Comprehensive Assessment of Fluid Status by Point-of-Care Ultrasonography

Eduardo R Argaiz\textsuperscript{1}, Abhilash Koratala\textsuperscript{2} and Nathaniel Reisinger\textsuperscript{3}

\textsuperscript{1}National Institute of Medical Sciences and Nutrition Salvador Zubirán, Mexico City, Mexico
\textsuperscript{2}Medical College of Wisconsin, Milwaukee, Wisconsin, USA
\textsuperscript{3}University of Pennsylvania, Philadelphia, Pennsylvania, USA

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**Corresponding Author:**

Abhilash Koratala, M.D.
Medical College of Wisconsin
8701 W Watertown Plank Rd,
Division of Nephrology, Room A 7633
Wauwatosa, WI, USA 53226
Email: akoratala@mcw.edu
Abstract:

The management of complex fluid and electrolyte disorders is central to the practice of nephrologists. The sensitivity of physical examination alone to determine fluid status is limited, precluding accurate clinical decision making. Point-of-Care Ultrasonography (POCUS) is emerging as a valuable non-invasive bedside diagnostic tool for objective evaluation of physiological and hemodynamic parameters related to fluid status, tolerance, and responsiveness. Rapid bedside sonographic evaluation can obtain qualitative data on cardiac function as well as quantitative data on pulmonary congestion. Advanced POCUS, including goal-directed Doppler echocardiography provides additional quantitative information including flow velocities and pressures across the cardiac structures. Recently, abnormal Doppler flow patterns in abdominal organs secondary to increased right atrial pressure have been linked to congestive organ damage adding another component to the hemodynamic assessment. Integrating POCUS findings with clinical and laboratory data can further elucidate a patient’s hemodynamic status. This drives decisions regarding crystalloid administration or, conversely, diuresis or ultrafiltration and allows tailored therapy for individual patients. In this article, we provide an overview of the focused assessment of cardiovascular function, pulmonary and venous congestion using POCUS and review relevant literature.
Case:

A 63-year-old man with medical history of hypertension, obesity, and heart failure with reduced ejection fraction presents to the nephrology clinic for assessment of an elevated serum creatinine. His baseline serum creatinine was ~0.6-0.8 mg/dL. He complains of abdominal distension, loose stools and dyspnea on exertion. Medications include carvedilol, lisinopril, isosorbide mononitrate, and hydralazine. Diuretics were held by the referring physician due to rise in creatinine. Physical examination demonstrates blood pressure of 92/59 mmHg with a heart rate of 65 bpm. Cardiopulmonary exam reveals pedal edema, but no obvious jugular venous distention, rales, or S3. Urine sediment is bland. Review of chest roentgenogram obtained in the primary care setting shows no evidence of pulmonary edema or pleural effusions. Laboratory studies show stable serum creatinine 1.4 mg/dL after discontinuation of diuretics. What is the next step?

Introduction:

Assessment of fluid and hemodynamic status is a critical skill for nephrologists, central to almost every consult from hypertension and electrolyte disorders to management of AKI and ESKD. Fluid status assessment has a storied tradition with physical exam signs of jugular venous distention, S3, rales, and peripheral edema learned and reproduced by generations of physicians. These signs are helpful in extreme cases but are insensitive for the detection of volume overload.¹ Radiographic signs of volume overload such as pleural effusions, and Kerley B lines aid in fluid status assessment, but themselves lack sensitivity.² Natriuretic peptides and pulmonary artery catheters also have limitations.³,⁴ In 30 years, point-of-care ultrasonography (POCUS) has
expanded from a niche subspecialty skill to a cornerstone of bedside diagnosis.\textsuperscript{5,6} Ultrasound allows us to directly visualize the body in a way that was previously inaccessible. POCUS involves answering focused clinical questions using bedside ultrasonography and increases the sensitivity of conventional physical examination\textsuperscript{7-12} As we move forward, ultrasonographic indicators of fluid status are being developed and validated. Some are intuitive to our understanding of physiology and others are novel markers of previously unknown significance [Figure 1]. Having said that, POCUS findings should be interpreted in conjunction with other clinical parameters such as vital signs, body weight, mucous membrane examination, capillary refill time, axillary moisture etc., and not viewed as an alternative to physical examination or standard imaging studies. We describe the most well-validated indices of fluid status: focused sonographic assessment of the heart, abdominal veins, and lungs (the pump-pipes-leaks approach)\textsuperscript{13} to gain insight into systemic hemodynamics and guide fluid management decisions.

