Point-of-care Ultrasound after Cardiac Surgery
Understanding the Congestion Cascade

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KEYWORDS

• Point-of-care ultrasound • POCUS • Venous congestion • Shock in cardiac surgery

KEY POINTS

- Point-of-care ultrasound (POCUS) enables rapid, bedside assessment of hemodynamics in perioperative and critically ill cardiac patients.
- The *sonographic congestion cascade* provides a systematic, physiology-based framework from heart valves to peripheral veins.
- Integrated cardiac, lung, and venous Doppler ultrasound improves shock differentiation and volume assessment.
- Venous Doppler patterns help identify congestion phenotypes beyond traditional pressure-based metrics.
- Serial POCUS guides timely, targeted interventions and escalation of care.

INTRODUCTION

Point-of-care ultrasound (POCUS) refers to the use of portable ultrasonography performed and interpreted by the clinician at the bedside to guide real-time diagnosis and management.[1] POCUS offers immediate, dynamic visualization of anatomic structures and physiologic processes, making it an invaluable tool to augment clinical decision-making in the acute and perioperative care setting.

The use of POCUS in perioperative and critical care settings has steadily grown but with variable adoption across various specialties, training backgrounds, institutions, and health care providers. Initially adopted for rapid assessment of trauma and shock, its utility has expanded into the perioperative environment, POCUS has become instrumental for guiding vascular access, assessing volume status, and evaluating cardiac function.[2] Its integration into cardiac surgery represents a natural progression, given the complexity of hemodynamic management and the need for timely decision-making in this high-risk population.

POCUS can be utilized to rapidly diagnose and monitor common postoperative cardiac complications such as tamponade, ventricular dysfunction, hypovolemic/hemorrhagic shock, and volume overload.[3] POCUS allows clinicians to promptly identify life-threatening conditions, optimize fluid therapy, and guide pharmacologic or mechanical support. We present a protocolized systematic approach to assess patients using advanced whole-body ultrasound to identify etiologies of shock states and congestion.

SONOGRAPHIC CONGESTION CASCADE

The cardiovascular system can be envisioned conceptually as a series of valves, pipes, and pumps in continuity that span across from the

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Abbreviations	
A4C	apical 4 chamber
AR	aortic regurgitation
ARDS	acute respiratory distress syndrome
AT	acceleration time
AVA	aortic valve area
CFV	common femoral vein
DI	dimensionless index
ICU	intensive care unit
IVC	inferior vena cava
LUS	lung ultrasound
LV	left ventricle
LVOT	left ventricular outflow tract
LVOTO	left ventricular outflow tract obstruction
MR	mitral regurgitation
PA	pulmonary artery
PASP	pulmonary artery systolic pressure
PCWP	pulmonary capillary wedge pressure
PEEP	positive end-expiratory pressure
PLAX	parasternal long-axis
POCUS	point-of-care ultrasound
PSAX	parasternal short-axis
PVR	pulmonary vascular resistance
RVOT	right ventricular out flow tract
SAM	systolic anterior motion
TAPSE	tricuspid annular plane systolic excursion
TDI	tissue Doppler imaging
TR	tricuspid regurgitation
VTIs	velocity time integrals

cardiovascular outlet (ie, aortic valve) proximally through the left heart, lungs, right heart, inferior venae cava, hepatic vein, portal vein, and common femoral vein (CFV). This "cardiopulmonary venous unit" should be scrutinized to determine the point of obstruction within this congestion cascade (Fig. 1) to ensure timely diagnosis and management of critically ill patients. Understanding Doppler flow patterns within different vascular beds provides a deep understanding of physiology and opportunities to identify critical pathology.

The initial step in assessing patients with undifferentiated shock should involve ruling out time-sensitive pathologies that are immediately reversible and require different treatment pathways (obstructive shock—tension pneumothorax, pericardial tamponade, and acute obstructive pulmonary embolism). A thorough history, physical examination, laboratory investigations, and imaging help identify causes such as septic shock, anaphylactic shock, and severe endocrinopathies. After this initial assessment, a more comprehensive evaluation of the congestion cascade to systematically assesses each congestion point should be undertaken.

Aortic Valve

The aortic valve is visualized using a combination of echocardiographic views (parasternal long-axis, parasternal short-axis, 5 chamber, and apical windows) to assess the structural anatomy and rule out significant pathology. A focused evaluation should be conducted to assess for severe or critical aortic stenosis that can often precipitate or compound shock states. Two-dimensional imaging reveals thickened, heavily calcified, immobile cusps with restricted opening and can be further confirmed using Doppler to estimate peak velocity, mean transvalvular gradients, and calculate aortic valve area. Echocardiographic findings consistent with severe aortic stenosis include (1) elevated transvalvular velocity (>4 m/s^2) and (2) gradients (>40 mm Hg), with a calculated (3) aortic valve area (AVA) less than 1 cm^2.[4] AVA can be calculated using the continuity equation incorporating left ventricular outflow tract (LVOT) diameter and LVOT and aortic valve velocity time integrals (VTIs). Use of a dimensionless index (DI) can be useful as it obviates potential error laden LVOT measurements. The DI is defined as the ratio between LVOT and aortic valve velocities or VTIs, with DI less than 0.25 being strongly suggestive of severe aortic stenosis. Reduced left ventricular function or ventricular cavity size may lead to flow-dependent decrease in stroke volume due to low-flow low-gradient or paradoxic low-flow low-gradient aortic stenosis, thus masking true severe aortic stenosis. Suspicion should prompt more comprehensive imaging and consultation. Identifying severe or critical aortic stenosis as the primary driver of low cardiac output in shock states is imperative to provide appropriate treatment modalities that may include surgical replacement or transcatheter therapies.

