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Narrative Review

A Critical Care Echocardiography—Driven Approach to Undifferentiated Shock

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Abstract

The clinical approach to undifferentiated shock in critically ill patients should be revised to use modern, point-of-care tools that are readily available. With the increasing availability of 2-dimensional ultrasonography and advanced Doppler capabilities, a quick, simplified, and integrated stepwise approach to shock using critical care echocardiography is proposed. Evidence supports the feasibility and usefulness of critical care echocardiography in enhancing diagnostic accuracy for shock, but there is a lack of systematic application of the technology in patients with undifferentiated shock. The proposed approach begins with the use of noninvasive ultrasonography with pulsed-wave Doppler capability to determine the flow state by measuring the velocity time integral of the left ventricular outflow tract. This narrative review explores the use left ventricular outflow tract velocity time integral, velocity time integral variation, limited visceral organ Doppler, and lung ultrasonography as a systematic approach for patients with undifferentiated shock.

Keywords: Critical care; echocardiography; shock, cardiogenic; hypovolemia; shock, septic; echocardiography, Doppler

Introduction

Shock occurs when there is a lack of oxygen supply compared with demand, which leads to insufficient oxygen delivery and dysoxia. In addition, various causes of shock may present simultaneously, resulting a complex shock state involving more than 1 type of shock. Each of the 4 types of shock can be present singularly or in combination at any time in critical illnesses.[1,2] Therefore, initial and subsequent hemodynamic monitoring at the bedside is necessary to guide the appropriate management of shock. Although they have their limitations, noninvasive measures of hemodynamic parameters, such as stroke volume and cardiac output, can help establish the nature of the shock, particularly when assessing patients in acute care settings.[3-5] Over the past decade, there has been a paradigm shift from invasive monitoring with static and dynamic measures to echocardiography at the point of care. Critical care echocardiography (CCE) can be defined as point-of-care echocardiography performed and interpreted by the treating clinician of critically ill patients to augment diagnosis, manage care, and guide invasive procedures.[6] Along with this paradigm shift, considerable evidence supports the feasibility and usefulness of CCE in enhancing diagnostic accuracy for shock. Given its growing usefulness at the bedside, CCE needs to be systematically applied as a monitor in patients with undifferentiated shock.

The current gold standard for hemodynamic monitoring is the pulmonary artery catheter because it is readily available to all team members, including cardiovascular disease specialists. The reliability of the device, however, and the benefit to harm ratio have been questioned.[7] Furthermore, the pulmonary artery catheter offers no visualization of the cardiac structures. Therefore, it cannot distinguish mechanisms of low- or high-cardiac-output shock or acute on

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chronic, right- or left-sided, or combined acute systolic heart failure.[8] An ideal approach would be noninvasive, continuous, repeatable, accurate, cost-effective, and easy to use, which are criteria that ultrasonography fulfills.[5]

Evidence shows a correlation between the noninvasive pulsed-wave Doppler estimation of stroke volume and cardiac output and pulmonary artery catheter–derived measurements in critically ill patients.[9] The American Society of Echocardiography also recommends transthoracic echocardiography or transesophageal echocardiography for assessing stroke volume and cardiac output.[10] Recent advances in ultrasonography have made CCE readily accessible to many physicians at the point of care. This narrative review presents a CCE-driven algorithm to systematically evaluate patients with undifferentiated shock (Fig. 1). Two example cases are also presented in which this algorithm has been used to diagnose and guide the management of undifferentiated shock.

Key Points

- An algorithm based on CCE is proposed to enhance the diagnosis of undifferentiated shock.
- This algorithm evaluates shock by using LVOT VTI, velocity time integral variability, limited visceral organ Doppler, and lung ultrasonography.
- The left heart, right heart, and lungs are evaluated in this approach to undifferentiated shock using CCE.

