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OPINION REVIEW

Point of care venous Doppler ultrasound: Exploring the missing piece of bedside hemodynamic assessment

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Abstract

Accurate assessment of the hemodynamic status is vital for appropriate management of patients with critical illness. As such, there has been a constant quest for reliable and non-invasive bedside tools to assess and monitor circulatory status in order to ensure end-organ perfusion. In the recent past, point of care ultrasonography (POCUS) has emerged as a valuable adjunct to physical examination in various specialties, which basically is a clinician-performed bedside ultrasound to answer focused questions. POCUS allows visualization of the internal anatomy and flow dynamics in real time, guiding apt interventions. While both arterial (forward flow) and venous (organ outflow or afterload) limbs of hemodynamic circuit are important for tissue perfusion, the venous side remains relatively under-explored. With recent data underscoring the deleterious consequences of iatrogenic volume overload, objective evaluation of venous congestion is gaining attention. Bedside Doppler ultrasound serves this purpose and aids in diagnosing and monitoring the congestion/venous blood flow pattern. In this article, we summarize the rationale for integrating this technology into routine care of patients with volume-related disorders, discuss the normal and abnormal waveforms, limitations, and future directions.

Key Words: Ultrasound; Point of care ultrasonography; Doppler; VExUS; Congestion; Hemodynamics; Heart failure; Nephrology; Critical care

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Core Tip: Point-of-care Doppler ultrasonography is emerging as a valuable bedside diagnostic tool for the assessment of venous congestion. Doppler interrogation of the abdominal veins such as the hepatic, portal, renal parenchymal veins in addition to inferior vena cava ultrasound provides useful insights into a patient's hemodynamics, when interpreted in conjunction with other sonographic parameters such as the cardiac pump function, lung ultrasound and conventional clinical assessment.

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INTRODUCTION

Objective assessment of hemodynamic status is fundamental to guide resuscitative efforts in a critically ill patient[1]. Among the myriad of methods used at the bedside, only a few have stood the test of time. Capillary refill time and passive leg raise with non-invasive cardiac output (CO) monitoring can be counted amongst these as both strategies have been shown to improve relevant patient outcomes in controlled trials[2, 3]. The success of these assessments seems to rely on avoiding unnecessary fluid loading thus mitigating fluid overload, which has been increasingly linked to adverse patient outcomes[4].

Inevitably, intensive care unit physicians will encounter over-resuscitated patients as well as those presenting with pre-existing volume overload[5]. While avoiding further fluid loading is important, efforts to actively decrease extracellular volume (de-resuscitation) have been shown to lead to potentially beneficial clinical outcomes[6]. De-resuscitation is especially relevant for patients presenting with or developing heart failure during the course of their critical illness as volume overload in this patient population results in increasing severity of venous congestion[7]. Increased left-sided filling pressures facilitate lung congestion and lead to worsening respiratory status[8]. Less appreciated however, are the consequences of systemic venous congestion secondary to increased right-sided filling pressures. Increased right atrial pressure (RAP) can be transmitted backwards across the venous tree and lead to congestive organ dysfunction[9]. This can manifest as elevated bilirubin from congestive hepatopathy[10], delirium from congestive encephalopathy[11], acute oliguric kidney injury from 'intra-capsular tamponade'[7], and gut edema resulting in increased endotoxemia[12,13].

The degree of congestive organ dysfunction is not only a function of absolute RAP, but also depends on the degree of transmission of such pressure to the peripheral organs. Increased RAP becomes initially attenuated along the venous vascular tree as a consequence of venous distensibility[14]. However, progressive increases in venous volume will eventually result in maximally stretched venous walls reaching the flat part of the venous compliance curve; At this point, pressure transmission will be greatly enhanced leading to peripheral organ congestion. Because of this, assessing congestion at the level of the organs can provide valuable information regarding the mechanisms of organ dysfunction[15]. Given venous congestion results in altered patterns of organ venous flow, Doppler point-of-care ultrasonography (POCUS) allows quantification of these alterations at the bedside[16].

INFERIOR VENA CAVA AS THE FIRST STEP IN THE ASSESSMENT OF CONGESTION

Sonographic assessment of the collapsibility/distensibility of inferior vena cava (IVC) to predict volume responsiveness has several caveats and, in our opinion, should not be used for such purpose[17]. However, a plethoric (> 20 mm) non collapsible IVC is

not normal and will only be seen in patients with pathological venous congestion[18]. Evaluation of the IVC using POCUS is a well-accepted surrogate of venous congestion as it mainly reflects RAP; However, many factors influence IVC size and collapsibility such as respiratory effort in spontaneously breathing patients[19] and the presence of intra-abdominal hypertension[20]. Another problem is inherent to the conventional long axis view of interrogation; Given the IVC is a three-dimensional structure with elliptical shape, evaluation of diameters in both long and short axes has been shown to be a better estimate of central venous pressure (CVP)[21].

