Use of Ultrasound to Assess Hemodynamics in Acutely Ill Patients

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Abstract
Early diagnosis of acute kidney injury (AKI) and preventive measures can likely decrease the severity of the injury and improve patient outcomes. Current hemodynamic monitoring variables, including blood pressure, heart and respiratory rates, temperature, and oxygenation status, have been used to identify patients at high risk for AKI. Despite the widespread use of such variables, their ability to accurately and timely detect high-risk patients has been questioned. Therefore, there is a critical need to develop and validate tools that can measure new and more kidney-specific hemodynamic and laboratory variables, potentially assisting with AKI risk stratification, implementing appropriate and timely preventive measures, and hopefully improved outcomes. The new ultrasonography techniques provide novel insights into kidney hemodynamics and potential management and/or therapeutic targets. Contrast-enhanced ultrasonography, Doppler flow patterns of hepatic veins (HV), portal vein (PV), and intra- kidney veins (iKV), and ultrasound elastography are among approaches that may provide such information, particularly related to vascular changes in acute kidney injury, venous volume excess or congestion, and fluid tolerance. This review summarizes the current state of these techniques and their relevance to kidney hemodynamic management.
Introduction
Managing hypotension in critically ill patients has traditionally focused on sustaining cardiac output via intravenous fluids or vasopressors (1). Administering intravenous fluids to patients with distributive or hypovolemic shock is a widely adopted treatment strategy (2). Rapid administration of crystalloids for hypotensive patients is a cornerstone of the surviving sepsis campaign (SSC) 1-hour bundle (3). SSC also suggests using volume responsiveness (VR) as a guide to administering fluids. VR is defined as an increase of the stroke volume (SV) of >10% in response to intravenous fluid challenges (4). However, there is a growing concern that these guidelines recommend fluid administration as not supported by substantial evidence (5). Using VR may lead to over-resuscitation resulting in iatrogenic volume overload. While fluid therapy is life-saving for appropriate indications, it could also be associated with significant adverse effects (6-8). One of the organs that is frequently impacted by volume overload is the kidney. Indeed, kidney congestion is considered one of the primary etiologies of decreased glomerular filtration rates(9, 10).

From a physiological perspective, fluid administration leads to an increase in right and left atrial pressures. Perfusion depends on the pressure gradient between the artery and vein supplying an organ, as do filtration and plasma resorption across the capillary bed. Increased venous pressure reduces the pressure gradient and thus decreases blood supply. At the same time, it causes increased interstitial fluid. Edema occurs when there is a mismatch between plasma filtration at the capillary arteriolar side and resorption at the venule side. When this mismatch exceeds lymphatic drainage, edema occurs (Figure 1). Further elevations in venous pressure from ongoing volume loading cause more edema formation, which increases interstitial pressure. Eventually, interstitial pressure exceeds arteriolar inflow. This effect is more prominent in encapsulated organs such as the kidneys (11), where increased venous pressure increases interstitial pressure leading to organ dysfunction and acute kidney injury (11, 12).

Patients in shock are particularly susceptible to kidney edema formation. Low oncotic pressure from disease and crystalloid boluses contribute to the filtration/resorption mismatch. Systemic vasopressors increase arterial and venous vascular tone, leading to increased filtration (higher mean arteriolar pressure) and decreased resorption (higher mean venule pressure), increasing the risk of organ edema.

When the shock is associated with respiratory failure, positive pressure ventilation results in right-sided venous congestion. Flow into the right atrium from the inferior vena cava (IVC) and superior vena cava (SVC) depends on the pressure gradient between the mean circulatory filling pressure (MCFP) and right atrial pressure (RAP). The MCFP is the pressure in the extrathoracic IVC and SVC and reflects the venous pressure at the end organ. The RAP is lower than the MCFP to allow blood flow into the thorax. Positive pressure ventilation increases thoracic pressure and, thus, right atrial pressure and central venous pressure increase without an increase in MCFP. This effect would lead to a decline in the pressure gradient, causing decreased venous return. When initially placed on positive pressure ventilation, many patients become acutely hypotensive and require fluid boluses to increase the MCFP. Among mechanically ventilated patients who need increased mean airway pressure to manage progressive respiratory failure, fluid boluses may be needed to protect the MCFP and facilitate venous return. In turn, the increased MCFP could increase the kidney venous pressure and may further worsen kidney congestion.