**Lung ultrasound**

Extravascular lung water (EVLW) or the fluid content in the lung interstitium is an important indicator of fluid status, often guiding management decisions in clinical practice. It essentially depends on LV filling pressures and permeability of the pulmonary vasculature. In the recent past, lung ultrasound (LUS) has emerged as a valuable bedside tool to detect pulmonary congestion even before it is clinically apparent.\textsuperscript{14} Moreover, LUS is technically least challenging of all the sonographic applications described in this review. There are data suggesting that nephrologists can be effectively trained to measure EVLW using LUS by an entirely internet-based program.\textsuperscript{15}
Interpretation

LUS primarily involves interpretation of the artifacts rather than visualization of the pulmonary parenchyma because air is strongly reflective to the ultrasound beam. In normal aerated lung, the only detectable structure is the pleura, which appears as a shimmering hyperechoic (bright) horizontal line in between the rib shadows. The shimmer or synchronous horizontal movement with respiration denotes pleural sliding. A-lines are equidistant hyperechoic horizontal lines seen on a normal LUS [Figure 2A]. These are reverberation artifacts formed due to multiple reflections of the ultrasound beam between the transducer and the pleura with underlying air-filled lung. When the air content in the lung decreases due to transudate or exudate in the interstitium, vertical hyperechoic artifacts are seen, termed the B-lines [Figure 2B]. These are ultrasound equivalent of the Kerley B lines seen on a chest radiograph and provide a semi-quantitative estimate of the amount of EVLW. B-lines arise from the pleural line, extend to the end of the image without fading, and move synchronously with lung sliding. A positive ‘B-line region or zone’ is defined as the presence of three or more B-lines in a longitudinal plane between two ribs. Two or more positive regions bilaterally constitutes ‘interstitial syndrome’ and indicates diffuse pulmonary edema. B-lines can also be seen in conditions other than cardiogenic pulmonary edema such as focal pneumonia, acute respiratory distress syndrome, pulmonary fibrosis, and contusion and hence should be interpreted in the appropriate clinical context. These conditions are typically associated with thickened/irregular pleural line and non-homogeneous distribution of B-lines. When the air content in the lung further decreases, such as in alveolar consolidation, lung parenchyma can be visualized on ultrasound similar to that of liver
and spleen [Figure 2C]. In contrast, pleural effusion appears as an anechoic (black) space above
the diaphragm, typically surrounding the atelectatic or consolidated lung [Figure 2D].

Most research studies, particularly in patients with end-stage kidney disease (ESKD) used a
twenty-eight-zone scanning technique to obtain the total B-line count, which involves scanning
of the anterior and lateral chest from the second to fourth (on the right side to the fifth)
intercostal spaces and from the parasternal to midaxillary lines on each side [Figure 3A].
However, in routine clinical practice, an eight-zone scanning technique is frequently employed
examining two anterior and two lateral areas on each hemithorax [Figure 3B]. Recently, Torino,
et al. showed that eight-zone technique correlates well with the classical twenty-eight zone
score and also retains its prognostic significance (discussed below).

