How I Do It

Risks and Benefits of Fluid Administration as Assessed by Ultrasound

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For patients in shock, decisions regarding administering or withholding IV fluids are both difficult and important. Although a strategy of relatively liberal fluid administration has traditionally been popular, recent trial results suggest that moving to a more fluid-restrictive approach may be prudent. The goal of this article was to outline how whole-body point-of-care ultrasound can help clarify both the possible benefits and the potential risks of fluid administration, aiding in the risk/benefit calculations that should always accompany fluid-related decisions.

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KEY WORDS: medical education; resuscitation; shock; ultrasound

For patients in shock, especially septic shock, decisions regarding IV fluid administration are both important and difficult. Fundamental practices such as early antibiotic administration and infectious source control have become widespread and consistent, and thus physicians caring for patients with sepsis are often left to struggle primarily with decisions regarding how much fluid to administer. Although a strategy of relatively liberal fluid administration was popular in previous decades, recent trial results suggest a more fluid-restrictive approach as the potential harms of over-resuscitation have become more apparent.[1]

The potential benefits of IV fluids are generally well understood. For patients in shock (defined as a state of inadequate oxygen delivery to meet cellular metabolic needs) who are also fluid responsive (FR), defined as a state in which the administration of a fluid bolus will increase stroke volume (SV),[2,3] there is a clear potential benefit to fluid administration.[4] Cardiac output can be increased, and oxygen delivery thereafter improved. Although a state of shock is relatively easy to diagnose by physical examination and laboratory assessment, an FR state is not. This situation is aggravated by the fact that critically ill patients consistently have a near "perfect" 50% probability of being in an FR state, indicating that we are typically operating in a zone of complete uncertainty.[5]

In the face of uncertain potential benefit, it becomes particularly important to consider potential harm. Fluid overload may cause deleterious effects in multiple organ systems, including pulmonary edema, renal dysfunction, intraabdominal hypertension, delirium, cerebral edema, and impaired

ABBREVIATIONS: CFTI = carotid flow time index; CSA = cross-sectional area; FR = fluid responsive; IVC = inferior vena cava; LV = left ventricular; LUS = lung ultrasound; LVOT = left ventricular outflow tract; POCUS = point-of-care ultrasound; PLR = passive leg raise; RV = right ventricular; SV = stroke volume; SVC = superior vena cava; VExUS = venous excess ultrasound; VTI = velocity time integral

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wound healing, among others.[6-8] Thinking more globally, a positive fluid balance has been associated with increased duration of mechanical ventilation, longer hospital stay, and higher mortality in several cohorts.[9-11] Despite this multitude of risks, current guidelines do not provide clear stopping points for fluid administration in sepsis and underemphasize the potential harms of over-resuscitation.[12]

Case Example

A 65-year-old man with hypertension, heart failure with preserved ejection fraction, and previous nephrolithiasis presents with fever, left-sided flank pain, and hypotension. His urinalysis is strongly positive for nitrites and leukocyte esterase, and his creatinine and lactate levels are elevated. The emergency physician has administered 3 L of IV balanced crystalloid and appropriate antibiotics, but the patient remains hypotensive, acidotic, and oliguric. Furthermore, the patient is tachypneic at 28 breaths/min and is now requiring 3 L of oxygen via nasal cannula to maintain an oxygen saturation of 93%. Should more fluid be administered?

General Advantages of Ultrasound

Even for those well versed in the dangers of excessive fluid administration, determining how much fluid is too much is complex. Volume status is extremely challenging to assess by physical examination, and although invasive hemodynamic monitoring could theoretically be helpful, these tools are not available to all patients and are not without their own inherent flaws and risks. Point-of-care ultrasound (POCUS), conversely, allows for the examination of multiple organ systems, noninvasively, in real time, to be integrated with other clinical parameters, and repeated serially.

Basic Principles

All of the techniques described in the following sections assume proficiency in basic critical care ultrasound, and a few require a more advanced skill set, including use of spectral Doppler.[13] As with all tools, these techniques perform optimally when used in combination and when integrated with other salient clinical parameters; each has multiple pitfalls and caveats. For each of the four techniques related to potential fluid benefit and the five

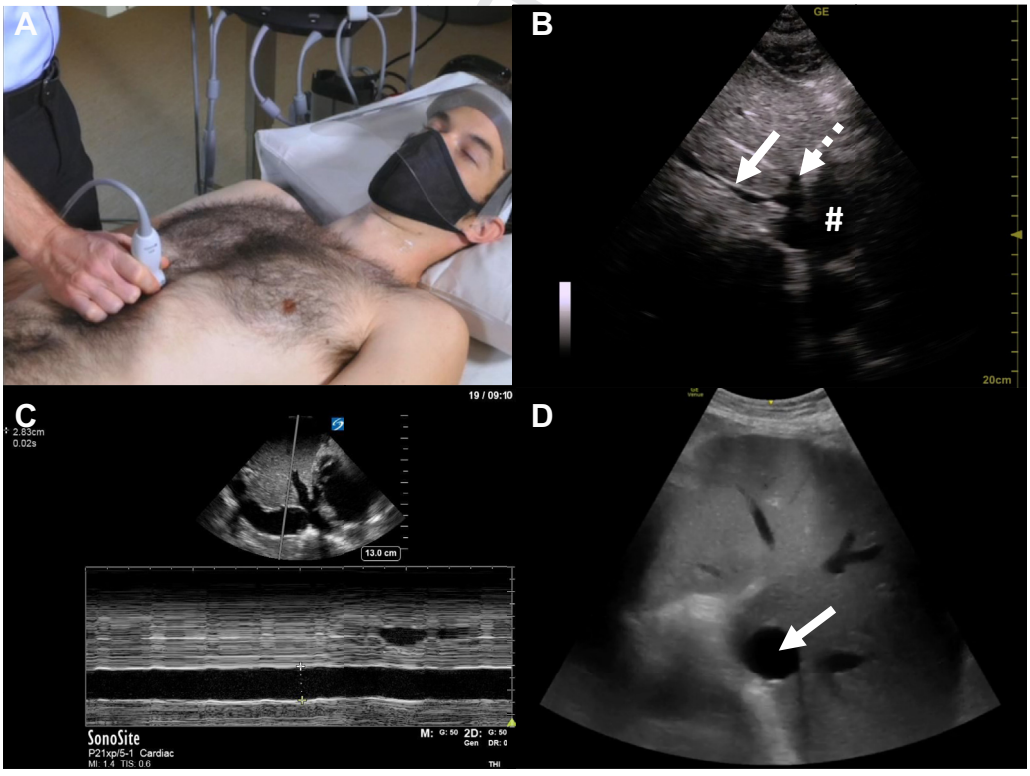

Figure 1 – *A-D, Analysis of the inferior vena cava (IVC) for fluid responsiveness. A, Typical transducer position to assess the long-axis view of the IVC; here a phased-array transducer is held just below the xiphoid process with the orientation marker oriented toward the patient's head. B, Long-axis view of a thin IVC (arrow) in B-mode; the hepatic vein (dashed arrow) and right atrium (number sign) can also be seen. C, Long-axis view of a dilated IVC, with the end-expiratory diameter (here 28 mm) measured in M-mode. D, Short-axis view of the IVC (arrow), which can be helpful with erroneous measurements due to foreshortening of the vessel.*

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related to fluid risk, the relevant associated figure and video should be consulted for a detailed technical description of how to perform the maneuvers. Some patients may not fall neatly into either category (of being either FR or fluid overloaded), and both states can exist simultaneously. These cases are particularly difficult, and here an informed risk/benefit calculation regarding fluids is both challenging and valuable.

