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Assessment of venous congestion using vascular ultrasound

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Brief Title: Assessment of venous congestion using VEXUS.

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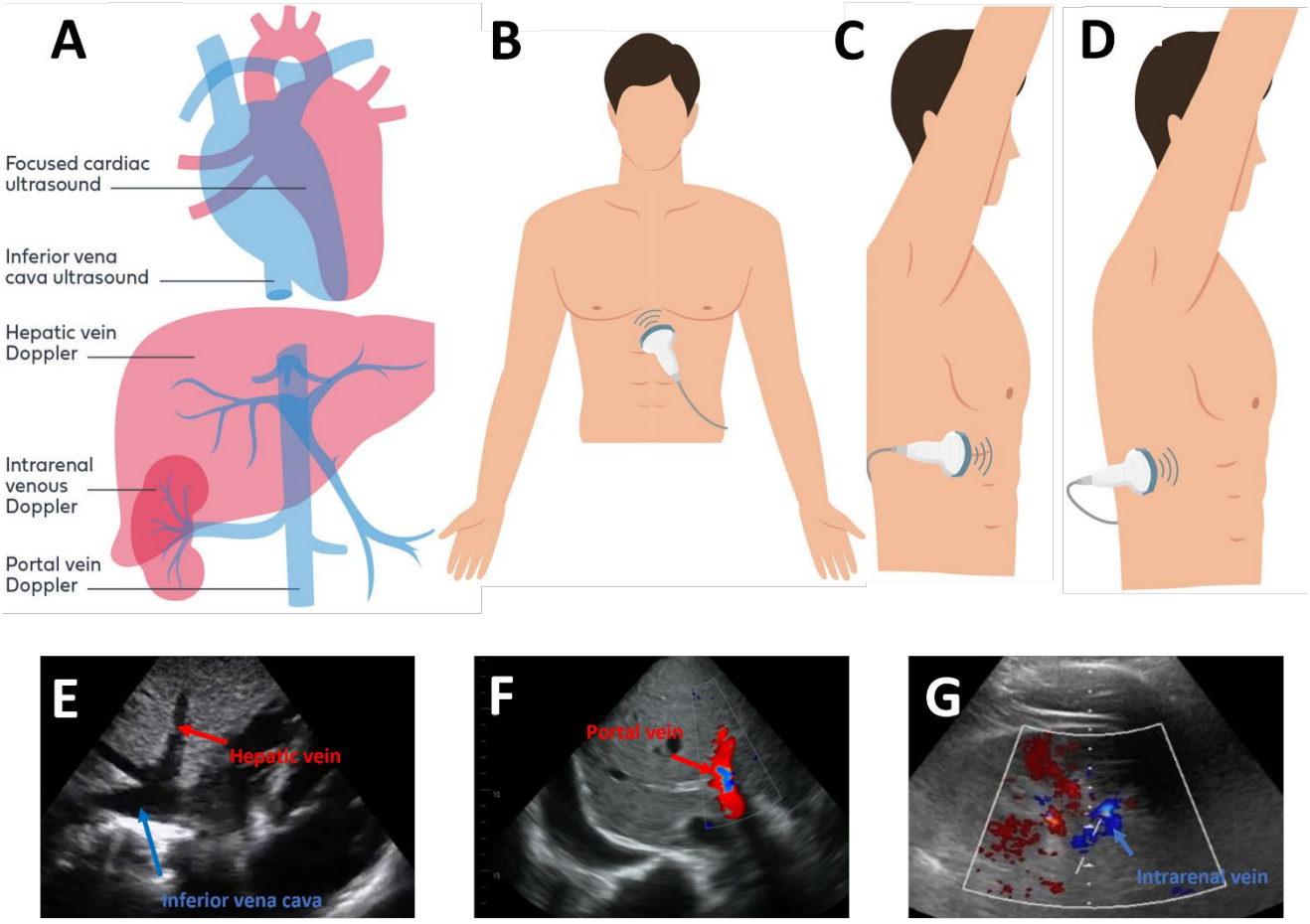
Introduction