**Diagnostic performance**

The diagnostic performance of LUS to detect pulmonary congestion is far superior compared to
auscultation. For example, in a study including 79 hemodialysis patients deemed to be at higher
cardiovascular risk, only about half of those with severe congestion on LUS (defined as >30 B-
lines on a 28-zone scan) had crackles on lung auscultation. Likewise, in patients with moderate
congestion on LUS (15 to <30 B-lines), the prevalence of crackles was only 31%. In

In patients with acute decompensated heart failure, LUS has shown to be more sensitive for
detection of pulmonary edema than chest radiography, which is the typical first-line imaging. In
addition, LUS has demonstrated substantial correlation with cardiac catheterization-derived LV
end-diastolic pressure, making it a valuable adjunct to echocardiography and clinical variables in the management of heart failure patients.\textsuperscript{19}

\textit{Prognostic significance}

LUS-detected pulmonary congestion is associated with adverse outcomes even in asymptomatic patients. For instance, in a multi-center observational study including 392 ESKD patients on hemodialysis, those with very severe congestion (> 60 B-lines on a 28-zone scan) had a 4.2-fold risk of death (hazard ratio [HR] 4.20; 95\% confidence interval, 2.45-7.23) and a 3.2-fold risk of cardiac events (HR 3.20; 95\% CI,1.75-5.88) after adjusting for heart failure class and other risk factors compared with those having mild or no congestion (<15 B-lines).\textsuperscript{20}

Analogously, in the context of heart failure, residual lung congestion at hospital discharge as well as in the outpatient clinic has shown to be a strong predictor of outcome.\textsuperscript{21-23}

\textit{Role in guiding the therapy}

As LUS provides dynamic information, the change in B-line count can be used to monitor the effectiveness of decongestive/ultrafiltration therapy and titrate it accordingly.\textsuperscript{24-27} In a recent randomized controlled trial including 71 “clinically euvolemic” ESKD patients those who underwent pre-hemodialysis LUS-guided dry weight reduction achieved a greater reduction in weight compared to those receiving standard care, correlating with the change in number of B-lines (–5.3 in active group vs. +2.2 in controls). In addition, the magnitude of 48-hour ambulatory blood pressure reduction was significantly greater in the active group at 8 weeks (systolic –6.61 vs. –0.67 mm Hg and diastolic –3.85 vs. –0.55, p = 0.033).\textsuperscript{28}
Similarly, in patients with acute exacerbation of HF, B-line count has shown to consistently decrease with diuretic therapy soon after presentation, thereby guiding further management.\textsuperscript{29} In ambulatory patients with chronic HF, an 8-zone LUS-guided diuretic therapy was associated with a significant reduction in HF-related urgent care visits compared to standard care.\textsuperscript{30} Clinical trials are in progress that may provide further insights into long-term outcomes of LUS-guided therapy.\textsuperscript{31}

**Focused Cardiac Assessment (FOCUS):**

Hemodynamic assessment is predicated on basic knowledge of cardiac function and the presence of pathologies. In contrast to referral echocardiography, FOCUS is a limited study aimed at answering specific questions such as evaluation of the left and right ventricular function and presence or absence of pericardial effusions. Most commonly, FOCUS consists of a series of five 2-dimensional echocardiographic clips without spectral Doppler obtaining images of the heart in orthogonal planes to provide the relevant physiologic information detailed in Figure 4.\textsuperscript{32,33} Further clues to fluid status can be gleaned from additional studies using Doppler ultrasonography. Table 1 Summarizes the image acquisition of basic echocardiographic views, common structures visualized, sonographic parameters, and pathologies assessed in each view. A brief note on common pathologies is provided below.

**Pericardial effusion**

Pericardial effusion is an important cause of hypotension and hemodynamic compromise, which can be quickly identified on FOCUS. It appears as an anechoic (black) space between the two
pericardial layers. Parasternal long axis and subcostal views allow better visualization of the effusion, though it is generally identifiable on all the standard FOCUS views [Figure 5A]. In terms of severity, separation between the pericardial layers in diastole of less than 1 cm is considered mild, whereas 1-2 cm is considered moderate and more than 2 cm severe effusion.34

**Left ventricular systolic dysfunction**

Qualitative estimation or ‘eyeballing’ of the left ventricular (LV) ejection fraction is another key component of FOCUS, which involves observing wall thickening and motion during cardiac cycle. In general, the LV walls should approximate by one-fourth or more in parasternal views. In patients with depressed LV function, both wall thickening and inward motion are decreased. Conversely, a hyperdynamic ventricle, that is where ventricular walls/papillary muscles in the parasternal short axis view almost touch at end-systole is indicative of volume depletion in the appropriate clinical context.

