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Point-of-Care Ultrasound and Volume Status: Embracing Physiology Over Proxies

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INTRODUCTION

Few challenges in nephron-critical care generate as much debate and quiet uncertainty as the assessment of "volume status". Despite decades of clinical experience and an expanding array of bedside technologies, clinicians continue to wrestle with a deceptively simple question: is this patient volume overloaded, volume depleted, or neither? The persistence of this uncertainty reflects a deeper truth. Volume status is not a single physiologic variable, and any attempt to reduce it to one measurement will inevitably fall short.

Point of care ultrasound (POCUS) has increasingly entered this debate, often accompanied by the expectation that it can directly measure volume. In particular, inferior vena cava (IVC) ultrasound is frequently treated as a kind of magic wand for determining volume status. It is not. Importantly, neither are the invasive tools that have long been regarded as definitive. Even pulmonary artery catheterization does not measure volume; it reports pressures and derived indices that function as surrogates for complex hemodynamic states. Hemodynamic assessment, whether ultrasound based or invasive, is therefore inherently inferential.

Right atrial pressure (RAP) illustrates this limitation particularly well. It is frequently invoked as a proxy for intravascular volume, yet it reflects far more than volume alone. The dissociation between intravascular volume and RAP has been well documented.[1] Venous pressures are influenced by global cardiac performance, pericardial constraint, intrathoracic pressure, pulmonary vascular resistance, valvular pathology, venous compliance, and intraabdominal pressure. Any interpretation of IVC ultrasound as a surrogate for RAP must account for this physiologic complexity (**Figure**). Even then, its accuracy is limited, particularly in patients receiving mechanical ventilation. Parkin's assertion remains profoundly relevant: *RAP, and by extension IVC ultrasound, reflects true volume state only when the heart is not beating*.[2] The remark may come across as provocative, yet it distills a central physiologic truth. Pressure should not be mistaken for volume. The broader literature on IVC ultrasound further complicates matters. It often blurs the distinction between its intended role as a tool for estimating RAP and its controversial use in predicting fluid responsiveness. The latter concept is itself fraught with physiologic and methodological limitations, and the field is increasingly shifting toward the more clinically grounded construct of fluid tolerance as detailed in prior commentary.[3]

In practical terms, identical RA pressures can represent profoundly different physiologic states. One patient with an elevated pressure and a plethoric IVC may be truly hypervolemic and benefit from diuresis or ultrafiltration. Another may have elevated pressure driven by a large pericardial effusion, where indiscriminate fluid removal could precipitate hemodynamic collapse. In yet other cases, modulation of pulmonary

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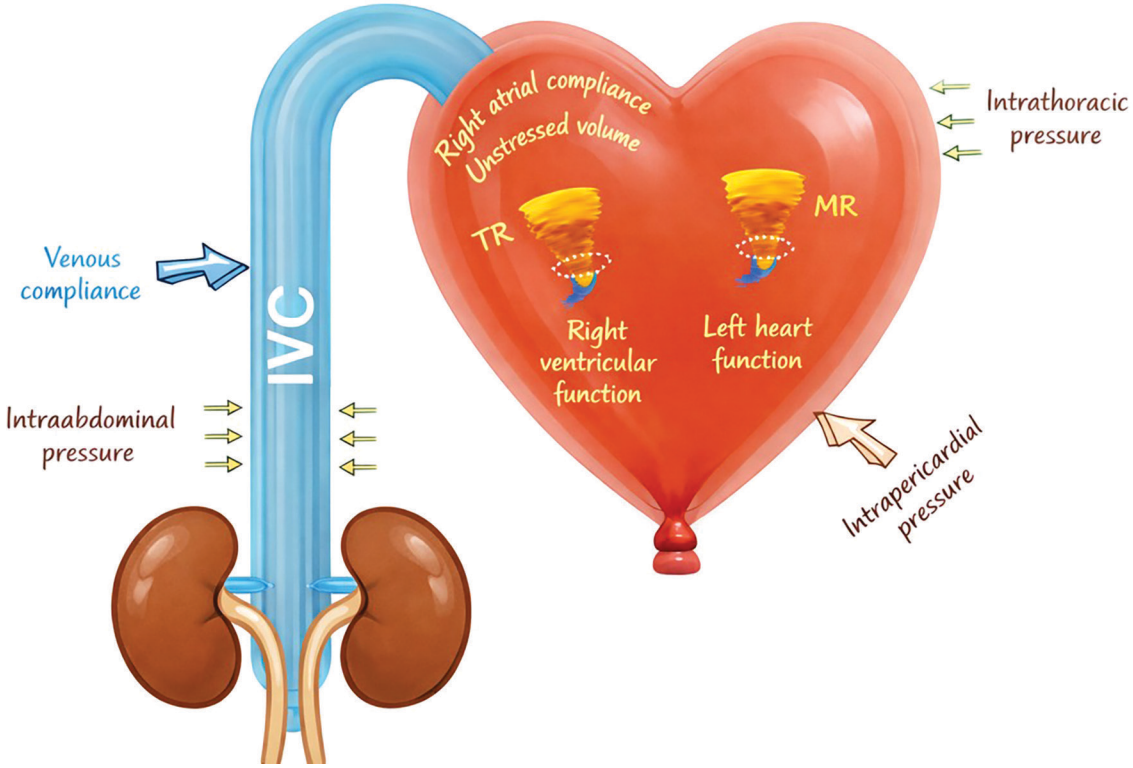

Conceptual framework illustrating physiologic determinants of inferior vena cava (IVC) dynamics during hemodynamic assessment, including right and left heart function, valvular regurgitation, venous compliance, intraabdominal and intrathoracic pressures, and pericardial constraint. TR denotes tricuspid regurgitation and MR denotes mitral regurgitation.

vascular resistance may be more appropriate than altering volume. Conversely, a small and collapsible inferior vena cava does not reliably distinguish hypovolemia from euvolemia or from vasodilatory states. As such, interpreting RAP in isolation risks conflating pressure with volume. That conceptual error can meaningfully distort clinical decision making and lead to inappropriate fluid administration or removal.