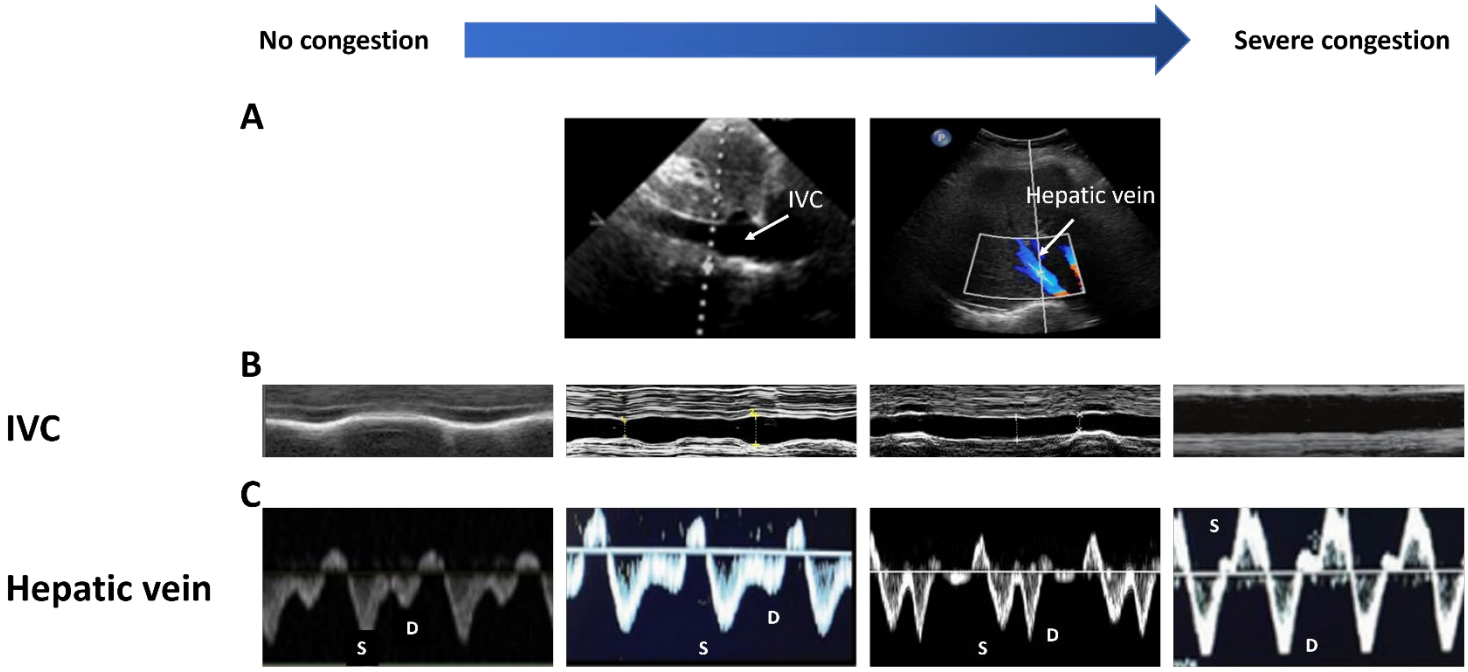
An accurate assessment of the venous pressure is crucial to optimize fluid management in patients with heart failure. Although evaluation of the inferior vena cava (IVC) is the most commonly used noninvasive method, it does not always represent a correct estimation of the patient's preload. Indeed, the IVC is a static parameter and can be dilated in healthy patients (e.g. athletes) or patients with specific comorbidities (e.g. pulmonary hypertension). As such, a dilated IVC does not necessarily indicate that your patient is "congested". Venous congestion not only results in a dilated IVC, but also reduces hepatic, intestinal and renal blood flow. Evaluation of the hepatic, portal, and intrarenal vein flow patterns (also called venous excess ultrasonography or 'VEXUS') may therefore help to identify venous congestion at an earlier stage and could be useful to optimize fluid management. The following article is intended to provide guidance on how to perform VEXUS and how it can be used as an add-on to transthoracic echocardiography.