**Relative chamber size**

A normal right ventricle (RV) cavity diameter is less than two-thirds of the LV and can quickly dilate with pressure or volume overload. Apical 4-chamber and parasternal short axis are good views to assess this. With volume overload, the RV becomes dilated, and the interventricular septum is flattened in diastole giving the appearance of ‘D’ to the LV in parasternal short axis view. This is called the ‘D-sign’ [Figure 5B]. While such patients are hypotensive, empirically administering intravenous fluids causes further compromise of the LV cavity and reduced cardiac output.
**Right atrial pressure**

Inferior vena cava (IVC) ultrasound is used to estimate right atrial pressure (RAP) and get an idea of the resistance to venous return. In spontaneously breathing patients, IVC collapses during inspiration due to negative intrathoracic pressure. An IVC diameter ≤ 2.1 cm and collapsibility >50% with a sniff indicates normal RA pressure of 3 mm Hg (0-5 mm Hg); IVC diameter > 2.1 cm with < 50% inspiratory collapse indicates high RA pressure of 15 mm Hg (10-20 mm Hg); scenarios in between correspond to an intermediate value of 8 mm Hg (5-10 mm Hg).\(^{35}\)  

**Figures 5C and 5D** demonstrate sonographic images of large and small IVC obtained from a patient with heart failure and volume depletion, respectively. On the other hand, these cut-offs cannot be applied in mechanically ventilated patients as the IVC is dilated at baseline due to positive pressure ventilation and may not collapse at all during respiration. While novice POCUS users are enthusiastic about IVC ultrasound as it is relatively easy to learn, interpreting it in isolation is subject to numerous pitfalls. For example, small collapsible IVC is seen in normal state of health and equating it with volume depletion without considering the clinical context leads to unnecessary fluid administration. Moreover, the magnitude of the respiratory effort significantly affects collapsibility (for example, a frail elderly woman versus a muscular young man), altering the interpretation. Furthermore, technical factors such as obesity, surgical dressings, increased intra-abdominal pressure, mistaking aorta, or dilated bowel for IVC etc. can result in errors. Depending on how the ultrasound beam is aimed, it may not depict the true diameter of the vessel. Notably, studies demonstrate moderate to poor inter-rater agreement between IVC measurements.\(^{36,37}\) While IVC is a good indicator of central venous pressure, it is not reliable to
assess fluid responsiveness. Therefore, IVC ultrasound should be interpreted in conjunction with other POCUS findings and overall clinical picture.

Users with additional training in Doppler echocardiography assess additional parameters such as cardiac output and its response to fluid administration, LV filling pressures and right ventricular filling pressures at the bedside.

**Venous Doppler:**

Organ dysfunction in heart failure is closely related to venous congestion. Central venous pressure (CVP) is the strongest hemodynamic determinant for the development of worsening renal function in patients with decompensated HF. Normal and abnormal patterns of flow in abdominal and central veins result from retrograde transmission of central venous pressure. POCUS can enhance the clinical evaluation of venous congestion using venous Doppler in addition to IVC ultrasound. Figure 6 illustrates the technique of obtaining the sonographic images of hepatic, portal and intra-renal veins.

**Hepatic Vein Doppler**

Blood flow in the hepatic veins (HV) is pulsatile and changes in its velocity reflect changes in RA pressure. Normal HV flow pattern consists of two antegrade waves (a larger “S” and a smaller “D” wave corresponding to CVP “X” and “Y” descent respectively) and one or two retrograde waves (a larger “A” wave and smaller “V” wave corresponding to CVP “A” and “V” waves respectively). Frequently, “V” wave is not seen. Understanding the origin of hepatic flow waves aids in understanding common pathological alterations. For example, severe
pulmonary hypertension can manifest as prominent “A” waves and or/ decreased “D” wave amplitude because of increase in RV end-diastolic pressure. Systolic dysfunction of the RV and tricuspid regurgitation (TR) both alter the RA pressure during ventricular systole leading to progressive decrease in the peak velocity of the “S” wave. In addition, severe TR can cause S wave reversal. [Figure 7B] Thus, hepatic vein Doppler provides relevant information about the filling pattern of the RA.