Acute hemodynamic instability may also be due to severe regurgitant aortic valvulopathy and should be considered as a potential etiology of shock. The American Society of Echocardiography Guidelines on Valvular Heart Disease states echocardiographic parameters associated with severe aortic regurgitation (AR), which includes vena contracta greater than 0.6 cm, pressure half time less than 200 milliseconds, regurgitant volume greater than 60 mL, and regurgitant fraction greater than 50%.[4] However, this may be difficult to assess in an acutely decompensating patient and requires more comprehensive echocardiographic assessment. A broad, central AR jet on parasternal long or short axis, especially occupying 65% or greater of the LVOT width, suggests severe regurgitation. In acute settings, such as intraoperative or intensive care unit (ICU)

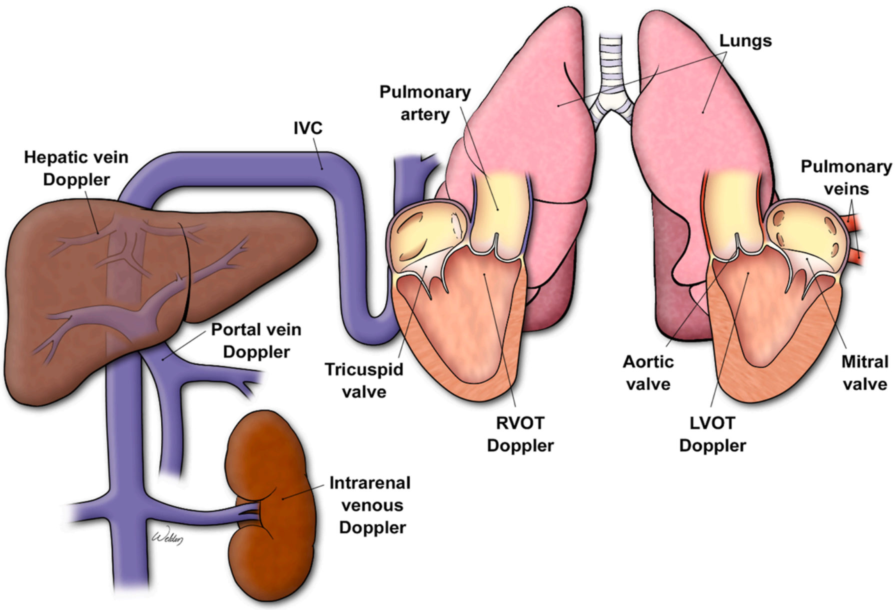


Fig. 1. Congestion cascade.

hypotension, recognition of a large AR jet necessitates prompt volume and afterload management to support forward cardiac output. Echo dense mobile masses should be sought that may represent infective endocarditis especially in the setting of regurgitation. Proximal aortic dissection should also be considered as an etiology of acute AR.

In the postoperative cardiac surgery patient, POCUS can assist in identifying complications related to surgical valve replacement such as paravalvular leak, valve thrombosis, or valve dehiscence but often requires further imaging with transesophageal echocardiography.

Left Ventricular Outflow Tract

Dynamic left ventricular outflow tract obstruction (LVOTO) is a significant and often underdiagnosed hemodynamic complication in the perioperative period, particularly following cardiac surgery.[5] Incidence varies but has been reported postaortic valve replacement, mitral valve surgery, and transcatheter aortic valve implantation. Echocardiography plays a central role in identifying, characterizing, and guiding management of this dynamic process.

Dynamic LVOTO arises due to a narrow LVOT, often due to interventricular septal hypertrophy and/or disrupted mitral valve structures, in the setting of altered loading conditions.[6] Postcardiac surgery, left ventricle (LV) volume and contractility can change rapidly because of volume shifts, adrenergic stimulation, or catecholamines. These changes lead to high-velocity flow through the LVOT, generating Venturi forces that draw the anterior mitral leaflet toward the septum—known as systolic anterior motion (SAM)—causing obstruction and often mitral regurgitation (MR). Characteristic echocardiographic features of dynamic LVOTO include

i. SAM of the anterior mitral leaflet: this can be easily detected by utilizing parasternal long-axis view of the outflow tract. Systolic motion can be detected using M-mode or observing septal contact during cardiac systole (aortic valve [AV] opening)
ii. LVOTO Doppler pattern showing dagger like appearance
iii. Late peaking systolic gradient
iv. Posteriorly directed MR

Early recognition is key to successful management, which requires volume resuscitation, use of pure vasoconstrictors, beta-blockade, and avoidance of inotropes and vasodilators.[7]

Left Ventricular Systolic and Diastolic Function

In POCUS, left ventricular systolic function is primarily assessed qualitatively rather than quantitatively, relying on visual estimation (“eyeballing”) of myocardial contractility. Multiple studies have demonstrated that visual estimation by trained clinicians correlates well with formal echocardiographic measurements, making it a reliable and efficient method especially in acute care settings.[8]

The parasternal long-axis (PLAX), parasternal short-axis (PSAX), apical 4 chamber (A4C), and subcostal views are used to evaluate and obtain an all-encompassing assessment of LV systolic performance. This provides a better understanding of both regional wall motion and global function. Care must be taken to obtain good acoustic views and to avoid foreshortening, which may be difficult in postoperative patients with less-than-ideal windows. Clinicians assess global contractility by observing (1) endocardial excursion, (2) myocardial thickening, and (3) overall ventricular size change between diastole and systole. Normal systolic function is characterized by vigorous inward motion and wall thickening, whereas reduced function presents as diminished contraction, minimal wall thickening, and reduced chamber emptying.