Fluid Benefit Techniques: Assessment of Fluid Responsiveness

Technique #1: Analysis of the Inferior Vena Cava: The size and variability of the inferior vena cava (IVC) have both been advanced as tools to help in determining FR (Fig 1, Video 1). Smaller IVC size is believed to make an FR state more likely and a larger size less so, but exact cutoffs have been challenging to identify. Similarly, a higher degree of IVC collapsibility (in spontaneously breathing patients) or distensibility (in patients who are mechanically ventilated) has been proposed to be associated with FR. However, the evidence supporting IVC analysis for FR is controversial, complex, and subject to interpretation; it has been reviewed in detail elsewhere.[14] For patients who are spontaneously breathing and those who are mechanically ventilated, many small, single-center studies have yielded contradictory results; recent meta-analyses have yielded variable but generally unsupportive results.[15] Perhaps most importantly, the largest single study to date (by a very large margin) was unsupportive of both IVC size and variability for determining FR.[16] In addition, there are numerous technical challenges associated with measuring the IVC accurately, including known inter- and intra-rater variability of measurements and a series of common confounding factors, including right ventricular (RV) dysfunction and intraabdominal hypertension.[14]

Given the challenges and caveats noted here, use of the IVC for determining FR must be approached with significant caution. As with many tests in medicine, it is likely to be most useful at extremes. Although the IVC may provide useful information as part of an integrated volume assessment, it is essential that the provider be well versed in the test characteristics and limitations, and that the data be considered as one piece of a holistic POCUS and clinical assessment.[17,18]

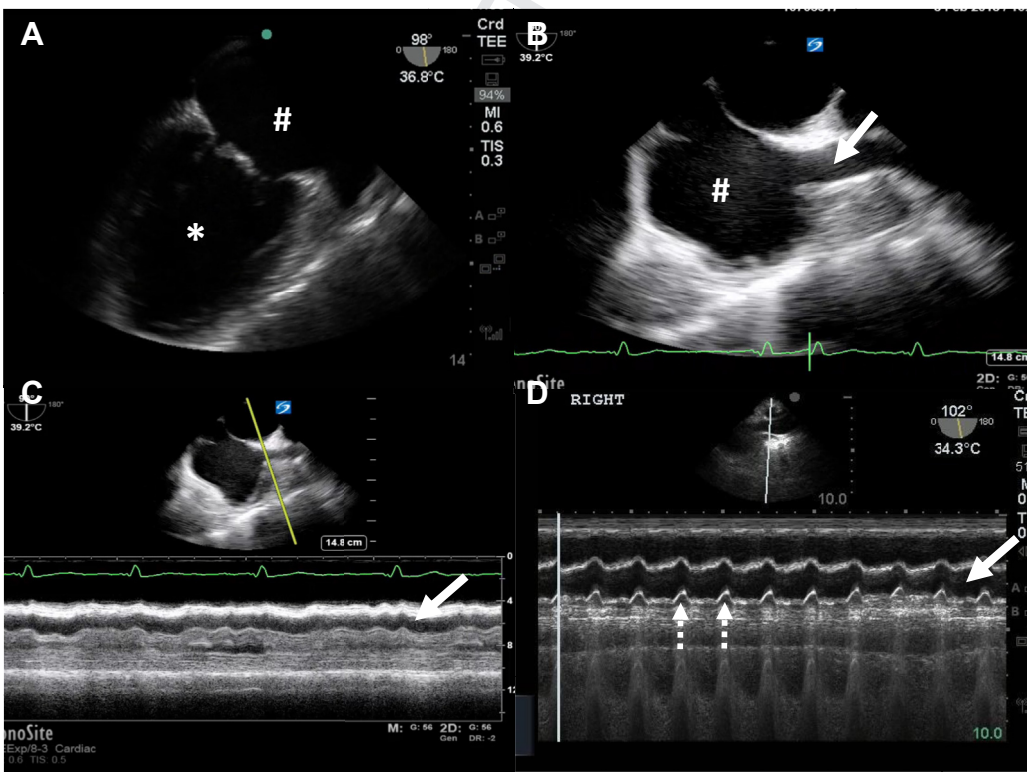

Figure 2 – *A-D, Analysis of the superior vena cava (SVC) for fluid responsiveness. A, From the starting mid-esophageal four-chamber view (not shown), the omniplane angle is rotated to approximately 90° (here 98°) to achieve a mid-esophageal two-chamber view; the left ventricle (asterisk) and left atrium (number sign) are seen. B, From the previous mid-esophageal two-chamber view, the physical transducer is rotated clockwise until the bi-caval view is achieved, showing the SVC (arrow) connecting to the right atrium (number sign). C, M-mode analysis of a small, collapsible SVC (arrow). D, M-mode analysis of a large, noncollapsible SVC (arrow); note the regular pulsatility here (dashed arrows) represents cardiac contractility, not respiratory variation.*

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Technique #2: Analysis of the Superior Vena Cava: Assessing respiratory variation in the size of the superior vena cava (SVC) is analogous to analyzing the IVC and offers a mix of advantages and disadvantages (Fig 2, Video 2). Its major disadvantage is that it requires a transesophageal approach and is therefore generally reserved for patients who are already intubated. It is an invasive technique, albeit with an excellent associated safely profile,[19] and one requiring a skill set that is less commonly available.[20]

On the positive side, use of the SVC avoids all confounding elements associated with changes in intraabdominal pressure and can be used in patients with irregular cardiac rhythms. In addition, given that SVC is almost universally deployed in patients who are intubated and sedated, concerns regarding spontaneous respiratory efforts are generally absent. Where studied, assessment of the SVC for respiratory variability performs better than the IVC, although the amount of data is limited.[16,21] A change in the size of the SVC of $> 36\%$ is a commonly cited cutoff,[21] although lower values have also been proposed.[16]

Technique #3: Measurement of Left Ventricular Outflow Tract Velocity Time Integral: The velocity time integral (VTI) can be measured via a transthoracic approach at the level of the left ventricular outflow tract (LVOT) (Fig 3, Video 3). If one imagines a patient's SV as a cylinder of blood passing through the aortic valve with each cardiac contraction, the VTI is the height of that cylinder. Because the cross-sectional area (CSA) of the cylinder at the level of the LVOT is essentially a fixed value, the SV is perfectly correlated to the height of the cylinder, the VTI. SV as estimated in this manner has been shown to correlate well with other established techniques such as Swan-Ganz catheterization. Once the VTI has been measured, several useful FR techniques can be deployed[22]; this technique, therefore, is better supported by evidence than many of the other tools described in this article. This tool can be applied to patients in atrial fibrillation, but an average of multiple measurements must be used to avoid overestimation or underestimation.