**Portal Vein Doppler**

As opposed to hepatic vein flow, the splanchnic circulation is an isolated vascular unit protected from the systemic circulation by the resistance of post-sinusoidal sphincters. Thus, normal portal flow is continuous or only mildly pulsatile [Figure 7A]. However, pathological increases in RA pressure can be transmitted through liver sinusoids into the portal vein. [Figure 7B] Portal vein pulsatility was originally described in patients with severe TR but has now been described in multiple conditions associated with increased RA pressure. Increased pulsatility in portal venous flow has been associated with a higher NT-pro-BNP, higher systolic pulmonary artery pressures, positive fluid balance, and RV dysfunction. Portal Vein flow alterations can be quantified by the pulsatility fraction (100 [(Vmax-Vmin)/Vmax]); a pulsatility fraction ≥ 30% is considered mild while ≥ 50% is considered severely elevated.

There are a few studies evaluating the relationship between portal vein pulsatility and patient outcomes in the setting of heart failure. In a landmark study by Beaubien-Souiligny, et al., a portal vein pulsatility fraction of > 50% and severe alterations in intra-renal venous flow were associated with an increased risk of AKI in cardiac surgery patients. The inclusion of portal vein
Doppler significantly improved AKI risk prediction. Furthermore, alterations in portal vein flow have been associated with the development of congestive hepatopathy, encephalopathy, major complications in cardiac surgery patients, and may be a useful prognostic marker in patients hospitalized for acute heart failure. Together, these data suggest that sonographic evaluation of portal vein pulsatility could become a useful tool for the diagnosis and management of venous congestion. In our experience, increased portal vein pulsatility fraction associated with volume overload often improves with diuretic treatment.

Even though a plethoric, non-collapsible IVC indicates venous congestion, caution must be exercised when interpreting it in patients with cardiac conditions impeding venous return (Chronic RV dysfunction/tricuspid regurgitation, RV myocardial infarction, Cardiac tamponade); These patients may be fluid responsive despite IVC plethora. The evaluation of hemodynamic AKI in these conditions can be enhanced by assessing portal vein flow; increased portal pulsatility is suggestive of congestive AKI, which can potentially improve with decongestive therapy. A case of portal vein flow normalization with diuresis even in the presence of persistent severe TR is presented in Figure 7C.

Portal venous flow cannot be relied upon in patients with cirrhosis as both absent pulsatility in the presence of severe congestion and increased pulsatility unrelated to RA pressure can occur. Occasionally, portal vein pulsatility can be seen in thin healthy individuals. Given these limitations, portal vein PF should not be interpreted in isolation. A recent study evaluating IVC size, hepatic, portal, and intra-renal vein Doppler flow patterns found increased specificity
using the combination of multiple POCUS markers to identify clinically significant venous congestion.

**Intra-Renal Venous Doppler**

Similar to the portal vein, flow pattern in intrarenal (arcuate and interlobar) veins depends on surrounding renal parenchymal histology as much as RA function. Iida et al. used Doppler imaging to evaluate intra-renal venous flow (IRVF) patterns in patients with heart failure. IRVF waveforms were divided into three flow patterns: continuous, biphasic, and monophasic. IRVF profile was altered by increases in RAP but was not associated with changes in cardiac index. The monophasic pattern was associated with significant TR. IRVF strongly correlated with clinical outcomes including death from cardiovascular disease or unplanned hospitalization for heart failure. This correlation was independent of RAP. Similar results were obtained by Puzzovivo and colleagues.

Supporting the role of IRVF alterations as a marker of venous congestion, experimental fluid expansion worsened IRVF pattern and correlated with less diuretic efficiency in patients with HFpEF. A recent study in patients with pulmonary hypertension also showed adverse outcomes associated with IRVF alterations. Three patients from this study who developed severe AKI with diuretic-resistant fluid overload and required renal replacement therapy exhibited a monophasic IRVF pattern.