POCUS allows simple categorization of LV systolic function into broad clinical groups—hyperdynamic, normal, mildly reduced, moderately reduced, or severely reduced. A hyperdynamic LV should be investigated thoroughly. Although often seen in distributive shock or hypovolemia, it is characterized by near-complete cavity obliteration during systole. Important differential diagnoses in the perioperative acute care setting should be excluded and include

1. Decreased venous return: Preload-dependent state such as hypovolemic or hemorrhagic shock
2. Decreased flow from right to left heart: Right heart dysfunction
3. Nonoutflow tract flow out of the LV: Acute MR, and large, hemodynamically significant ventricular septal defect.
4. Increased contractility: Use of inotropic agents
5. Decreased afterload: Distributive shock, vasodilators, high output state

While qualitative assessment is the cornerstone of POCUS, semiquantitative methods may enhance accuracy in select settings. Fractional shortening, derived from M-mode measurements in the PLAX view, can provide an estimate of systolic function but is limited by geometric assumptions and regional wall motion abnormalities. Measurement of stroke volume and cardiac output using LVOT VTI can be used to monitor stroke volume/cardiac output and aid in titrating inotropic agents or fluid resuscitation. However, this requires advanced echocardiographic skillsets and is less commonly performed in routine POCUS due to time constraints and technical challenges.

Diastolic dysfunction is common in the critically ill and elderly patients and is associated with heart failure with preserved ejection fraction, hypertensive heart disease, and ischemia. Although formal diastolic assessment can be complex, POCUS offers simplified approaches that provide clinically meaningful insights.[9]

Surrogate markers of LV filling pressures and relaxation are used in POCUS rather than formal grading for rapid assessment. Key elements include mitral inflow Doppler patterns, tissue Doppler imaging (TDI) of the mitral annulus, left atrial size, and pulmonary venous congestion, often inferred from lung ultrasound (LS) findings.

Mitral inflow Doppler, obtained from the A4C view, measures early (E wave) and late (A wave) ventricular filling velocities during diastole. While interpretation of E/A ratios can be challenging in isolation, certain patterns are suggestive of abnormal filling. For example, a dominant E wave with rapid deceleration may indicate elevated left atrial pressure, whereas impaired relaxation is associated with reduced E velocity. However, mitral inflow alone is highly preload-dependent and should not be interpreted without additional data.[10]

TDI of the septal or lateral mitral annulus provides a relatively preload-independent measure of myocardial relaxation (e′ velocity). A reduced e′ velocity suggests impaired relaxation, while an elevated E/e′ ratio correlates with increased LV filling pressures. E/e’ ratio greater than 15 provides a simple binary cutoff that is useful in clinical practice. Although TDI requires additional training and appropriate machine settings, it is increasingly feasible with modern POCUS devices and adds significant diagnostic value.

Left atrial enlargement, assessed qualitatively in the A4C view, serves as a marker of chronically elevated filling pressures and long-standing diastolic dysfunction. In acute settings, LS complements cardiac POCUS by identifying B-lines, which reflect pulmonary interstitial edema and indirectly support the presence of elevated left-sided filling pressures. Rather than formal diastolic grading, POCUS emphasizes binary or pragmatic questions: Is diastolic dysfunction likely present? Are left ventricular filling pressures elevated? These answers directly influence management decisions, particularly fluid administration. Aggressive fluid resuscitation, in the presence of normal systolic function with evidence of elevated filling pressures and pulmonary congestion, may lead to clinical deterioration thus highlighting the importance of diastolic assessment in shock and respiratory failure.

Mitral Valve

In the immediate postoperative period and in acutely ill patients, mitral valve dysfunction may present abruptly and contribute to hypotension, pulmonary edema, hypoxemia, or low cardiac output state. Etiologies include ischemia-related papillary muscle dysfunction, volume shifts leading to functional MR, exacerbation of pre-existing valve disease, atrial arrhythmias, or complications following cardiac and noncardiac surgery.[11] POCUS enables clinicians to rapidly assess whether mitral valve pathology is present and whether it plausibly explains the patient's hemodynamic or respiratory instability.

In these settings, the goal of POCUS is not comprehensive valve quantification but rather early recognition of clinically significant mitral valve disease, differentiation of cardiac from noncardiac causes of deterioration, and guidance of immediate management of fluid administration, vasopressor or inotrope selection, comprehensive echocardiography and escalation to mechanical circulatory support.

Color Doppler is used to evaluate mitral valve function and should be carefully optimized to avoid artifact or misinterpretation. In postoperative and critically ill patients, image acquisition may be challenging due to dressings, mechanical ventilation, or patient positioning.