A baseline measure of VTI can be followed by a dynamic maneuver to predict an FR state. If the patient's VTI increases with passive leg raise (PLR), they are likely to

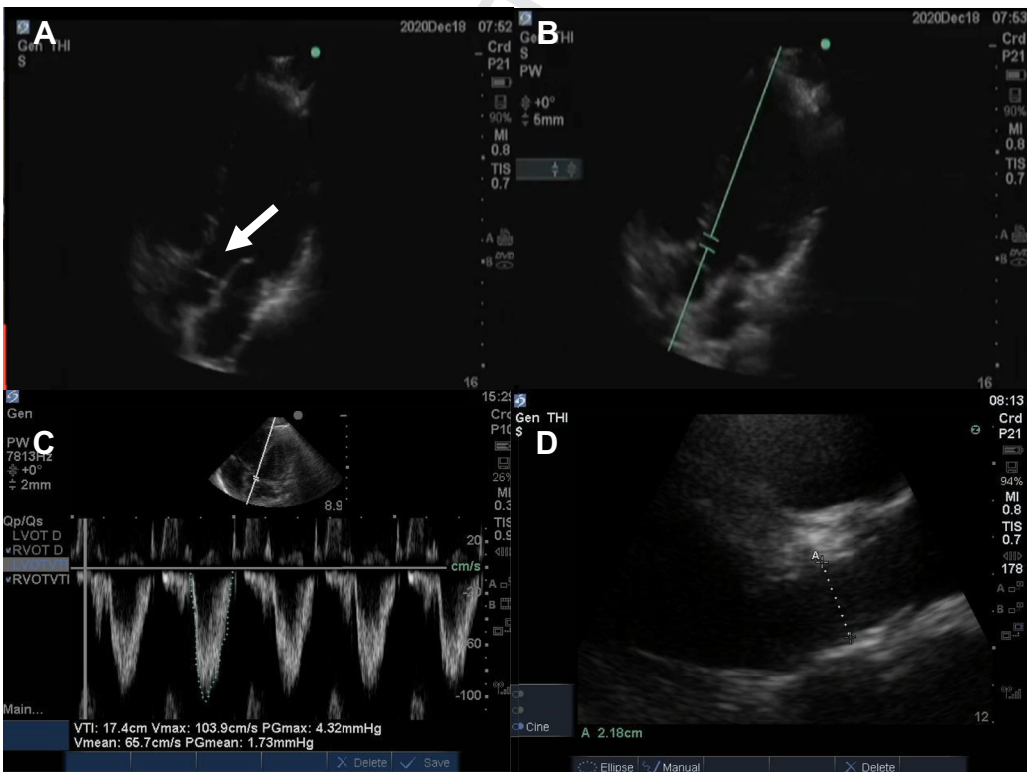

Figure 3 – *A-D, Measurement of left ventricular outflow tract (LVOT) velocity time integral to help in determining fluid responsiveness. A, Apical five-chamber view, with visualization of the LVOT (arrow). B, Pulsed wave Doppler interrogation of the LVOT. The interrogation window is placed just above the aortic valve, and the line of interrogation is positioned parallel to the long-axis of the LVOT itself. C, Measuring the area under the curve of the LVOT Doppler waveform to derive the velocity time integral (here 17.4 cm). D, Diameter of the LVOT, measured from a parasternal long-axis view (zoomed-in; here 2.18 cm).*

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be FR; a cutoff of 10% or 15% is typically used.[5] When PLR is contraindicated and the risks of fluid administration are high, a change in VTI after 50 mL of crystalloid is rapidly infused over 10 s or a VTI difference noted between an end-inspiratory and end-expiratory hold can also be used to test the effect of alterations in preload to predict fluid responsiveness,[23,24] although these newer techniques can be technically challenging to perform and have not yet been robustly studied. Otherwise, the VTI can be measured prior to and following a fluid bolus (generally 500 mL). If the VTI increases (again a cutoff of either 10% or 15% is typically used), this action suggests that the patient was in an FR state, and the process can be repeated until the VTI no longer increases with a bolus.

A related technique involves using the change in the maximum velocity of blood flow at the LVOT with respiration. This tool is analogous to systolic pressure variation as typically measured by using an arterial catheter and can be of interest in patients who do not have an invasive BP-monitoring device.

If an estimate of SV is wanted, the diameter (d) of the LVOT is measured from a parasternal long-axis view, and this value is used to calculate the CSA:

$$CSA = \pi \left(\frac{d}{2}\right)^2$$

The CSA is then multiplied by the VTI to yield an estimated SV:

$$SV = CSA \times VTI$$

Technique #4: Estimation of Fluid Responsiveness Via Carotid Flow Time: Although using LVOT VTI to estimate SV is a very well-established and validated technique, it can be time-consuming (Fig 4, Video 4). As such, efforts have been made to identify more straightforward techniques that are not as technically difficult. One such option is the corrected carotid flow time index (CFTI), a noninvasive surface measurement of systolic blood flow at the level of the carotid artery, a structure that is typically easy to image.[25]

Here, a pulsed-wave Doppler waveform of carotid blood flow is generated, and the flow time between the onset of