While hepatic vein Doppler mainly reflects the RA filling pattern, portal and intra-renal venous Doppler provide additional information about RA filling pressure and its correlation with
congestive organ injury.\textsuperscript{43} Whether interventions aimed at addressing abnormal organ flow patterns can improve relevant outcomes in patients with venous congestion remains unknown. Moreover, the cause-effect relationship between sonographic markers of venous congestion and AKI remains elusive at this time and must be evaluated by larger studies. POCUS alterations that occur in venous congestion are summarized in Figure 8.

**Going back to the case:**

FOCUS revealed severely decreased LV EF and dilated RV with significantly reduced function. Numerous bilateral B-Lines were present on LUS. IVC was 2.4 cm in diameter with no respiratory variation and there was no significant ascites [Figure 9A]. These findings were compatible with HFrEF with severe RV failure, as well as moderate pulmonary congestion. To evaluate for the presence of systemic venous congestion, a bedside Doppler ultrasonography was performed. Hepatic vein Doppler demonstrated decreased diastolic (D) wave amplitude (seen in severe pulmonary hypertension and abnormal RV relaxation), portal vein pulsatility fraction of 100%, and a monophasic IRVF [Figure 9B]. Although a plethoric IVC was suggestive of venous congestion, the presence of severe RV dysfunction and significant pulmonary hypertension makes it less reliable (can be chronically dilated) and patient might still be fluid responsive. However, both portal and intra-renal venous flow patterns indicated that backward transmission of RAP was significant enough to lead to abdominal organ congestion suggesting congestive kidney injury.

**Treatment and outcome**
Given these findings, diuretic therapy was restarted with dose intensification. On follow up, the patient showed steady decrease in weight (~10 kg) and noticeable improvement in symptoms. While serum creatinine worsened from 1.4 to 1.8 mg/dL initially, it improved and stabilized at 0.6 mg/dL with continued diuresis. Follow up POCUS evaluation of venous congestion showed markedly improved flow patterns on both portal vein (PF = 32%) and IRVF (Biphasic pattern) \[Figure 9C].

**POCUS education and program development:**

Despite stated advantages of POCUS and growing interest among nephrologists to acquire this skill, training opportunities remain sparse at this time. Workshops and short courses organized by professional societies such as the National Kidney Foundation and the American Society of Nephrology provide an introduction to the technique and interpretation but continued practice, preferably under supervision is vital to achieve mastery. Longitudinal curricula are shown to have a favorable impact on long-term skill retention\(^{75,76}\); as such, integrating POCUS training into the 2-year nephrology fellowship offers a conceivable advantage. Nevertheless, only a few nephrology programs currently train their fellows beyond kidney ultrasound\(^77\), the key problem being lack of trained faculty. We suggest that nephrology divisions identify faculty interested in learning/teaching POCUS and support them to pursue a structured multi-component certification program such as the one offered by the American College of Chest Physicians\(^{78}\). Thereafter, multi-specialty collaboration at the institutional level with specialties such as emergency medicine and radiology facilitates organizing the curriculum, training more faculty as well as quality assurance. In addition, taking advantage of the online POCUS educational tools.
built for nephrologists such as NephroPOCUS.com minimizes the need to create new didactic material by individual fellowship programs. Furthermore, forming an expert panel and putting forward consensus guidelines by Nephrology professional organizations would help standardize POCUS training.

