dysfunction IVC distensibility in spontaneously breathing patients SVC collapsibility in ventilated patients Hepatic vein systolic filling fraction	
LV diastolic function and filling pressures	E/A ratio e' E/e' PCWP ( <i>Nagueh Formula</i> ) Predominant B-profile on lung ultrasound	<ul style="list-style-type: none"> <li>• <b>LA pressure</b> (not always related to diastolic function): <ul style="list-style-type: none"> <li>-Volume overload: high LA pressure despite normal LV diastolic function</li> <li>-Hypovolemia: low LA pressure despite diastolic dysfunction due to poor LV compliance</li> </ul> </li> <li>• <b>LUS:</b> helpful to evaluate LA pressure and haemodynamic overload, (especially with uncertain TTE data). [<i>Normal aeration pattern on LUS: PASP &lt; 18 mmHg [51].</i>]</li> <li>• <b>Mitral annular velocities:</b> often show poor correlation with LV filling pressures (<i>e.g. CAD affecting basal septal or lateral segments, significant mitral annular calcification, surgical rings or prosthetic MV, LBBB, RV pacing, CRT, pericardial disease</i>)</li> </ul>

(continued)

Table 1. Continued

	Parameters	Pitfalls
Cardiac tamponade	<ul style="list-style-type: none"> <li>• RV dilation with interventricular septum shift (and consequent LV underfilling)</li> <li>• Large amount of pericardial effusion with heart movement within the pericardial space (the “swinging-heart”)</li> <li>• Early diastolic RV collapse (the most specific sign)</li> <li>• Systolic right atrial wall invagination (for more than one-third of the cardiac cycle) <ul style="list-style-type: none"> <li>• Respiratory transvalvular Doppler variation: (in spontaneous breathing patients): trans-mitral E-wave increases &gt;25% on expiration and reduces on inspiration, the opposite happens for RV inflow (for patients with mechanical ventilation these phasic changes are reversed)</li> </ul> </li> <li>• Dilated IVC without collapsibility during inspiration (which has low specificity especially in mechanically ventilated patients).</li> </ul>	<ul style="list-style-type: none"> <li>• Early after cardiac surgery, cardiac tamponade may not present with the classical clinical and echocardiographic features and can present with a localized rather than global pericardial effusion [13].</li> <li>• Extracardiac factors, such as a massive pleural effusion, tension pneumothorax or lung hyperinflation due to positive pressure ventilation, can mimic tamponade physiology [14] [15] Echocardiography combined with LUS can confirm such a diagnosis and prevent the detrimental consequences of an unnecessary pericardiocentesis.</li> </ul>

(continued)



**Table 1. Continued**

	Parameters	Pitfalls
LVOTO	<ul style="list-style-type: none"> <li>• small LV cavity, basal septal hypertrophy, elongated anterior mitral leaflet and the presence of systolic anterior motion (SAM) of the anterior mitral leaflet → increased systolic velocity across the LVOT with late peaking by CWD (Dagger-shaped)</li> <li>• Eccentric posteriorly directed MR</li> </ul>	<p>Several predisposing factors that are not uncommon in the critically ill patients make them prone to dynamic LVOTO or intracavitary obstruction:</p> <ul style="list-style-type: none"> <li>• Anaemia</li> <li>• Sepsis</li> <li>• Concentric left ventricular hypertrophy</li> <li>• Stress Cardiomyopathy (Takotsubo)</li> <li>• Following mitral valve surgery (especially in the presence of an elongated anterior mitral leaflet).</li> <li>• Profound vasoplegia associated with RV failure after cardiopulmonary bypass.</li> </ul>
Acute aortic pathologies	<ul style="list-style-type: none"> <li>• <b>intimal flap</b> separating the true and false lumens (<i>diagnostic finding</i>)</li> <li>• Distinction between true and false lumen using color-flow mapping.</li> <li>• Aortic valve regurgitation resulting from aortic dissection and its mechanism (accurately determined by TOE)</li> </ul>	<ul style="list-style-type: none"> <li>• Low sensitivity of TTE for the diagnosis of aortic lumen dissection (better performance for <i>Stanford Type A</i>)</li> <li>• When evaluating ascending aorta with TOE, <b>artifactual echoes</b> should be encountered: <ul style="list-style-type: none"> <li>–<b>true dissection</b> -&gt; random mobility and constant echo intensity through its course</li> <li>–<b>artifact</b> -&gt; rigid and fixed location, echo intensity gradually reduces from its origin (sino-tubular junction) toward the lumen of the aorta and does not alter the distribution of color-flow signal.</li> </ul> </li> <li>• Limited ability of TOE to visualize the distal ascending aorta and proximal arch because of interposition of the air-filled trachea and the main bronchus.</li> <li>• Adequate sedation of the patients and experienced operator are required to perform bedside TOE</li> <li>• Consider TOE contraindications (esophageal and cervical spine disease) and rare complications (aspiration, hematemesis, arrhythmias, esophageal perforation, laryngospasm)</li> </ul>