Although a plethoric non-collapsible IVC establishes the presence of venous congestion, this information alone is not always adequate to guide management for two important reasons: Firstly, obstructive pathologies acutely leading to venous congestion need immediate resolution by specific interventions that have nothing to do with extracellular volume (cardiac tamponade, tension pneumothorax, massive pulmonary embolism). In these cases, focused cardiac ultrasound is necessary to establish diagnosis and management[22]. The second reason is that certain cardiac pathologies (chronic severe pulmonary hypertension, right ventricular failure, severe valvulopathies, restrictive cardiomyopathy or constrictive pericarditis) require elevated RAP in order to maintain CO; as such, excessive volume removal targeting a normal IVC diameter and collapsibility is not in the best interest of these patients[23]. However, progressive volume overload beyond what is needed to maintain CO will lead to an excessive increase in RAP, which can be transmitted to peripheral organs resulting in their dysfunction[7]. Thus, in this particular setting, evaluating pressure transmission using Doppler ultrasonography is a valuable non-invasive adjunct to overall clinical assessment.

NORMAL VENOUS DOPPLER FLOW PATTERNS

To assess the venous system with Doppler ultrasound, it is important to understand that flow pattern is the main variable being measured. Flow is generated by a pressure differential between two points, given a relatively constant vessel diameter, this pressure differential will determine flow velocity. Equilibration of pressures will cause flow to cease. When assessing flow with pulsed wave Doppler ultrasound, the direction is represented by positive or negative deflections from the baseline, while speed will be represented by the deflection amplitude. If the flow moves away from the transducer, the image will show a negative deflection (analogous to 'blue' on color Doppler). A positive deflection will be seen if flow is directed towards the transducer (analogous to 'red' on color Doppler)[24]. The normal venous flow patterns are determined by the changes in RAP throughout the cardiac cycle and modified by venous compliance and distance from the heart[25]. Therefore, the flow patterns will be different depending on the site being evaluated. Normal waveforms can be pulsatile with discernable flow corresponding to the phases of cardiac cycle as in the case of hepatic vein (HV), or continuous as with portal and intra-renal veins. Moreover, respirophasic changes in amplitude can be demonstrated reflecting the increased venous return during inspiration in spontaneously breathing patients.

HV flow pattern

In a normal CVP trace, atrial systolic contraction results in a rise in RAP represented as the A-wave. After the tricuspid valve closes (C-wave), the right atrium relaxes, and the ventricular systole pulls down the annulus towards apex resulting in a fall of RAP represented as the X-descent. The RA filling from the venous system during ventricular systole causes a progressive rise in RAP and forms the V wave. The Y-descent is then caused by tricuspid valve opening.

Since HV directly joins the IVC, their flow pattern is a mirror reflection of RAP variations throughout the cardiac cycle. Normal HV flow pattern consists of a positive/retrograde wave (A) that represents atrial systolic contraction analogous to A-wave of the CVP, and two negative/antegrade systolic (S) and diastolic (D) waves that represent the X and Y-descents of the CVP, respectively. Since X-descent is deeper than Y-descent, the HV S-wave usually has a larger amplitude than the D-wave (S > D) [26]. Figure 1 illustrates the normal time-correlated electrocardiographic (ECG) findings, CVP tracing and HV Doppler waveform.

Intra-renal and femoral venous flow patterns

In more distal vascular beds such as intra-renal and femoral veins, the tracing will not directly reflect RAP variations; this is explained by the high compliance of the venous