**Conclusion:**

Physical examination is limited in determination of fluid status. Augmented examination using POCUS is gaining popularity as a non-invasive bedside tool that provides a detailed insight into cardiovascular physiology and hemodynamic determinants of fluid status. Evaluation of cardiac function by FOCUS, pulmonary congestion by LUS, and systemic venous congestion by abdominal venous Doppler can guide management by providing information on fluid tolerance and responsiveness. Thorough multi-organ assessment should be undertaken to offset the limitations of individual POCUS applications. Integrating POCUS findings with clinical and laboratory data allows decisions regarding crystalloid administration or diuresis to be tailored according to the individual physiology with a risk-benefit analysis of the preferred therapeutic strategy. Further studies are needed to determine whether management guided by abnormalities detected on POCUS translates to improved clinical outcomes. On a note of caution, POCUS is not a replacement to clinical judgement and is susceptible to limitations like any other technology. Improper technique, wrong interpretation and over-reliance on an isolated finding can potentially lead to mismanagement of the patient. Therefore, the nephrology community should focus on developing POCUS training standards and a robust certification process to ensure quality.
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A Koratala: Conceptualization; Supervision; Writing - original draft; Writing - review and editing
N Reisinger: Conceptualization; Writing - original draft; Writing - review and editing
All the authors participated in the design, drafting, and proofreading the manuscript. All authors approved the final submission.
References:


Table 1: Summary of the basic echocardiographic views: image acquisition, common structures, sonographic parameters, and pathologies assessed in each view.

<table>
<thead>
<tr>
<th>FOCUS view</th>
<th>Image acquisition</th>
<th>Structures</th>
<th>Key assessments</th>
<th>Common pathologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSLA</td>
<td>Parasternal 3rd-5th intercostal space, indicator to patient’s R shoulder</td>
<td>RV, LV, LA, LVOT, AV, MV, descending aorta</td>
<td>Visual assessment of LV EF</td>
<td>LV dysfunction, pericardial effusion, left-sided pleural effusion posterior to descending aorta</td>
</tr>
<tr>
<td>PSSA</td>
<td>2nd-4th intercostal space, indicator to L shoulder</td>
<td>RV, LV, interventricular septum</td>
<td>Visual assessment of LV EF and LV shape</td>
<td>LV dysfunction, septal flattening, or shift indicative of RV dysfunction, Pericardial effusion</td>
</tr>
<tr>
<td>Apical</td>
<td>Point of maximum impulse, indicator to L axilla</td>
<td>RA, LA, RV, LV, LVOT, MV, TV</td>
<td>RV/LV comparison, Color Doppler of tricuspid and mitral valves</td>
<td>RV failure, pericardial effusion, valvular regurgitation</td>
</tr>
<tr>
<td>SX</td>
<td>Below the xiphoid process, indicator to patient’s L</td>
<td>RA, LA, RV, LV</td>
<td>Pericardium</td>
<td>Pericardial effusion, LV dysfunction</td>
</tr>
</tbody>
</table>
PSLA parasternal long axis view; PSSA parasternal short axis view; SX subxiphoid view; RV right ventricle; LV left ventricle; LA left atrium; RA right atrium; LVOT left-ventricular outflow tract; AV aortic valve; MV mitral valve; TV tricuspid valve; EF ejection fraction; R = right; L = left

*Note: List of assessments/pathologies is not exhaustive, and each pathology can be seen in multiple views*

**Figure legends:**

**Figure 1:** Key components of the comprehensive sonographic evaluation of the fluid volume status using the B-mode and Doppler ultrasound.

**Figure 2:** Sonographic images demonstrating (A) Normal lung with A-lines (arrows); (B) Vertical B-lines (arrows) indicating interstitial edema. Arrowhead points to the pleural line; (C) Consolidated lung (arrow); (D) Pleural effusion (asterisk) above the diaphragm (arrow).

**Figure 3:** (A) 28 and (B) 8-zone Lung ultrasound scanning techniques. Lower panel demonstrates the transducer position, which should be perpendicular to the chest wall following its curvature to obtain optimal images. Human body photograph obtained from Shutterstock®; photo credit: Albina Glisic. Illustration made using Biorender®