A, late diastolic transmitral velocity; CAD, coronary artery disease; CRT, cardiac resynchronization therapy; CWD; continuous-wave doppler; E, early diastolic transmitral velocity; E' medium velocity in the three points of mitral annular descent by TDI; IVC, inferior vena cava; LA, left atrium; LBBB, left bundle-branch block; LUS; lung ultrasound; LV, left ventricle; LVEDA; left ventricular end-diastolic area; LVOT, left ventricular outflow tract; MAPSE, mitral annular plane systolic excursion; MPAP, mean pulmonary artery pressure; MPI, myocardial performance index; MV, mitral valve; PADP, pulmonary artery diastolic pressure; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PEEP; positive end-expiratory pressure; PVR; pulmonary vascular resistance; RA, right atrium; RV, right ventricle; RV FAC; right ventricular fractional area change; S' systolic wave velocity by TDI; SAM; systolic anterior movement; SV; stroke volume; SVC, superior vena cava; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue doppler imaging; TOE, transoesophageal echocardiography; TR; tricuspid regurgitation; TTE, transthoracic echocardiography; VTI, velocity time integral; LVOTO, Left Ventricular Outflow Tract Obstruction.



there are concerns that limit its usefulness in these patients [4] as it is greatly influenced by LV geometry, heart rate and loading conditions [17, 18].

Therefore, estimation of cardiac output (CO) by echocardiography represents a validated tool for assessing ventricular function in critically ill patients [19]. This has been well correlated to other standard invasive methods of CO estimation [20].

In the standard method, stroke volume (SV) is determined by the LVOT area (obtained by measuring the LVOT diameter in parasternal long-axis view in mid-systole then calculating the LVOT area by the equation  $(LVOT\ area = \pi \times LVOT\ radius^2)$  multiplied by the LVOT Velocity Time Integral (VTI) – which is the distance the blood travels across the LVOT – and is automatically calculated after tracing the LVOT pulsed-wave doppler (PWD) spectral display. Normal LVOT VTI is higher than 18–20 cm and it could be used as a surrogate for SV. By TOE, this can be calculated by obtaining the deep trans-gastric five-chamber view at  $0^\circ$  or the modified trans-gastric long-axis view at  $120^\circ$  as both provide fair alignment with the LVOT flow and the PWD beam.

The assessment of mitral annulus systolic velocity using tissue-doppler imaging TDI is suggested to be less load dependent [21]. Moreover, the use of advanced echocardiographic techniques, such as strain imaging, allows the quantification of LV twisting and torsion properties as well [22].

## Assessment of volume status and fluid responsiveness

Typically, resuscitation of hypotensive patients requires the administration of fluids to increase the circulating volume. Echocardiography has an important role in the prediction of fluid responsiveness, which is determined by an increase in the SV by 15% or more after an intravenous bolus of fluid. It has been shown that 40–70% of cases of shock will respond to volume expansion [23] (Fig. 1).

## Static indices

The assessment of the systolic obliteration of LV cavity can be performed by tracing the endocardium in end-diastole in parasternal short axis (SAX) view by TTE or in the deep trans-gastric SAX view by TOE. The inferior vena cava (IVC) diameter and the LV end-diastolic area (LVEDA) are not considered reliable indices of fluid responsiveness [24]; however, they can be used cautiously as a guide at the extremes of cardiac filling and function. IVC size may be used to estimate volume status and right atrial pressure (RAP). An IVC diameter  $<2.1$  cm that collapses  $>50\%$  with inspiration suggests a normal RAP = 3 mmHg, while an IVC diameter  $>2.1$  cm that collapses  $<50\%$  with inspiration suggests a high RAP = 15 mm Hg [25].