Abbreviations and Acronyms

CCE	critical care echocardiography
CVP	central venous pressure
IVC	inferior vena cava
LiVOD	limited visceral organ Doppler
LVOT	left ventricular outflow tract
LUS	lung ultrasonography
RAP	right atrial pressure
VTI	velocity time integral

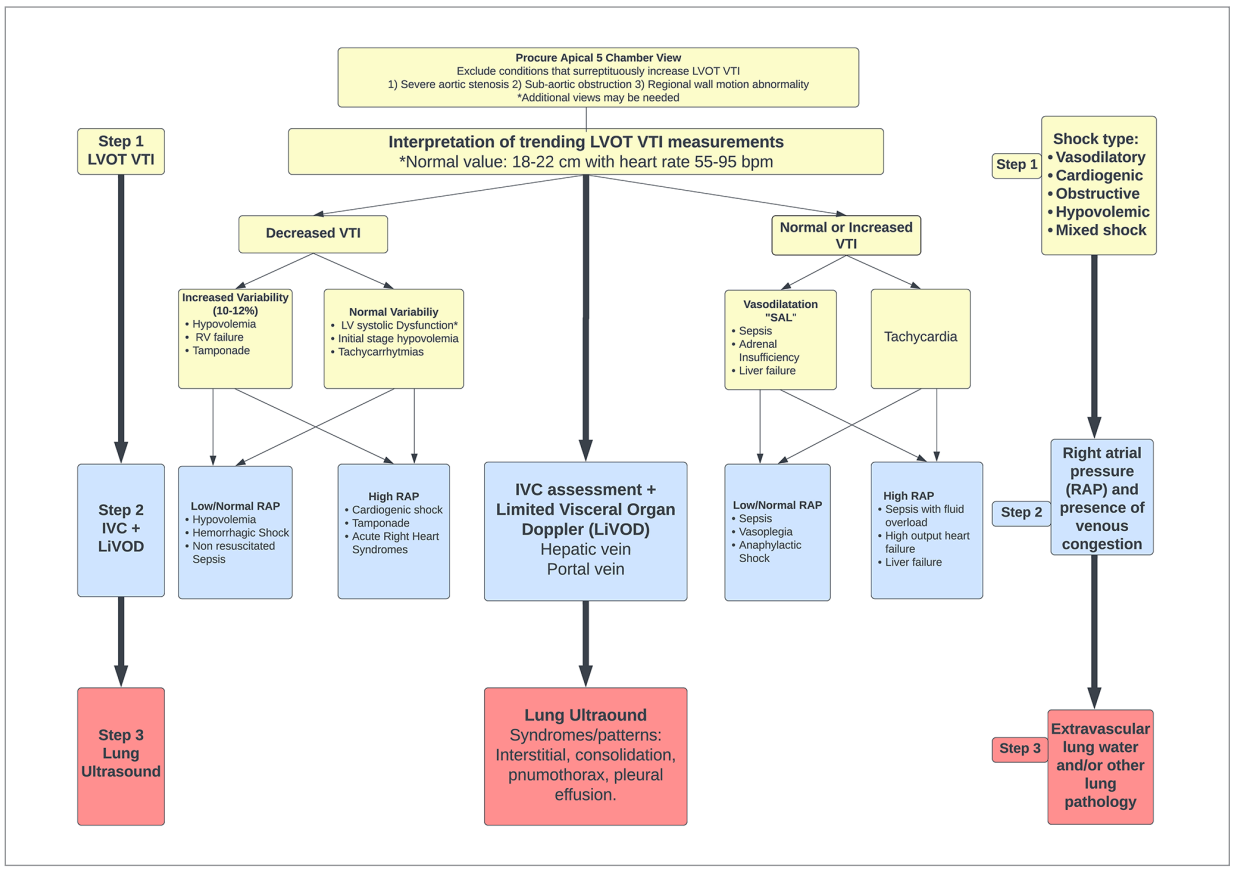

Fig. 1 Critical care echocardiography–driven algorithm to navigate undifferentiated shock using left ventricular outflow tract velocity time integral, velocity time integral variation, right atrial pressure, visceral organ Doppler, and lung ultrasonography.

*Except in cardiac alternans phenomena with a characteristic pattern of alternating VTI.

IVC, inferior vena cava; LiVOD, limited visceral organ Doppler; LV, left ventricle; LVOT, left ventricular outflow tract; RAP, right atrial pressure; RV, right ventricle; VTI, velocity time integral.