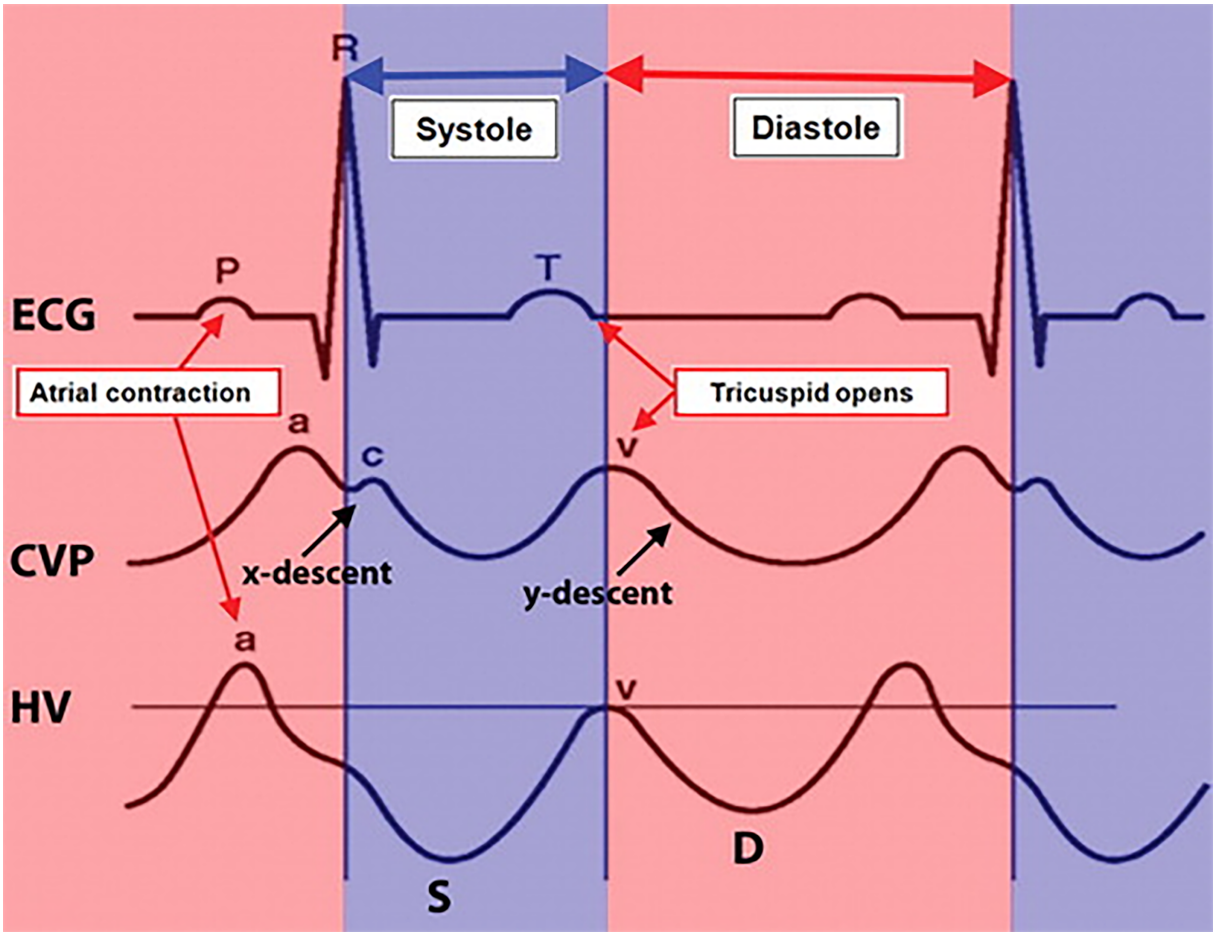

Figure 1 Normal time-correlated electrocardiographic findings, central venous pressure tracing, and hepatic venous waveform. The peak of the retrograde a wave corresponds with atrial contraction, which occurs at end diastole. The trough of the antegrade S wave correlates with peak negative pressure created by the downward motion of the atrioventricular septum during early to mid-systole. The peak of the upward-facing v wave correlates with opening of the tricuspid valve, which marks the transition from systole to diastole. The peak of this wave may cross above the baseline (retrograde flow) or may stay below the baseline (*i.e.*, remain antegrade). The trough of the antegrade D wave correlates with rapid early diastolic right ventricular filling. ECG: Electrocardiographic; CVP: Central venous pressure; HV: Hepatic venous. Citation: McNaughton DA, Abu-Yousef MM. Doppler US of the liver made simple. Radiographics 2011; 31: 161-188. Copyright © The Authors 2020. Published by Radiological Society of North America (RSNA®).

system and the attenuation of RAP variations with increasing distance from the heart. The flow pattern in normal distal veins will be predominantly continuous with no discernible waves, although low amplitude S and D-waves may be seen[27,28]. It is of note that the intra-renal Doppler is usually obtained at the level of interlobar vessels, which pass through the renal parenchyma and hence thought to better reflect organ perfusion compared to the main renal vein. Intra-renal venous trace is often accompanied by arterial trace above the baseline as the Doppler sample volume overlies both interlobar vein and artery, which are much smaller compared to other vessels such as HV.

Portal vein flow pattern

The portal vein (PV) is part of a distinct venous system that it is isolated from central veins by the hepatic sinusoids and from the arterial system by splanchnic capillaries. Therefore, the Doppler waveform of the normal PV will not reflect RAP variations unlike that of HV and appears as a characteristic positive (flow towards the transducer), continuous (or mildly pulsatile) flow[29].

ALTERED FLOW PATTERNS IN VENOUS CONGESTION

Hepatic vein Doppler alterations

When the RAP increases, the characteristic ascending and descending waves formed within the RA will change. As the RA filling pressure increases, the X-descent

decreases in amplitude while the Y-descent amplitude increases. This is due to loss of RA compliance and decreased right ventricular systolic pull of the tricuspid valve annulus. Right ventricular overload will eventually cause tricuspid annular dilation and tricuspid regurgitation, leading to obliteration of the X descent and fusion of C-V waves of the CVP waveform. All of this will be reflected in the HV flow; initially, the amplitude of the S-wave decreases compared to that of D-wave (S < D pattern)[30-32]. With worsening congestion, the S-wave can be obliterated or become reversed/retrograde if severe tricuspid regurgitation if present[33-35]. HV alterations have been shown to correlate with increased PV pulsatility, abnormal intra-renal venous flow and adverse kidney events including acute kidney injury (AKI) in recent studies[16,36-38].