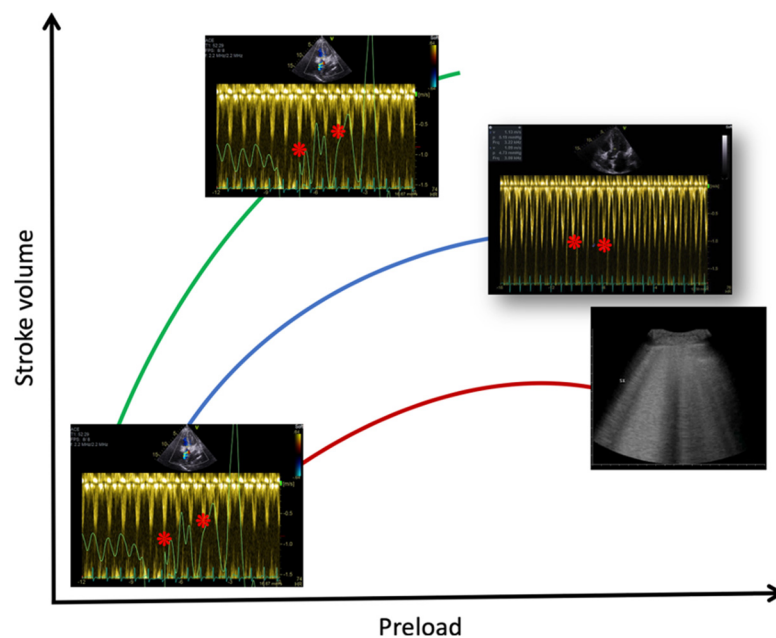


Fig. 1. Illustration of the Frank-Starling curve showing different states of cardiac stroke volume in response to increasing preload by an intravenous fluid challenge. The initial starting point shows a state of fluid responsiveness with a significant VT variability (28%) with a notable difference between maximum and minimum peak velocity. In the red curve, the state of the failing heart is demonstrated by the appearance of abundant B-lines which reflect an increase in the extravascular lung water (EVLW) denoting interstitial-alveolar pulmonary edema. In the blue curve, the disappearance of the significant variability in stroke volume reflects the state of neutrality in which further increase in preload doesn't lead to an increase in stroke volume which is what we would see with the normal cardiac function. In the green curve, there is still significant variability in stroke volume despite further increase in preload which is a depiction of hyperdynamic states (vasoplegia, sepsis etc.)

## Dynamic indices

A dynamic assessment of the heart and circulation may be achieved by assessing the response to a fluid bolus, or heart-lung interactions in either spontaneous or mechanically ventilated patients or passive leg raising (PLR) which provides 300–500 ml of a reversible auto-bolus of blood. It is based on assessing the dynamic changes on IVC or superior vena cava (SVC) diameter with cyclic respiration [26, 27] and SV variations with postural changes [28, 29].

Changes in caval dimensions and collapsibility have been extensively studied. On the one hand, the IVC as an extra-thoracic structure tends to become distended by positive pressure ventilation. On the other hand, the SVC is an intrathoracic structure, therefore, it collapses during the positive pressure inspiratory phase. The IVC distensibility index can be useful in predicting fluid responsiveness if it's equal to or more than the cutoff value of 18% in mechanically ventilated patients [27] and 40% in spontaneously breathing patients [30].

$$\text{IVC distensibility index} = \left( \frac{\text{IVC}_{\text{maximum diameter}} - \text{IVC}_{\text{minimum diameter}}}{\text{IVC}_{\text{minimum diameter}}} \right) \times 100\%$$

If TOE is readily available, The SVC collapsibility index could be used with a cut off of more than or equal to 36% as an indicator of fluid responsiveness [31].

$$\text{SVC collapsibility index} = \left( \frac{\text{SVC}_{\text{maximum diameter}} - \text{SVC}_{\text{minimum diameter}}}{\text{SVC}_{\text{maximum diameter}}} \right) \times 100\%$$

SVC collapsibility index is considered the most reliable index of fluid responsiveness among other indices [32]. While PLR is the most reliable parameter in spontaneously breathing patients and in those with irregular heart rhythm.

Importantly, changes in caval distension or SV employing heart-lung interactions should be applied only under strict conditions: Patients should be sedated/paralysed on controlled mechanical ventilation with tidal volume of around 8 mL kg<sup>-1</sup>, with no evidence of right HF and in normal sinus rhythm.

## Estimation of LV diastolic function and filling pressures

Estimation of diastolic function is an essential part of CCE to allow the early detection of respiratory failure due to cardiogenic pulmonary edema [33]. LV diastolic function assessment is performed by assessing the trans-mitral flow by PWD and then estimating mitral inflow E (early diastolic relaxation) and A (late atrial contraction) waves. TDI is then used to assess the mitral annulus diastolic velocities (e' and a'). Analysis of pulmonary venous PWD waveforms, LA size and tricuspid regurgitation jet are also required for a comprehensive LV diastolic function assessment [34].

For a given state of LV relaxation, an increase of LA pressure will lead to an increase in the E wave while e' remains reduced and unaffected by the elevated LA pressure in the presence of myocardial disease. Therefore, with good reproducibility, the E/e' ratio is considered an important load-independent marker of LV filling pressure [35] (Fig. 2):

$E/e' < 8$  = low LV filling pressure,  $E/e' > 14$  = high LV filling pressure [36].

$$\text{Pulmonary capillary wedge pressure (PCWP)} \\ = 1.24 * (E/E') + 1.9$$

Mourad et al. [36] demonstrated that  $e' < 8$  cm s<sup>-1</sup> was associated with increase mortality in the critically ill patients. Ritzema et al. [37] found that E/e' could reliably detect increased LA pressure >15 mmHg measured with an implanted monitor.