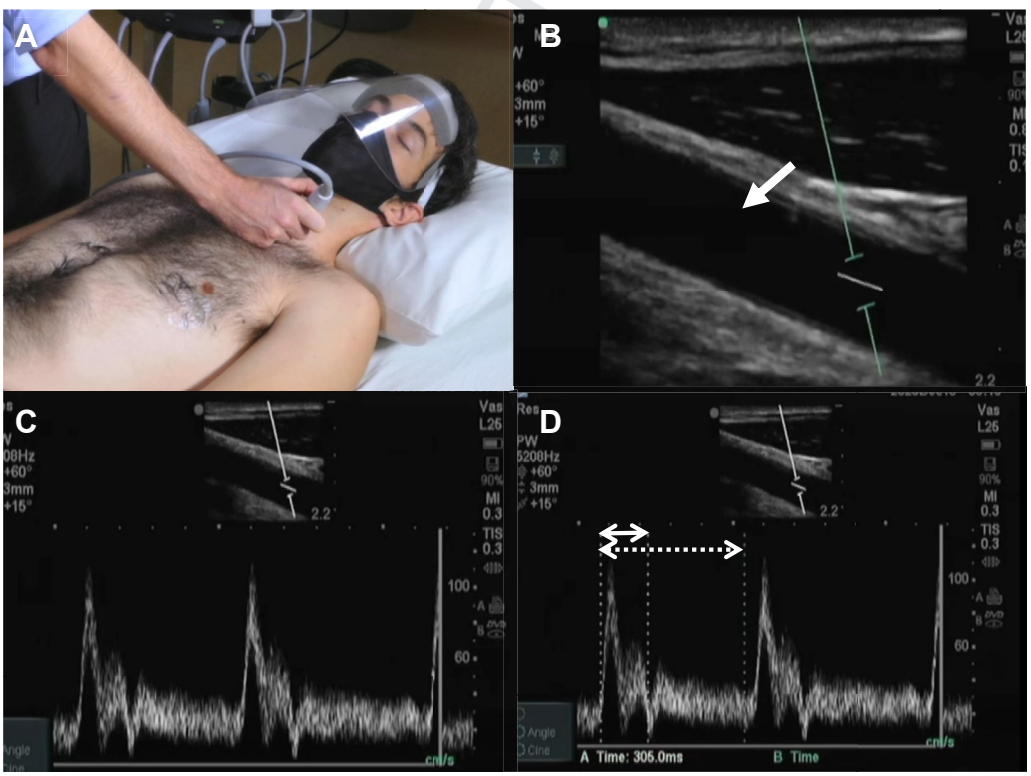

Figure 4 – *A-D, Estimation of fluid responsiveness by using carotid flow time measurement. A, Transducer position for carotid analysis. The linear transducer is placed at approximately the level of the thyroid cartilage, with the orientation marker pointed toward the patient's head. B, Carotid artery (arrow) in long-axis. The transducer has been angled to avoid the artery laying perfectly horizontally, the Doppler interrogation line (green) has also been angled to make it more parallel to the long-axis of the artery, and angle correction software (white line) has been applied. C, Carotid artery Doppler waveform. D, Measuring the systolic time (solid arrow; here 305 milliseconds) and total cycle time (dashed arrow).*

systole and the closure of the aortic valve (the dicrotic notch) is measured, as is the duration of the full cardiac cycle. The value of the CFTI is calculated as:

$$CFTI = \frac{Systolic\ flow\ time}{\sqrt{Cardiac\ cycle\ time}}$$

Once this value is obtained, a PLR maneuver may be performed, and the CFTI is re-measured. An FR state would be associated with an increase in the CFTI value by virtue of a longer systolic flow time, due to a slightly longer time required to eject the additional blood. This time is "corrected" for any changes in heart rate by indexing it to the total cycle time.[25] Administration of an actual fluid bolus could be substituted for a PLR, analogous to the process described earlier for the LVOT VTI. The primary difficulty lies in determining the ideal cutoff value to determine a positive test; when a relatively high value is used (eg, a change in CFTI of 25% following PLR[26]), it results in a very high specificity but a low sensitivity. Cutoff values of 10% to 15% are more typical.[25,27,28] There are a paucity of trials assessing the performance of carotid flow time measurements,[27] and more research is required.

Fluid Risk Techniques: Assessment of Venous Congestion

Technique #5: IVC Size and Variability: The evaluation of the IVC for the purpose of determining FR was discussed earlier. On the flip side of this coin, a large, static IVC may suggest congestion and make harm from fluid administration more likely, but precise cutoffs vary widely[29] (Fig 5, Video 5). A modest correlation exists between larger IVC size and central venous pressure; elevated central venous pressure, in turn, is associated with worse outcomes.[11,30,31] A plethoric IVC is also a prerequisite for solid organ assessment of venous congestion, as described later in Techniques 8 and 9.

In spontaneously breathing patients, a dilated IVC without respiratory variation may point to venous congestion, but this does not necessarily apply to patients who are mechanically ventilated, particularly those with high positive end-expiratory pressures.

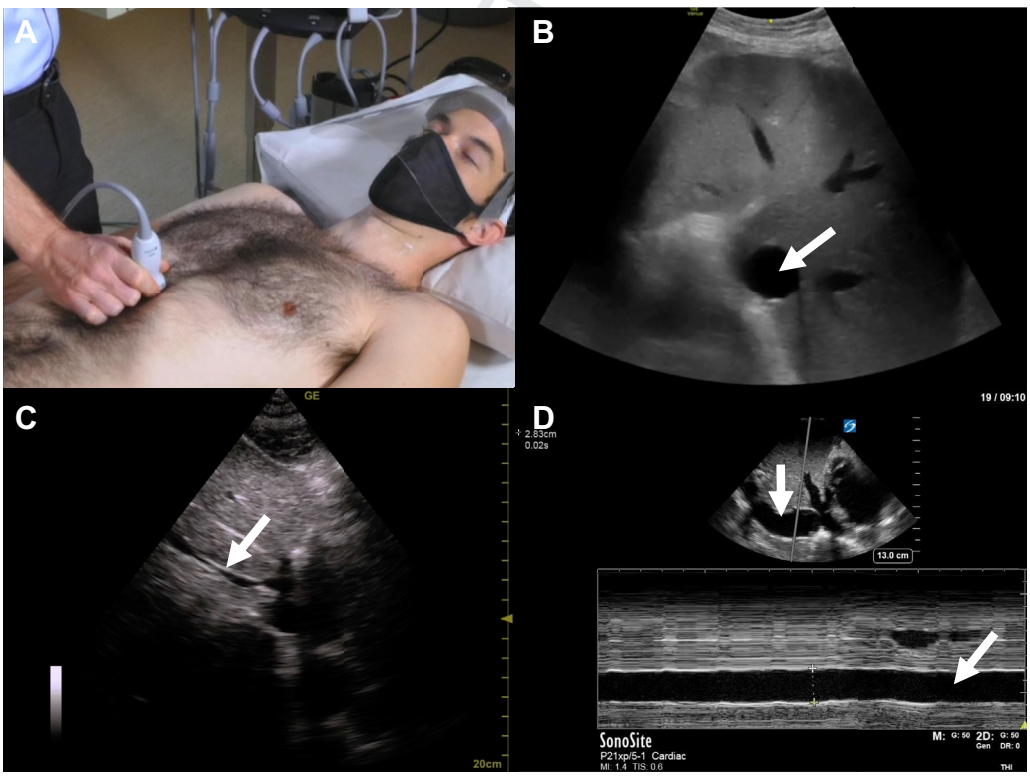

Figure 5 – *A-D, Analysis of inferior vena cava (IVC) size and variability for venous congestion. A, Typical transducer position to assess the long-axis view of the IVC; here a phased-array transducer is held just below the xiphoid process with the orientation marker oriented toward the patient's head. B, Long-axis view of a thin IVC (arrow) in B-mode; the hepatic vein (dashed arrow) and right atrium (number sign) can also be seen. C, Long-axis view of a dilated IVC, with the end-expiratory diameter (here 28 mm) measured in M-mode. D, Short-axis view of the IVC (arrow), which can be helpful with erroneous measurements due to foreshortening of the vessel.*