Methods

The CCE-driven algorithm for undifferentiated shock requires basic and advanced cardiac and noncardiac views. The first step of this algorithm (Fig. 1) involves obtaining an apical 5-chamber view to assess left ventricular outflow tract (LVOT) velocity time integral (VTI). The pulsed Doppler sample is positioned in the LVOT just below the aortic valve. It is crucial to align the LVOT parallel with the beam to avoid underestimating the proper stroke volume. The VTI is measured by tracing the spectral Doppler contour. Using the LVOT VTI instead of the calculated stroke volume can eliminate a potential source of the error caused by measuring the LVOT diameter. The normal VTI measurement range is between 18 and 22 cm, and VTI is considered to be severely decreased when it is less than 16 cm. The variability of the VTI waveform must also be assessed for differential diagnosis of shock states.[5] The LVOT VTI variability is calculated by subtracting the maximal LVOT VTI from the minimal LVOT VTI divided by the mean LVOT VTI. Aortic peak velocity can also be used to assess variation in LVOT VTI. If the variation is not visualized, this step can be omitted and the VTI assumed to have no variability. Variability greater than 10% is considered abnormal.[11,12]

The second step in the algorithm is to evaluate the right atrial pressure (RAP) (Fig. 1). The RAP evaluation consists of the inferior vena cava (IVC) assessment and limited visceral organ Doppler (LiVOD). The RAP is considered to be low or normal when the IVC diameter is less than or equal to 2.1 cm and has greater than 50% collapsibility (estimated central venous pressure [CVP], 0-5 mm Hg), and it is considered high when the IVC diameter is greater than 2.1 cm with less than 50% collapsibility (estimated CVP, 10-20 mm Hg).[13] Performing LiVOD interrogation is appropriate when intermediate values are seen for the IVC diameter and collapsibility (estimated CVP, 5-10 mm Hg).[14,15] The LiVOD assesses the hepatic venous flow pattern, which is closely related to the central venous flow pattern.[15] In individuals without severe tricuspid regurgitation, atrial fibrillation, or intrahepatic diseases that can alter Doppler findings, the S wave is more prominent than the D wave. The interpretation and limitations of hepatic vein Doppler are beyond the scope of this review, but a simplified interpretation can be used to understand the algorithm. An increase in RAP is considered when the S wave becomes less prominent than the D wave, and even a reversal of the S wave can be seen with severe RAP elevation.[16]

The third step of this algorithm incorporates lung ultrasonography (LUS) findings that include patterns such as pneumothorax, interstitial syndrome, pleural effusion, and alveolar syndrome. The correlation between B lines and extravascular lung water and pulmonary congestion has been well documented, and the same principle can be applied in this third step of the algorithm. For example, an increased number of B lines, usually greater than 3, can suggest pulmonary edema and elevated left-sided filling pressures.[17-19]

Case 1

A 60-year-old man who recently recovered from SARS-CoV-2 infection presented with 2 weeks of progressive orthopnea, paroxysmal nocturnal dyspnea, and dyspnea with minimal exertion. In the emergency department, he received 5 mg of intravenous metoprolol for atrial fibrillation, with a rapid ventricular rate of 170 bpm. The patient then became hypotensive, with a blood pressure of 88/56 mm Hg, and his extremities were noted to be cold to the touch. He was transferred to the intensive care unit in overt respiratory distress. Critical care echocardiography was performed, which revealed poor biventricular systolic function, determined by visual estimation of ejection fraction. The LVOT VTI was reduced to 12 cm (Fig. 2A). The IVC diameter was 1.9 cm without respirophasic variation, consistent with a CVP of approximately 5 to 10 mm Hg. The hepatic veins revealed systolic flow reversal consistent with increased RAP because there was a lack of severe tricuspid regurgitation as a confounder (Fig. 2B). The LUS showed extensive B lines in upper lung fields, which suggested pulmonary edema and elevated left-sided filling pressures (Fig. 2C, Fig. 2D). Cardiogenic shock resulting from acute on chronic systolic heart failure, volume overload, and fluid intolerance was diagnosed. The patient received medical therapy with intravenous digoxin, milrinone, amiodarone, and furosemide infusion. During the first 24 hours, his urine output was greater than 3 liters, serum lactic acid levels normalized, and his extremities became warm. Ten days after the resolution of heart failure symptoms and organ dysfunction and the restoration of sinus rhythm, cardiac magnetic resonance imaging was done for strong suspicion of myocarditis. This imaging showed evidence of myocardial edema and nonischemic myocardial injury consistent with the diagnosis of SARS-CoV-2 myopericarditis.