Besides, there are several limitations of this index to consider: an E/e' value between 8 and 14 cannot reliably predict LV filling pressure, and the majority of critically ill patients have an E/e' in this "grey zone", [29]; mitral stenosis and severe MR invalidate the measurement of mitral inflow velocities; sinus or arrhythmic tachycardia, particularly atrial fibrillation, and prolonged atrioventricular nodal conduction may lead to fusion of the E and A waves [10]; positive pressure ventilation could influence LV filling pressure in several ways, making these indices unreliable [26].

The evaluation of LV and LA strain by speckle tracking imaging is far more sensitive to acute changes in cardiac loading conditions than conventional echocardiography, therefore, it could represent an alternative approach to define treatment responsiveness among different clinical acute HF phenotypes [38, 39] (Fig. 3).

## Assessment of RV function and pulmonary artery pressures

### RV function

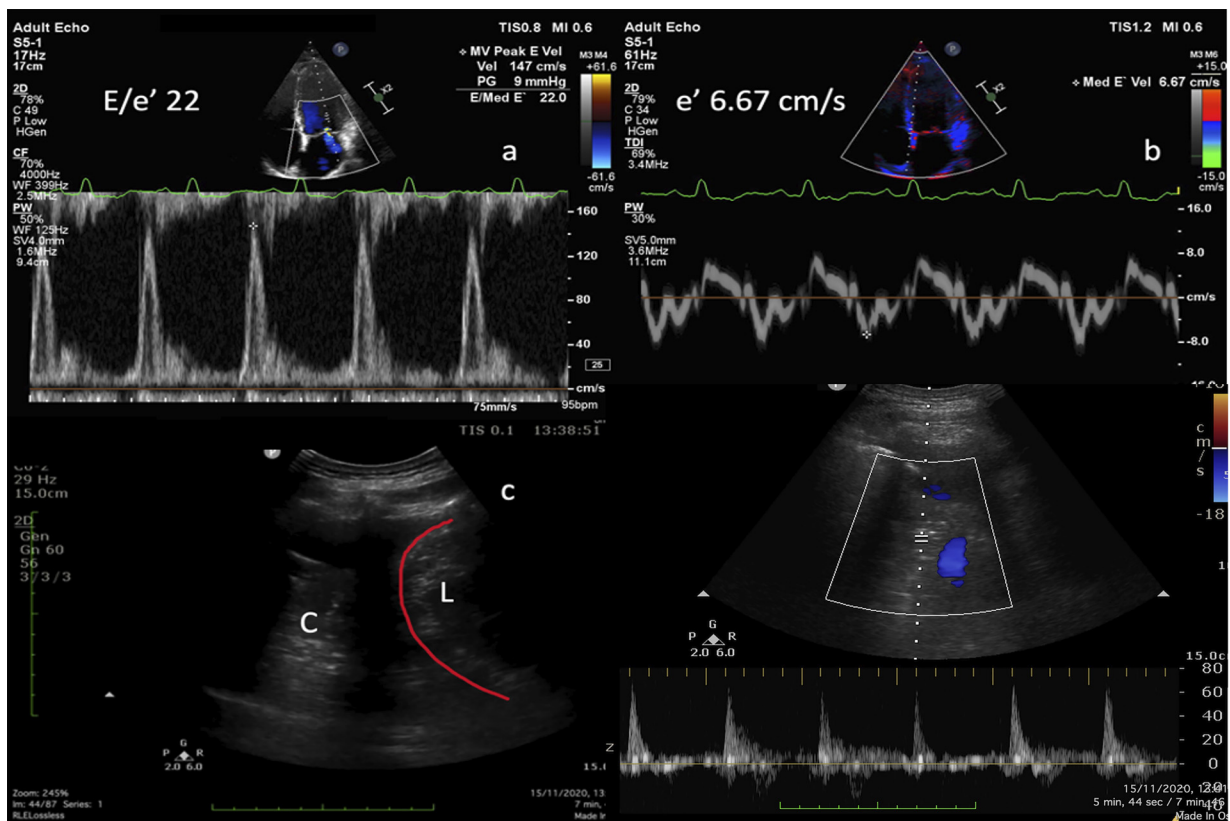
The RV is particularly vulnerable to different stresses that critically ill patients encounter as RV is less tolerant to large increases in preload or afterload. Besides positive pressure ventilation, RV function could also be worsened by hypoxemia, acidemia and tachyarrhythmias. Inferior myocardial infarction could lead to RV myocardial injury, leading to right HF (Fig. 4). RV dilatation and ventricular septal wall motion abnormalities are common signs of RV infarction and failure which could lead to apparent hyperdynamic LV (underfilled) due to ventricular interdependence.