Figure 1. Components of an echocardiographic assessment to evaluate venous congestion



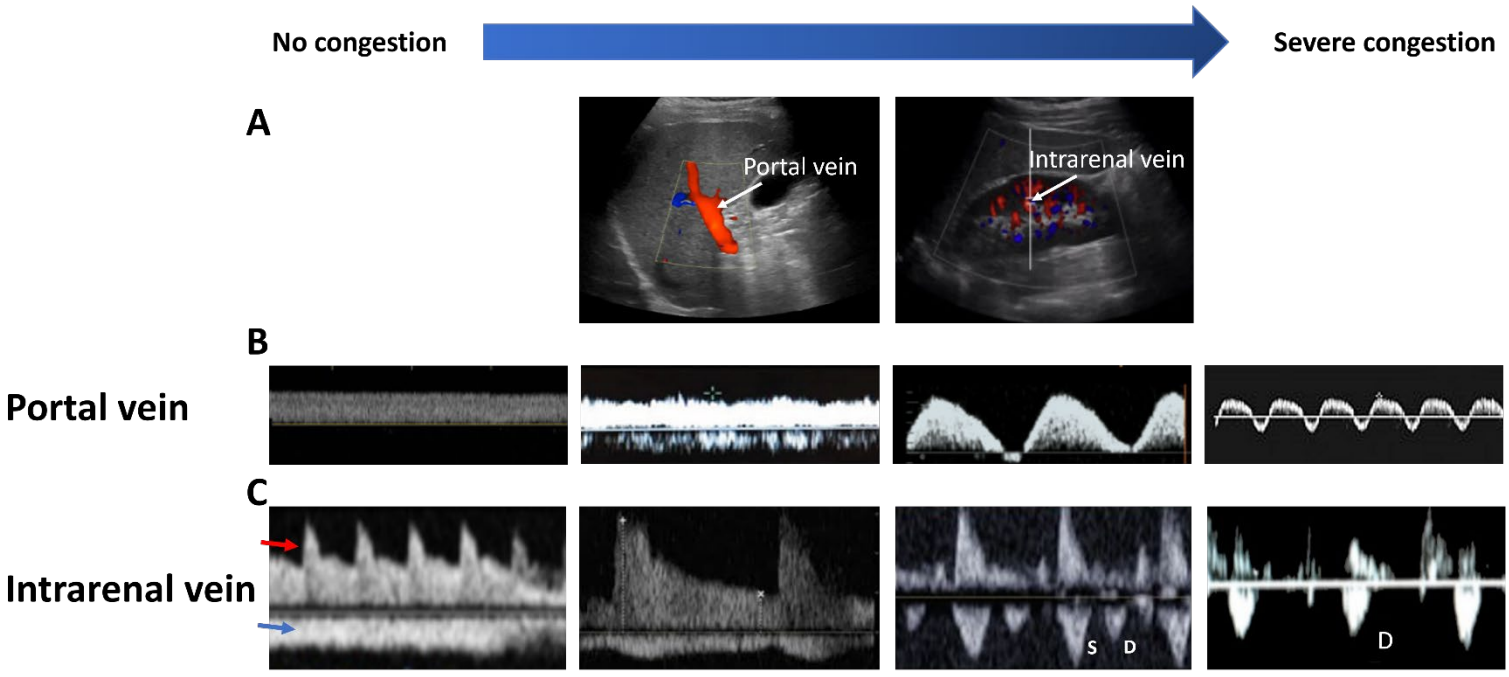
A comprehensive evaluation of venous congestion with vascular ultrasound consists of an evaluation of the inferior vena cava and a pulsed wave Doppler assessment of the portal vein, hepatic vein and intrarenal veins. These examinations can be performed as an add-on to transthoracic echocardiography **(A)**. The inferior vena cava and hepatic veins can be visualized from the subxiphoid position **(B, E)**. The portal vein can be visualized by positioning the probe along the anterior axillary line, tilting the tip of the probe slightly to anterior **(C, F)**. From this position, both the hepatic and portal veins can be visualized simultaneously by means of a clockwise rotation of the probe. The intrarenal veins can be evaluated by placing the probe along the mid-axillary line with the tip in a superior-posterior position **(D, G)**.