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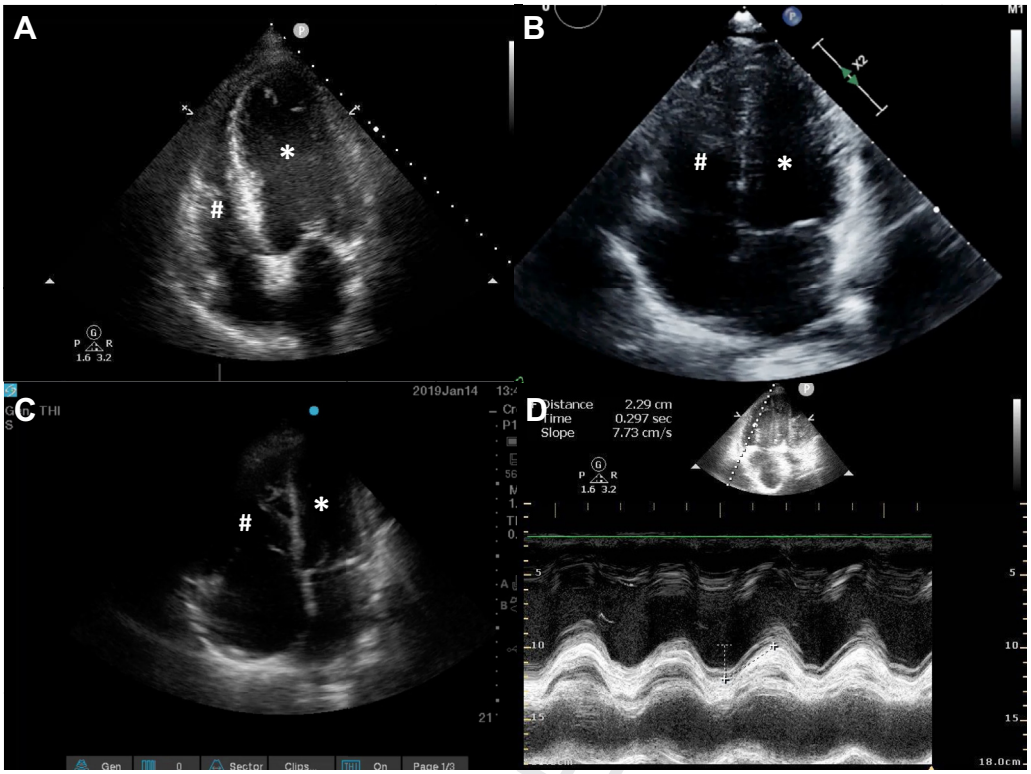

Figure 6 – *A-H, Right ventricular assessment. A, Normal right ventricular size, apical four-chamber view; the right ventricle (RV) (number sign) is smaller than the left ventricle (asterisk). B, Moderately dilated RV, apical four-chamber view; the RV (number sign) is roughly the same size as the left ventricle (asterisk). C, Severely dilated RV; apical four-chamber view. The RV (number sign) is significantly larger than the left ventricle (asterisk). D, Normal tricuspid annular plane of systolic excursion (here approximately 23 mm), apical four-chamber view. E, Abnormal tricuspid annular plane of systolic excursion (here approximately 11 mm), apical four-chamber view. F, D-shaped septum (arrow), parasternal short-axis view. G, Normal right ventricular free wall thickness (between arrows; here approximately 5 mm), sub-xiphoid four-chamber view. H, Abnormal right ventricular free wall thickness (between arrows; here approximately 18 mm), sub-xiphoid four-chamber view.*

Technique #6: RV Assessment: Unlike the left ventricle, the right ventricle is thin-walled and ill-equipped to deal with acute increases in pressure or volume, whether due to fluid administration or acute insults such as hypoxemia or pulmonary embolism (Fig 6, Video 6). Ultrasound evaluation of the right ventricle, therefore, plays a key role in determining potential harms from fluid therapy. A dysfunctional right ventricle will respond poorly to additional volume with further dilation, worsening systolic function, and decreased stroke volume, and will subsequently impair LV filling. This can lead a deadly spiral of systemic hypotension, right ventricular (RV) ischemia, and further worsening RV function.[32] Video 6 describes techniques for evaluating RV size and function.

The primary caveat with RV assessment as it relates to the decision to give or withhold IV fluids is that it is very challenging, and often impossible, to determine the chronicity of RV changes. Patients with chronic RV pathology and elevated right-sided pressures may tolerate and even potentially benefit from fluids despite a dilated right ventricle, as they have had the opportunity to adapt over time and shift their individual Frank-Starling curves. RV hypertrophy can be helpful in signaling a degree of chronicity, although it remains challenging to exclude an acute-on-chronic insult. Analogously, high systolic pulmonary artery pressures (a cutoff of 60 mm Hg is often used) suggest a more chronic process. It is conceptually helpful, in these cases, to return to the principle of a risk/benefit analysis: in the presence of an impaired right ventricle, the potential risk of IV fluids is undoubtedly higher, and thus the perceived benefit would need to be more substantial to justify a trial of fluid therapy.

Finally, it should be noted that other cardiac ultrasound techniques may help inform the potential risks of fluid therapy. In particular, the evaluation of LV systolic and diastolic function may help clarify how wide or narrow a therapeutic window may be present; a heart with significant systolic and/or diastolic dysfunction will more quickly exhibit signs of organ congestion and fluid harm.

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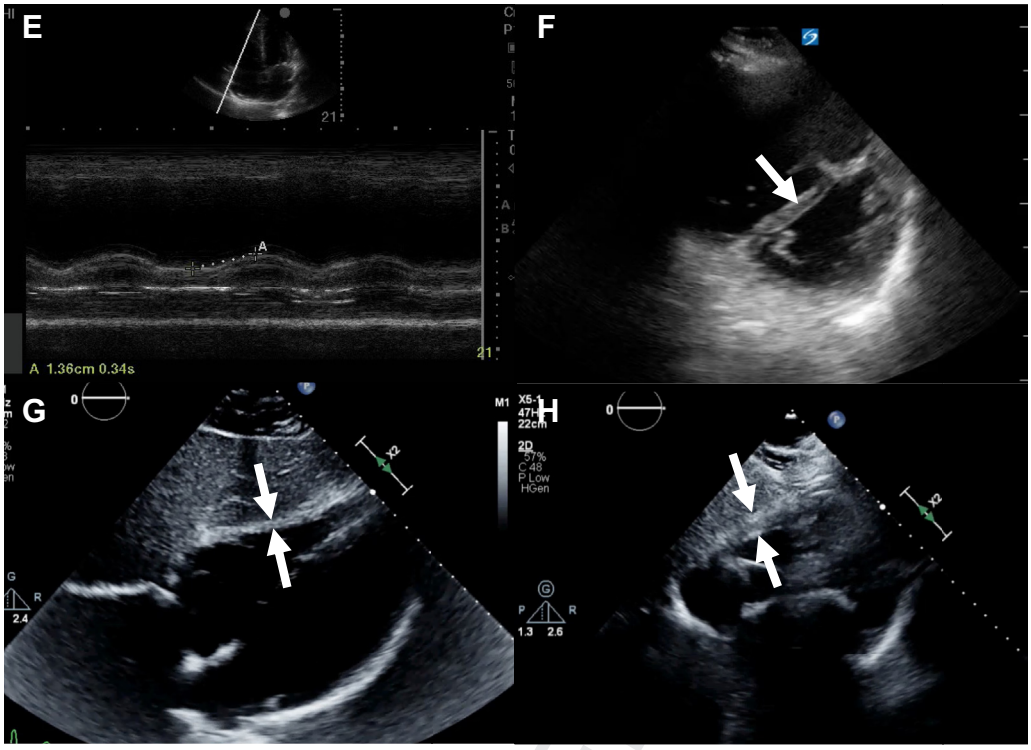

