Journal Pre-proof

Demystifying Volume Status: An Ultrasound-Guided Physiologic Framework

Juliana YL. Kan, MMed, Shane Arishenkoff, FRCPC, Katie Wiskar, FRCPC

PII: S0012-3692(24)05735-0

DOI: https://doi.org/10.1016/j.chest.2024.12.026

Reference: CHEST 6669

To appear in: *CHEST*

Received Date: 3 September 2024

Revised Date: 9 December 2024

Accepted Date: 19 December 2024

Please cite this article as: Kan JY, Arishenkoff S, Wiskar K, Demystifying Volume Status: An Ultrasound-Guided Physiologic Framework, *CHEST* (2025), doi: https://doi.org/10.1016/j.chest.2024.12.026.

Text: 3447
Abstract: 245

Title Page

Title of Paper: Demystifying Volume Status: An Ultrasound-Guided Physiologic Framework

Authors and Affiliations: Juliana Kan YL[1] (MMed), Shane Arishenkoff[2] (FRCPC), Katie Wiskar[2] (FRCPC).

[1]Department of Internal Medicine, Singapore General Hospital.

[2]Department of Medicine, University of British Columbia.

Corresponding author information:

Name: Katie Wiskar

Mailing address: 2775 Laurel St, 7th floor, Stn 3; Vancouver, BC, Canada. V5Z 1M9

Email: katiewiskar@gmail.com

Conflict of interest statement: All authors declare that they have no conflicts of interest.

Acknowledgement: All authors contributed substantially to the writing and review of the manuscript. SA conceptualized the idea of the manuscript. KW organized the sections, provided a factual review, and revised the paper substantially. JK performed the literature review, created the tables, and curated the ultrasound images.

Demystifying Volume Status: an Ultrasound-Guided Physiologic Framework

Abstract

Topic Importance:

Accurate assessment of a patient's volume status is crucial in many conditions, informing decisions on fluid prescribing, vasoactive agents, and decongestive therapies. Determining a patient's volume status is challenging, due to limitations in examination and investigations and the complexities of fluid homeostasis in disease states. Point-of-care ultrasound (POCUS) is useful in assessing hemodynamic parameters related to volume status, fluid responsiveness, and fluid tolerance. It requires understanding several physiologic concepts to accurately interpret and integrate POCUS findings into volume-related clinical decision-making.

Review findings:

The following concepts serve as a scaffold for a comprehensive volume status assessment: central venous pressure, right heart function, left heart assessment, extravascular volume, and venous congestion. POCUS allows us access to these hemodynamic and structural data points as an extension and refinement of the physical exam. Often, multiple POCUS applications are employed, and findings must be integrated with the rest of the clinical evaluation. We illustrate this using three common scenarios: hypotension, hypoxia, and acute kidney injury. Clinicians must be aware of the strengths and weaknesses of findings in different physiologic states, and potential pitfalls of image acquisition and interpretation. Further studies are necessary to determine the benefits and clinical outcomes of a POCUS-directed volume status assessment.

Summary:

Volume status assessment is ubiquitous, yet challenging to perform. This review summarizes foundational physiologic concepts relevant to volume status evaluation, and highlights how multiorgan POCUS informs on hemodynamic parameters that can be combined with the conventional clinical assessment to make fluid-related decisions.

Key words list:

Fluid status

Hemodynamics

Physiology

Point of Care Ultrasound

Volume status

Abbreviations list:

AKI: Acute kidney injury

CO: Cardiac output

CVP: Central venous pressure

HR: Heart rate

IVC: Inferior vena cava

JVP: Jugular venous pressure

LAP: Left atrial pressure

LV: Left ventricle

MAP: Mean arterial pressure

POCUS: Point-of-care ultrasound

RV: Right ventricle

SV: Stroke volume

SVR: Systemic vascular resistance

TR: Tricuspid regurgitation

VEXUS: Venous excess ultrasound

Accurate assessment of a patient's volume status is crucial in multiple clinical scenarios such as heart failure, kidney disease, cirrhosis, and sepsis.[1–4] With the end goal of optimizing organ perfusion, decisions on administration of fluids, vasoactive agents, and decongestive therapy rely heavily on an accurate volume status assessment.[5] Although the notion of *volume status* is a familiar concept among physicians, the term remains nebulous. Classification using the terms hypervolemia, euvolemia, and hypovolemia lack the precision necessary to adequately describe the complex relationship between fluid compartments and hemodynamic factors in the cardiovascular circuit. Volume status is contextual and dynamic, rather than a uniform state with a fixed definition.

Unfortunately, a patient's volume status is notoriously challenging to assess at the bedside, and there is no standardized list of findings that defines a volume status assessment.[6,7] Techniques such as assessing the jugular venous pressure (JVP), lower extremity edema, and the hepatojugular reflex can be challenging, are limited by suboptimal test characteristics, and are not direct measures of volume: rather, these are hemodynamic factors that can be altered independent of volume.[5,8–10] Available laboratory investigations commonly associated with volume overload such as serum brain natriuretic peptide levels have limited specificity.[11] Furthermore, much of the cardiovascular circuit is not accessible to examination, forcing clinicians to make inferences about volume based on an incomplete data set.

Adding to the difficulty of a volume status assessment is the complexity of the cardiovascular circuit itself. This does not behave anatomically or functionally as a simple uniform compartment; with intravascular blood volume unequally distributed among the arterial and venous systems and significant volumes stored in venous reservoirs.[12] Regional differences in compliance, resistance, and capacitance render it near impossible to draw generalized

conclusions based on findings in a single compartment. Increasing attention is also being paid to the potential harms associated with venous congestion and the need to balance fluid *responsiveness* (forward flow) with fluid *tolerance* (back pressures from the venous system). Furthermore, hemodynamic congestion may or may not be associated with systemic fluid overload, once again underscoring the disctinction between pressure and volume. Overall, accurate assessment of volume status is fraught with difficulties, and misinterpretations may lead to errors in management.[13,14]

Point-of-care ultrasound (POCUS) is a bedside ultrasound examination performed as an extension of the physical exam to help guide clinical management, and can be useful in assessing volume status.[8,15] POCUS allows for evaluation of physiologic and hemodynamic parameters related to volume status, fluid responsiveness, and fluid tolerance.[16] Used in combination with other clinical parameters, ultrasound examination of the heart, lungs, and vasculature can help answer complex volume-related questions and is particularly helpful in challenging physiologic situations such as cirrhosis, kidney disease, and cardiorenal syndrome.[5,16–19]

However, like any tool in medicine, POCUS has pitfalls and caveats. POCUS-derived data points are inextricably linked to the individual patient's physiology; and failure to consider the impact of important anatomical and hemodynamic-related factors leads to misinterpretation. An understanding of several fundamental physiologic concepts is imperative for physicians who are integrating POCUS findings in volume-related clinical decision-making: (1) central venous pressure, (2) right heart function, (3) left heart assessment, (4) extravascular volume, and (5) venous congestion. This narrative review aims to clarify these concepts and their role in performing comprehensive ultrasound-assisted volume status assessments, including a discussion of the strengths and weaknessnes of POCUS findings in different physiologic states. This paper presumes a basic understanding of POCUS applications.

Discussion of image acquisition techniques and their challenges are beyond the scope of this review and are well described elsewhere.[20]

Literature Review

This review draws on seminal works known to the authors related to ultrasound assessment of volume status and related physiologic concepts; citation searching was applied to draw in updated studies. In addition, we performed a targeted literature search of PubMed and Medline using keywords such as POCUS, ultrasound, volume status, central venous pressure, hemodynamics, and echocardiography. Papers considered by the authors to be most relevant to the concepts in this paper were included in the review.

Evidence Review

1) Central venous pressure

POCUS findings (Table 1, section 1a-1b)

Central venous pressure (CVP) can be inferred from the

1) Internal jugular vein (IJV)[21–24]
2) Inferior vena cava (IVC)[25–27]

CVP is defined as the mean vena caval pressure or right atrial pressure; which, in the absence of tricuspid stenosis, is equal to the right ventricular end-diastolic pressure. Though widely discussed, several key aspects of the CVP are often poorly understood and bear clarifying.