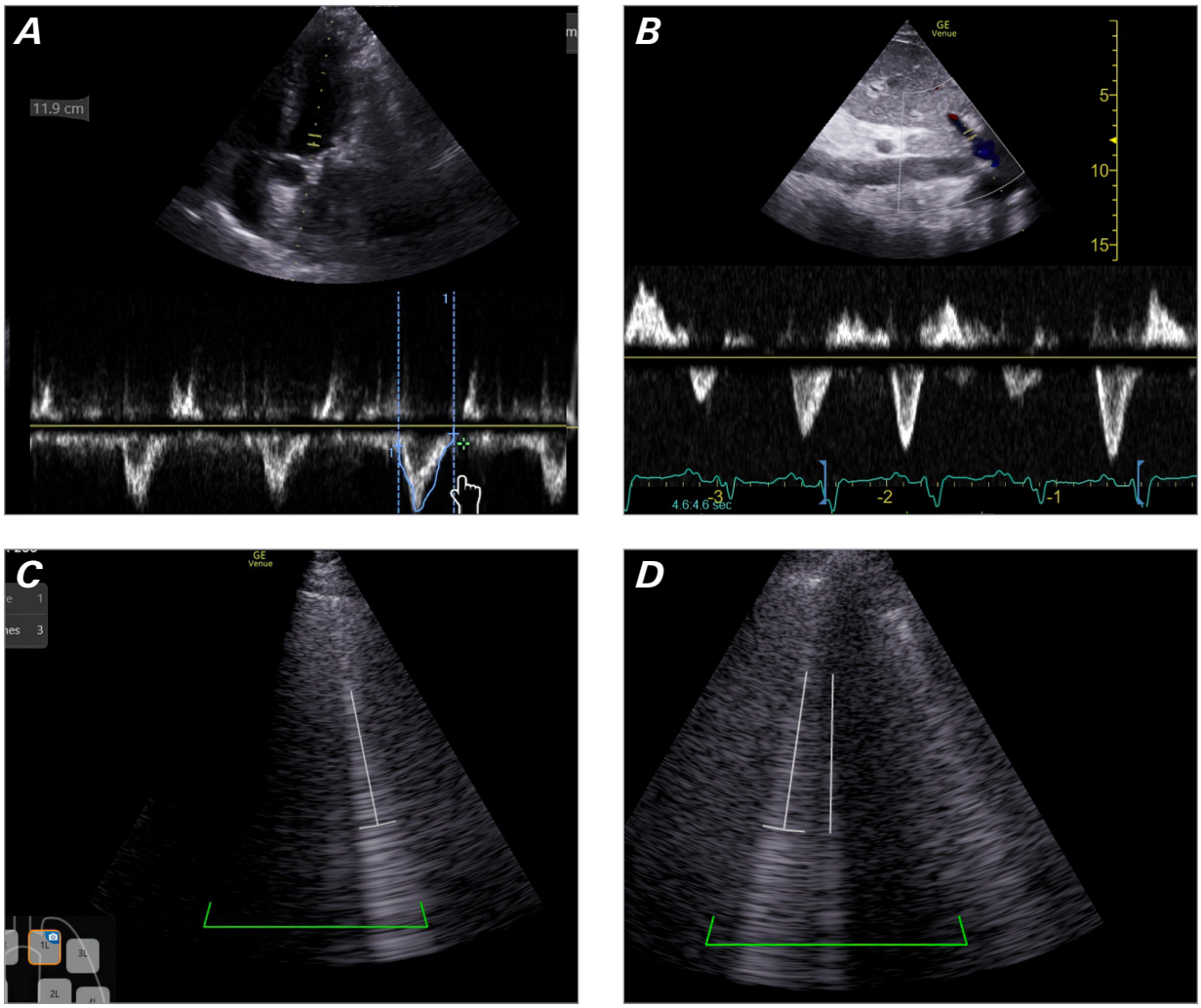

Fig. 2 Transthoracic echocardiogram from case 1. **A**) Pulsed-wave Doppler in apical 5-chamber view with left ventricular outflow tract velocity time integral measuring 12 cm, with correct position and angle. **B**) Hepatic vein Doppler showing systolic flow reversal. **C,D**) Lung ultrasonography views demonstrating increased B lines in bilateral upper lung fields.