MR is the most common and clinically relevant mitral valve abnormality encountered in acute and postoperative care.[12] POCUS assessment of MR is based on the identification of systolic regurgitant flow into the left atrium using color Doppler. Features suggestive of hemodynamically significant MR include a large, holosystolic jet extending deep into the left atrium, a broad jet origin at the valve plane, and associated signs of elevated left atrial pressure such as pulmonary edema on LS.

Acute MR may occur due to papillary muscle ischemia or dysfunction, transient myocardial stunning, or abrupt changes in loading conditions following surgery. Unlike chronic MR, acute MR may present as severe respiratory failure and hypotension despite a nondilated left atrium, making rapid POCUS recognition particularly valuable. Identification of new or worsening MR in this context should prompt urgent cardiology consultation and comprehensive echocardiographic evaluation. Jet directionality may also indicate the underlying etiology of the regurgitant jet.[13] Posterior jets indicating either restricted posterior MV leaflets or prolapse of anterior leaflets. Conversely, anterior jets may indicate restricted anterior or prolapsed posterior leaflets. Large central jets may be indicative of annular dilation and functional MR while disruption or perforation of leaflets can create eccentric jets in association with vegetations. Eccentric jets and in patient with acute hypotension, the severity of MR can be severely underestimated and may require more comprehensive transesophageal echocardiography to better assess valve structure.[14]

Although less common in postoperative and acute care settings, mitral stenosis has important implications for fluid and heart rate management. On POCUS, mitral stenosis is suggested by restricted leaflet opening, thickened or calcified leaflets, and turbulent diastolic flow across the valve on color Doppler. Elevated diastolic inflow velocities may be appreciated with pulsed-wave Doppler by experienced operators.[4]

Recognition of significant mitral stenosis is critical in postoperative patients who develop pulmonary edema or hypotension in response to tachycardia or modest volume administration, as these patients are particularly sensitive to changes in diastolic filling time.[15]

Mitral valve POCUS should be integrated with the assessment of left ventricular systolic and diastolic function, left atrial size, and pulmonary findings. Functional MR related to left ventricular dilation or systolic dysfunction is common in critically ill patients and may improve or worsen dynamically with changes in preload, afterload, or contractility. Serial POCUS examinations are, therefore, particularly valuable in postoperative and ICU settings.

Lung and Pleural Ultrasound

LUS has become a core component of POCUS in perioperative and acute care settings, offering a rapid, bedside, radiation-free method to assess pulmonary pathology and guide real-time clinical decision-making.[16]

Perioperative and acutely ill patients frequently develop respiratory complications, including atelectasis, pulmonary edema, pneumonia, pneumothorax, and pleural effusion. Traditional imaging modalities such as chest radiography and computed tomography may be delayed, insensitive in early disease, or impractical in unstable patients.

LUS is highly sensitive at detecting interstitial and alveolar pathology and often outperforms chest radiography in acute settings. Its ability to be performed serially makes it well suited for perioperative monitoring and evaluation of evolving respiratory conditions.

LS is typically performed using a systematic approach, scanning anterior, lateral, and posterior lung zones when feasible. LS involves determining the presence of lung sliding, the predominant artifact profile and its distribution, and ruling out effusions and consolidations.[17] High-frequency linear probes are useful for pleural and superficial assessment, while curvilinear or phased-array probes allow deeper evaluation of lung, pleural, and diaphragmatic structures.

Interpretation of LS is pattern-based rather than anatomic. Key findings include

- Pleural sliding: Indicates lung apposition of the visceral and parietal pleural interface, which may assist in ruling out pneumothorax although lack of lung sliding may be due to a variety of reasons such as pleural blebs, previous surgery, and main stem intubation.
- A-lines: Horizontal reverberation artifacts consistent with normal aeration or hyperinflation.
- B-lines: Vertical, comet-tail artifacts arising from the pleura that indicate increased lung density due to interstitial or alveolar fluid. These lines emerge from the hyperechoic pleural line, they cast downward and efface intervening A-lines, and move with lung sliding.
- Consolidation: Tissue-like echotexture representing alveolar collapse or pneumonia, often with dynamic air bronchograms and irregular and thickened pleural lines.
- Pleural effusion: Anechoic or hypoechoic fluid collections, best visualized in dependent regions.

In the perioperative setting, LS is particularly useful for identifying common postoperative pulmonary complications. Atelectasis is frequent after general anesthesia and appears as dependent consolidations and can be monitored in response to recruitment maneuvers or changes in positive end-expiratory pressure (PEEP).[18] LUS can also rapidly confirm endotracheal tube position by assessing bilateral lung sliding.[19]

Postoperative hypoxemia is a common diagnostic challenge. LS allows rapid differentiation between atelectasis, pulmonary edema, pleural effusion, pneumothorax, and aspiration.[20] This facilitates targeted therapy and may reduce unnecessary imaging or empiric treatments.

In acute care and critical care settings, LS plays a central role in the evaluation of acute respiratory failure, shock, and undifferentiated dyspnea. Diffuse bilateral B-lines suggest cardiogenic pulmonary edema or fluid overload, whereas focal B-lines or consolidations are more consistent with pneumonia or lung contusion.