Figure 2. Alterations that occur in the inferior vena cava and hepatic vein with progressive venous congestion



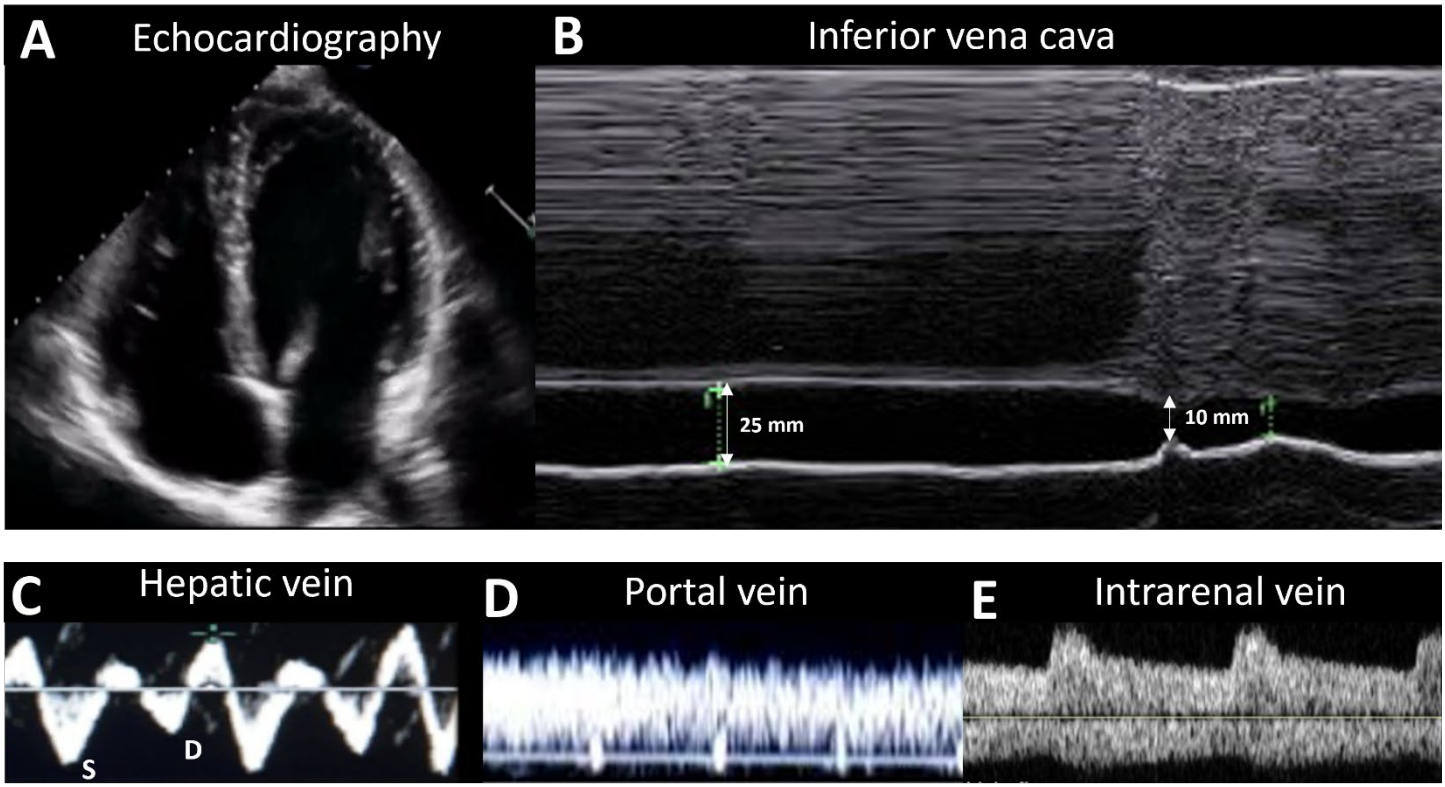
Echocardiography of the inferior vena cava and hepatic vein **(A)**. The size of the inferior vena cava increases and the respiratory variability reduces with progressive venous congestion **(B, left to right)**. Pulsed wave Doppler assessment of the hepatic vein in a patient without congestion shows a systolic (S) and diastolic (D) component with S>D. With progressive venous congestion, the S/D ratio reverses (S<D), and eventually the systolic component reverses **(C, left to right)**. By convention, flow towards the probe is shown above the baseline, whereas flow away from the transducer is shown below the baseline.