Figure 6 – *Continued.*

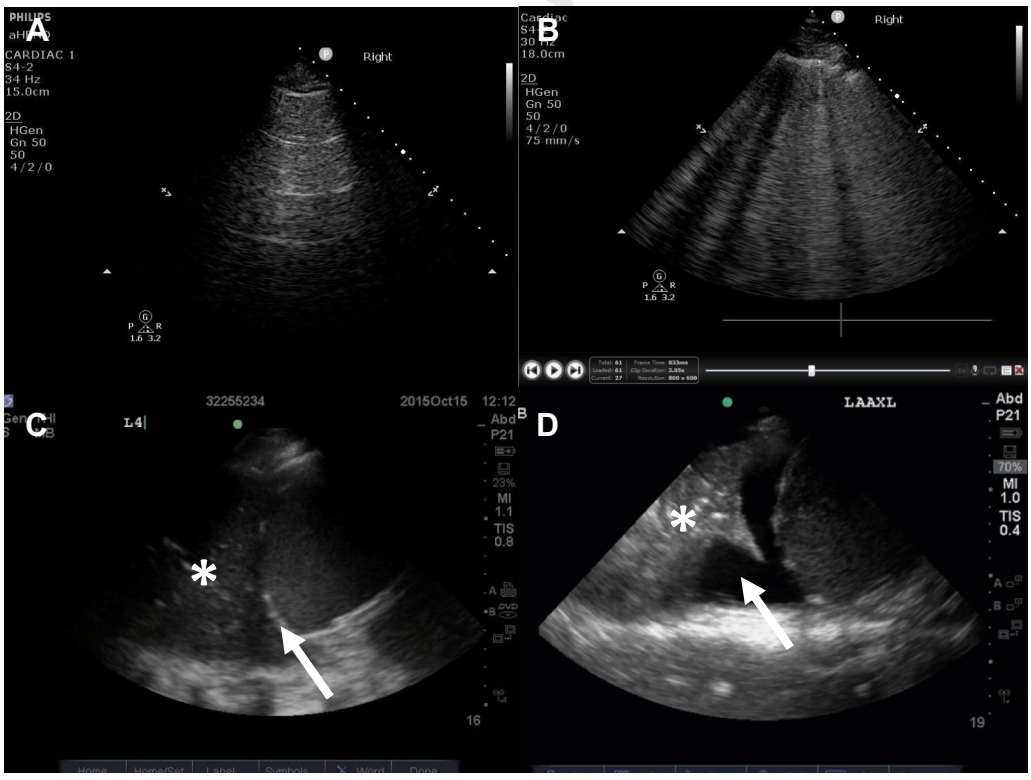

Figure 7 – *A-D, Thoracic ultrasound for lung congestion. A, Typical A-lines, here generated with the phased-array transducer oriented perpendicular to the rib and placed at the level of the second intercostal space in the mi-clavicular line. B, Typical B-lines; here the transducer is in the same position but oriented parallel to the ribs. C, Lung consolidation (asterisk) above the diaphragm (arrow). D, Pleural effusion (arrow) with associated consolidated lung (asterisk).*

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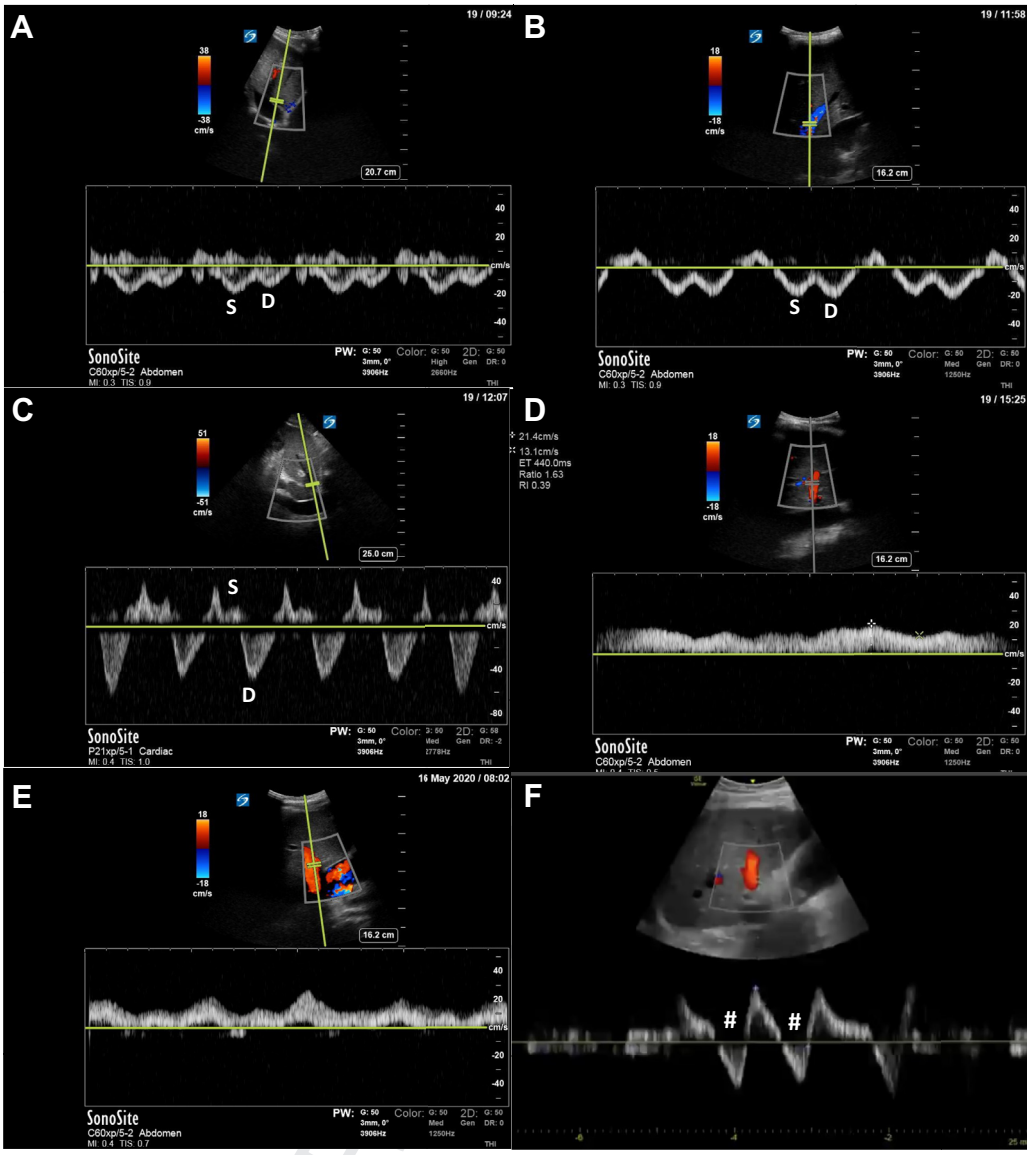

