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IMAGING VIGNETTE

Manifestations of Cardiovascular Disorders on Doppler Interrogation of the Hepatic Veins

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SPECTRAL (PULSED-WAVE) DOPPLER INTERROGATION OF FLOW IN THE HEPATIC VEINS (HVs) IS A PART of any comprehensive echocardiographic examination. Analysis of the direction, velocity, and duration of the Doppler waveforms and their phasic response to respiration allows distinguishing normal from abnormal flow patterns and provides diagnostic insights into disorders that affect the function of the right heart.

Together with the superior vena cava, the HVs are the standard conduits for the assessment of systemic venous filling of the right heart due to the wide angle of interrogation of inferior vena caval flow from transthoracic windows. Blood flow in the HVs is dependent on the cardiac cycle and on the function of the right heart and is influenced by the respiratory cycle and the compliance of the liver parenchyma. Therefore, the HV Doppler becomes altered in disease states that either affect right heart function or disturb the cardiac rhythm. Additionally, respiratory and hepatic disorders may incur characteristic changes to the flow in the HVs.

Normal flow in the HVs is phasic and bidirectional, predominantly antegrade, and fluctuations in flow direction and velocity reflect changes in right atrial pressure. As the Eustachian valve does not restrict blood flow into the right atrium, flow in the HVs reflects right atrial filling throughout the cardiac cycle and right ventricular filling during diastole.

We present a series of cases illustrating characteristic HV flow patterns associated with various cardiovascular and related disorders (**Figures 1 to 6**, Video 1). The requirements for optimal recording of the hepatic vein Doppler are listed in **Table 1**.

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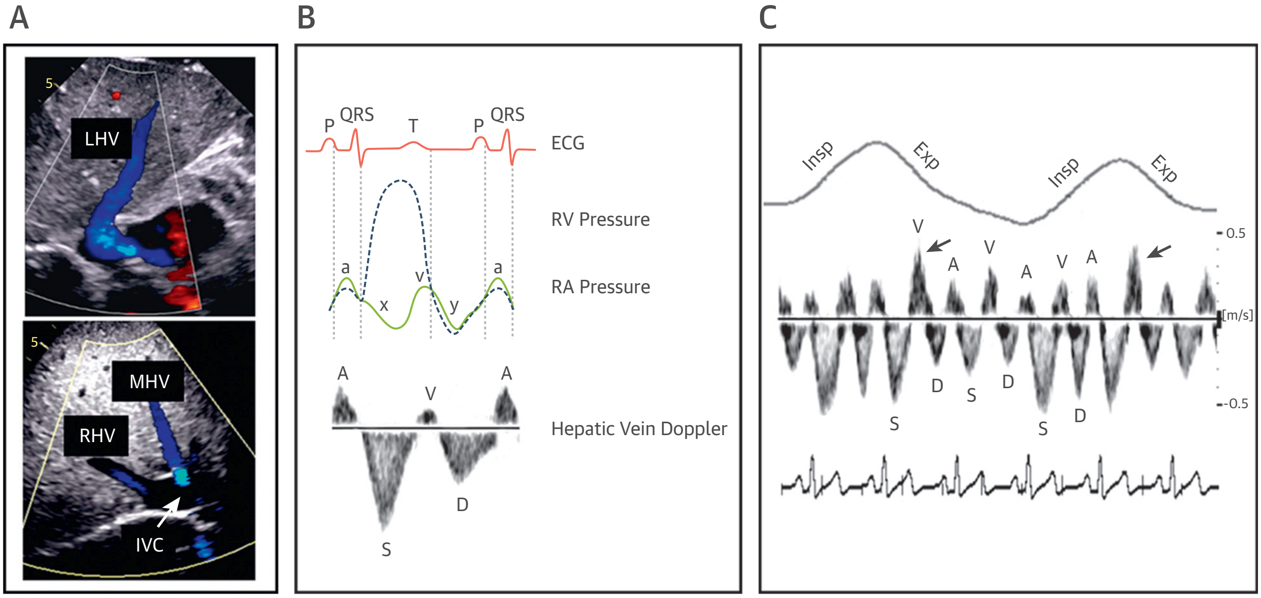
FIGURE 1 HV Anatomy and Normal Flow Profile