Figure 3. Alterations that occur in the portal vein and intrarenal veins with progressive venous congestion



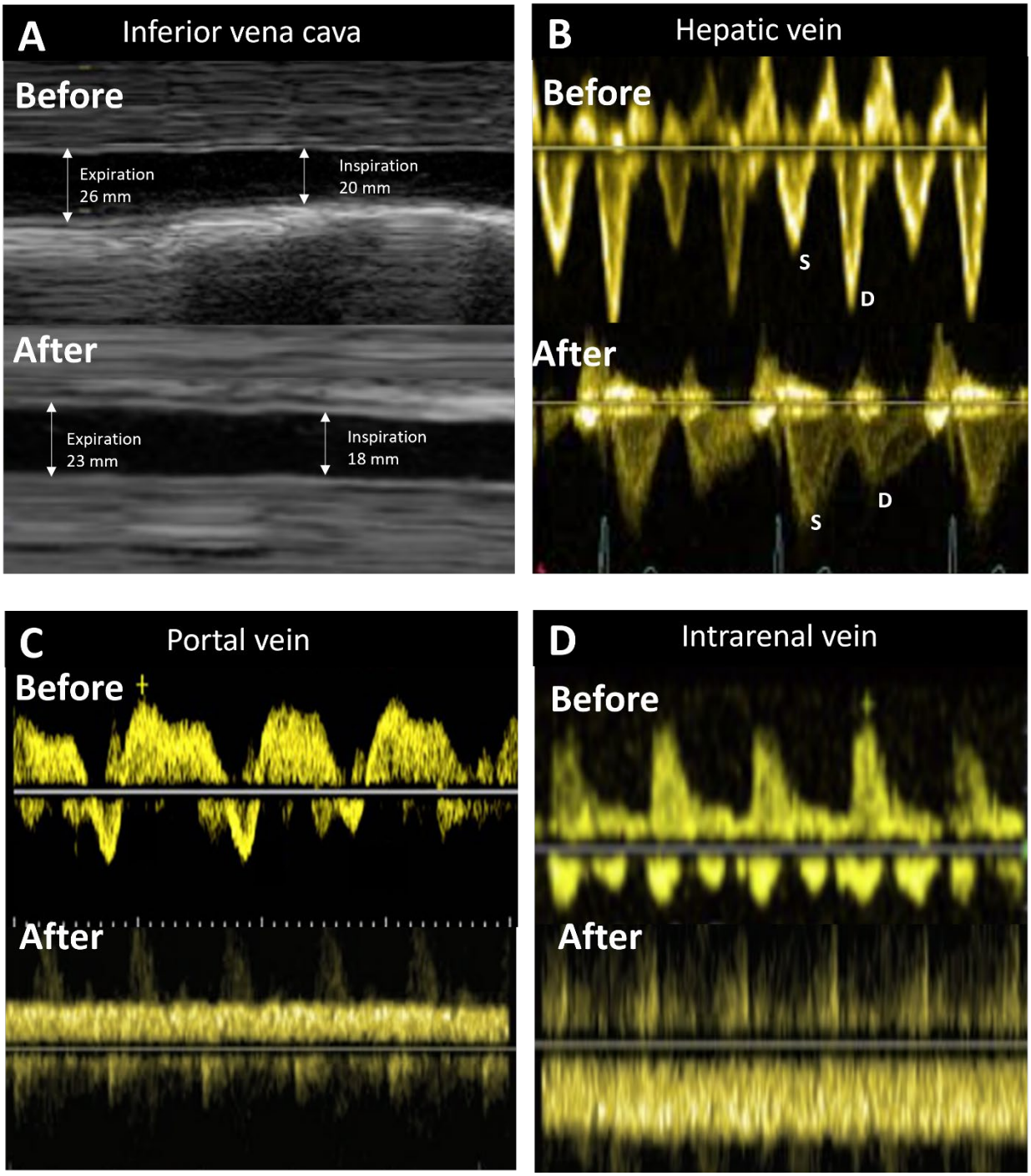
Echocardiography of the portal vein and intrarenal veins **(A)**. Pulsed wave Doppler assessment of the portal vein in a patient without congestion shows a continuous, non-pulsatile flow above the baseline. With progressive venous congestion, the flow becomes pulsatile and flow reversal can occur during systole in patients with severe congestion **(B, left to right)**. Pulsed wave Doppler assessment of the intrarenal vein also shows a continuous, non-pulsatile flow, but below the baseline (blue array). The intrarenal arterial waveform is often displayed above the baseline (red array), which helps to identify the phases of the cardiac cycle. With progressive venous congestion, the flow becomes pulsatile and biphasic, showing separate systolic (S) and diastolic (D) waves. In severe congestion, a diastolic-only pattern is seen, and the flow is completely dependent on right ventricular filling (**C, left to right**).