First, although volume *affects* pressure, one must not conflate the two. An increase in CVP may be secondary to either a primary *pressure-related* disorder and/or a *volume-related* disorder. Elevated CVP may be an indication of a number of non-volume related

pathologies, as listed in Table 2.[28] Although CVP is not itself a measure of volume, it provides information about the right heart's compliance and performance, which reflects its ability to accommodate volume.

Secondly, an elevated CVP is a precursor to the development of clinically significant venous congestion. Venous return to the right heart is governed by the pressure gradient between the right atrium and peripheral veins, and by venous resistance.[12,29] This pressure gradient is small, with peripheral venous pressure usually 8-10mmHg, and normal CVP ranging from 0-8mmHg.[30,31] Therefore, as CVP increases, there is a requisite rise in peripheral venous pressures in order to maintain right heart filling.

Third, CVP is impacted by the peripheral venous system. The venous system is a low pressure, high capacitance system containing approximately 70% of intravascular circulating blood volume. Venous volume can be physiologically divided into *stressed* and *unstressed* volume. Under basal conditions, only about 30% of the intravascular blood volume is *stressed volume*; that is to say, it produces tension in the venous walls contributing to pressure in the vessels. The proportion of volume contained within the stressed and unstressed compartments varies depending on venous tone and is independent of total volume. The venous system can respond to physiologic demands by recruiting and de-recruiting volume from the unstressed compartment by means of sympathetic-mediated venous constriction, converting unstressed blood volume to stressed blood volume.[12]

<u>Key points for CVP</u>

1. CVP is influenced by non-volume variables, physiologic conditions, and hemodynamic responses to disease states, rather than being a true reflection of whole-body volume or fluid responsiveness.[14]
2. A non-elevated CVP is the normal physiologic state; and does not necessarily equate to a need for additional fluids.

3. An elevated CVP may represent volume intolerance and/or pathologies listed in Table 2.
4. When the IJV and IVC findings on POCUS are discordant there should be careful examination for caveats or confounders (Table 1, section 1a-1b).[32–35]

2) Right heart function

POCUS findings (Table 1, section 2a-2d)

1) Right ventricle (RV) size and function
2) Interventricular septum flattening
3) Presence of tricupid regurgitation (TR)
4) Presence of pericardial effusion

Pathology in the RV inherently compromises fluid tolerance; thus, an understanding of RV physiology and assessment is imperative when interpreting volume-related problems.

The right heart is a thin-walled compliant chamber with the capacity to accommodate wide ranges of volume, allowing for increases in cardiac output (CO) while maintaining a low filling pressure and optimizing venous return.[28,29] Beyond certain limits, RV dilation is eventually limited by the constraints of the pericardium. Additional volume loading past this point will lead to elevated RV diastolic pressure, resulting in an increased CVP which limits venous return. Septal flattening and ventricular interdependence will also result in impaired left ventricular filling in cases of RV pressure or volume overload.[35]

In settings of chronically elevated RV afterload, such as pulmonary hypertension, the RV adapts to higher pressures over time with cardiomyocyte hypertrophy, which increases contractility and RV systolic pressure without chamber dilation or CVP elevation. Once

contractile adaptations are overwhelmed, the RV dilates to preserve stroke volume (SV), leading to eventual impairment of RV function and right heart failure.[36] This stage is often associated with significant functional tricuspid regurgitation and chronically elevated filling pressures.

Lastly, it is worth describing the impact of the pericardial space on RV filling in the absence of RV dilation. In cardiac tamponade and constrictive pericarditis, increased pericardial pressure compromises RV filling due to extrinsic forces, which reduces preload.[37] This underscores the distinction between volume and pressure, and demonstrates that RV filling can be affected by non-volume variables.

Key points for right heart function

1. RV dilation and poor systolic function may represent excessive RV preload, excessive RV afterload, or intrinsic RV failure. Extra volume is tolerated poorly.
2. In significant TR and chronic pulmonary hypertension, an elevated CVP may not represent a volume overload state.

3. Left heart assessment

POCUS findings (Table 1, section 3a-3d)

1) Left atrial pressure (LAP)
2) LV systolic function
3) LV diastolic function
4) Calculated CO [38–41]

Overview

The end goal of optimizing a patient's volume status and hemodynamic parameters is to ensure adequate perfusion. The primary function of the LV is to provide sufficient CO to the

organs. CO is dependent on heart rate and LV stroke volume (SV), which is governed by preload, contractility, and afterload.[42] Intuitively, the left heart can only pump out what it receives; so left heart preload is further dependent on intravascular volume and right heart output.

Left Atrial Pressure

The left atrium is a reservoir, conduit, and pump to modulate LV filling.[43] LAP correlates with LV end-diastolic pressure; it is a surrogate for LV end-diastolic volume and thus may be used to assess LV preload.[44] Elevated LAP may be seen in LV systolic and/or diastolic dysfunction, left-sided valve pathology, or disease states such as sepsis and volume overload.[45] While it remains important not to conflate pressure and volume, in the setting of a volume assessment, elevated LAP should raise concerns about a patient who is fluid intolerant and at risk of pulmonary congestion.[44] Although commonly used as a surrogate of pulmonary congestion, it is important to recall that CVP is a right-sided parameter and that discordance between left and right sides may occur, rendering direct LAP asssessment useful in certain scenarios.

LV Systolic Function

Reduced LV systolic function is frequently encountered in patients with chronic heart failure, or more acutely as a consequence of a systemic illness (ie. septic cardiomyopathy). LV ejection fraction (EF) may be visually estimated, or calculated using end-systolic volume minus end-diastolic volume.[46] While a useful global parameter, EF is less reflective of the patient's current physiologic milieu compared with velocity-time integral-derived SV/CO as discussed below. A patient with a dilated LV may have a normal CO despite significant systolic dysfunction. Furthermore, EF may be confounded by valve lesions such as mitral regurgitation, leading to overestimation of forward flow.

LV Diastolic Function

Patients with isolated diastolic dysfunction are similarly at risk of complications related to cardiac decompensation and dysvolemia. Diastolic dysfunction may occur acutely in conditions such as sepsis; or chronically in patients with predisposing comorbidities. Recognition of impaired myocardial relaxation and filling is important as the therapeutic window for optimal fluid management in these patients is likely narrower.[47] Although a comprehensive diastolic assessment is beyond the skillset of casual POCUS users, clues to diastolic performance are available and should be incorporated into a holistic volume status assessment (Table 1: 3c).[48]

Cardiac output

When considering the relationship between volume and CO, the following equation provides important clues about the physiology associated with forward flow:

Mean arterial pressure (MAP) = CO x Systemic vascular resistance (SVR) + CVP

Fundamentally, the purpose of administering a fluid bolus is to increase SV.[49] The aim of increasing SV is to improve CO, with the end goal of optimizing tissue perfusion. Changes in a patient's SV and/or CO after a passive leg raise or small fluid bolus can be helpful in determining fluid responsiveness, and serial measurements can help monitor response to fluid, diuretic, vasoactive therapies.[50–52] However, decisions about fluid administration must look beyond fluid responsiveness alone. Fluid therapy impacts both the macrocirculation, which is concerned with the pulsatile pressure flow in large arteries; and the microcirculation.[53] For example, in sepsis, microcirculatory dysfunction, capillary occlusion, and capillary leakage causing tissue edema is common. Although fluid administration may improve SV, such benefits may be eclipsed by worsening microcirculatory tamponade and increased venous back-pressure, decreasing tissue oxygenation.[54] In other words, patients may be both volume responsive and volume intolerant at the same time. Therefore, although CO and maintenance of arterial pressure is crucial to perfusion, maximizing CO and

extinguishing volume responsiveness should not be the sole goal of resuscitative efforts, and consideration must be given to the overall impact that therapeutic interventions have on both the macro- and microcirculation.

Lastly, it is worth emphasizing that CO does not equal MAP, as SVR may vary widely in disease states. As it is relatively straightforward to measure MAP, CO and CVP at the bedside, these values can be used to make clinically useful inferences about a patient's SVR.

Key points for left heart assessment

1. In the presence of elevated LAP, caution must be exercised during fluid administration.
2. SV/CO measurement is useful in determining etiology of shock.
3. LV EF does not equate to CO; CO is a more useful dynamic variable to follow in acutely unwell patients.
4. SVR is an important aspect of the hemodynamic profile that impacts the interpretation of other volume-related data points.