Echocardiographic assessment of the RV in the critically ill patients should include the assessment of the RV size and systolic function as evaluated by RV Fractional Area Change (FAC), Tricuspid Annular Plane Systolic Excursion (TAPSE) and the tricuspid lateral plane systolic velocity by TDI (S').

RV restrictive physiology has been previously described after the surgical repair of Tetralogy of Fallot [40]. The presence of antegrade flow across the pulmonary artery at the end of diastole develops as the RV end-diastolic pressure becomes higher than pulmonary artery diastolic pressure, therefore, the RV will act as a conduit which will not be able to tolerate further increases in pressure and hence, atrial contraction is transmitted to the pulmonary artery. However, data are scarce in adult population regarding this parameter.







**Fig. 2.** Assessment of hypoxemia in a 71 years old patient who underwent a recent tissue aortic valve replacement. **a,b:** Transmitral pulsed-wave doppler (PWD) flow showing restrictive filling pattern and septal mitral annular early diastolic velocity by tissue-doppler showing evidence of diastolic dysfunction and elevated left atrial filling pressures as demonstrated by E/e' ratio = 22. **c:** Lung ultrasound assessment of the right costophrenic angle at the mid-axillary line is showing a hyperechoic shadow (C = consolidation) above the diaphragm (red line). L = Liver. **d:** Evidence of pulmonary arterial flow inside the pulmonary consolidation as shown by color flow doppler and PWD which confirms the presence of intrapulmonary shunting as a cause of the patient deterioration of oxygenation

An RV/LV diameter ratio <0.6 [41] and an RV sphericity index <0.5 [42] are normal reference values. TAPSE <15 mm measured by M-mode has shown to be 59% sensitive and 92% specific in detecting RV dysfunction [43].

$$\text{RV sphericity index} = \frac{\text{RV}_{\text{basal diameter}}}{\text{RV}_{\text{longitudinal diameter}} / \text{RV}_{\text{basal diameter}}}$$

### Pulmonary artery pressures (PAP)

Echocardiography is the most widely used non-invasive tool that can be used at the bedside for estimating PAP [44]. Systolic PAP (PASP) can be measured by estimating the peak systolic velocity of tricuspid regurgitation (TR) by CWD and then PASP is derived by adding the RAP to the TR peak systolic gradient. Measurement of the PA acceleration time (AcT) could indicate an elevation in the pulmonary vascular resistance (PVR) which is demonstrated by shortening of the AcT (Fig 5) with or without mid-systolic notching. Echocardiographic measurement of PVR is not fully validated to initiate or monitor treatment of pulmonary

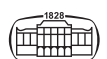
hypertension [45]. Therefore, this method should not replace invasive measurement by right heart catheterization.

$$\begin{aligned} \text{mPAP} &= 4(\text{PR peak velocity})^2 + \text{RAP} \\ \text{mPAP} &= \text{mean}\Delta P + \text{RAP} \\ \text{mPAP} &= 90 - (0.62 * \text{AT}_{\text{RVOT}}) \\ \text{mPAP} &= 2/3\text{rd of PADP} + 1/3\text{rd of PASP} \end{aligned}$$

$$\text{PVR} = (\text{Peak TR velocity (m/s)} / \text{VTIRVOT (cm)} * 10) + 0.16(60)$$

### Lung ultrasound

Lung ultrasound (LUS) can be extremely useful in patients in CICU, especially when coupled with echocardiography. An integrated cardiopulmonary ultrasound is highly informative in patients with acute HF of any etiology and, more generally, in patients with acute respiratory failure and hypotension/shock [46].