Figure 4. Vascular ultrasound in a patient without venous congestion



A 67-year-old female presented with bilateral malleolar edema. Transthoracic echocardiography showed a non-dilated right ventricle without tricuspid regurgitation **(A)**. The inferior vena cava was dilated (25mm), but showed a >50% reduction with inspiration **(B)**. Pulsed wave Doppler assessment of the hepatic vein showed a separate systolic (S) and diastolic (D) component with S>D **(C)**. Echocardiographic evaluation of the portal vein **(D)** and the intrarenal vein **(E)** showed a continuous, non-pulsatile flow. Despite the dilated inferior vena cava, there was no further evidence of systemic congestion. Lymphoscintigraphy was performed and confirmed the diagnosis of lymphedema.

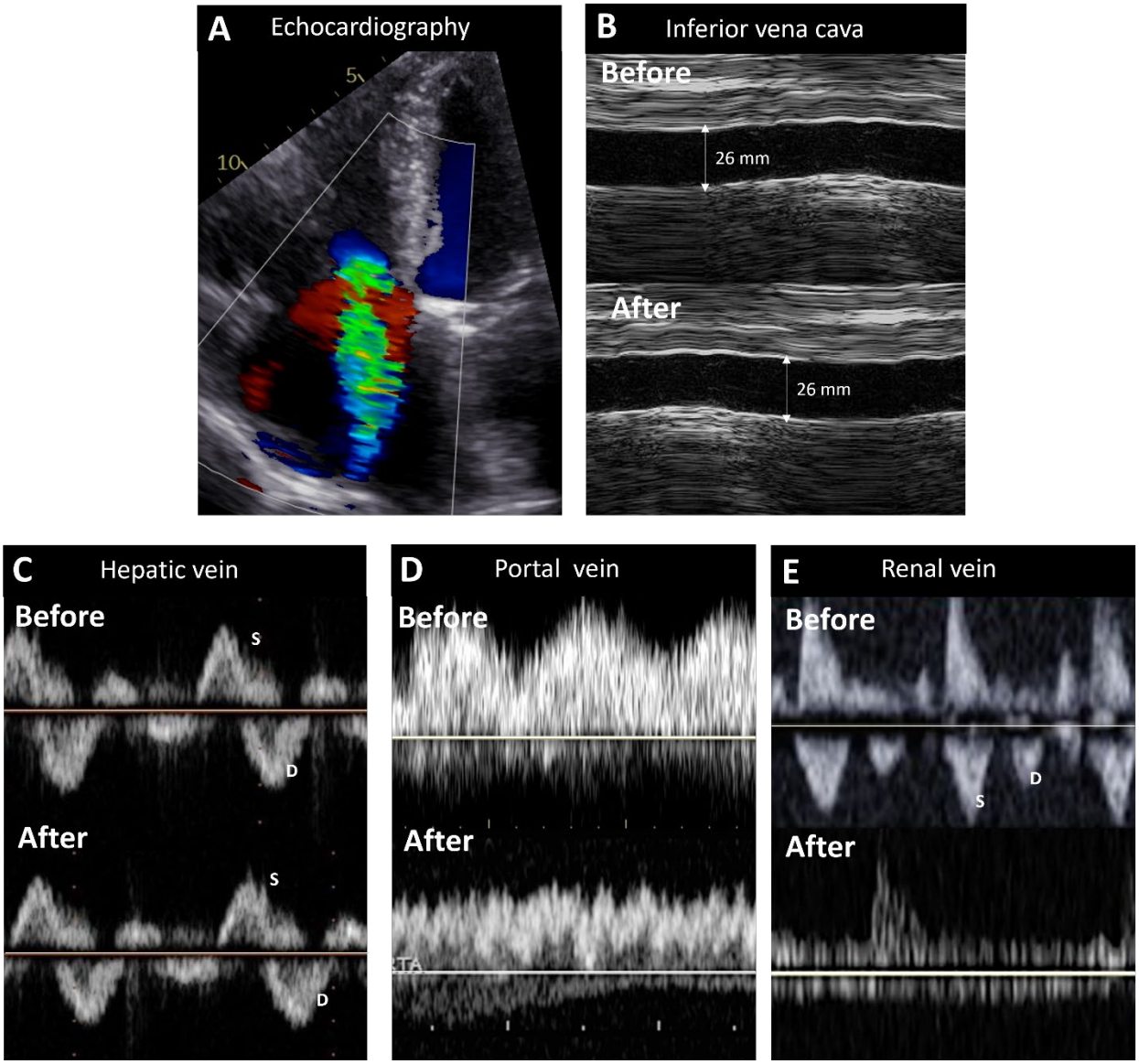
Figure 5. Vascular ultrasound in a patient with right heart failure and venous congestion