4. Extravascular volume

POCUS findings (Table 1, section 4a-4e)

1) Pulmonary edema
2) Pleural effusion
3) Pericardial effusion
4) Ascites
5) Subcutaneous edema[55–57]

Despite myths and traditional practice patterns to the contrary, the development of extravascular volume including tissue edema may or may not coexist with increased intravascular fluid volume. Deciphering the etiology of extravascular fluid, and its relationship to intravascular volume, requires attention to the following physiologic principles.

Accumulation of extravascular fluid is the result of an imbalance between capillary filtration and lymphatic reabsorption. Broadly speaking, increased capillary filtration is the result of an increased trans-capillary hydrostatic pressure gradient, trans-capillary oncotic pressure differences, and increased capillary permeability.[58,59] Increased hydrostatic pressure may be due to an increase in total intravascular volume, volume redistribution, or venous insufficiency. This is commonly associated with neurohormonal dysregulation in conditions such as heart failure and cirrhosis.[60,61] Furthermore, the hydrostatic pressure gradient may also be impacted by reduced interstitial fluid pressure.[58] Finally, a disruption to the endothelial glycocalyx and/or increase in capillary permeability can also lead to increased movement of protein-rich fluid from intravascularly to the interstitium.[59,62]

The primary mechanism for fluid and protein reabsorption from the extravascular space is through the lymphatic system. Problems with reabsorption may arise from lymphatic obstruction or dysfunction. Lymphatic obstruction may occur via compression due to mass effect from tumors, compartment syndrome, or resistance to thoracic duct outflow secondary to increased CVP, which limits return of fluid to the central veins. Lymphatic dysfunction may be secondary to valvular incompetence, or a reduction in contractility of lymphatic smooth muscle cells as seen in systemic inflammatory conditions.[58] The above mechanisms of extravascular fluid accumulation demonstrate how tissue edema can occur without substantial increase in intravascular volume and/or pressure.

Extravascular fluid can accumulate in tissues, including the lungs and abdominal viscera, and in body cavities, including the pleural, peritoneal, and pericardial spaces. In general,

accumulation of extravascular fluid in multiple spaces is associated with a systemic process leading to total body volume overload, whereas regional edema may represent local disease processes or redistribution of volume from other compartments.

Key points for extravascular volume

1. Extravascular volume accumulation in multiple spaces suggests total body volume overload and volume intolerance.
2. Increased extravascular volume alone (ie. pedal edema) is not necessarily a reflection of elevated intravascular volume.
3. Extravascular fluid accumulation may be related to an increase in pressure, volume, and/or redistribution of volume from one compartment to another.

5. Venous congestion

POCUS findings (Table 1, section 5a-5c)

1) Hepatic vein waveform
2) Portal vein waveform
3) Intrarenal vein waveform

In combination with the IVC, this is termed the venous excess ultrasound (VEXUS) exam.[63–68]

Instead of pursuing euvolemia as it pertains to absolute fluid volume, *perfusion* should be the hemodynamic focal point of the volume assessment. Historically, cardiovascular and critical care research has focused on improving perfusion by optimizing forward flow, prioritizing CO and MAP in hemodynamically unstable patients. Guidelines have focused on aggressive fluid administration as a cornerstone therapy, together with inotropic and vasopressor support. Until recently, little attention was afforded to the detrimental effects of elevated venous pressures and increase resistance to venous return.[69,70] More recent studies have

clearly demonstrated the negative consequences of venous congestion in shock, cardiorenal syndrome, and hepatorenal syndrome. Volume overload, systemic venous hypertension, and tissue edema have deleterious effects on multiple organ systems, and are associated with increased mortality.[71,72]

Perfusion takes place at the level of the microcirculation, where the delta between capillary pressure and venous pressures is small (Figure 1). Excessive fluid administration leading to elevated venous pressures and reduced arteriovenous gradient across vital organs may hamper adequate perfusion.[63] In addition, for encapsulated organs such as the kidneys, interstitial edema may cause a significant increase in interstitial pressure, tubular compression, and intracapsular tamponade, further reducing organ perfusion.[72,73] Clinicians must consider the potential consequences of elevated venous pressures and compartment pressures when administering fluids to increase CO, as a disproportionate rise in upstream venous pressure causing organ congestion may outweigh the benefits of an increased MAP.

While an elevated CVP identifies elevated right heart filling pressures, this pressure may not necessarily be transmitted upstream causing tissue congestion. Conversely, focal congestion can occur without an associated increase in CVP. Ultrasound evaluation of specific venous waveforms upstream from the central veins has been shown to correlate with congestion at the organ level, and may provide additional specificity in this regard.[63] However, as with CVP, changes in venous waveforms are a pressure phenomenon which may or may not be related to volume.

<u>Key points for venous congestion</u>

1. Fluid resuscitation end points should not only focus on MAP, but consider the impact of venous congestion on tissue perfusion.
2. Abnormalities in venous waveforms suggest that organ impairment (congestive nephropathy and hepatopathy) may be secondary to venous congestion.[64,74]

Clinical Applications

A physiology-based evaluation incorporating central venous pressure, right heart function, left heart assessment, extravascular volume, and venous congestion serves as the scaffold for a comprehensive volume status assessment. POCUS allows us access to these hemodynamic and structural data points (Table 1). Because volume status is inextricably linked to an individual's hemodynamic profile, the POCUS exam must account for the patient's unique and often dynamic physiologic state. Findings must be integrated with a comprehensive clinical evaluation, including a review of relevant history and medications, clinical course including response to treatment, physical exam, laboratory results, and imaging investigations.[5,8,16]

The combination of POCUS applications most relevant to a volume-related problem depends on the clinical question at hand. We illustrate this using three common clinical scenarios: hypotension, hypoxia, and acute kidney injury (AKI) (Table 3). Certain POCUS applications, such as assessing for other causes of hypoxia and for obstructive uropathy, are not covered in this paper.

Despite the multitude of additional data point accessible with POCUS, bedside evaluation of fluid in the cardiovascular system remains limited. Clinicians should avoid making assumptions about a patient's intravascular volume status based on the findings of a single compartment. Using CVP as an example, a systematic review demonstrated a very poor relationship between CVP and blood volume, as well as the inability of CVP to predict fluid responsiveness in a wide spectrum of clinical conditions.[13,14] This weak relationship of CVP as a surrogate of preload and volume responsiveness is due to the unaccounted variables of vascular and cardiac compliance, non-volume variables, and the unequal distribution of total body volume among the various cardiovascular compartments.[4] Lastly, an awareness of the

potential caveats and pitfalls in image acquisition, interpretation, and clinical integration is paramount.

Future Directions

As the number of POCUS applications expand and become more advanced, the integration of multiorgan findings with a patient's unique physiology becomes increasingly complex, raising the risk of errors. Consequently, comprehensive training and oversight in POCUS training programmes are essential. Research is needed to explore the clinical outcomes of POCUS-guided volume assessments in different disease states, supported by a systematic scanning protocol. These outcomes should be compared with existing non-invasive hemodynamic monitoring methods.

Summary

Volume status is a ubiquitous yet nebulous clinical concept that is frequently oversimplified and superficially addressed in medical education. Unfortunately, an accurate volume status assessment remains elusive despite traditional physical examination techniques. This narrative review summarizes foundational physiologic tenants in the evaluation of volume status; and highlights how multiorgan POCUS can serve as a window into these hemodynamic parameters. In combination with conventional clinical assessment, these principles can help clinicians make challenging fluid-related decisions across a wide range of pathophysiologies.