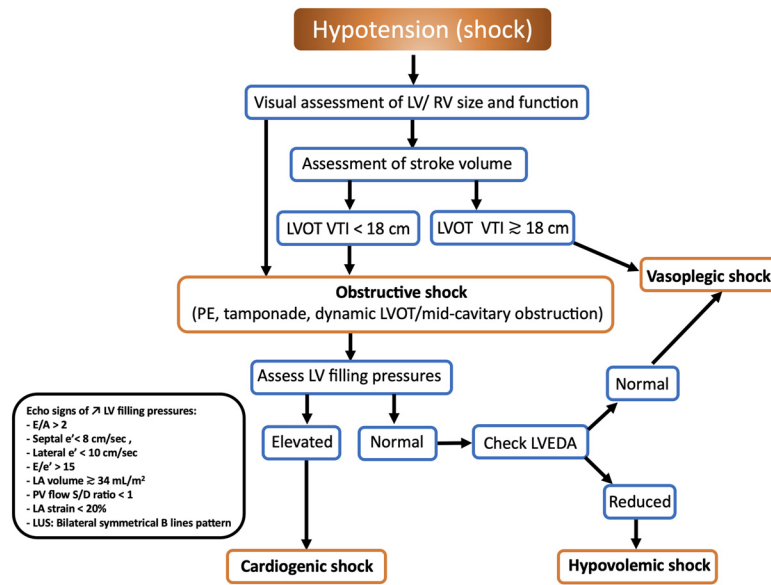


Fig. 3. Proposed algorithm for the Echo-guided management of patients with hypotension. E/A, early-diastolic wave by transmitral pulsed-wave doppler/late diastolic wave by transmitral pulsed-wave doppler; LA, left atrial; LUS, lung ultrasound; LV, left ventricle; LVEDA, left ventricular end-diastolic area; LVOT, left ventricular outflow tract; PE, pulmonary embolism; PV, pulmonary veins; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; tIVT, total isovolumic time; VTI, velocity time integral

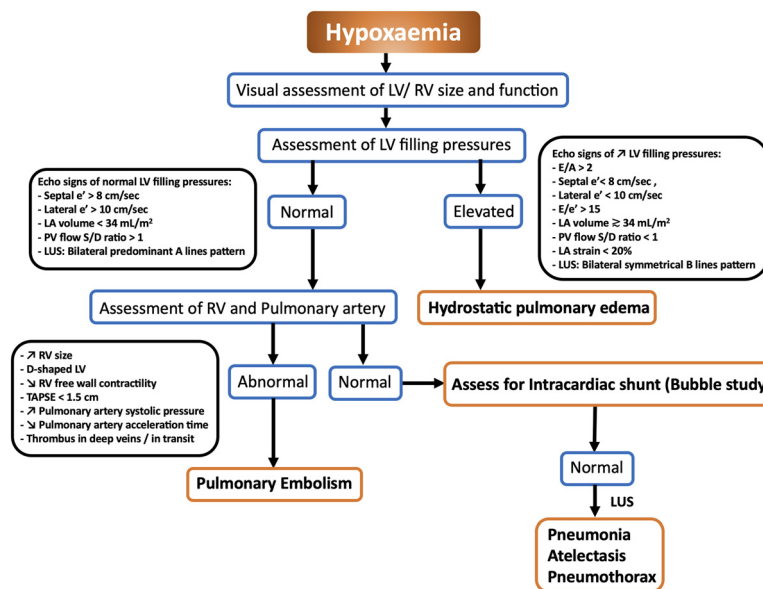


Fig. 4. Proposed algorithm for the Echo-guided assessment of patients with hypoxemia. ASD, atrial septal defect; E/A, early-diastolic wave by transmitral pulsed-wave doppler/late diastolic wave by transmitral pulsed-wave doppler; E', mitral annular velocity by tissue doppler imaging; LA, left atrial; LUS, lung ultrasound; LV, left ventricle, PFO, patent foramen ovale; PV, pulmonary veins; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion

LUS can support the diagnosis of acute HF, both in ruling it out, thanks to its high negative predictive value (close to 100%) [47], but also ruling in this condition, when the pattern of multiple, diffuse, bilateral B-lines is present [48]. In patients with hypotension/shock as well as in acute respiratory failure, both at admission and during ICU stay,

LUS combined with echocardiography can quickly confirm the clinical diagnosis, or exclude life-threatening conditions, such as cardiogenic shock, hypovolemia, cardiac tamponade, pulmonary embolism, pneumothorax, and may offer crucial information also in distributive shock in the context of pneumonia and sepsis [49, 50].