LUS is a key component of integrated POCUS protocols for shock, where it complements cardiac and vascular ultrasound. For example, the presence of diffuse B-lines alongside elevated cardiac filling pressures supports a cardiogenic etiology and argues against aggressive fluid resuscitation. Conversely, a predominantly A-line pattern may suggest hypovolemia or obstructive lung pathology. Changes in LS patterns over time provide insight into disease progression and response to therapy.

One of the most impactful uses of LS in perioperative and acute care is guiding fluid therapy. The appearance or progression of B-lines serves as a marker of extravascular lung water and reduced fluid tolerance. Serial LUS examinations allow clinicians to balance resuscitation needs against the risk of pulmonary edema, particularly in patients with cardiac dysfunction, sepsis, or renal failure.

Despite its strengths, LS has limitations. It cannot visualize pathology deep within normally aerated lung and is operator-dependent. Interpretation must account for patient positioning, mechanical ventilation, and chronic lung disease, which may alter baseline findings. LS should be integrated with clinical assessment and other POCUS findings rather than used in isolation.

Right Ventricular Out Flow Tract Doppler

Right ventricular out flow tract (RVOT) Doppler measures blood flow velocity across the right ventricular outflow tract into the pulmonary artery during systole, reflecting the interaction among right ventricle (RV) contractility, preload, and afterload (pulmonary vascular resistance, impedance, and pulmonary artery pressure). Pulsed-wave Doppler is typically obtained in the PSAX or modified PLAX (right ventricular outflow) at the level of the great vessels, with the sample volume placed just proximal to the pulmonary valve.[21] Proper alignment of the Doppler beam with flow is essential for accurate waveform interpretation.

Unlike isolated pressure or volume metrics, RVOT Doppler waveforms capture dynamic RV–pulmonary artery coupling, making them particularly useful in conditions where pulmonary vascular load changes rapidly.[22]

A normal RVOT Doppler waveform has a smooth, symmetric, parabolic systolic envelope with peak velocity occurring in midsystole. The acceleration time (AT)—defined as the interval from onset of flow to peak velocity—is relatively prolonged, reflecting low pulmonary vascular resistance and normal RV ejection.

A shortened RVOT AT is a key marker of elevated pulmonary vascular resistance (PVR) or pulmonary hypertension. As RV afterload increases, peak velocity is reached earlier in systole, resulting in a triangular or sharply peaked waveform. In critically ill patients, a markedly shortened AT suggests acute increases in PVR, such as pulmonary embolism, acute respiratory distress syndrome (ARDS), hypoxic pulmonary vasoconstriction, or high intrathoracic pressures from mechanical ventilation.

Notching reflects reflected pressure waves from the pulmonary circulation returning during systole before pulmonary valve closure and opposing RV ejection. In acute care settings, early systolic notching can be associated with acute pulmonary embolism, with mid or late notching being seen in advanced ARDS or severe pulmonary hypertension of any cause. The presence of notching correlates with worse RV–pulmonary artery coupling and poorer prognosis and outcomes.

RVOT VTI can serve as a surrogate for RV stroke volume. A reduced RVOT VTI suggests decreased RV forward flow, which may result from impaired RV contractility, severe RV afterload, hypovolemia, or tamponade physiology. In shock states, a low RVOT VTI indicates compromised RV output and may explain systemic hypotension or low cardiac output. In RVOT Doppler patterns with notching, high post notch velocities can be indicative of preserved RV ejection while lower postnotch velocities may be indicative of a poor RV systolic function with inability to generate high velocities and is associated with poor outcomes (Fig. 2).

RVOT VTI can also be used serially to assess response to therapeutic interventions, such as fluid administration, vasopressor or inotrope initiation, pulmonary vasodilators, or ventilator changes. An increase in RVOT VTI following intervention suggests improved RV forward flow, whereas lack of change may indicate persistent afterload mismatch or contractile failure.

Right Ventricular Function and the Tricuspid Valve

The RV is highly sensitive to changes in preload, afterload, and intrathoracic pressure, RV dysfunction frequently contributes to shock, hypoxemia, and perioperative instability. A focused POCUS approach allows clinicians to rapidly assess RV size and systolic performance, identify clinically significant tricuspid valve disease, and estimate pulmonary artery (PA) pressures to guide timely management.

The RV should be evaluated systematically for size, wall thickness, systolic function, and interaction with the LV, while the tricuspid valve is assessed for leaflet morphology, coaptation, and regurgitation using 2 dimensional imaging and color Doppler.[23]

RV size provides critical insight into acute or chronic pressure and volume loading. In the A4C view, RV dilation is assessed relative to the LV. A normal RV is smaller than the LV; an RV-to-LV end-diastolic ratio approaching or exceeding 1 suggests significant RV dilation. In the PSAX view, interventricular septal flattening resulting in a “D-shaped” LV indicates RV pressure or volume overload and reflects adverse RV–LV interaction that can impair cardiac output.

Qualitative visual assessment of RV free wall motion remains the cornerstone of RV systolic evaluation in POCUS. Reduced inward motion or global hypokinesis suggests RV dysfunction. Semiquantitative measures can enhance assessment when feasible.

- Tricuspid annular plane systolic excursion (TAPSE):
  TAPSE is measured using M-mode through the lateral tricuspid annulus in the A4C view and reflects longitudinal RV shortening. Reduced TAPSE indicates impaired RV systolic function. Although loading conditions influence TAPSE, it is simple, reproducible, and widely used in acute care.
- Tricuspid annular systolic velocity (S′):
  S′ is obtained using TDI at the lateral tricuspid annulus and provides a velocity-based assessment of RV longitudinal contraction. Reduced S′ supports the presence of RV systolic dysfunction and complements TAPSE, particularly when M-mode alignment is suboptimal.