Figure 8 – *A-F, Hepatic and portal vein assessment for congestion. A, Normal hepatic vein Doppler waveform. The S-wave is larger than the D-wave. B, Abnormal hepatic vein Doppler waveform. The D wave is larger than the S wave. C, Very abnormal hepatic vein Doppler waveform; S-wave reversal. D, Normal portal vein Doppler waveform; continuous hepato-petal flow with low variability. E, Abnormal portal vein Doppler waveform; increased pulsatility. F, Very abnormal portal vein Doppler waveform; absent diastolic flow (number sign).*

A detailed description of the techniques used to evaluate the left heart is beyond the scope of the current article.

Technique #7: Thoracic Ultrasound for Lung Congestion: Lung ultrasound (LUS) is a well-established tool for the detection of extravascular lung water, displaying excellent test characteristics and easily outperforming chest radiograph, with a reported sensitivity of 88% and specificity of 90% in a meta-analysis[33] (Fig 7, Video 7). The LUS examination for pulmonary edema is centered on the detection of B-lines: these well-defined, hyperechoic, vertical ultrasound artifacts originate from the pleural line and vary with respiration, extend the length of the ultrasound screen, and obliterate horizontal A-lines. Other features that may be suggestive of fluid overload include pleural effusions, particularly if bilateral. Specific LUS scanning protocols vary, and the precise protocol is less important than obtaining a representative sample of the upper, middle, and lower aspects of both lungs.[34-36]

It is important to note that B-lines are reflective of an interstitial process and may be seen in a variety of conditions other than cardiogenic pulmonary edema, including pneumonia, interstitial lung disease, pulmonary hemorrhage, and non-cardiogenic

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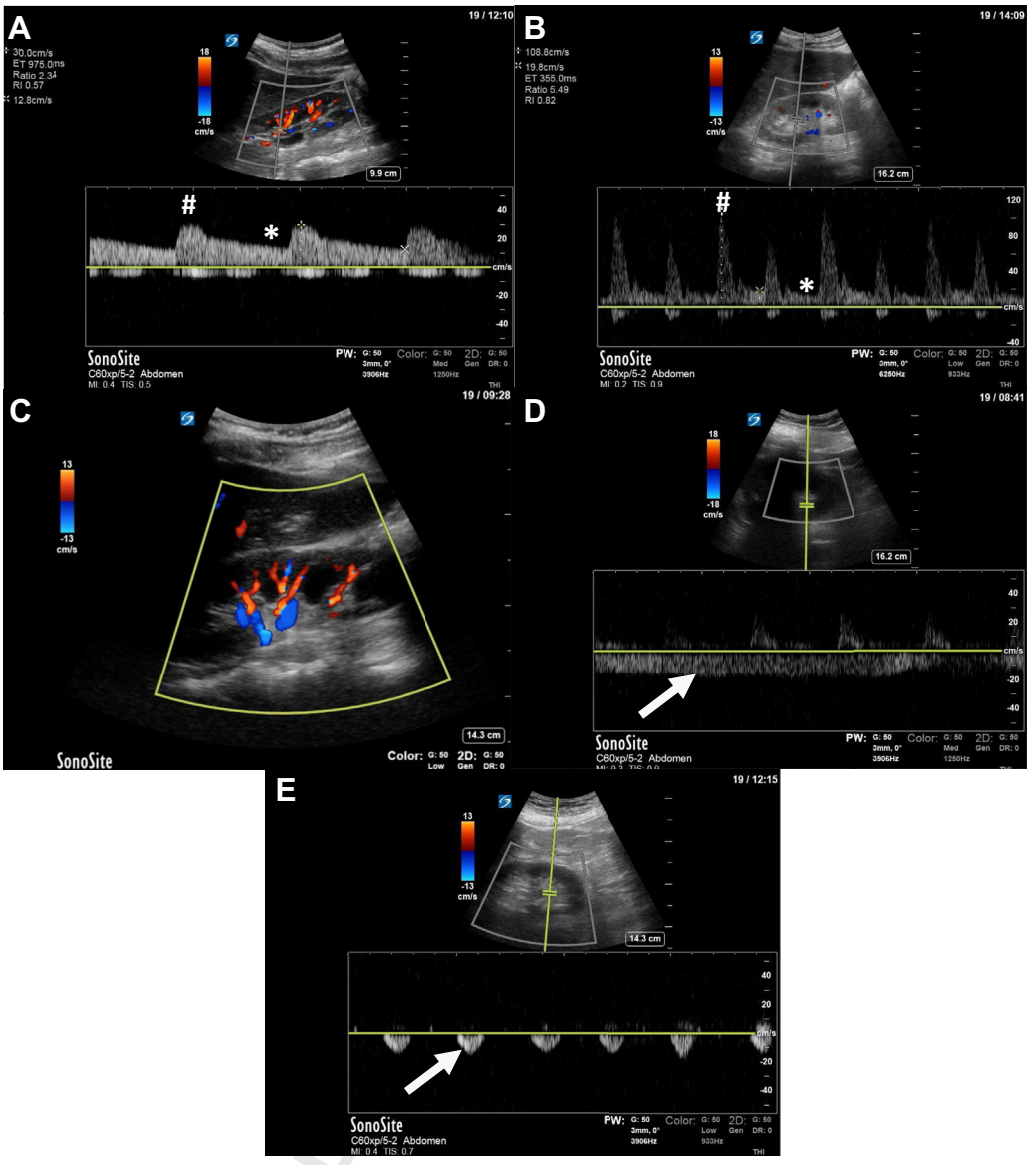

Figure 9 – *A-E, Renal vascular assessment for congestion. A, Normal intrarenal arterial Doppler waveform, with gentle systolic upstroke (number sign) and preserved end-diastolic flow (asterisk). B, Abnormal intra-renal arterial Doppler waveform with higher velocity in systole (number sign) and lower velocity in diastole (asterisk). C, Kidney in long-axis, with color Doppler to assist in locating vessels. D, Normal intra-renal venous Doppler waveform, with continuous flow below the baseline (arrow). E, Very abnormal intra-renal venous Doppler waveform, with monophasic waveform (arrow).*

pulmonary edema, among others.[37] B-lines consistent with pulmonary edema are typically diffusely symmetrical with a dependent gradient, arise from a smooth pleural line, and are often accompanied by small simple pleural effusions. The presence of B-lines consistent with pulmonary edema should be taken as an indication that the patient already has an elevated left atrial pressure and that further IV fluid will likely worsen pulmonary congestion. Worsening B-lines with further fluid therapy can reinforce this concept. Patients with B-lines from other causes, or with other evident pulmonary pathology such as consolidations or significant effusions, will also be at higher risk when receiving fluids, given their already deranged respiratory physiology.