References

1. Murray CM, Abbas Agha S, Rathi S, Germany RE. The evaluation and monitoring of volume status in congestive heart failure. *Congestive Heart Failure*. 2008;14(3):135-140.
2. Zhu F, Rosales L, Kotanko P. Techniques for assessing fluids status in patients with kidney disease. *Curr Opin Nephrol Hypertens*. 2016;25(6):473-479.
3. Davenport A, Argawal B, Wright G, et al. Can non-invasive measurements aid clinical assessment of volume in patients with cirrhosis? *World J Hepatol*. 2013;5(8):433-438.
4. Kalantari K, Chang JN, Ronco C, Rosner MH. Assessment of intravascular volume status and volume responsiveness in critically ill patients. *Kidney Int*. 2013;83(6):1017-1028.
5. Kearney D, Reisinger N, Lohani S. Integrative Volume Status Assessment. *POCUS Journal*. 2022;7(Kidney):65-77.
6. Maw A, Ortiz-lopez C, Morris M, et al. Hospitalist Perspectives of Available Tests to Monitor Volume Status in Patients With Heart Failure: A Qualitative Study. *Cureus*. Published online June 26, 2020.
7. EISENBERG PR, JAFFE AS, SCHUSTER DP. Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients. *Crit Care Med*. 1984;12(7):549-553.
8. Elhassan MG, Chao PW, Curiel A. The Conundrum of Volume Status Assessment: Revisiting Current and Future Tools Available for Physicians at the Bedside. *Cureus*. Published online May 26, 2021.
9. Chakko S, Woska D, Martinez H, et al. Clinical, radiographic, and hemodynamic correlations in chronic congestive heart failure: conflicting results may lead to inappropriate care. *Am J Med*. 1991;90(3):353-359.
10. Stevenson LW. The Limited Reliability of Physical Signs for Estimating Hemodynamics in Chronic Heart Failure. *JAMA: The Journal of the American Medical Association*. 1989;261(6):884.
11. Peacock WF, Braunwald E, Abraham W, et al. National heart, lung, and blood institute working group on emergency department management of acute heart failure: Research challenges and opportunities. *J Am Coll Cardiol*. 2010;56(5):343-351.
12. Persichini R, Lai C, Teboul JL, Adda I, Guérin L, Monnet X. Venous return and mean systemic filling pressure: physiology and clinical applications. *Crit Care*. 2022;26(1).
13. Marik PE, Baram M, Vahid B. Does Central Venous Pressure Predict Fluid Responsiveness?*: A Systematic Review of the Literature and the Tale of Seven Mares. *Chest*. 2008;134(1):172-178.
14. Marik PE, Cavallazzi R. Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense*. *Crit Care Med*. 2013;41(7):1774-1781.
15. Narula J, Chandrashekhar Y, Braunwald E. Time to Add a Fifth Pillar to Bedside Physical Examination. *JAMA Cardiol*. 2018;3(4):346.
16. Argaiz ER, Koratala A, Reisinger N. Comprehensive Assessment of Fluid Status by Point-of-Care Ultrasonography. *Kidney360*. 2021;2(8):1326-1338.

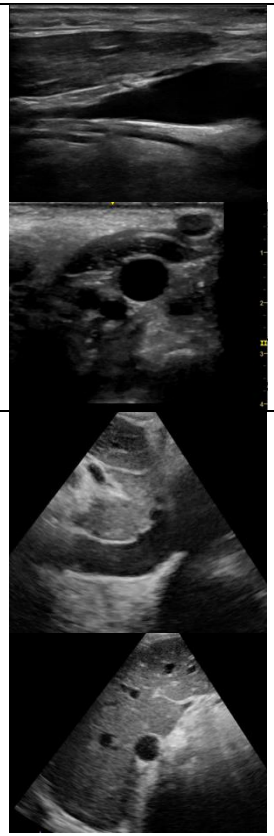
17. Koratala A, Kazory A. Point of Care Ultrasonography for Objective Assessment of Heart Failure: Integration of Cardiac, Vascular, and Extravascular Determinants of Volume Status. *Cardiorenal Med*. 2021;11(1):5-17.
18. Batool A, Chaudhry S, Koratala A. Transcending boundaries: Unleashing the potential of multi-organ point-of-care ultrasound in acute kidney injury. *World J Nephrol*. 2023;12(4):93-103.
19. Argaiz ER, Romero-Gonzalez G, Rola P, Spiegel R, Haycock KH, Koratala A. Bedside Ultrasound in the Management of Cardiorenal Syndromes: An Updated Review. *Cardiorenal Med*. 2023;13(1):372-384.
20. Soni N, Arntfield R, Kory P. *Point of Care Ultrasound*. 2nd ed. Elsevier; 2019.
21. Lipton B. Estimation of central venous pressure by ultrasound of the internal jugular vein. *Am J Emerg Med*. 2000;18(4):432-434.
22. Hossein-Nejad H, Mohammadinejad P, Ahmadi F. Internal jugular vein/common carotid artery cross-sectional area ratio and central venous pressure. *Journal of Clinical Ultrasound*. 2016;44(5):312-318.
23. Bano S, Qadeer A, Akhtar A, et al. Measurement of Internal Jugular Vein and Common Carotid Artery Diameter Ratio by Ultrasound to Estimate Central Venous Pressure. *Cureus*. Published online March 5, 2018.
24. Keller AS, Melamed R, Malinchoc M, John R, Tierney DM, Gajic O. Diagnostic accuracy of a simple ultrasound measurement to estimate central venous pressure in spontaneously breathing, critically ill patients. *J Hosp Med*. 2009;4(6):350-355.
25. Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography. Endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *Journal of the American Society of Echocardiography*. 2010;23(7):685-713.
26. Seo Y, Iida N, Yamamoto M, Machino-Ohtsuka T, Ishizu T, Aonuma K. Estimation of Central Venous Pressure Using the Ratio of Short to Long Diameter from Cross-Sectional Images of the Inferior Vena Cava. *Journal of the American Society of Echocardiography*. 2017;30(5):461-467.
27. Huguet R, Fard D, d'Humieres T, et al. Three-Dimensional Inferior Vena Cava for Assessing Central Venous Pressure in Patients with Cardiogenic Shock. *Journal of the American Society of Echocardiography*. 2018;31(9):1034-1043.
28. Berlin DA, Bakker J. Starling curves and central venous pressure. *Crit Care*. 2014;19(1).
29. Mebazaa A, Karpati P, Renaud E, Algotsson L. Acute right ventricular failure - From pathophysiology to new treatments. *Intensive Care Med*. 2004;30(2):185-196.
30. De Vecchis R, Baldi C, Giandomenico G, Di Maio M, Giasi A, Cioppa C. Estimating Right Atrial Pressure Using Ultrasounds: An Old Issue Revisited With New Methods. *J Clin Med Res*. 2016;8(8):569-574.
31. Tansey EA, Montgomery LEA, Quinn JG, Roe SM, Johnson CD. SOURCEBOOK OF LABORATORY ACTIVITIES IN PHYSIOLOGY Understanding basic vein physiology and venous blood pressure through simple physical assessments. *Adv Physiol Educ*. 2019;43:423-429.
32. Via G, Tavazzi G, Price S. Ten situations where inferior vena cava ultrasound may fail to accurately predict fluid responsiveness: a physiologically based point of view. *Intensive Care Med*. 2016;42(7):1164-1167.