Case 2

A 70-year-old man with a history of hypothyroidism presented with tachycardia, hypotension, and right flank cellulitis. He received 5 L of intravenous crystalloids for suspected septic and hypovolemic shock. Critical care echocardiography showed increased left and right ventricular systolic function (hyperdynamic) and LVOT of 20 cm (Fig. 3A). Variability in the LVOT VTI was not appreciated by automated ultrasonography or manual tracing, which suggested no VTI variability (Fig. 3B). The IVC was 2.5 cm in diameter and noncollapsible, which was consistent with RAP greater than 15 mm Hg (Fig. 3C). The LiVOD of the portal vein, however, showed a normal, continuous, and monophasic waveform (Fig. 3D). The LUS assessment revealed a bilateral B lines pattern on posterobasal lung zones, suggesting increased extravascular lung water and fluid intolerance (Fig. 3E, Fig. 3F). Therefore, no further fluid administration was provided. The patient was started on norepinephrine and broad-spectrum antibiotics. Computed tomography imaging of the abdomen revealed extensive fat stranding of the anterior abdominal wall and right flank. Because of his unresolved septic shock, the patient required an exploratory laparotomy, which confirmed severe necrotizing infection with frank purulence of fascia, muscle, and subcutaneous tissue that tested positive for methicillin-resistant *Staphylococcus aureus.* The patient was discharged home after a complicated 40 days of hospitalization with acute kidney injury, encephalopathy, anemia, and urinary tract infection.