Both TAPSE and S′ should be interpreted in conjunction with visual assessment and clinical context, as they represent longitudinal rather than global RV function.

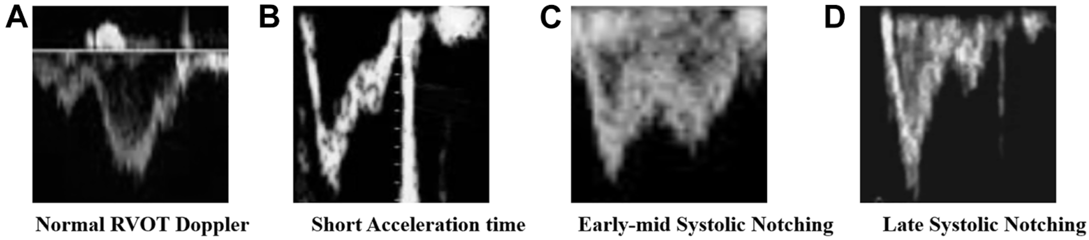


Fig. 2. RVOT doppler patterns.

The tricuspid valve should be evaluated for leaflet mobility, thickness, masses, and coaptation. Tricuspid regurgitation (TR) is common in critically ill patients and may be functional, resulting from RV dilation and annular enlargement. Color Doppler is used to identify and qualitatively assess TR severity. While precise quantification is beyond the scope of POCUS, recognition of moderate-to-severe TR is clinically important, as it influences RV preload, venous congestion, and interpretation of Doppler-based pressure estimates.

POCUS allows noninvasive estimation of pulmonary artery systolic pressure (PASP) using Doppler assessment of the tricuspid regurgitant jet. Continuous-wave Doppler is aligned with the TR jet in the A4C or parasternal view to measure peak TR velocity. PASP is estimated using the modified Bernoulli equation and an estimate of right atrial pressure, often inferred from inferior vena cava (IVC) size and collapsibility.[4]

Elevated estimated PA pressures suggest increased RV afterload due to pulmonary hypertension, acute pulmonary embolism, hypoxic pulmonary vasoconstriction, or high intrathoracic pressures. In the critically ill, these estimates are most useful for identifying significantly elevated pressures rather than precise quantification.

RV and tricuspid valve assessment with POCUS is limited by image quality, operator experience, and the influence of loading conditions and mechanical ventilation. Estimation of PA pressures depends on the presence and quality of a TR jet and accurate right atrial pressure estimation. POCUS should be viewed as a focused, bedside assessment rather than a substitute for comprehensive echocardiography.

Inferior Vena Cava

Assessment of IVC is one of the most misunderstood aspects of POCUS with many false assumptions being made regarding volume status with IVC measurements, collapsibility, and distensibility. The IVC represents right-sided filling pressures and may or may not correlate with volume status. For example, in obstructive physiology, marked increase in right-sided filling pressures can occur despite no change in volume status and hence represent a congestion point along the cardiovascular system. Volume removal in response to a large IVC or elevated right side filling pressures may be catastrophic in these instances of preload dependence for forward flow.

IVC ultrasound can be used to estimate right atrial pressure but may not be accurate in critically ill patients. Furthermore, IVC collapsibility index greater than 50%, in spontaneously breathing patients, may suggest low right atrial pressures and volume responsiveness. In mechanically ventilated patient, IVC distensibility index greater than 20%, may indicate volume responsiveness. However, IVC may be affected by a multitude of factors including PEEP, intra-abdominal pressure, pericardial tamponade, volume status, right atrial pressure, TR, right ventricular dysfunction, and elevated pulmonary pressures. Hence, it is crucial not to equate IVC dimensions to volume status and approach IVC assessment by understanding its clear limitations. IVC POCUS is most helpful in extremes of pathology (eg, small, collapsible IVC in mechanically ventilated patients which may be reflective of hypovolemic shock), hence we suggest always to incorporate IVC findings with hepatic and portal vein Doppler.

Hepatic Vein Doppler

The hepatic veins drain directly into the right atrium in a hepato-fugal manner. The hepatic veins, thus, provide insightful information on hemodynamics of the right-sided heart structures.[24] Thus, a complete understanding of Doppler waveform patterns in this vascular structure can help diagnose and manage perioperative critically ill patients and can be especially useful in patients with cardiac comorbidities.

With atrial depolarization, the right atrium contracts leading to retrograde flow into the hepatic vein, which is represented by an initial atrial reversal wave. With right ventricular contraction during cardiac systole (occurring between peak of the R-wave and end of T-wave), the tricuspid valve is displaced downward (given the downward twisting movement of the right ventricle caudally). Thus, increasing right atrial volumetric dimensions leading to a suction force that creates antegrade flow from the hepatic veins to the right atrium, this initial predominant forward flow is known as the S-wave (a systolic event). As the right atrium fills with blood the pressure builds up until a point where atrial pressure is greater than right ventricular pressure leading to the opening of the tricuspid valve and forward flow during diastole. Abnormalities of atrial reversal wave, S-wave, and D-wave are summarized in **Figs. 3** and **4**.