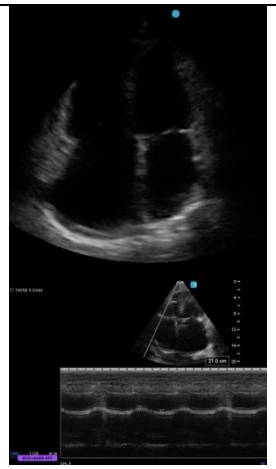
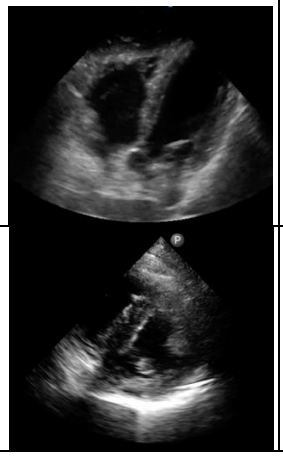
33. Millington SJ. Ultrasound assessment of the inferior vena cava for fluid responsiveness: easy, fun, but unlikely to be helpful. *Canadian Journal of Anesthesia*. 2019;66(6):633-638.
34. Wachsberg RH, Levine CD, Maldjian PD, Simmons MZ. Dilatation of the inferior vena cava in patients with cirrhotic portal hypertension. *Clin Imaging*. 1998;22(1):48-53.
35. Istrail L, Kiernan J, Stepanova M. A Novel Method for Estimating Right Atrial Pressure With Point-of-Care Ultrasound. *Journal of the American Society of Echocardiography*. 2023;36(3):278-283.
36. Rako ZA, Kremer N, Yogeswaran A, Richter MJ, Tello K. Adaptive versus maladaptive right ventricular remodelling. *ESC Heart Fail*. 2023;10(2):762-775.
37. Yuriditsky E, Horowitz JM. The physiology of cardiac tamponade and implications for patient management. *J Crit Care*. 2024;80:154512.
38. Čelutkienė J, Lainscak M, Anderson L, et al. Imaging in patients with suspected acute heart failure: timeline approach position statement on behalf of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail*. 2020;22(2):181-195.
39. Mok KL. Make it SIMPLE: Enhanced shock management by focused cardiac ultrasound. *J Intensive Care*. 2016;4(1).
40. Vermeiren GLJ, Malbrain MLNG, Walpot JMJB. Cardiac Ultrasonography in the critical care setting: A practical approach to asses cardiac function and preload for the "non-cardiologist." *Anaesthesiol Intensive Ther*. 2015;47(1):s89-s104.
41. Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Journal of the American Society of Echocardiography*. 2016;29(4):277-314.
42. Vincent JL. Understanding cardiac output. *Crit Care*. 2008;12(4).
43. Thomas L, Marwick TH, Popescu BA, Donal E, Badano LP. Left Atrial Structure and Function, and Left Ventricular Diastolic Dysfunction. *J Am Coll Cardiol*. 2019;73(15):1961-1977.
44. Bowcock EM, Mclean A. Bedside assessment of left atrial pressure in critical care: a multifaceted gem. *Crit Care*. 2022;26(1).
45. Orde S, Slama M, Hilton A, Yastrebov K, McLean A. Pearls and pitfalls in comprehensive critical care echocardiography. *Crit Care*. 2017;21(1).
46. Foley TA, Mankad S V, Anavekar NS, et al. Measuring Left Ventricular Ejection Fraction – Techniques and Potential Pitfalls. *European Cardiology Review*. 2012;8(2):108.
47. Sanfilippo F, Scolletta S, Morelli A, Vieillard-Baron A. Practical approach to diastolic dysfunction in light of the new guidelines and clinical applications in the operating room and in the intensive care. *Ann Intensive Care*. 2018;8(1):100.
48. Kattel S, Memon S, Saito K, Narula J, Saito Y. An effect of left ventricular hypertrophy on mild-to-moderate left ventricular diastolic dysfunction. *Hellenic Journal of Cardiology*. 2016;57(2):92-98.
49. Marik PE, Lemson J. Fluid responsiveness: An evolution of our understanding. *Br J Anaesth*. 2014;112(4):617-620.
50. Wu Y, Zhou S, Zhou Z, Liu B. A 10-second fluid challenge guided by transthoracic echocardiography can predict fluid responsiveness. *Crit Care*. 2014;18(3).

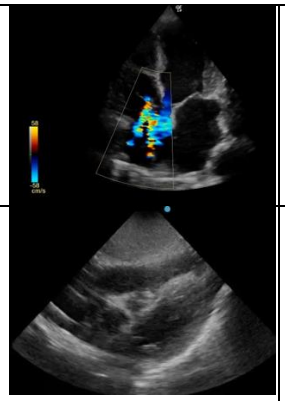
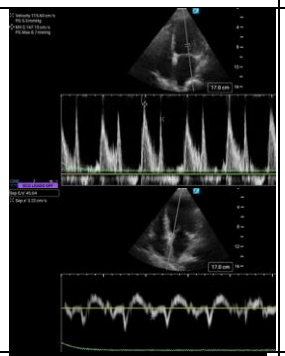
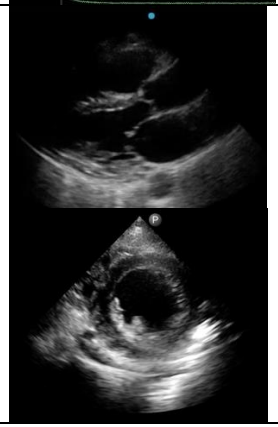
51. Cherpanath TG V., Hirsch A, Geerts BF, et al. Predicting Fluid Responsiveness by Passive Leg Raising. *Crit Care Med*. 2016;44(5):981-991.
52. Cavallaro F, Sandroni C, Marano C, et al. Diagnostic accuracy of passive leg raising for prediction of fluid responsiveness in adults: Systematic review and meta-analysis of clinical studies. *Intensive Care Med*. 2010;36(9):1475-1483.
53. Khanna AK, Karamchandani K. Macrocirculation and Microcirculation: The “batman and Superman” Story of Critical Care Resuscitation. *Anesth Analg*. 2021;132(1):280-283.
54. Bennett VA, Vidouris A, Cecconi M. Effects of Fluids on the Macro- and Microcirculations. *Crit Care*. 2018;22(1).
55. Hubert A, Girerd N, Le Breton H, et al. Diagnostic accuracy of lung ultrasound for identification of elevated left ventricular filling pressure. *Int J Cardiol*. 2019;281:62-68.
56. Wiener-Kronish JP, Matthay MA. Pleural Effusions Associated with Hydrostatic and Increased Permeability Pulmonary Edema. *Chest*. 1988;93(4):852-858.
57. Platz E, Merz AA, Jhund PS, Vazir A, Campbell R, McMurray JJ. Dynamic changes and prognostic value of pulmonary congestion by lung ultrasound in acute and chronic heart failure: a systematic review. *Eur J Heart Fail*. 2017;19(9):1154-1163.
58. Dull RO, Hahn RG. Hypovolemia with peripheral edema: What is wrong? *Crit Care*. 2023;27(1).
59. Lent-Schochet D, Jialal I. *Physiology, Edema*. StatPearls Publishing; 2023.
60. Miller WL. Fluid volume overload and congestion in heart failure. *Circ Heart Fail*. 2016;9(8).
61. Arroyo V. *Pathophysiology, Diagnosis and Treatment of Ascites in Cirrhosis*. Vol 1.; 2002.
62. Siddall E, Khatri M, Radhakrishnan J. Capillary leak syndrome: etiologies, pathophysiology, and management. *Kidney Int*. 2017;92(1):37-46.
63. Beaubien-Souligny W, Rola P, Haycock K, et al. Quantifying systemic congestion with Point-Of-Care ultrasound: development of the venous excess ultrasound grading system. *Ultrasound Journal*. 2020;12(1).
64. Styczynski G, Milewska A, Marczewska M, et al. Echocardiographic Correlates of Abnormal Liver Tests in Patients with Exacerbation of Chronic Heart Failure. *Journal of the American Society of Echocardiography*. 2016;29(2):132-139.
65. Gallix BP, Taourel P, Dauzat M, Bruel JM, Lafortune M. Flow pulsatility in the portal venous system: a study of Doppler sonography in healthy adults. *American Journal of Roentgenology*. 1997;169(1):141-144.
66. Scheinfeld MH, Bilali A, Koenigsberg M. Understanding the Spectral Doppler Waveform of the Hepatic Veins in Health and Disease. *RadioGraphics*. 2009;29(7):2081-2098.
67. Singh S, Koratala A. Utility of Doppler ultrasound derived hepatic and portal venous waveforms in the management of heart failure exacerbation. *Clin Case Rep*. 2020;8(8):1489-1493.
68. Gómez-Rodríguez C, Tadeo-Espinoza H, Solis-Huerta F, et al. Hemodynamic Evaluation of Right-Sided Congestion With Doppler Ultrasonography in Pulmonary Hypertension. *Am J Cardiol*. 2023;203:459-462.
69. Van Diepen S, Katz JN, Albert NM, et al. Contemporary Management of Cardiogenic Shock: A Scientific Statement from the American Heart Association. *Circulation*. 2017;136(16):e232-e268.
70. Seymour CW, Rosengart MR. Septic Shock. *JAMA*. 2015;314(7):708.