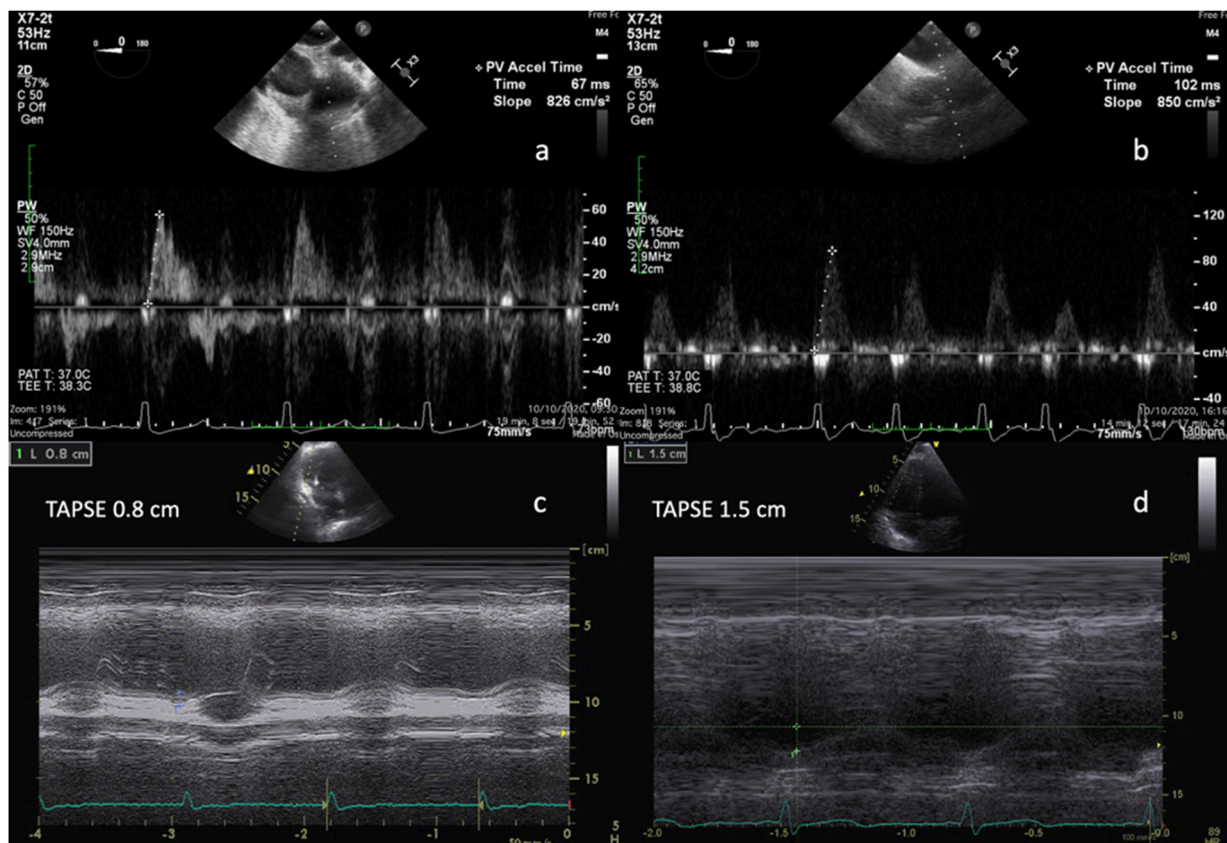


Fig. 5. The effect of pulmonary vasodilators (inhaled Nitric Oxide and Milrinone) on the pulmonary vascular resistance (PVR) and the right ventricular longitudinal function in a patient after mitral and aortic valve replacement.

- Upper oesophageal ascending aorta with transoesophageal echocardiography (TOE) view showing a pulsed wave doppler (PWD) tracing of the pulmonary artery with a very short acceleration time (67 ms) with short ejection time which demonstrates elevated PVR prior to receiving pulmonary vasodilators.
- Upper oesophageal ascending aorta TOE view showing a PWD tracing of the pulmonary artery in the same patient after pulmonary vasodilators showing an increased acceleration time (102 ms).
- Apical four chamber transthoracic echocardiography (TTE) view from the same patient showing a severely reduced RV longitudinal function (TAPSE: 0.8 cm) prior to pulmonary vasodilators.
- Apical four chamber TTE view showing a significantly improved RV longitudinal function (TAPSE: 1.5 cm) after treatment with pulmonary vasodilators

LUS is also valuable for monitoring and prognostic stratification of patients. In acute HF, it is possible to closely follow-up the dynamic variations of B-lines to better manage and titrate diuretic therapy [51]. Moreover, the residual number of B-lines at discharge, as a sign of persistent sub-clinical congestion, has a high prognostic impact in predicting new hospitalizations for acute HF in the following months [52]. Integrating echocardiography with LUS allows the simultaneous assessment of the cause of acute HF and the degree of decompensation, both in terms of hemodynamic congestion (with LV and LA volume, degree of MR, E/e' and other parameters of diastolic dysfunction), and of pulmonary congestion/extravascular lung water with estimation of B-lines numbers and distribution [53].

Similarly, LUS can monitor the evolution of pneumothorax, as well as the extension of pulmonary consolidations; it can be used to titrate ventilation parameters in intubated patients and to help understanding the correct timing of weaning [54]; the appearance of B-lines during fluid

administration without clinical improvement in hemodynamics, can also be a sign of distributive shock [50]. LUS has also been used widely during the COVID-19 pandemic as a safe, easy tool in monitoring the changes in aeration/deaeration [55]. Moreover, Transesophageal lung ultrasound (TELUS), as part of a TOE scan, has been recently proposed as an effective way of imaging the dorsal segments of the lung which are often missed in the supine ventilated patients [56].