Blunting of the S-wave can indicate loss of atrial contraction, TR, right ventricular dysfunction, volume overload, and/or pulmonary hypertension. Complete flow reversal of the S-wave can be seen in extreme forms of TR and right heart dysfunction.[25] With predominately valvular regurgitation, systolic flow reversal is late. With right ventricular systolic dysfunction, flow reversal is early. However, when combined pan or holosystolic

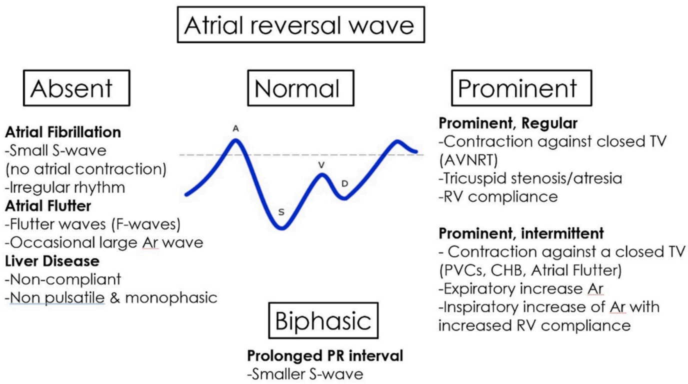


Fig. 3. Atrial reversal wave abnormalities in hepatic vein Doppler.

flow reversal can be seen. HV Doppler, thus, allows bedside assessment and monitoring of congestion and right heart function.[26]

Portal Vein Doppler

Portal vein can be easily imaged using a transhepatic lateral view. The portal vein can be viewed anterior to the hepatic vein structures. It can be distinguished from other vascular structures with its hyperechoic rim, hepato-pedal flow, proximity to portal triad, and bifurcation to left and right portal veins. The portal vein drains mesenteric vessels toward the liver in a hepato-pedal manner. Doppler interrogation of portal vein flow in euvolemic patients shows monophasic flow with minimal phasicity or pulsatility. Portal vein (PV) Doppler may be a better reflection of central volume overload and indicate splanchnic congestion. With increased central volume overload the splanchnic circulation becomes more pressurized and hence more pulsatile. The degree of pulsatility can be quantified and categorized as mild, moderate, or severe by use of a pulsatility index. More severe forms of volume overload may be seen as pulsatile and biphasic waveforms indicating retrograde flow within the portal vein.

Postal vein flow is somewhat buffered by the liver sinusoids and hence why it better reflects mesenteric flow patterns.[27] However, very high right atrial pressures can still cause backward congestion and lead to pulsatile portal vein flow.[28] Noncompliance of the liver may also blunt these transmissions. For example, a patient who received over judicious volume resuscitation may have normal hepatic vein morphology and right atrial pressures but exhibit portal vein pulsatility due to central volume overload. Conversely, patients with extreme right-sided filling pressures can also have pulsatile PV waveforms due to both backward congestion and volume overload. Understanding congestion phenotypes becomes imperative in the perioperative and acute care setting.

Intrarenal Venous Doppler

Intra-renal venous doppler (IRVD) evaluates venous blood flow patterns within the kidney, typically using the interlobar veins. The waveform reflects how pressure is transmitted from the right heart and venous system into the kidney.[29] Typical flow patterns include

- *Continuous flow*: Normal or low venous pressure
- *Pulsatile flow*: Mild-to-moderate congestion
- *Biphasic or monophasic flow*: Severe congestion, associated with worse renal outcomes

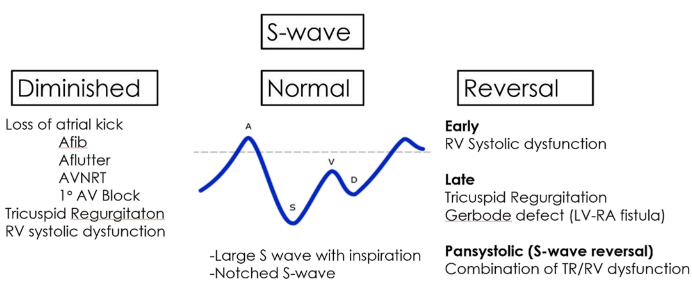


Fig. 4. S-wave abnormalities in hepatic vein Doppler.

Use of IVRD may be more technically challenging but complements and can be integrated with IVC ultrasound, hepatic/portal venous Doppler, and echocardiography as part of congestion cascade assessment.

Common Femoral Vein Pulsatility

CFV Pulsatility assessed by bedside ultrasound is an emerging, noninvasive marker of systemic venous congestion with growing relevance in the perioperative setting. CFV pulsatility refers to the transmission of right-sided cardiac pressure changes into the femoral vein, visible as phasic or pulsatile Doppler waveforms rather than continuous venous flow.[30] Assessment of the CFV is easily performed with easy accessibility and repeatability. Cardiac transmission or pulsatility into the CFV represents severe forms of venous congestion. When assessing the CFV with Doppler care must be taken not to confuse respiratory phasicity or transmission of pulsatility from the adjacent common femoral artery, with transmitted backward congestion from high right atrial pressures. CFV Doppler can be useful when IVC or hepatic/portal venous Doppler is not feasible (eg, surgical dressings and obesity). Waveforms can be influenced by mechanical ventilation, intra-abdominal pressure, and arrhythmias and should not be assessed in isolation.