**Figure 4:** Basic Echocardiographic Views. A. Parasternal Long Axis B. Parasternal Short Axis C. Apical 4-Chamber D. Subxiphoid E. Inferior Vena Cava. Green arrows indicate the direction of the transducer orientation marker. Abbreviations: LV, Left Ventricle; RV, Right Ventricle; LA, Left Atrium; RA, Right Atrium; IVC, Inferior Vena Cava. Human body illustration obtained from Shutterstock®; photo credit: Albina Glisic
Figure 5: (A) Pericardial effusion (asterisk) surrounding the heart seen from the subcostal window. (B) D-sign: interventricular septal flattening seen from the parasternal short axis view. (C) Plethoric IVC (D) Small IVC. Abbreviations: LV, Left Ventricle; IVC, inferior vena cava

Figure 6: (A) IVC and hepatic veins (arrowheads) in long and transverse axes visualized from the subxiphoid scan position. (B) Upper: Hepatic veins (arrowheads) and portal vein from the lateral scan plane obtained by placing the transducer along the anterior axillary line with orientation marker supero-anteriorly. Lower: Color Doppler image of the kidney obtained by placing the transducer along the mid-axillary line with orientation marker supero-posteriorly. Abbreviations: IVC, inferior vena cava. Illustrations made using Biorender®

Figure 7: (A) CVP Waveform, HV and PV Doppler in a healthy person and (B) in a patient with severe biventricular failure, TR and venous congestion. HV flow mirrors changes in CVP waveform. Transmission of RAP to PV is only seen in the presence of severe venous congestion. (C) HV and PV Doppler in a patient with PAH and severe TR before and after diuretic therapy. Note improvement on PV pulsatility fraction while HV alterations from severe TR remained unchanged. (D) IRV flow alterations in venous congestion. Waveform above the baseline corresponds to intra-renal arterial flow while the waveform below the baseline depicts intra-renal venous flow. Abbreviations: CVP, Central Venous Pressure; HV, Hepatic Vein; PV, Portal Vein, TR, Tricuspid Regurgitation; PAH = pulmonary arterial hypertension; IRV Intra-renal Venous.

Figure 8: Summary of POCUS alterations that occur in venous congestion. IVC (inferior vena cava) size increases with increase in right atrial pressure and reaches its maximum compliance after a certain point. Hepatic vein waveform initially shows both systolic and diastolic components and
as the congestion worsens, the systolic component reverses leaving only the diastolic wave below the baseline. By convention, above the baseline represents flow towards the transducer and below the baseline represents flow away from the transducer (towards the heart in this case). Normal portal vein waveform is continuous and above the baseline. As the severity of congestion increases, it becomes pulsatile and flow reversal (below the baseline) is seen in systole with further elevations in right atrial pressure. Normal intra-renal vein Doppler is similar to that of portal vein except that it is below the baseline (flow away from the transducer). As the congestion worsens, it becomes increasingly pulsatile leaving only diastolic component below the baseline in severe cases. Above the baseline tracing represents intra-renal arterial flow, which is helpful to identify systolic and diastolic phases.

**Figure 9**: Case POCUS findings. A) IVC, LUS, PSLA, and A4ch views. B: B-Lines, RV: Right Ventricle. Ao: Ascending Aorta. LA: Left Atrium. LV: Left Ventricle. B) Venous Doppler obtained before restarting diuretics. C) IVC and Venous Doppler on follow-up after restarting diuretics. Abbreviations: IVC, Inferior Vena Cava; LUS, Lung Ultrasound; PSLA, Parasternal Long Axis; A4ch, Apical 4 chamber view; HV, Hepatic Vein; PV, Portal Vein; IRV, Intra-renal Vein.
POCUS-assisted hemodynamic assessment

- Lung ultrasound
- Focused cardiac ultrasound
- Inferior vena cava ultrasound
- Hepatic vein Doppler
- Intrarenal venous Doppler
- Portal vein Doppler

Figure 1
Figure 7

A CVP

B Severe Tricuspid Regurgitation

C Before Diuresis

D Not Congestive

Moderate Congestion

Severe Congestion

Hepatic Vein (HV)

Portal Vein (PV)

Intra-renal vein

Continuous

Biphasic

Monophasic
Figure 8

Not Congestive → Severe Congestion

IVC

Hepatic Vein

Portal Vein

Intra-Renal Vein