A 72-year-old male patient with a reduced left ventricular ejection fraction of 41% and a dilated right ventricle with reduced systolic function, complained of dyspnea and abdominal distension. The inferior vena cava (IVC) was dilated (26mm) with no significant respiratory

variation **(A)**. Although a dilated IVC is suggestive for venous congestion, the IVC is often chronically dilated in the presence of RV dysfunction and the patient can still be fluid responsive. Doppler echocardiography demonstrated a hepatic vein with a S<D pattern **(B)**, a pulsatile flow in the portal vein **(C)** and separate S and D waves in the intrarenal veins **(D)**. These patterns showed that the right atrial pressure was high enough to lead to end-organ congestion. Diuretic therapy was started and a significant improvement in the flow patterns of the hepatic vein, portal vein and intrarenal vein was observed, even though the IVC remained dilated with no significant respiratory variation (**A-D**).

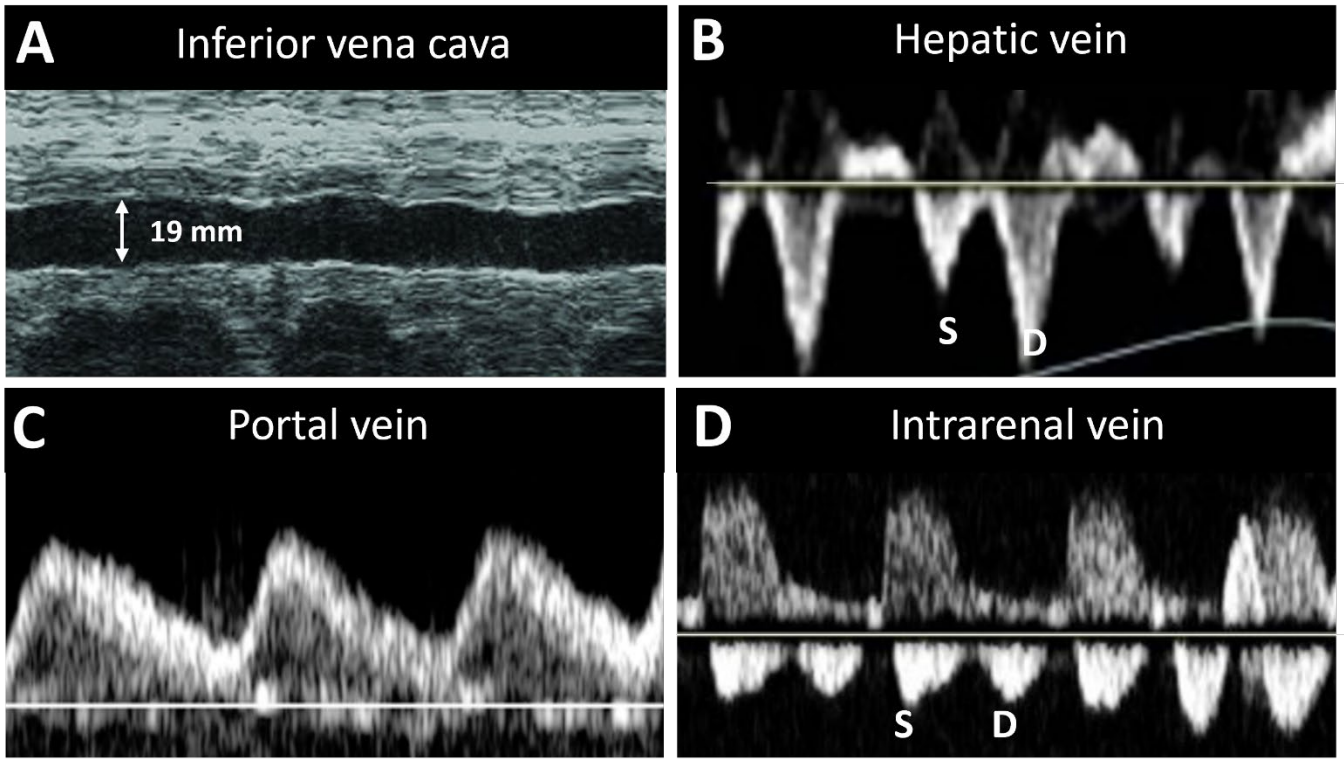
Figure 6. Vascular ultrasound in a patient with significant tricuspid regurgitation



An 83-year-old female with heart failure and preserved ejection fraction presented with severe, secondary tricuspid regurgitation **(A)**. The inferior vena cava was dilated and demonstrated no respiratory variation **(B)**. Pulsed-wave Doppler echocardiography showed systolic flow reversal in the hepatic vein, consistent with severe tricuspid regurgitation **(C)**. In addition, there was a pulsatile flow in the portal