Portal vein Doppler alterations

The main alteration in the PV waveform is progressive increase in pulsatility with elevated RAP. This can be quantified by the pulsatility fraction [(Vmax-Vmin/Vmax) × 100]; a pulsatility fraction ≥ 30% is considered mild elevation while ≥ 50% is considered severe. Further increases in RAP may lead to flow reversal (below the baseline) during systole[39-42]. The physiological explanation of pulsatility is the reduction of flow velocity during systole secondary to retrogradely transmitted waves from the right atrium during this phase of the cardiac cycle.

Most Clinical studies evaluating PV Doppler have been performed in the context of decompensated heart failure and cardiac surgery. PV pulsatility has been correlated with elevated RAP, clinical features of congestion[40], pulmonary wedge pressure, pulmonary artery resistance, right ventricular end diastolic pressure[39], left and right ventricle size[41], mean pulmonary artery pressure and peripheral vascular resistance [42]. Similar to HV, the recent focus has been to study the impact of PV pulsatility on clinical outcomes. In patients with decompensated heart failure, increased PV pulsatility was associated with worse clinical outcomes if present at discharge and predicted response to diuresis at admission[43,44]. In cardiac surgery patients, PV pulsatility was associated with congestive encephalopathy and delirium[11], AKI[45] and right ventricular dysfunction[46].

Intra-renal vein Doppler alterations

The intra-renal venous Doppler (IRVD) pattern is continuous, sometimes with a brief interruption during atrial systole. This pattern becomes biphasic as RAP increases and two distinct waves (S and D) can be observed. These waves are analogous to the normal hepatic waveform and represent increased pressure transmission from the heart to the interlobar renal veins[27]. With worsening congestion (intracapsular tamponade) the S-wave can either become reversed or disappear (obscured in the arterial trace). Though venous impedance index [(maximum flow velocity−minimum diastolic flow velocity)/maximum flow velocity] is frequently reported in studies to quantify renal venous pulsatility, pattern recognition described above is simpler. Moreover, when the waveform is discontinuous, the impedance index becomes 1 as the minimum velocity is zero and does not differentiate between biphasic and monophasic patterns. In this regard, renal venous stasis index (RVSI) proposed by Husain-Syed *et al*[47] better reflects the full continuum of renal congestion. It indicates the proportion of the cardiac cycle during which there is no venous outflow and is calculated as: Cardiac cycle time−venous flow time/cardiac cycle time. Therefore, monophasic pattern has a higher RVSI than biphasic pattern.

Multiple studies have shown that IRVD alterations are not merely a reflection of increased RAP, but also strong predictors of adverse clinical outcomes in patients with compensated[27] and decompensated heart failure[48], those undergoing cardiac surgery[45], patients with pulmonary hypertension and right heart failure[47].

In cardiac surgery patients, altered intra-renal Doppler pattern was shown to be a strong predictor of AKI. However, this was not replicated in less selected populations of critically ill patients[38,49]. Given the multitude of etiologies of AKI in addition to venous congestion (such as tubular injury) in such patients, this lack of association is not surprising. A visual summary of normal and altered venous flow patterns in the above-described veins is shown in Figures 2-4.

Femoral vein Doppler alterations

As opposed to intra-renal and PV, the common femoral vein is directly connected to the IVC facilitating the quick transmission of pressure waves as RAP increases. Flow in the normal femoral vein is relatively continuous with respiratory variability although a *low amplitude* positive/retrograde wave (A-wave) and anterograde S and D-waves maybe appreciated depending on the angle of insonation[28]. With elevations in RAP,