The sequelae of elevated left atrial pressure are relatively easy to assess clinically. It often leads to pulmonary edema, which results in hypoxia and is frequently detected on plain x rays or
oxygenation indicators like PaO2/FiO2. In contrast, identifying an increase in right atrial pressure is more challenging (12). Central venous pressure monitors and pulmonary artery catheters assess right atrial pressure. Still, they are invasive, confounded by positive pressure ventilation, and have not improved outcomes despite years of study (13, 14). Point-of-care ultrasonography (POCUS) is helpful in this area, as increasing pressure in the right atrium from venous excess or congestion leads to distinct changes in Doppler venous return flow patterns. Doppler flow patterns of hepatic veins (HV), portal vein (PV), and intra-kidney veins (iKV) are noninvasive and can identify early stages of right-sided venous congestion (15). Indeed, abnormal waveforms are associated with AKI in post-cardiac surgery patients (16, 17) and may help predict early AKI in the general ICU population (12).

In addition to Doppler flow patterns, other ultrasonography techniques are suggested to delineate the kidney hemodynamics, including contrast-enhanced ultrasonography (CEUS) (18) and ultrasound elastography (19).

In this review article, we present POCUS techniques to assess kidney hemodynamics and describe their clinical implications.

**Ultrasound Terminology**

The ultrasound examination is usually performed in three consecutive phases (20). The first step is a grayscale 2D image of the organ of interest. This mode is called B or brightness mode. B-modes are followed by the color Doppler to produce a colored representation of blood flow dynamics. Finally, a small portion of the vessel is selected, and a spectral Doppler waveform is obtained for a more focused assessment of the blood flow rates. Doppler is an eponym named after the Austrian physicist who first described the "Doppler effect" in the 19th century (21). Pulsed wave Doppler (PWD), the main form of Doppler ultrasound described in this article, is based on sending pulsed signals allowing sampling at a specific location by the cursor.

**Inferior Vena Cava (IVC)**

The inferior vena cava (IVC) is a compliant tear-shaped vessel. IVC size and shape fluctuate with variations in CVP and intravascular volume (22, 23). Several factors may affect the IVC size. Under normal physiologic conditions, IVC diameter decreases, and venous return increases during inspiration due to negative intrathoracic pressure and positive intra-abdominal pressure. This relationship is reversed in positive pressure mechanical ventilation (24). IVC diameter also decreases during ventricular systole in spontaneously breathing patients. Moreover, the patients' position alters IVC diameter. However, measuring IVC in the supine position is now recommended in the American Society of Echocardiography guidelines (25).

The IVC diameter assessment is usually considered a noninvasive tool to measure central venous pressure (CVP) (22). Measuring CVP was described several decades ago (20) and has since become a standard method to assess volume status and guidance for intravenous fluid therapy. However, many subsequent studies indicate a poor association between CVP and blood volume and the inability of CVP and/or its changes to predict the hemodynamic response to a fluid challenge (2, 26). Hence, relying on CVP for fluid management should not be the only deciding factor. However, IVC diameter and IVC variability may be helpful as a marker of the right ventricular (RV) function in some clinical circumstances, such as acute RV failure (26). The IVC is best examined using a subcostal view with a longitudinal section (24). The diameter is best measured in M-mode coupled to the two-dimensional image, just upstream of the hepatic vein entry or approximately 1-2 cm caudal to the cavoatrial junction. Measurements are most accurate when the M-mode tracing is perpendicular to the IVC (27).
The IVC diameter variation through the respiratory cycle can be quantified by measuring its collapsibility index (IVCCI). The operator measures the maximum (IVC-max) and minimum IVC diameters (IVC-min) during the respiratory cycle. IVCCI is then calculated as \[(IVC\text{-}max-IVC\text{-}min)/IVC\text{-}max\]. In a spontaneously breathing patient, an IVC diameter of <2 correlates with CVP <10 cmH\text{2}O (28). An IVC diameter of >2 cm and IVCCI of <50% indicates a CVP of >10 cmH\text{2}O (29). It is important to note that IVCCI has not been validated in patients with respiratory failure requiring positive pressure mechanical ventilation.