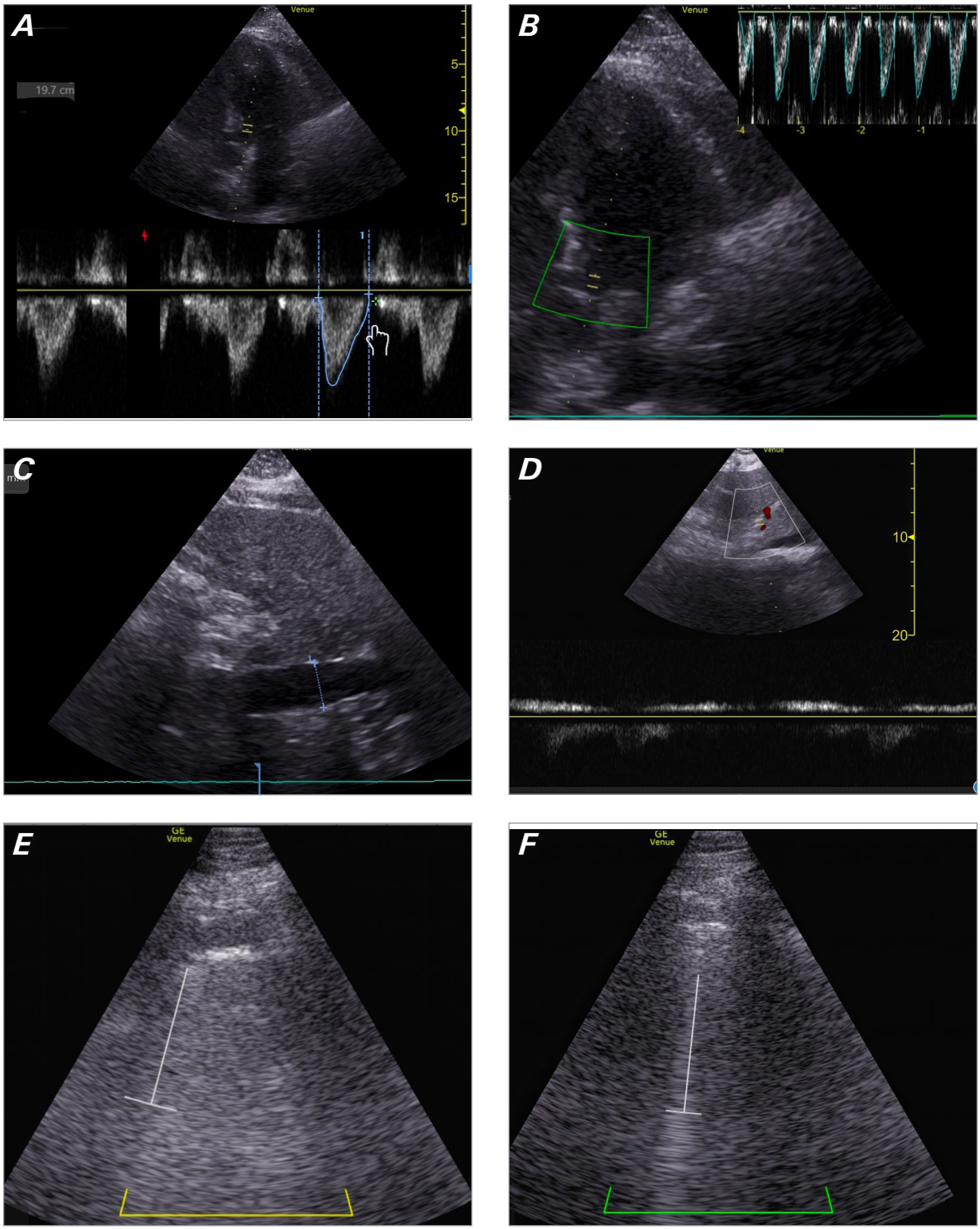

Fig. 3 Transthoracic echocardiogram from case 2. **A**) Left ventricular outflow tract velocity time integral measuring 20 cm, with correct position and angle. **B**) Automated ultrasonography velocity time integral variability tracing shows no variability. **C**) The inferior vena cava diameter was 2.5 cm with minimal collapsibility, consistent with elevated central venous pressure of more than 15 mm Hg. **D**) Portal vein Doppler image showing normal continuous and monophasic flow. **E,F**) Posterobasal lung zones showing a bilateral B-line pattern suggestive of increased extravascular lung water.

Discussion

This narrative review describes the systematic use of LVOT VTI, VTI variation, LiVOD, and LUS to approach patients with undifferentiated shock. This algorithm is especially applicable if there is a concomitant acute respiratory failure or suspected multifactorial shock. This approach provides guidance for identifying the cause of shock by assessing both the left and right heart and initiating subsequent tailored hemodynamic management.

In case 1, an initial structural assessment of the patient's ventricular function suggested potential biventricular systolic failure. Then, following the algorithm, the LVOT VTI was severely reduced, suggesting a low cardiac output state. The variability measurement of the LVOT VTI further indicated the volume status of the left ventricle. In this case, normal variability suggested a differential diagnosis of left ventricular systolic dysfunction, the initial stage of hypovolemia, or tachyarrhythmias. It is important to note that the variability of LVOT VTI is affected by a complex interaction of intrathoracic pressure changes. Therefore, the variability of LVOT VTI can be affected by spontaneous or positive pressure ventilation, changes in lung volumes, interventricular dependence phenomena, various loading conditions of the cardiac chambers, and pericardial constraints.[20,21]

In case 1, further evaluation of the right heart by assessing the IVC and visceral organ showed dilated IVC without respirophasic changes, and the hepatic vein Doppler interrogation revealed systolic flow reversal. This finding was consistent with RAP elevation and further solidified the differential diagnosis of cardiogenic shock, as shown in the algorithm (Fig. 1). Including LUS assessment in the algorithm can additionally help evaluate and exclude peripheral lung pathology contributing to respiratory failure and state of shock (eg, lung consolidation or pleural effusions) and assess for increased extravascular lung water from elevated left-sided filling pressures. As demonstrated in case 1, B lines in all lung fields were consistent with interstitial pulmonary edema, which is most likely hydrostatic and associated with elevated left-sided filling pressures and congestion. This finding further narrowed the diagnosis to cardiogenic shock, which can be confirmed with other clinical, hemodynamic, and laboratory data.

In case 2, the LVOT VTI was 22 cm without variability. The VTI without variability indicates a high cardiac output state with decreased systemic vascular resistance, suggesting septic shock etiology. The IVC evaluation was consistent with elevated RAP but without abnormalities on LiVOD, suggesting adequate volume resuscitation without severe venous congestion. The LUS showed increased B lines compatible with increased extravascular lung water, confirming the presence of pulmonary edema and intolerance to further fluid resuscitation.

In both cases, the evaluation of LiVOD and LUS guided the team in differentiating shock and assisted in assessment of volume status and fluid tolerance. The concurrent assessment of CVP and central volume status is designed to corroborate the shock state by characterizing the right side of the heart. Furthermore, the interrogation of hepatic and splanchnic veins aids in determining the impact of elevated CVP on visceral organ congestion. The LUS is also used to evaluate the transmission of high left-sided filling pressures and alternative shock diagnoses in patients with respiratory failure.[22] The discussion of fluid tolerance is an evolving one. The concept examines various fluid compartments and organs concerning congestion (fluid overload) leading to dysfunction or failure.[23] In this algorithm, fluid intolerance refers to a surrogate for the right-sided and left-sided filling pressures and the patient's ability to tolerate further fluid administration without greater damage to the vital organs.

As 2-dimensional ultrasonography and advanced Doppler capabilities have become more widely available, a quick, simplified, and integrated stepwise approach to shock using point-of-care ultrasonography is proposed. The authors recognize the need for further study to determine the impact of this novel approach on clinical outcomes, but this algorithm provides the beginning platform for an organized approach to complex, undifferentiated shock using CCE.

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Correction: The online article and PDF were corrected 26 October 2023. The title was incorrect and has been replaced.

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