## Assessment of acute aortic pathologies by TTE/TOE

Patients in CICU may present with acute chest pain due to acute aortic syndromes, which represents diagnostic and management challenges for clinicians. Aortic dissection represents 80–90% of the acute aortic syndromes, with

Stanford Type A (involving the ascending aorta) carrying high mortality risks and requiring timely surgical treatment.

Also, intramural hematoma (a hemorrhage into the medial layer, often located in the descending aorta) and penetrating aortic ulcer (an out-pouching in the aortic wall with jagged edges, usually in presence of significant atheroma) can break through the adventitia to form pseudoaneurysm or rupture into the mediastinum [57].

Rapid imaging is essential for the timely diagnosis and treatment of these potentially life-threatening conditions, considering atypical presentation, the often missed or delayed diagnosis and the time-dependent morbidity and mortality [58].

### Transthoracic echocardiography

Useful for a primary immediate scan, TTE may show the intimal flap and thickened aortic wall if involving the proximal 4–8 mm of ascending aorta which could be visualized in parasternal long- and short-axis views, or the part of descending aorta that could be seen in the apical 2-chambers view and sometimes in parasternal long-axis view (as a circular structure behind the LA) or the aortic arch, visualized from the suprasternal view. Moreover, complications of aortic dissection could be detected with TTE and regarded as indirect signs that could raise the suspicion for diagnosis: aortic regurgitation, pericardial effusion and/or tamponade, dilated ascending aorta.

The presence of normal aortic dimensions and geometry and the absence of aortic regurgitation on TTE suggest the absence of an ascending aortic dissection [59].

However, the sensitivity of TTE for the diagnosis of aortic dissection is 59–83% overall and 63–93% compared with other modalities. Its performance is better for Type A dissection (78–100% sensitivity vs. 31–55% for Type B dissection), even if a negative TTE could not fully exclude the diagnosis [60, 61].

### Transoesophageal echocardiography

Bedside TOE has become necessary to confirm the diagnosis of acute aortic syndromes in CICU, showing similar sensitivity and specificity to CT and MRI [62] with greater availability, and portability. TOE provides a real-time visualization of both ascending and descending aorta, due to the close proximity of the esophagus to thoracic aorta, with high spatial resolution and accuracy. The proximal 5–10 cm of the ascending aorta are visualized by scanning at a 120° imaging plane, descending aorta is visualized at 0° imaging plane advancing the transducer towards the gastro-esophageal junction and rotating it 180° posteriorly, then it could be withdrawn slowly to obtain a complete scanning of the aorta. The arch should be studied at the level of subclavian artery, with a 90° rotation to obtain an elongated view.

The dissection flap could be assessed (being careful to discriminate it from artefactual echoes) as a mobile intimal separation from the aortic wall with constant echo intensity along its course; color-flow imaging will also show margination of flow by a true dissection flap [63].

Aortic dissection is confirmed when two lumens separated by an intimal flap are visualized within the aorta; also, the identification of the false lumen sometimes is essential for surgical therapeutic planning:

1. In case of aortic arch involvement, surgeon needs to know whether the supra-aortic vessels arise from the false lumen [64].
2. In case of visceral arteries involvement in descending aorta dissection, with ischemic complications prior to surgery or endovascular therapy, since intimal fenestration may be an option when the main artery branches arise from the false lumen

TOE can reach a diagnostic sensitivity of 99% for aortic dissection with a specificity of 89%, positive predictive value of 89%, and negative predictive value of 99%. It has also shown to detect variants of acute aortic syndromes such as intraluminal hematoma [65].

Notably, TOE has shown 100% sensitivity in detecting aortic valve regurgitation resulting from aortic dissection and could provide information on its mechanism (dilatation of aortic sinus with cusps mal-coaptation or dissection flap reaching the sinus of Valsalva with cusps base disruption), that is crucial to decide whether valve-sparing surgery could be performed. Echocardiography is useful in evaluating pericardial effusion and wall motion abnormalities as consequences of aortic dissection.

Furthermore, bedside TOE in CICU could be useful to confirm post-operative aortic competence, when the aortic valve is preserved, and possible displacement of aortic prosthesis [66].

## Conclusions

CCE is one of the most useful tools in the evaluation of patients in cardiac intensive care units. Its unique ability as a non-invasive hemodynamic tool could empower the cardiac intensivist in addressing bedside clinical challenges. Moreover, it enables the delivery of a personalized management for each individual patient. Integrated cardiopulmonary ultrasound should be part of the daily assessment of patients as an extension to the bedside clinical examination.

Structured and comprehensive training programs are essential to ensure practitioners are well trained and formal certification and accreditation programmes are crucial as well as quality control and compliance with the local governance systems for the safe delivery of high-quality care for the critically ill patients.

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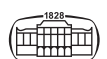
We are deeply grateful to our colleagues and the wider multidisciplinary team for their ongoing support and to our patients for being our everyday teachers.





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