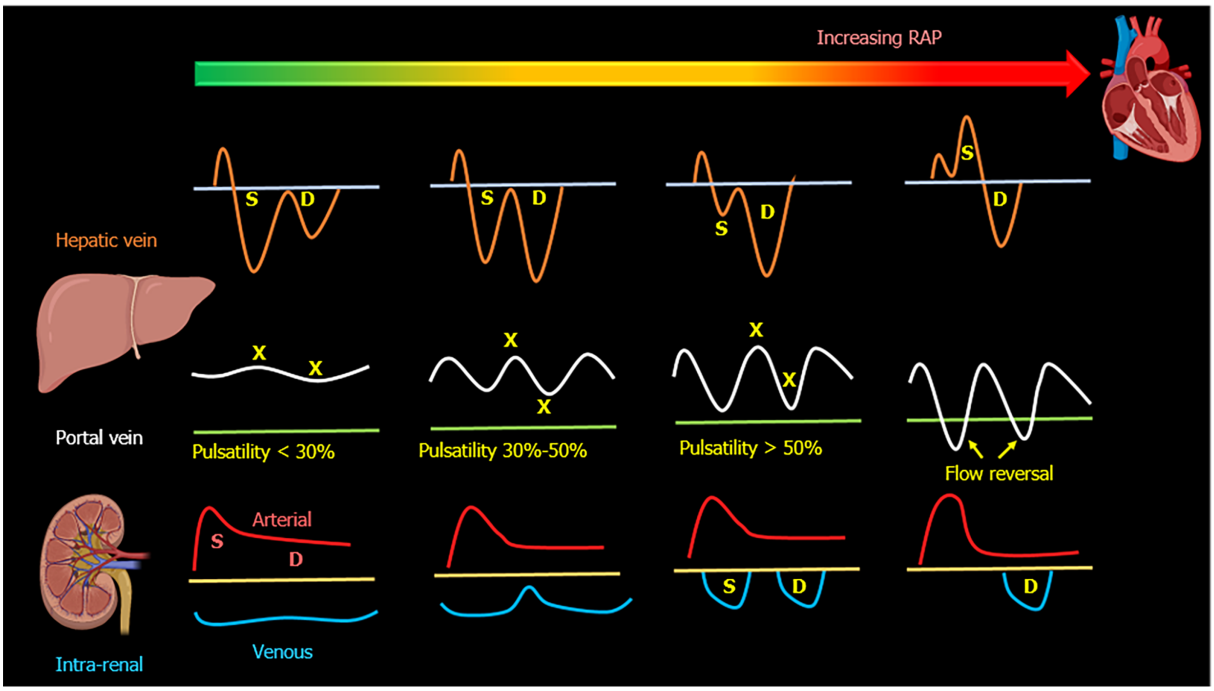

Figure 2 Transformation of the hepatic, portal, and intra-renal Doppler waveforms with increasing right atrial pressure. Asterisks on the portal waveform represent the highest and lowest points during a cardiac cycle used to calculate pulsatility fraction. RAP: Right atrial pressure.

the retrograde A-wave increases in amplitude or it will fuse with a reversed S-wave if severe tricuspid regurgitation is present. A retrograde wave velocity of ≥ 10 cm/s is considered abnormal and indicative of high RAP[50].

Few studies have integrated femoral vein Doppler flow into diagnostic algorithm; in a recent study including 47 patients with pulmonary thromboembolism, changes in the pulsatility pattern of the femoral vein were seen in all patients with right ventricular dysfunction[51]. Recently, it has been proposed as a quick way to diagnose right ventricular dysfunction in patients undergoing cardiac surgery[28]. Indeed, it is an attractive option in the emergency settings, where femoral vein is often sonographically assessed for central venous catheter placement. Table 1 summarizes the advantages and limitations of the Doppler evaluation of above-discussed vessels.

LIMITATIONS OF DOPPLER EVALUATION OF VENOUS CONGESTION

Doppler evaluation of venous congestion does not come caveat-free; first of all, the evaluation is operator dependent, meaning that the experience of the observer effects the image acquisition and interpretation. It is not unexpected because Doppler ultrasonography requires a higher skill level than for basic greyscale POCUS applications. Interobserver agreement has been reported mainly with experienced operators. For the HV, the kappa index was 0.95[52]; for the intra-renal venous Doppler and PV, the interobserver agreement was 87% and 95% respectively[45]; and for the femoral vein Doppler, the reproducibility of readings was 80%-98%[50]. Secondly, clinicians must be aware of the false negative and false positive findings that can interfere with interpretation. The HV Doppler should be accompanied by a simultaneous ECG as much as possible; otherwise, the observer can incorrectly identify A-wave as a retrograde S-wave and vice versa. Similarly, S and D waves can be confused for one another. Notably, pulsatile PV flow can be found in young healthy individuals with low body mass index, without elevations in RAP[53]. On the other hand, reduced PV pulsatility despite elevated RAP has been reported in patients with parenchymal liver disease[54-56]. Intra-renal venous Doppler is technically challenging to obtain and more time consuming; it can also be altered by obstructive urological pathologies[57]. Doppler interrogation of the femoral vein may be altered by application of excessive transducer pressure. Due to these limitations, isolated interpretation of individual waveforms may lead to incorrect conclusions. Therefore, assessing IVC and multiple venous sites including HV, PV, IRVD in an organized stepwise manner could enhance diagnostic performance. Corroborating this notion, a recent study employing a