(A) Anatomy of hepatic veins (HVs). The HVs drain blood from the liver posteriorly into the retrohepatic inferior vena cava (IVC), 2 to 3 cm caudal to its junction with the right atrium (RA). The left HV (LHV) and middle HV (MHV) drain the left lobe, whereas the right HV (RHV) drains the right lobe of the liver. Most often, the MHV offers the best alignment of flow with the Doppler beam from the subxiphoid window. Video 1 demonstrates blood flow in the MHV (small arrow) and RHV (large arrow). **(B)** Normal HV Doppler. The normal flow profile in the HVs demonstrates phasic and bidirectional waveforms that are temporally related to the waves obtained on pressure recording in the RA. Four distinct Doppler waveforms are often visualized: 1) a large antegrade waveform (negative velocity) noted during early and mid-systole (S-wave) that corresponds to the "*x*" descent on RA pressure. It occurs in response to the fall in RA pressure caused by the increase in volume as a result of atrial relaxation and systolic displacement of the tricuspid annulus towards the RV apex; 2) a small retrograde wave (positive velocity) in late systole (V-wave), that is occasionally absent, and corresponds to the "*v*" wave on RA pressure; 3) an antegrade wave noted in early and mid-diastole (D-wave) that is less prominent than the S-wave and relates to the "*y*" descent in the RA. This wave results from the fall in RA pressure that follows tricuspid valve (TV) opening and emptying of the RA into the right ventricle (RV); and 4) a retrograde wave noted in late diastole (A-wave) that corresponds to the "*a*" wave in the RA. This wave is caused by atrial contraction with rise in RA pressure that exceeds IVC pressure, thus leading to flow reversal in the HVs. **(C)** Effect of respiration on HV flow. Respiration imparts physiological changes to flow in the HVs. Inspiration (Insp), as noted on the respirometer, is associated with a decrease in intrathoracic and intracavitary pressure leading to an increase in blood flow from the vena cava to the right heart. The corresponding HV Doppler shows an increase in the velocity of the forward S and D waves with no increase in flow reversals because the normally compliant right heart chambers are able to accommodate the augmented preload without increase in filling pressures. In contrast, expiration (Exp) is associated with a rise in intrathoracic pressure, leading to a decrease in systemic venous return and thus a reduction in the velocity of the HV forward S and D waves together with an increase in diastolic flow reversals **(arrows)**. The latter are most pronounced on the first cardiac cycle following the onset of expiration due to the rapid shift and sudden increase of the intrathoracic pressure that is generated during early expiration. ECG = electrocardiogram.

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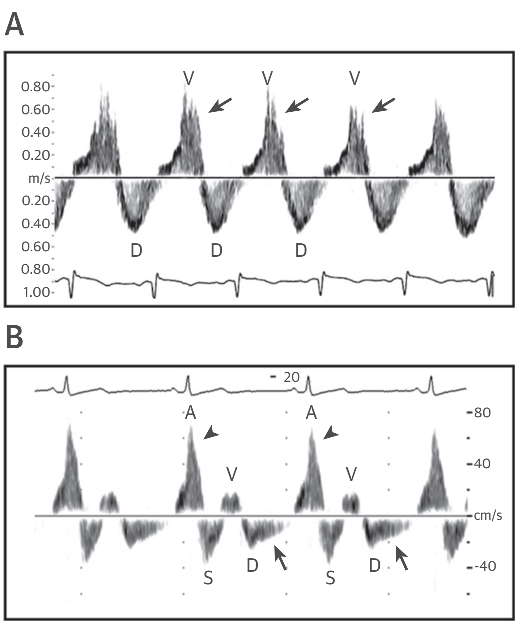