CONGESTION PHENOTYPES

After systematically assessing the congestion cascade, it is important to contextualize sonographic findings in a whole-body approach to undifferentiated shock, respiratory failure, and assessment of volume status. Patients can be categorized into congestion phenotypes.[31] We present several illustrative examples that highlight key differentiating points between these phenotypes that would affect management and therapeutic strategies. **Table 1** summarizes the key aspects.

Hemodynamic Congestion

In this phenotype there are high filling pressures within the heart but no sonographic evidence of congestion. This is highlighted for example, in cases of hypertensive urgency (high afterload state), or acute elevations of left-sided filling pressures. If an invasive PA catheter is placed, there will be evidence of high pulmonary capillary wedge pressure (PCWP). LS would reveal predominant b-line pattern or profile indicating pulmonary congestion. Right-sided filling pressures may be elevated, however, only when left-sided filling pressures are sustained and prolonged and "spill" over to the right side. HV Doppler may be slightly abnormal and PV Doppler usually monophasic with minimal pulsatility. In these cases, aggressive diuresis in response to a high PCWP would be detrimental as there is no sonographic evidence of venous congestion. The patient would be better served addressing high afterload and receiving positive end expiratory pressure as a temporizing measure for increased extravascular lung water.

Volume Overload

This phenotype can be illustrated in a patient with normal biventricular and valvular function who receives over judicious fluid administration. Due to preserved heart function even excessive amounts of volume will not lead to exorbitantly high filling pressures. The hepatic vein (HV) Doppler may be slightly abnormal (never showing complete flow reversal as this necessitates moderate-to-severe TR and or RV dysfunction usually in combination), however PV Doppler will show pulsatility. Hence, despite only a slightly elevated PCWP, this patient should be diuresed given evidence of venous congestion. This optimizes forward flow and often results in improvement in mean arterial pressure. Hence, diuresis or volume removal would be recommended even if the patient is on pressors if there is significant sonographic venous congestion. Reliance on PCWP would be misleading.

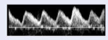
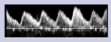
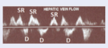
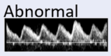
Systemic Congestion

Invasive PA catheter hemodynamics, unlike the earlier 2 phenotypes, is congruent with sonographic venous congestion findings in these patients. This can be seen in patients with severe ventricular dysfunction and valvular regurgitation who develop systemic venous congestion. These patients have elevated PCWP, elevated right-sided filling pressures, systolic flow reversal in HVs, and marked pulsatility in PV. In these cases, expedient diuresis or renal replacement therapy in combination with inotropic support and timely escalation of MCS is required.

Right Heart Apparatus Dysfunction

In these patients, left filling pressures may be normal or even low. Right-sided filling pressures are markedly elevated. Since the HVs reflect right filling pressures, HV Doppler will be invariably abnormal. However, PV Doppler can be used to determine volume status. Marked pulsatility indicates not only high right atrial pressures but also central volume overload. Nonpulsatile waveforms indicate a fluid tolerant state. In these circumstances, where patients are preload sensitive (pulmonary hypertension, severe aortic stenosis,

Table 1
Congestion phenotypes

Phenotypes	Left Sided filling Pressures	Right Sided Filling Pressures	Hepatic Vein Doppler	Portal Vein Doppler	Treatment
Hemodynamic	Elevated	Elevated	Normal/Mildly abnormal	Normal/Mildly Abnormal	?Afterload reduction
Volume Overload	Normal/ Slightly elevated	Normal/ Slightly elevated	Mildly abnormal 	Abnormal 	Volume removal
Systemic Venous Congestion	Markedly Elevated	Markedly Elevated	Abnormal 	Abnormal 	Inotropy + Volume removal
Right Heart Apparatus Dysfunction	Normal/Low	Markedly Elevated	Abnormal 	Abnormal  Normal 	• RV Support + gentle diuresis • RV Support – optimize preload

dynamic LVOTO, and so forth), keeping PV waveforms slightly pulsatile is preferred. Continued volume removal in these patients will lead to predictable decrease from marked pulsatility to minimal pulsatility however further volume removal once PV Doppler waveforms are near normalized or monophasic may lead to hypovolemia and compromise stroke volume.

SUMMARY

POCUS has become an essential tool in the postoperative management of cardiac surgery patients, offering rapid, bedside, and repeatable assessment of hemodynamics, cardiac function, pulmonary function, and volume status. Understanding the congestion cascade allows systematic evaluation of congestion points along the cardiopulmonary venous unit in patients with undifferentiated shock and respiratory failure.

CLINICS CARE POINTS

- Do not rely on a single POCUS variable—integrate cardiac, lung, and venous findings to accurately define hemodynamics and guide therapy.
- In patients with hypoxemia or undifferentiated shock - apply a systematic approach utilizing an understanding of the 'congestion cascade' and examining each congestion point.
- IVC size and collapsibility/distensibility reflects right atrial pressure—not volume status—misinterpretation can lead to harmful fluid or diuretic decisions.
- Venous Doppler (HV, PV, IRVD) and invasive filling pressures can be combined to determine congestion pheontypes (hemodynamic congestion, volume overload, systemic congestion, and right heart apparatus dysfunction) and hence appropriate treatment.

DISCLOSURE

None.

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