Table 1 Advantages and limitations of the Doppler evaluation of various veins

	Advantages	Limitations
Hepatic vein	Easy to acquire images from the same window used to assess IVC.	Prone to erroneous interpretation without simultaneous EKG tracing.
		Influenced by arrhythmias (*e.g.*, S-wave can be smaller in atrial fibrillation), right ventricular systolic dysfunction.
		May never normalize in chronic pulmonary hypertension, structural tricuspid regurgitation irrespective of fluid status.
Portal vein	Easy to assess without EKG.	Not reliable in cirrhosis.
	Reliably changes with decongestive therapy - can monitor response to diuresis/ultrafiltration in real time.	Can be pulsatile in young, thin individuals without raised RAP.
	Tends to improve with decongestion, if not normalize even in chronic pulmonary hypertension.	
Renal parenchymal vein	Simultaneous arterial tracing functions as a built-in EKG.	Difficult to obtain optimal images.
		Not studied in chronic kidney disease/patients with structural renal abnormalities.
		Interstitial edema may hamper improvement with decongestive therapy in real time (improves but lags behind decongestion).
		May never normalize in chronic pulmonary hypertension, structural tricuspid regurgitation irrespective of fluid status.
Femoral vein	Technically easier to acquire images of the vein.	Susceptible to excessive transducer pressure.
		Dependent on correct Doppler angle if measuring absolute velocities (pattern evaluation is less angle dependent).

IVC: Inferior vena cava; RAP: Right atrial pressure; EKG: Electrocardiogram.

protocolized venous Doppler examination termed "VExUS" (venous excess ultrasound score) has shown greater specificity for organ injury than any individual assessments [16].

INTERNAL JUGULAR VEIN AND SUPERIOR VENA CAVA ULTRASOUND

Similar to IVC, internal jugular vein (IJV) ultrasound can also be used to estimate RAP non-invasively. In one study, < 17% increase in right IJV cross sectional area with Valsalva maneuver predicted an elevated RAP (≥ 12 mmHg) with 90% sensitivity and 74% specificity[58]. In patients who cannot follow instructions, assessment of IJV diameter at the end of inspiration and expiration can provide a rough idea of CVP. For example, in a study of 34 spontaneously breathing patients, mean IJV diameter was 7 mm in those with CVP < 10 cm H_2O [95% confidence interval (CI): 5.7-8.3] *vs* 12.5 mm (95%CI, 11.2-13.8) in those with CVP of ≥ 10 cm H_2O[59]. In intubated patients, it is of limited utility to predict CVP but an IJV distensibility of > 18% prior to volume challenge has shown to predict response to fluids[60]. While IJV ultrasound appears easy to perform, the amount of information it can provide is limited and cannot be used in lieu of VExUS. Moreover, it is subject to erroneous interpretations due to inadvertent application of excess transducer pressure, limited access to the neck because of the presence of central venous catheters, tracheostomy collars, braces *etc.* On the other hand, superior vena cava ultrasound has been studied in the context of predicting fluid responsiveness and shown to perform better than IVC[61]. However, transesophageal echocardiography is required to reliably access the vessel, which is not routinely performed in all clinical settings.

INTEGRATION OF BEDSIDE DOPPLER ULTRASOUND INTO GLOBAL HEMODYNAMIC ASSESSMENT

Venous Doppler ultrasound should not be used to 'determine' fluid status or assess fluid responsiveness. This novel bedside tool should be viewed as another piece of