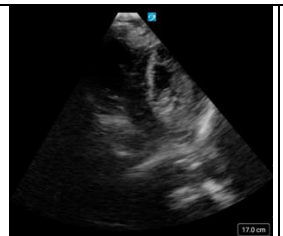
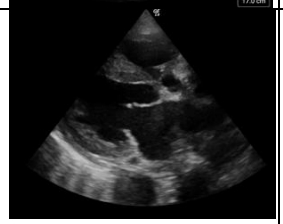
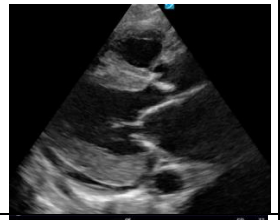
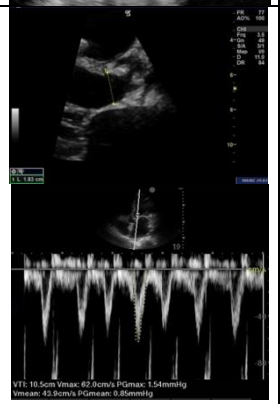
71. Jaffee W, Hodgins S, McGee WT. Tissue Edema, Fluid Balance, and Patient Outcomes in Severe Sepsis: An Organ Systems Review. *J Intensive Care Med*. 2018;33(9):502-509.
72. Rosenkranz S, Howard LS, Gomberg-Maitland M, Hoeper MM. Systemic Consequences of Pulmonary Hypertension and Right-Sided Heart Failure. *Circulation*. 2020;141(8):678-693.
73. Cruces P, Salas C, Lillo P, Salomon T, Lillo F, Hurtado DE. The renal compartment: a hydraulic view. *Intensive Care Medicine Experimental* . 2014;2(1):1-9.
74. Iida N, Seo Y, Sai S, et al. *Clinical Implications of Intrarenal Hemodynamic Evaluation by Doppler Ultrasonography in Heart Failure*.; 2016.

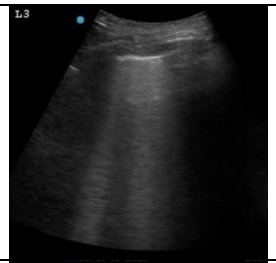
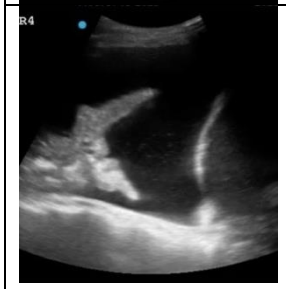
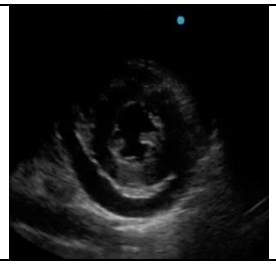
Table 1. POCUS Applications in the Volume Status Assessment

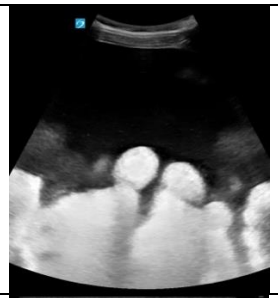
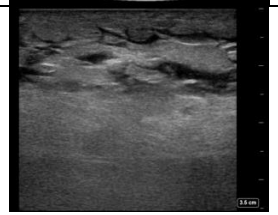
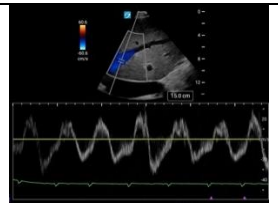
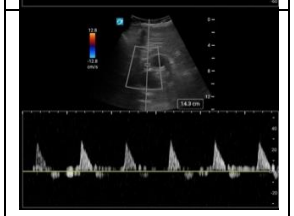
Physiological Concept	POCUS Application	POCUS findings	Interpretation	Example	Caveats or Confounders
1. Central venous pressure	(a) IJV	The height of the JVP is taken as the point of maximal tenting of the IJV in the neck, measured in centimetres above the sternal angle. Assess if IJV is ovoid or circular. Assess the ratio of the IJV to CCA diameter.	An elevated JVP, a circular IJV, and a higher ratio of IJV to CCA diameter correlates with an elevated CVP.		Increased right atrial depth may lead to an underestimation of CVP if purely based on JVP height. IJV stenosis/thrombus, SVC stenosis, pulmonary hypertension, and TR may to an elevated, distended, or circular IJV that is unrelated to volume.
	(b) IVC	Measure the diameter of the IVC. Assess if the IVC is ovoid or circular. Assess the respiratory variability of the IVC with a sniff test.	A distended, plethoric, spherical IVC with minimal inspiratory collapse corresponds with an elevated CVP in spontaneously breathing patients.		TR may result in a chronically distended IVC. In cirrhosis, the IVC may be dilated due to portosystemic collaterals, or constricted by a hypertrophied caudate lobe. In the setting of raised intra-abdominal pressure, the IVC size may be underestimated.

2. Right heart function	(a) RV size and function	Assess RV size.	The RV is dilated (on qualitative assessment) when it is >2/3 the size of the LV.		In the presence of LV dilation, RV dilation may be underestimated.
		Assess RV systolic function qualitatively, or measure the TAPSE.	Reduced TAPSE suggests reduced RV systolic function.		
			A dilated RV with reduced systolic function favours volume intolerance.		
		RV free wall thickness.	A thickened RV wall suggests chronic pulmonary hypertension.		It is challenging to distinguish between acute and chronic right heart failure.
	(b) IVS	Identify flattening of IVS i.e D-shaped septum.	Presence of D-shaped septum suggests RV volume or pressure overload.		Volume and pressure overload may co-exist

	(c) TR	Identify TR.	The presence of significant TR affects interpretation of CVP when making volume-related decisions.		In RV failure, TR may be underestimated due to failure of RV to generate sufficient contractile forces.
	(d) Pericardial space	Identify pericardial effusion and its impact on RA/RV filling.	The presence of RA and/or RV collapse during diastole suggests compromised right heart filling and tamponade physiology.		Hemodynamic effect of pericardial effusions are related to the volume, rate of accumulation, and patient's intravascular volume status.
3. Left heart assessment	(a) LAP	Assess MV inflow velocity (E), and mitral annular excursion (e').	E/e' > 14 suggests elevated LAP and can be seen in impaired diastolic function. An elevated LAP favours volume intolerance.		E/e' measurements are affected by intrinsic mitral valve pathologies.
	(b) LV systolic function	LV systolic function can be estimated visually.	Reduced myocardial thickening, myocardial excursion, and chamber emptying suggests reduced LV systolic function.		Recall that EF is not always congruent with CO, and the latter is much more reflective of the patient's current hemodynamic milieu. Valve disease such as MR can further confound EF assessment.

			A hyperdynamic LV may suggest intravascular hypovolemia or reduced SVR in the appropriate clinical context.		
	(c) LV diastolic function	Assess LA size. Assess LV thickness.	The LA is dilated (on qualitative assessment) when it is larger than the diameter of the RVOT and the aortic outflow tract in the parasternal long-axis view. LVH is associated with an increased probability of diastolic dysfunction.	 	LA dilation and LVH occur in chronically elevated LAP. In acutely elevated LAP (eg. sepsis), LA dilation and LVH may not be apparent. In such cases, E/e' can be helpful. Increased LV wall thickness on qualitative assessment via POCUS should trigger formal assessment for LVH.
	(d) Calculate cardiac output	Obtain the LVOT diameter and use it to calculate the CSA of the LVOT. Obtain the LVOT VTI. Calculate SV by multiplying the LVOT CSA and the LVOT VTI.	SV is a measure of forward flow out of the LV. CO is calculated using SV multiplied by heart rate. Changes in SV can be monitored in response to fluid and decongestive therapies.		Consider that SV is affected by preload, contractility, and afterload.