Of note, there is controversy around the utility of IVC measurements in mechanically ventilated patients (30), especially those who have undergone abdominal surgery or severe respiratory failure requiring high mean airway pressure ventilation. In this situation, the IVC diameter and collapsibility changes may reflect the positive pressure ventilation rather than CVP. Therefore, many intensivists no longer use this method of assessment as a measure of volume status. It is also worth noting that studies investigating the utility of IVC measurements use a wide variety of measurements and different percentages as the threshold for collapsibility. In addition, each study has used a different comparator to assess the validity of IVC measurements in determining fluid responsiveness.

While IVC measurements can estimate CVP and its variability, they are not strong predictors of fluid responsiveness. A meta-analysis examined 19 studies looking at the relationship between CVP and its variability and a change in cardiac performance following a fluid challenge. The pooled correlation coefficient between baseline CVP and change in the cardiac index was 0.18. The pooled area under the receiver operating characteristic curve (ROC) was 0.56. The pooled correlation between CVP variability and change in the cardiac index was 0.11 (26).

Stroke volume variation (SVV) is a better predictor of fluid responsiveness when compared to IVC variation in critically ill patients. A meta-analysis of clinical trials investigated the diagnostic value of SVV in predicting fluid responsiveness. A total of 568 patients from 23 studies were included. Baseline SVV was correlated to fluid responsiveness with a pooled correlation coefficient of 0.718 and pooled area under ROC of 0.84 (31). While POCUS can quantify SVV, the discussion of the technique is beyond the scope of the article.

**Liver Doppler Waveforms**

The hepatic arteries, hepatic veins, and portal veins are the principal vascular bundles of the liver. The changes in blood flow patterns in hepatic and portal veins are important in volume overload and tolerance assessment. Each one of these vessels has a distinctive "signature" waveform appearance.

**Hepatic Vein**

The hepatic vein can be examined using a phased array probe. The middle hepatic vein is identified from a mid-subcostal (Figure 2) or lateral view. Hepatic venous waveforms are obtained via applying PWD in about 2-4 cm from its junction to the IVC. The hepatic venous waveform reflects the pressure changes in the right atrium during the cardiac cycle. Unlike hepatic venous flow that correlates with the right atrial pressure changes and is pulsatile, the portal flow is continuous and reflects visceral venous pressure. The hepatic vein Doppler evaluates how blood flows into the RA, so theoretically, it is not confounded by positive pressure ventilation.
Typically, the hepatic vein waveform has one primary retrograde wave, i.e., flow directed towards the transducer and appears above the baseline, along with two major antegrade waves, i.e., flow directed away from the transducer and appears below the baseline. The retrograde wave (a) peaks corresponding with atrial contraction at the end of the diastole. The antegrade wave (S) corresponds with peak negative pressure generated by the tricuspid valve annulus downward motion in early systole. The antegrade wave (D) relates to RV filling in early diastole. In between the S and D waves, there is sometimes a (v) wave with a peak that marks the transition between systole and diastole, and it corresponds with the tricuspid valve opening. The v wave is rarely seen in hepatic vein Doppler.

The hepatic vein S and D waveforms remain antegrade when the right atrial pressure is not elevated, indicating the hepatic vein blood flow towards the right atrium during the cardiac cycle. The S wave is normally larger than the D wave as the antegrade flow is more prominent during systole.

In mild venous congestion, the S wave becomes smaller than the D wave due to increased right atrial pressure. In severe venous congestion, the S wave turns to retrograde (flow directed towards the transducer) or fuses with the (a) wave to become a sizeable retrograde wave (above the baseline). These changes are illustrated in (Figure 3).

Abnormal hepatic waveforms were shown to predict adverse kidney outcomes in a prospective study in adult patients admitted to the ICU. S to D reversal in hepatic vein flow had an OR of 4.0 for an increase in the likelihood of a significant kidney event (12).

**Portal vein**

Portal vein images are obtained in the supine position using a phased array transducer placed in mid-costa (Figure 2) or a right posterior-axillary coronal view between the ninth and eleventh intercostal space (32). Portal vein walls are echogenic (white) that differentiate them from the hepatic veins. The portal vein flow is mono- or biphasic. As the portal vein blood flow velocity is low (i.e., 10-30 cm/s), the velocity gate should be adjusted to a lower range (i.e., 20-40 cm/s) to acquire appropriate images. Typically, the monophasic to biphasic portal vein waveforms are completely retrograde (flow directed towards the transducer) and have small variations throughout the cardiac cycle. Respiratory variation is often observed.