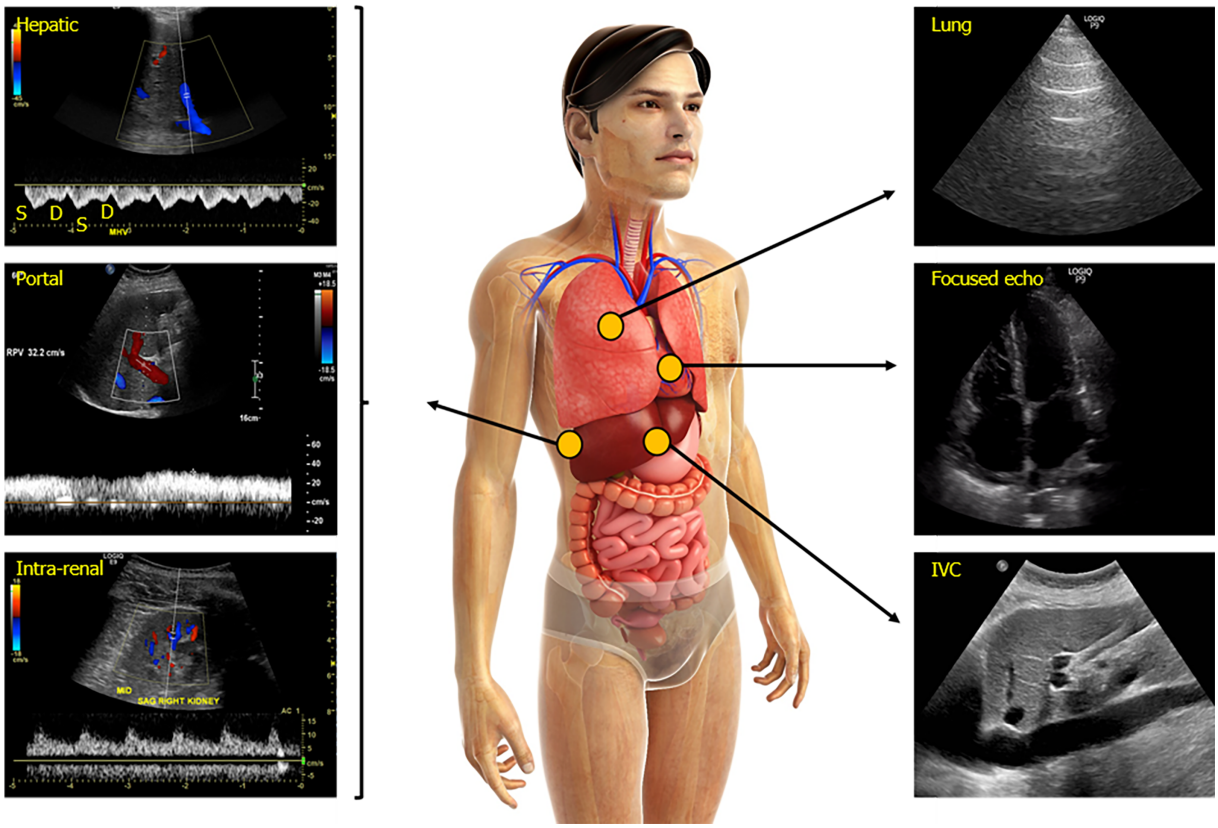

Figure 3 Figure illustrating the integration of venous Doppler with other vital pieces of sonographic assessment including focused cardiac and lung ultrasound. Normal waveforms shown. IVC: Inferior vena cava. Human body image licensed from Shutterstock®.

information in the global hemodynamic assessment of the critically ill patient in addition to other sonographic and clinical parameters. Since the information it yields might be particularly relevant for patients with oliguric AKI, the following discussion will center on the resuscitative efforts aimed at restoring renal perfusion (*renoresuscitation*). The first step in evaluating oliguric kidney injury is excluding obstructive pathology by kidney and urinary bladder ultrasound[62]. Also, looking for the cues to intrinsic kidney injury such as acute tubular necrosis or acute interstitial nephritis is of paramount importance as resuscitative efforts are unlikely to restore renal function in this situation[63]. Intrinsic AKI should be suspected when the clinical and laboratory data point to tubular dysfunction (exposure to nephrotoxins, prolonged hypotension, isosthenuria, high fractional excretion of sodium, abundant muddy brown casts on urine microscopy)[64]. A furosemide stress may help assessing renal tubular integrity as well as bears prognostic significance[63]. While acute glomerulonephritis is uncommon in patients with hospital-acquired AKI, finding of dysmorphic red blood cells on urine sediment examination should prompt nephrology consultation for investigation of glomerular causes of AKI.

On the other hand, evidence of preserved tubular function should lead to presumptive diagnosis of hemodynamic AKI caused by renal hypoperfusion. Evidence of global hypoperfusion (increased capillary refill time, skin mottling, altered mental status) increases the likelihood that resuscitative interventions could result in improved urine output. It is important to understand that renal perfusion pressure is proportional to the difference between mean arterial blood pressure (MABP) and renal venous pressure, and inversely proportional to renal arteriolar resistance[65]. Traditional resuscitative efforts have focused on increasing MABP (vasopressors) or increasing CO (fluids, inotropes). However, less attention has been paid to renal venous pressure even though this is an equally important determinant of renal perfusion. Measurement of intra-abdominal pressure should be performed if there is a suspicion of abdominal compartment syndrome, particularly in patients with trauma or tense ascites[66]. In addition, Doppler evaluation of venous congestion can point to renal congestion (intra-capsular tamponade) as the cause of renal hypoperfusion by demonstrating the effects of raised RAP on venous outflow[16,67]. This previously missing piece of the hemodynamic puzzle can add valuable information as oliguric