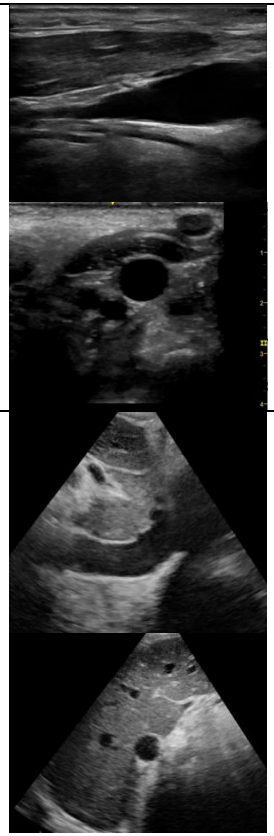
4. Extravascular volume	(a) Pulmonary edema	Assess distribution and quantity of B-lines in lungs.	Bilateral symmetrical B lines with a dependent gradient and smooth pleural line suggests cardiogenic pulmonary edema. The presence of pulmonary edema and pleural effusions is associated with elevated LV filling pressures and the permeability of pulmonary vasculature. Serial exams allow semi-quantitative measurements of the degree of pulmonary edema, and monitoring of response to decongestive therapies.		B-lines may have inflammatory etiologies, such as pneumonia and interstitial lung disease.
	(b) Pleural effusions	Assess quantity and character of fluid in pleural space.			Pleural effusions, pericardial effusions, and ascites may have inflammatory etiologies, including infection and malignancy.
	(c) Pericardial effusion	Assess quantity and character of fluid in pericardial space.	Pericardial effusions related to total body volume overload are typically circumferential and anechoic.		Hemodynamic effect is related to the volume, rate of accumulation, and patient's intravascular volume status.

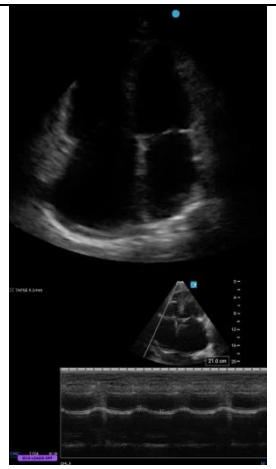
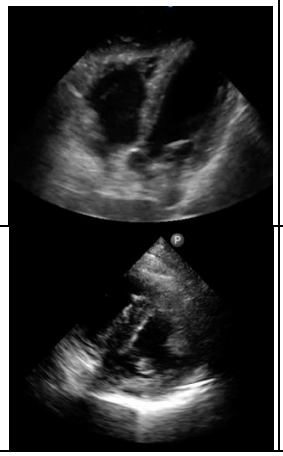
	(d) Ascites	Assess quantity and character of free fluid in peritoneal space.	Serial exams allow for monitoring of response to decongestive therapies.		Ascites may have inflammatory etiologies, including infection and malignancy.
	(e) Subcutaneous edema	Assess for presence of subcutaneous edema.	Subcutaneous edema leads to a "cobblestone" appearance. In total body volume overload, this accumulates in dependent regions of the body.		Subcutaneous edema can also be seen in inflammatory etiologies, such as cellulitis. These tend to be more localized.
5. Venous congestion	(a) Hepatic vein waveform	Assess pattern of venous waveform, in particular the S wave (systole) and D wave (diastole).	Venous congestion is suspected when the - hepatic vein waveform demonstrates S<D or S wave reversal. - portal vein waveform demonstrates >30% pulsatility. - intrarenal vein waveform is interrupted. Serial exams allow monitoring of response to treatment, as venous waveform abnormalities improve with decongestive therapy. This is particularly helpful in patients with chronic		Waveforms may be affected by non-volume related variables, including the presence of cirrhosis, tricuspid regurgitation, low body mass index, and intrinsic renal pathologies; as well as congestion due to primary pressure-related disorders.
	(b) Portal vein waveform	Assess pattern of venous waveform for pulsatility.			
	(c) Intrarenal vein waveform	Assess pattern of venous waveform for interrupted flow.			

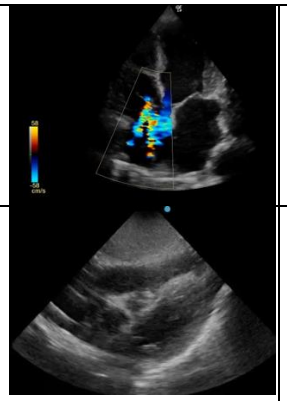
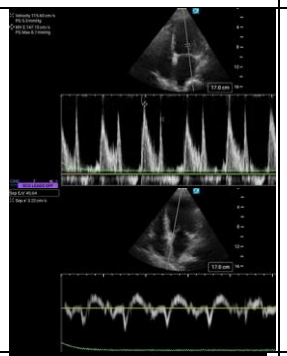
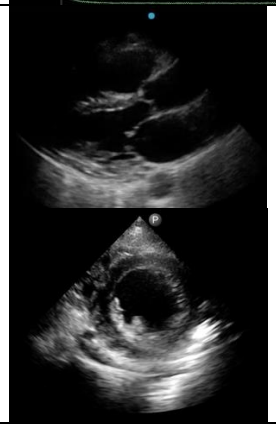
			cardiac pathology in whom parameters such as CVP may never normalize despite adequate decongestion.		

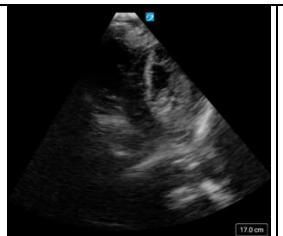
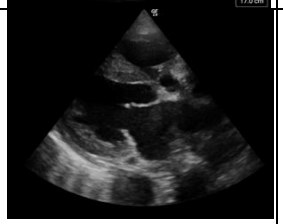
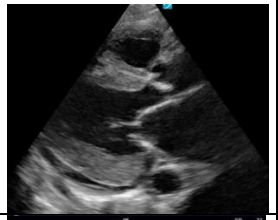
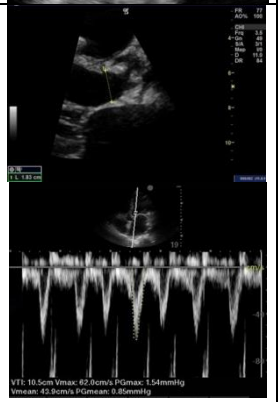
IJV: Internal jugular vein; JVP: Jugular venous pressure; CCA: Common carotid artery; CVP: Central venous pressure; SVC: Superior vena cava; TR: Tricuspid regurgitation; IVC: Inferior vena cava; RV: Right ventricle; TAPSE: Tricuspid annular plane systolic excursion; IVS: Interventricular septum; LAP: Left atrial pressure; LA: Left atrium; MV: Mitral valve; RVOT: Right ventricle outflow tract; LV: Left ventricle; LVH; Left ventricular hypertrophy; SVR: Systemic vascular resistance; EF: Ejection fraction; CO: Cardiac output; LVOT: Left ventricle outflow tract; CSA: Cross-sectional area; VTI: Velocity time integral; SV: Stroke volume.
See Corresponding Videos in the online supplemental material.

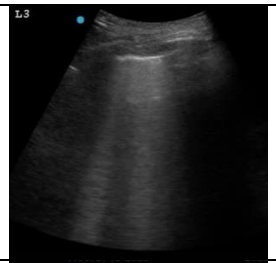
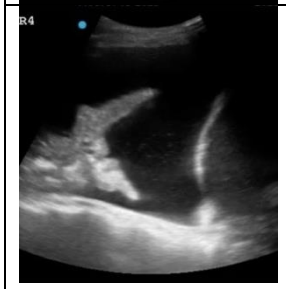
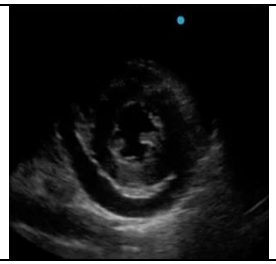
Table 1. POCUS Applications in the Volume Status Assessment