The waveforms are consistently biphasic with increased "pulsatility" due to a systolic flow reduction in mild congestion. In severe liver congestion, the flow completely disappears during systole. In more severe cases, the flow even can reverse to antegrade (flow directed away from the transducer), giving the waveform a "to and fro" pattern (Figure 3).

Measuring the portal vein pulsatility index ([maximum flow velocity - minimum flow velocity]/maximum flow velocity) quantifies the flow variation through the cardiac cycle, hence liver congestion. An index of >30% is highly suggestive of liver congestion and is associated with a higher incidence of adverse kidney outcomes (OR 2.2) (33). When measured pre- or postoperatively, Portal vein pulsatility is associated with a higher AKI incidence after cardiac surgery (16, 17). Portal vein pulsatility is also a measure of portal hypertension and can be found in patients with end-stage liver disease. In these patients, it may not represent elevated right atrial pressure.

**Kidney Doppler Waveforms**

Intra-kidney Doppler ultrasonography images can be obtained in supine (Figure 4) or lateral decubitus positions. Color Doppler velocity range should be set to approximately 16 cm/s (15). The color Doppler images can locate the inter-lobar vessels. As the interlobar arteries and veins...
run in proximity, PDWs of the interlobar arteries and veins can be recorded simultaneously. This approach can detect the venous waveform systolic and diastolic phases easier, even if EKG leads are not available. Normally, the iKV waveform is continuous and antegrade (flow directed away from the transducer). This reflects blood flow from the iKV towards the IVC in both systole and diastole.

In mild venous congestion, the flow pattern is biphasic with visible (S) and (D) waveforms (Figure 3). As venous congestion increases, the (S) waveform size decreases. In severe venous congestion, the (S) waveform completely disappears, giving the waveform a monophasic pattern. In a study by Iida et al., death and unplanned admission for heart failure were progressively higher when the waveform patterns changed from continuous to biphasic and monophasic patterns (15).

One method to quantify the discrepancy between systolic and diastolic iKV flow is calculating the kidney venous impedance index (VII) as \[
\frac{\text{maximum flow velocity} - \text{minimum flow velocity}}{\text{maximum flow velocity}}.
\]
VII has been shown to correlate with the expansion and removal of intravascular fluids among patients with heart failure with preserved (0.2 pre volume expansion to 0.7 post volume expansion), or reduced (0.4 pre volume expansion to 0.7 post volume expansion) ejection fraction (34).

### The Venous Excess Ultrasound (VexUS) Score

The VExUS grading system was developed to combine a qualitative assessment of venous Doppler examination of the HV, PV, iKV, and IVC diameter into grading the severity of venous congestion (35). The Doppler flow patterns are rated as normal, mildly abnormal, or severely abnormal (Figure 5). The IVC diameter is converted to a binary variable with 2 cm as the cutoff. This information was combined into a grading scheme.

In post-cardiac surgery patients, VexUS was used to predict acute kidney injury (AKI) (35). The authors evaluated several grading schemes, and VExUS C scheme, grade 3, which is defined by the presence of severe flow abnormalities in at least two vascular beds along with dilated IVC more than 2 cm, was most strongly associated with subsequent AKI (HR: 3.69 CI 1.5–8.24 p=0.001). The association remained significant after adjusting for baseline risk factors of AKI and the need for vasopressor/inotropic support (HR: 2.82 CI 1.21–6.55 p=0.02). It is worth noting that while IVC dilation alone had poor predictive performance for AKI, its inclusion within the VExUS grading system resulted in a slight increase in the VexUS specificity (35). Also, one should keep in mind that cardiac surgical patients are a specific subset of critically ill patients. Thus, some of these metrics may be different in a general ICU population. Table 1 describes two cases that ultrasonography examination of hemodynamics delineated clinical findings that led to improved care.
Assessment of Left and Right Ventricle Function