vein **(D)** and a biphasic flow pattern with S and D waves in the intrarenal vein **(E)**, consistent with end-organ congestion. After diuretic therapy, the size of the vena cava inferior did not change and systolic flow reversal in the hepatic vein remained present (as can be expected in a patient with persistent severe tricuspid

regurgitation after recompensation), but flow patterns in the portal vein and intrarenal vein normalized, indicating improvement of venous congestion **(B-E)**.

Figure 7. Vascular ultrasound in a patient with hyponatremia receiving diuretics



A 69-year-old male patient with a medical history of heart failure with preserved ejection fraction presented with elevated serum creatinine (1.5 mg/dl; baseline creatinine: 0.8-1.0 mg/dl) and hyponatremia (129 mmol/l). He had been on diuretics for 4 days because of dyspnea on exertion. Chest X-ray showed no signs of pulmonary edema. Based on physical examination and laboratory tests, it was unclear whether the patient had dilutional or depletion hyponatremia. The inferior vena cava was not dilated, but showed no respiratory variation **(A)**. Doppler echocardiography showed an S<D pattern in the hepatic vein **(B)**, pulsatile flow in the portal vein **(C)** and a biphasic flow pattern in the intrarenal vein **(D)**, suggesting congestive kidney injury with dilutional hyponatremia. Water intake was limited and diuretic therapy was continued. His serum creatinine initially worsened to 1.8 mg/dl, but then improved to 0.9 mg/dl with continued diuresis.