Lung POCUS findings are equally susceptible to oversimplification. B lines are sensitive indicators of increased extravascular lung water, yet they are not specific for cardiogenic pulmonary edema. Bilateral B lines may reflect a broad spectrum of conditions, including acute respiratory distress syndrome, diffuse interstitial pneumonia or pneumonitis, pulmonary fibrosis, and other chronic interstitial lung diseases. In contrast, localized or unilateral B lines more often point toward focal processes such as pneumonia, pulmonary contusion following trauma, or neoplastic involvement.[4] Interpretation therefore hinges on clinical context. Even with careful attention to structural and physiologic cues, overlap between cardiogenic and noncardiogenic causes of B lines is common and requires integration with cardiac structure and function. In some settings, lung POCUS is treated as a standalone measure of volume status, with enthusiasm that extends well beyond the evidence derived from hemodialysis cohorts. Accordingly, the absence of B lines may be misinterpreted as evidence of normal volume status, overlooking right sided congestion, or even construed as a risk factor for intradialytic hypotension. This reasoning can lead to suboptimal decongestion or fluid removal only in the presence of lung congestion. Such logic is no more defensible than arguing that a dialysis patient with a normal lung examination should not undergo ultrafiltration.[5]

These limitations underscore a central principle that is often underemphasized in discussions of volume status: the assessment must always include the heart. Whether through focused cardiac ultrasound or thoughtful interpretation of formal echocardiography, cardiac function provides the framework within which all other findings acquire

meaning. Left and right ventricular performance, diastolic function, chamber interactions, and valvular pathology directly shape filling pressures, venous congestion, and ultimately organ perfusion. POCUS is most informative when cardiac evaluation anchors the examination, allowing pulmonary and venous findings to be understood as downstream manifestations of upstream physiology. In its ideal form, POCUS based "hemodynamic status" assessment, a more accurate term than volume status assessment, encompasses the entire circulatory circuit, including forward flow, left sided filling pressures and their consequences such as mitral inflow patterns and lung ultrasound findings, as well as right sided pressures and their sequelae, including trans tricuspid gradients, IVC dynamics, and markers of venous congestion such as VExUS.[6]

Beyond ultrasound, clinicians rely on a range of complementary tools to estimate volume status, each with distinct strengths and important limitations. Physical examination remains universally accessible, and certain components, such as capillary refill time, have experienced renewed interest in recent years.[7] Capillary refill offers a rapid, inexpensive bedside estimate of peripheral perfusion; however, inconsistent measurement techniques and the absence of a uniform definition of normal have limited its clinical reliability and interpretability. More broadly, heavily relied-upon physical examination findings such as jugular venous pressure and pulmonary crackles have limited sensitivity and frequently fail to identify clinically significant congestion. Daily weights and intake-output records provide longitudinal trends but neither reflect compartmental fluid shifts nor escape the limitations of imperfect documentation. Bioimpedance techniques estimate total and extravascular body water but cannot reliably distinguish intravascular volume from interstitial or third spaced fluid, nor do they inform hemodynamic tolerance. Continuous hematocrit monitoring during renal replacement therapy reflects relative changes in intravascular volume but offers no insight into tissue congestion or cardiac reserve. Even invasive hemodynamic monitoring yields pressures and flows without clarifying where excess fluid resides or how well it is tolerated at the organ level. Advanced technologies aimed at assessing tissue perfusion, such as near infrared spectroscopy for direct measurement of tissue hemoglobin oxygen saturation and visualization of the sublingual microcirculation using polarized or darkfield microscopy, are not widely available and currently remain largely confined to research settings. More importantly, *each modality illuminates a different facet of volume physiology, but none resolves the problem in isolation.* The limitation is not primarily technological but conceptual. Volume status emerges from the dynamic interplay of cardiac function, vascular tone, venous compliance, and tissue characteristics. Any approach that compresses this complexity into a single metric will inevitably misclassify some patients.

In this context, POCUS is best understood not as a standalone solution but as a modality that helps reconcile discordant signals. Its value lies in bedside pattern recognition and physiologically grounded hypothesis testing, integrated with clinical history, laboratory data, and other available information. Ultimately, the assessment of volume status in nephrocritical care remains an art informed by science. Precision does not arise from pursuing a single metric, whether derived from ultrasound, invasive monitoring, or laboratory values. It emerges from synthesizing multiple strands of data within the unique clinical context of the individual patient. POCUS does not eliminate uncertainty; rather, when anchored in physiology and used alongside traditional assessment, it enables clinicians to navigate that uncertainty with greater clarity and humility.

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