A complete description of echocardiographic techniques is beyond the scope of this article. However, a focused echocardiographic examination is frequently performed in critically ill patients to determine "overall global function of the heart" and rule out acute conditions that can cause hypotension, such as large pericardial effusions or pneumothorax (36, 37). The examination could clarify if the extent of left and right ventricle contraction or the presence of decompensated heart failure, pulmonary hypertension, or pulmonary embolism. The basic views for echocardiography are 1) parasternal long-axis view (PLAX), which is obtained by placing the probe between the 3rd and 4th ribs left of the sternum (Figure 6A). The indicator points to the patient's right shoulder. An ideal image includes the left atrium, mitral valve, left ventricle, aortic valve, aortic root, and right ventricle. The apex of the left ventricle will often be outside of the image; 2) parasternal short-axis view (PSAX), acquired after obtaining PLAX after the probe is rotated 90 degrees clockwise to point to the patient's left shoulder. The image captured will usually be at the level of the mid-ventricle or papillary muscles. The levels captured as the operator tilts the transducer towards the apex and then towards the base include aortic valve level, mitral valve level, papillary muscle level, and apical level (Figure 6B); 3) Apical 4-Chamber View (A4C) that is obtained by placing the probe at the point of maximum cardiac impulse, preferably with the patient in the left lateral decubitus position (Figure 6C). The indicator is pointed between the patient's left shoulder and 3 o'clock. The probe is lifted upwards to allow a good view of the four chambers, and by lifting the probe more, the operator can open up the left ventricle outflow tract and aortic valve, obtaining an apical 5-chamber view (A5C).

Contrast-Enhanced Ultrasonography and Ultrasound Elastography

New ultrasonography technologies may open additional venues that kidney hemodynamics could be assessed independently and with higher accuracy. Contrast-enhanced ultrasonography (CEUS) and ultrasound elastography (USE) are among these emerging technologies. While the clinical utilization of these new technologies remains to be investigated, being familiar with this field's progress would potentially provide perspectives in research or clinical applications of ultrasonography.

Ultrasound contrast agents are used to enhance the resolution of cardiac and vascular images. Agitated saline contains microbubbles, and its use for higher resolution of aortic images was introduced in 1968 (38). As the agitated saline air microbubbles had a short half-life, the next generation of microbubbles includes a layer of albumin or galactose palmitic acid as a shell. Further, heavy molecular weight gas agents, e.g., sulfur hexafluoride, to fill the microbubbles and surfactant, as the shell, improved the agent stability (39). CEUS use for kidney hemodynamic assessment has been reported in the literature. For instance, Schneider et al. described kidney microvascular cortical perfusion changes in response to norepinephrine infusion, angiotensin II and captopril using CEUS (18, 40). Recent advances made in image resolution and ultrasound penetration depth have led to the development of high-resolution images of the kidney microvessels (e.g., super-resolution ultrasound microvessel imaging; SR-UMI (33, 41-43). While these technologies remain in preclinical investigation phases, they provide a vast potential for kidney hemodynamic assessment.

The other new technology in development with potential in enhanced ability in kidney hemodynamic assessment is USE. As kidney elasticity changes with hemodynamic alterations, elastography can provide a unique perspective on kidney hemodynamic alterations (44). In a
swine model, investigators showed that using USE correlates with intraabdominal pressure and pressure inside the kidney capsule significantly more than bladder pressure (19). This imaging technique can potentially enhance our ability to manage fluid therapy, particularly identifying kidney congestion.

**Challenges and benefits of Using POCUS**

Factors that may limit POCUS usability include the patients’ body habitus, bowel gas, and mechanical ventilation, making it harder to obtain ultrasonographic windows. However, many of these challenges exist for other diagnostic modalities. In addition, other diagnostic modalities raise challenges that do not exist for POCUS. For example, transporting obese patients and patients on mechanical ventilation to radiology for diagnostic imaging poses a safety issue, including infectious control issues, especially in pandemics.

Point-of-care ultrasound for hemodynamic evaluation is clearly feasible, but interobserver variability is poorly studied. Its accuracy depends on the skill of the person obtaining the images. It is best used by a well-trained individual provider as a point in time assessment or to track changes over time. LV EF and RV function can be assessed in > 90% of critically ill patients using a protocol that obtains all four cardiac windows (36, 37). Further, Speigal et al. on venous flow patterns showed that hepatic vein flow assessments could be completed in >90%, the portal in > 80%, and kidney in about 75% of ICU patients (12).