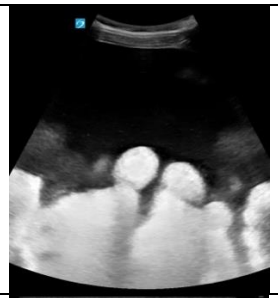
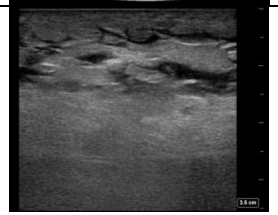
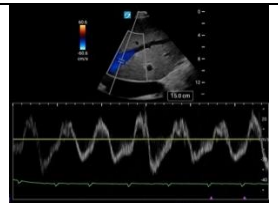
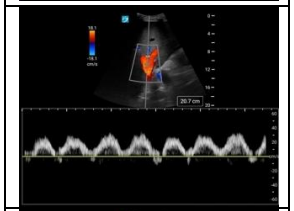
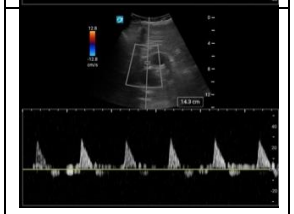
Physiological Concept	POCUS Application	POCUS findings	Interpretation	Example	Caveats or Confounders
1. Central venous pressure	(a) IJV	The height of the JVP is taken as the point of maximal tenting of the IJV in the neck, measured in centimetres above the sternal angle. Assess if IJV is ovoid or circular. Assess the ratio of the IJV to CCA diameter.	An elevated JVP, a circular IJV, and a higher ratio of IJV to CCA diameter correlates with an elevated CVP.		Increased right atrial depth may lead to an underestimation of CVP if purely based on JVP height. IJV stenosis/thrombus, SVC stenosis, pulmonary hypertension, and TR may to an elevated, distended, or circular IJV that is unrelated to volume.
	(b) IVC	Measure the diameter of the IVC. Assess if the IVC is ovoid or circular. Assess the respiratory variability of the IVC with a sniff test.	A distended, plethoric, spherical IVC with minimal inspiratory collapse corresponds with an elevated CVP in spontaneously breathing patients.		TR may result in a chronically distended IVC. In cirrhosis, the IVC may be dilated due to portosystemic collaterals, or constricted by a hypertrophied caudate lobe. In the setting of raised intra-abdominal pressure, the IVC size may be underestimated.

2. Right heart function	(a) RV size and function	Assess RV size.	The RV is dilated (on qualitative assessment) when it is >2/3 the size of the LV.		In the presence of LV dilation, RV dilation may be underestimated.
		Assess RV systolic function qualitatively, or measure the TAPSE.	Reduced TAPSE suggests reduced RV systolic function. A dilated RV with reduced systolic function favours volume intolerance.		
		RV free wall thickness.	A thickened RV wall suggests chronic pulmonary hypertension.		It is challenging to distinguish between acute and chronic right heart failure.
	(b) IVS	Identify flattening of IVS i.e D-shaped septum.	Presence of D-shaped septum suggests RV volume or pressure overload.		Volume and pressure overload may co-exist

	(c) TR	Identify TR.	The presence of significant TR affects interpretation of CVP when making volume-related decisions.		In RV failure, TR may be underestimated due to failure of RV to generate sufficient contractile forces.
	(d) Pericardial space	Identify pericardial effusion and its impact on RA/RV filling.	The presence of RA and/or RV collapse during diastole suggests compromised right heart filling and tamponade physiology.		Hemodynamic effect of pericardial effusions are related to the volume, rate of accumulation, and patient's intravascular volume status.
3. Left heart assessment	(a) LAP	Assess MV inflow velocity (E), and mitral annular excursion (e').	E/e' > 14 suggests elevated LAP and can be seen in impaired diastolic function. An elevated LAP favours volume intolerance.		E/e' measurements are affected by intrinsic mitral valve pathologies.
	(b) LV systolic function	LV systolic function can be estimated visually.	Reduced myocardial thickening, myocardial excursion, and chamber emptying suggests reduced LV systolic function.		Recall that EF is not always congruent with CO, and the latter is much more reflective of the patient's current hemodynamic milieu. Valve disease such as MR can further confound EF assessment.

			A hyperdynamic LV may suggest intravascular hypovolemia or reduced SVR in the appropriate clinical context.		
	(c) LV diastolic function	Assess LA size. Assess LV thickness.	The LA is dilated (on qualitative assessment) when it is larger than the diameter of the RVOT and the aortic outflow tract in the parasternal long-axis view. LVH is associated with an increased probability of diastolic dysfunction.	 	LA dilation and LVH occur in chronically elevated LAP. In acutely elevated LAP (eg. sepsis), LA dilation and LVH may not be apparent. In such cases, E/e' can be helpful. Increased LV wall thickness on qualitative assessment via POCUS should trigger formal assessment for LVH.
	(d) Calculate cardiac output	Obtain the LVOT diameter and use it to calculate the CSA of the LVOT. Obtain the LVOT VTI. Calculate SV by multiplying the LVOT CSA and the LVOT VTI.	SV is a measure of forward flow out of the LV. CO is calculated using SV multiplied by heart rate. Changes in SV can be monitored in response to fluid and decongestive therapies.		Consider that SV is affected by preload, contractility, and afterload.

4. Extravascular volume	(a) Pulmonary edema	Assess distribution and quantity of B-lines in lungs.	Bilateral symmetrical B lines with a dependent gradient and smooth pleural line suggests cardiogenic pulmonary edema. The presence of pulmonary edema and pleural effusions is associated with elevated LV filling pressures and the permeability of pulmonary vasculature. Serial exams allow semi-quantitative measurements of the degree of pulmonary edema, and monitoring of response to decongestive therapies.		B-lines may have inflammatory etiologies, such as pneumonia and interstitial lung disease.
	(b) Pleural effusions	Assess quantity and character of fluid in pleural space.			Pleural effusions, pericardial effusions, and ascites may have inflammatory etiologies, including infection and malignancy.
	(c) Pericardial effusion	Assess quantity and character of fluid in pericardial space.	Pericardial effusions related to total body volume overload are typically circumferential and anechoic.		Hemodynamic effect is related to the volume, rate of accumulation, and patient's intravascular volume status.

	(d) Ascites	Assess quantity and character of free fluid in peritoneal space.	Serial exams allow for monitoring of response to decongestive therapies.		Ascites may have inflammatory etiologies, including infection and malignancy.
	(e) Subcutaneous edema	Assess for presence of subcutaneous edema.	Subcutaneous edema leads to a “cobblestone” appearance. In total body volume overload, this accumulates in dependent regions of the body.		Subcutaneous edema can also be seen in inflammatory etiologies, such as cellulitis. These tend to be more localized.
5. Venous congestion	(a) Hepatic vein waveform	Assess pattern of venous waveform, in particular the S wave (systole) and D wave (diastole).	Venous congestion is suspected when the - hepatic vein waveform demonstrates S<D or S wave reversal. - portal vein waveform demonstrates >30% pulsatility. - intrarenal vein waveform is interrupted. Serial exams allow monitoring of response to treatment, as venous waveform abnormalities improve with decongestive therapy. This is particularly helpful in patients with chronic		Waveforms may be affected by non-volume related variables, including the presence of cirrhosis, tricuspid regurgitation, low body mass index, and intrinsic renal pathologies; as well as congestion due to primary pressure-related disorders.
	(b) Portal vein waveform	Assess pattern of venous waveform for pulsatility.			
	(c) Intrarenal vein waveform	Assess pattern of venous waveform for interrupted flow.			

			cardiac pathology in whom parameters such as CVP may never normalize despite adequate decongestion.		

IJV: Internal jugular vein; JVP: Jugular venous pressure; CCA: Common carotid artery; CVP: Central venous pressure; SVC: Superior vena cava; TR: Tricuspid regurgitation; IVC: Inferior vena cava; RV: Right ventricle; TAPSE: Tricuspid annular plane systolic excursion; IVS: Interventricular septum; LAP: Left atrial pressure; LA: Left atrium; MV: Mitral valve; RVOT: Right ventricle outflow tract; LV: Left ventricle; LVH; Left ventricular hypertrophy; SVR: Systemic vascular resistance; EF: Ejection fraction; CO: Cardiac output; LVOT: Left ventricle outflow tract; CSA: Cross-sectional area; VTI: Velocity time integral; SV: Stroke volume.
See Corresponding Videos in the online supplemental material.

Table 2. Causes of elevated CVP that do not reflect total body volume overload

Pathology	**Example**
Elevated juxtacardiac pressure impeding venous return	Cardiac tamponade Tension pneumothorax
RV outflow tract obstruction	Pulmonary stenosis
Elevated pulmonary vascular resistance	Pulmonary embolism Acute Respiratory Distress Syndrome
Intrinsic RV failure	RV infarct with RV systolic dysfunction
Acute valve failure	Damage to the tricuspid valve associated with infective endocarditis

RV: Right ventricle