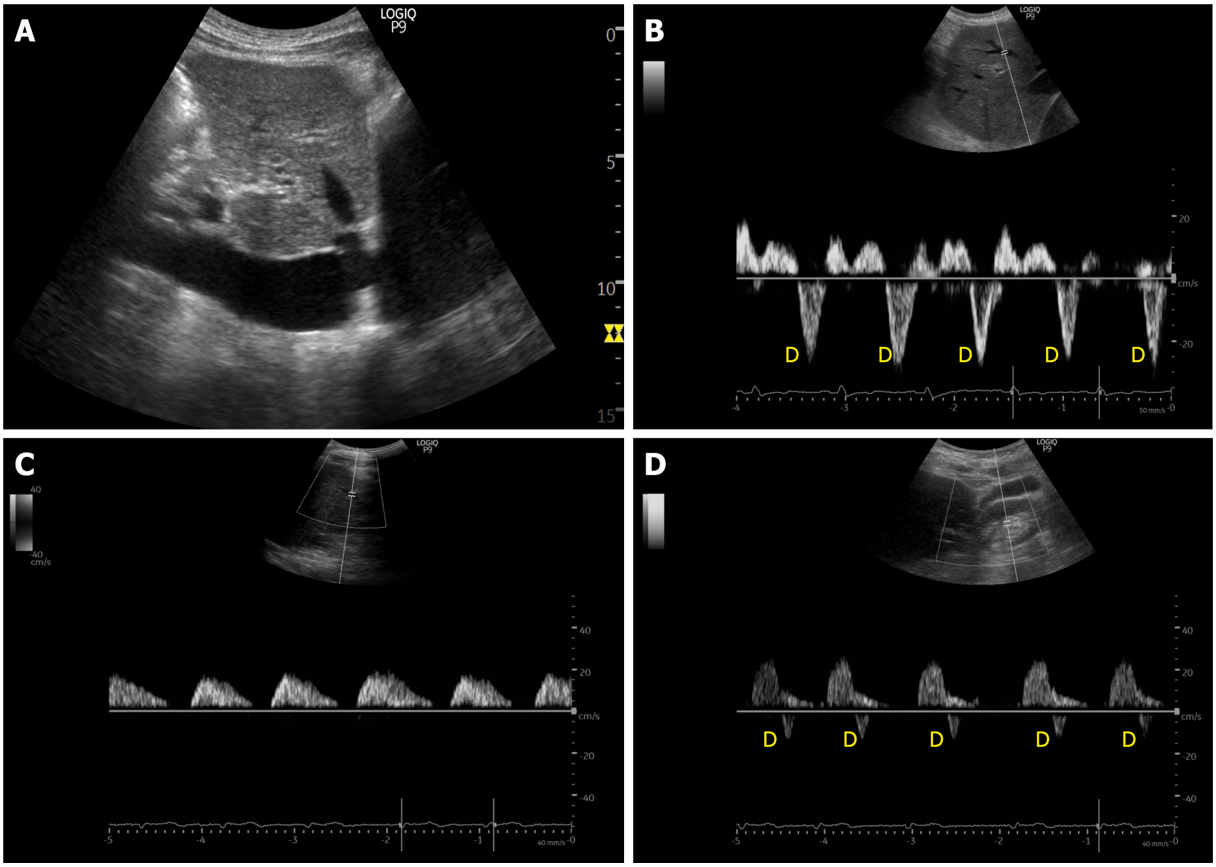

Figure 4 Example of ultrasound stigmata of severe venous congestion obtained from a patient with congestive heart failure exacerbation and tricuspid regurgitation. A: Dilated inferior vena cava; B: Hepatic vein Doppler demonstrating only D-wave below the baseline; C: Pulsatile portal vein with flow pauses in between the cardiac cycles; D: Ontra-renal vein demonstrating only D-wave below the baseline.

AKI in the presence of severe venous congestion will likely worsen with fluid administration but is likely to improve following decongestion[67-69]. Finally, it is also important to recognize that microvascular alterations underly many cases of sepsis associated AKI[70]. These alterations are an important determinant of glomerular hydrostatic pressure regardless of macrohemodynamics and as such, are not likely to improve with conventional resuscitative efforts.

Performing a comprehensive hemodynamic assessment using POCUS in addition to conventional evaluation is vital in the management of critically ill patients as multiple hemodynamic alterations might be present simultaneously (the so-called pump, pipes, leaks strategy)[70]. For example, a septic patient with pre-existing heart failure can display both vasodilation (low peripheral vascular resistance) and severe venous congestion. In this setting, vasopressors and diuretics can be used together to address these alterations. In summary, venous Doppler provides valuable information regarding a patient's hemodynamic status, when used in combination with multi-organ POCUS as well as clinical and laboratory data.

CONCLUSION

Multi-point Doppler evaluation of the venous system allows clinicians to assess the downstream effects of elevated RAP on peripheral organs. This tool should not be used as a marker of fluid status or volume responsiveness but rather as a means to determine if congestion is contributing to organ dysfunction and gauge the response to decongestive therapy. This information should be integrated into a comprehensive hemodynamic evaluation in order to choose the appropriate resuscitative strategy. Future studies should focus on investigating whether incorporating venous Doppler ultrasound in the diagnostic and treatment algorithms translates into better clinical outcomes. Furthermore, as most of the current data are from patients with heart failure, research should be undertaken in other subsets of patients susceptible to fluid overload such as those with liver cirrhosis and chronic kidney disease.

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