POCUS has been successfully applied within telemedicine. Tele-ultrasound has advanced in recent years, both in high-income settings and in resource-limited countries where demand for tests often outpaces the access to diagnostic modalities needed to identify disease. According to the World Health Organization (WHO), imaging is required for diagnosis in 20–30% of clinical cases, and ultrasound and/or plain radiographs are sufficient for 80–90% of those cases (45).

**Conclusion**

As POCUS has become an integrated part of patient care in inpatient and outpatient settings, understanding potential ultrasonography techniques to provide additional information about kidney-related hemodynamics is essential. Assessing right-sided venous flow patterns using POCUS is feasible and informative, not only for evaluating venous congestion but also for estimating organ congestion, particularly liver and kidney. The advent of novel ultrasonography technologies like CEUS and USE may open this window even further. We predict that ultrasound devices will continue to get smaller, more portable, and less expensive while providing higher resolution images. This progress is in concert with other technological advances that will make ultrasound more feasible and invaluable in many clinical scenarios.
Disclosures

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Author Contributions

S Safadi: Writing - original draft
S Murthi: Writing - review and editing
K Kashani: Conceptualization; Supervision; Writing - review and editing
References


### Table 1. Case prototypes

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<th>POCUS findings</th>
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<td>A 75-year-old female is hospitalized after undergoing elective cholecystectomy. Her surgery was complicated by bowel perforation. She is now in atrial fibrillation with RVR with borderline blood pressure. She has a net fluid balance of 6 liters since admission and bilateral pleural effusions on her chest x-ray. She requires 4 liters of oxygen via nasal cannula. Her kidney function is normal. The team wants to assess for intravascular volume overload and the need for diuresis. Her hepatic, portal, and intra-kidney vein doppler patterns are shown in figure TODO. She has an IVC diameter of 2.1 cm with minimal variation during the respiratory cycle.</td>
<td>In this case, the patient had clinical evidence of volume overload. However, her borderline blood pressure posed concerns about whether she was intra-vascularly depleted. The hepatic waveform pattern shows an inversion of the (S) wave indicating severe congestion. It is worth noting that the (a) waves are usually absent when the patient is in atrial fibrillation. The portal waveform pattern shows mild pulsatility with the cardiac cycle, and the intra-kidney waveform patterns look normal. Based on these findings and the IVC diameter, the patient was deemed to have venous congestion and was treated with diuresis. She remained hemodynamically stable with the diuresis. (Figures 7A, 7B, 7C)</td>
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<td>A 54-year-old female is hospitalized in the ICU after a gastrectomy. She has a complicated postoperative course, including prolonged mechanical ventilation, hypotension, and kidney failure. She has a net fluid balance of 12 liters since admission. She remains hypotensive on vasopressors. She was &quot;deemed volume-responsive&quot;—the team questions additional administration of IVF.</td>
<td>Despite volume responsiveness, the patient has clear evidence of volume congestion. The hepatic Doppler waveform looks slightly abnormal, with the (S) wave being smaller than the (D) wave. However, the portal waveform is abnormal with significant pulsatility, and the intra-kidney waveform is abnormal, showing discontinuous (S) and (D) waves. In this case, we suggested titrating up vasopressors and maintaining volume status if end-organ hypoperfusion is the concern. However, if pulmonary edema is the concern, then we suggested careful removal of volume. (Figures 8A, 8B, 8C)</td>
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Figure legends:

**Figure 1.** Edema formation from increased venous pressure  
**Figure 2.** Hepatic and Portal Vein Probe Placement  
**Figure 3.** Normal and Abnormal Venous Waveforms  
**Figure 4.** Probe Placement to obtain kidney Doppler waveforms  
**Figure 5.** Doppler Flow Patterns and VexUS Score  
**Figure 6.** Echocardiography, A) Parasternal Long Axis (PLAX) View; B) Parasternal Short Axis (PSAX) View; C) Apical 4-Chamber View (A4C), and Apical 5-Chamber View (A5C) Views  
**Figure 7.** Hepatic, Portal, and Intra-kidney Doppler Waveforms for Case 1 Respectively  
**Figure 8.** Hepatic, Portal, and Intra-kidney Doppler Waveforms for Case 2 Respectively
Normal RAP

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<td>Pulsatile &gt; 50%</td>
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* Includes flow wrapping around the baseline
Figure 6