Technique #8: Hepatic and Portal Vein Assessment for Congestion: Interrogating intraabdominal solid organ vessels with pulsed-wave Doppler to assess venous congestion is a relatively new and advanced application of POCUS (Fig 8, Video 8). Although based on solid physiological rationale, many unanswered questions remain, and the populations in which it has been studied

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are primarily limited to heart failure and cardiac surgery patients. Recently, a scanning protocol called Venous Excess Ultrasound (VExUS) has been proposed, and was found to be predictive of acute kidney injury in a postcardiac surgery population.[38] While waiting for further studies in broader patient populations, this tool can be considered a useful addition to the POCUS assessment for venous congestion, perhaps serving as an early warning sign for stopping further fluid therapy. It should be noted that these techniques, along with interrogation of the intrarenal vessels (Technique #9), are advanced applications and should be undertaken with appropriate training and an understanding of the pitfalls and caveats associated with each examination.

A technical description of hepatic venous Doppler analysis,[39] focusing on the S- and D-waves, is presented in Video 8. An explanation of portal venous Doppler analysis is also provided, with a focus on the pulsatility fraction (PF):

$$PF = \frac{maximum\ blood\ velocity - minimum\ blood\ velocity}{maximum\ blood\ velocity}$$

Given the caveats and potential confounders for both the hepatic and portal waveforms, they are best performed as a group, along with the intrarenal venous interrogation described in the following section, and interpreted holistically. Signs of congestion in multiple solid organ tracings paint a much stronger picture of congestion, which is a potential harm with additional fluid therapy, than a single abnormal waveform.

Technique #9: Renal Vascular Assessment for Congestion: The second half of the solid organ Doppler assessment for venous congestion can include evaluation of the intrarenal vessels[40,41] (Fig 9, Video 9). Although both the intrarenal arteries and intrarenal veins can be interrogated in a manner similar to the hepatic and portal veins, this is typically more challenging given the small size of the target vessels. Because of the lack of available evidence, they are talked about only briefly; Video 9 presents a discussion.

Doppler analysis of the renal arterial inflow is particularly discouraged, partly due to technical limitations common to critically ill patients, and partly due to the fact that measurements (most commonly the renal resistive index) can be confounded by intrinsic renal pathology. Renal venous Doppler, although also difficult, is at least a component of the aforementioned VExUS methodology.

Case Resolution

Ultrasound analysis revealed an IVC that measured 22 mm at end-expiration, with a 10% collapse on inspiration. Cardiac examination revealed a moderately dilated right ventricle with grossly normal function and a normally positioned interventricular septum. LVOT VTI was calculated prior to and following a PLR, with minimal (5%) change. LUS examination revealed

TABLE 1] Summary of Techniques for Determining Fluid Responsiveness

Technique	Key Point	Findings Suggestive of Fluid Responsiveness	Caveat
IVC	Potentially useful in extremes (eg, tiny IVC, very large IVC)	Small IVC (< 1.5 cm commonly used) High variability of IVC with respiration (> 50% commonly used)	Many technical challenges and confounding factors
SVC	Limited evidence suggests better performance than IVC	High variability of SVC with respiration (> 36% commonly used)	Required trans-esophageal echocardiography
VTI at LVOT	Correlates well with stroke volume as calculated by other methods	Significant change in VTI with passive leg raise or fluid bolus (> 10% commonly used)	Labor intensive to perform and repeat, more advanced skill set required
Carotid flow time integral	Generally easier to perform than LVOT VTI	Significant change in CFTI with passive leg raise or fluid bolus (> 10%-15% commonly used)	Poor evidence base, concerns about reproducibility

Relevant references are given in the text. CFTI = carotid flow time index; IVC = inferior vena cava; LVOT = left ventricular outflow tract; SVC = superior vena cava; VTI = velocity time integral.

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TABLE 2] Summary of Techniques for Assessing Potential Harms of Fluid Therapy

Technique	Key Point	Findings Suggestive of Congestion	Caveat
IVC	Potentially useful in extremes (eg, tiny IVC, very large IVC)	Large IVC (> 2.5 cm commonly used) Low variability of IVC with respiration (< 50% commonly used)	Many technical challenges and confounding factors
Cardiac ultrasound (focus on right heart)	Complex and somewhat subjective examination	Dilated RV Dysfunctional RV Shift in interventricular septum toward the left Low TAPSE	Difficult to separate acute from chronic findings
Lung ultrasound	Thorough examination covering upper, middle, and lower regions on both sides is essential	B-lines, especially worsening with fluids and in a pattern typical for cardiogenic pulmonary edema	Presence of B-lines is nonspecific
Hepatic and portal veins	Described as part of the VExUS examination	D wave > S wave (hepatic vein) Pulsatility fraction > 0.5 (portal vein)	Technically difficult, poor evidence base
Intrarenal vein	Described as part of the VExUS examination	Pulsatile, biphasic, and eventually monophasic renal vein flow	Technically very difficult, poor evidence base

Relevant references are given in the text. IVC = inferior vena cava; RV = right ventricle; TAPSE = tricuspid annular plane of systolic excursion; VExUS = venous excess ultrasound.

extensive B-lines present bilaterally with spared apices. The hepatic vein waveform was abnormal, exhibiting a D > S pattern. Portal venous assessment revealed increased pulsatility. Assessment of the renal parenchymal vessels was unsuccessful.

After repeating the patient's physical examination and laboratory assessment, the treating physician remained convinced that the state of shock had not resolved. Data derived from the ultrasound examination suggested that the patient was less likely to be FR, and, conversely, several features indicated a higher risk with additional fluid therapy. Consequently, the treating physician elected not to give further IV fluids, to begin vasoactive agents, and to reassess the situation frequently.

Conclusions

The appropriate titration of fluid therapy is one of the most challenging aspects of caring for acutely unwell patients. Given the increasing recognition of the harms associated with over-resuscitation, clinicians must be thoughtful in their prescription of IV fluids. Three questions should be addressed sequentially: First, is the patient in a form of shock, with evidence of end-organ hypoxia, that would benefit from an increased cardiac output to increase tissue oxygen delivery? Second, is the patient in a fluid-responsive state whereby the administration of IV fluids and preload augmentation will in fact result in an increased in cardiac output? Finally, is there evidence of multiorgan venous congestion suggesting that fluid therapy could cause the patient harm?

Overall, the decision to give IV fluids, like any decision in medicine, comes back to careful consideration of the possible risks and benefits. By using POCUS to better understand the potential harms associated with fluid administration, we can make more informed clinical decisions and improve the care of acutely unwell patients.

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