Imaging portal venous flow to aid assessment of right ventricular dysfunction

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Pulsatile portal venous blood flow is a sign of portal hypertension originally described as a sign of severity in patients with congestive heart failure (CHF) (Figure). In

this population, it is associated with increased central venous pressure (CVP), worse functional class, and elevated bilirubin, suggesting an impairment in liver

Figure A) To obtain an image of portal venous flow (PVF) using transesophageal echocardiography (TEE), a view of the inferior vena cava (IVC) is obtained using a lower mid-esophageal view with the omniplane transducer at 90° (with permission of CAE Healthcare, Ville Saint-Laurent, QC, Canada). B) The TEE probe is then slowly advanced in a transgastric position while maintaining the liver under the ultrasound beam. Transducer rotation between 50° and 70° will

typically align the right portal vein (PV) with the centre of the ultrasound beam.³ C) A normal portal vein flow velocity is 20 ± 5 cm·sec⁻¹ with minimal pulsatility. D) Abnormal PVF in a 78-yr-old male before mitral valve repair with peak and trough velocities of 18 and 4 cm·sec⁻¹, respectively, resulting in a pulsatility fraction of 78%. A video of the technique to acquire an image of the portal vein can be seen in the video that is available as Electronic Supplementary Material

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function from venous congestion. Portal hypertension resulting from CHF begins with an elevation of the CVP and dilatation of the inferior vena cava (IVC) including its main tributaries such as the hepatic veins. When the dilatation becomes severe, the venous compliance of the IVC is decreased and pressure variations occurring in the right atrium during the cardiac cycle are transmitted through the hepatic sinusoids to the portal system. This results in a decrease in velocities in the portal system or, when severe, in a complete absence or reversal of portal flow during the period of the cardiac cycle corresponding to the v wave on the CVP waveform. Accordingly, Doppler evaluation of the portal flow could be used as a marker of end-organ venous congestion.

The severity of velocity variations in the portal vein correlates with indices of right ventricular dysfunction¹ and is associated with postoperative renal failure in cardiac surgery, possibly through venous congestion.² For the portal flow to be representative of central venous congestion, other causes of portal hypertension such as cirrhosis and portal thrombosis must be absent. Additionally, a pulsatile fraction ($[\text{max-min}] \text{ velocity}/\text{max}$

velocity) of more than 50% has been reported in some individuals with low body mass indices and normal cardiac function. Consequently, other signs of elevated CVP such as IVC dilatation/non-collapsibility and abnormal hepatic vein flow waveform should support this finding.

Conflict of interest Dr. Denault is a Speaker for CAE Healthcare and Masimo.

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