FIGURE 2 **TV Disease**

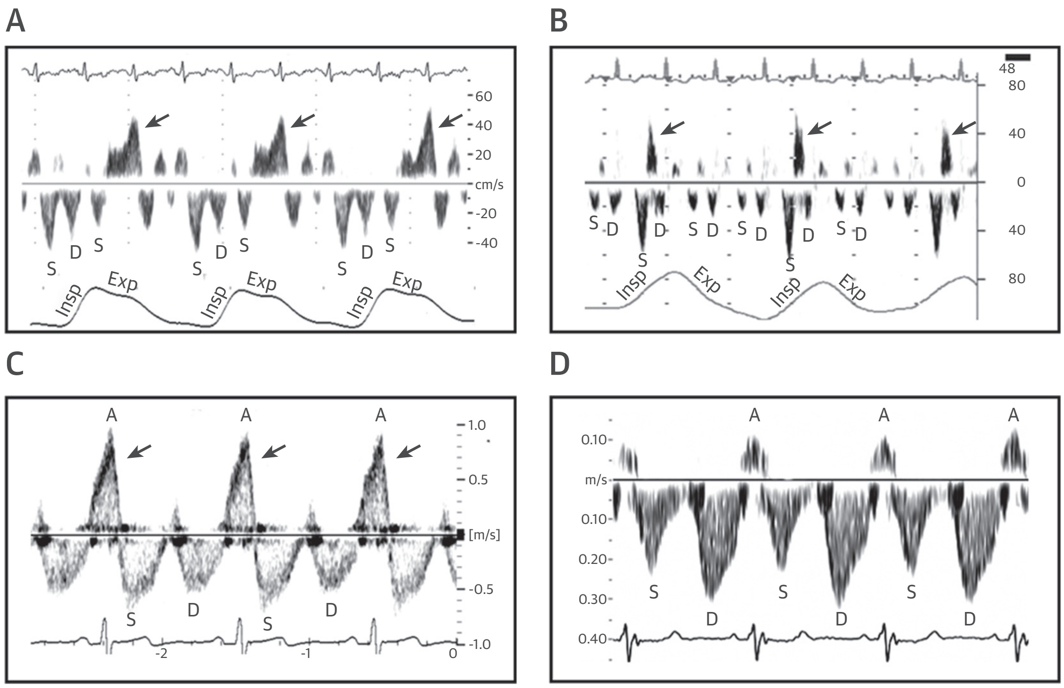
(A) Severe tricuspid regurgitation. The HV Doppler demonstrates holosystolic flow reversal that replaces the forward S-wave and is evident with every cardiac cycle **(arrows)**. This reversal typically peaks in late systole. It results from the transmission of the RV systolic pressure into the RA, a reflection of a prominent "*v*" wave on RA pressure recording. This phenomenon underlies the finding of a pulsatile liver on physical examination. **(B)** Tricuspid stenosis. Significant obstruction to RV inflow is associated with a prolonged deceleration time of the D-wave **(arrows)**, a reflection of the abnormally slow pressure decay across the TV during diastole. Additionally, a prominent A-wave can be evident **(arrowheads)**, indicative of a forceful RA contraction against the stenotic TV. Abbreviations as in Figure 1.

TABLE 1 **Requirements for Optimal Recording of the HV Doppler**

Ensure optimal ECG tracing showing P and QRS waves
Position ultrasound transducer in the subxiphoid region
Visualize the IVC along its long axis by medial angulation of the transducer
Identify the middle and left HVs by counterclockwise rotation of transducer
If the middle and left HVs are not well visualized, use the mid-clavicular or mid-axillary window to identify the right HV
Visualize blood flow in the HVs by color Doppler
Ensure alignment of Doppler signal with flow
Optimize pulse wave Doppler settings
Sample volume size: 2 mm
Sample volume location: 1-2 cm inside the HV
Gain and filter settings
Adjust recording speed 25 mm/s to assess effect of respiration on HV flow 50-100 mm/s to assess waveform morphology and duration
Activate the respirometer if needed to determine the phase of the respiratory cycle (inspiration, expiration, apnea)

ECG = electrocardiography; HV = hepatic vein; IVC = inferior vena cava.

FIGURE 3 Diseases of the Pericardium and Myocardium



Some disease states that typically involve the pericardium and RV myocardium affect the HV flow in a manner that is dependent on the respiratory cycle. Concomitant use of a respirometer is useful to determine the phase of the respiratory cycle (inspiration, expiration, or apnea) in relation to flow. **(A)** Constrictive pericarditis. This condition is characterized by exaggerated interdependence between the left ventricle (LV) and RV due to the noncompliant pericardium. Inspiration leads to augmented RV filling with increase in forward flow in the HVs, leftward shift of the interventricular septum, and subsequent decrease in LV filling. During expiration, the combination of decreased systemic venous return and increase in LV ventricular filling lead to a prominent rightward septal shift. This phenomenon results in a significant expiratory decrease in tricuspid flow and RV filling with associated flow reversals in the HVs. As noted on the respirometer, the onset of expiration leads to prominent diastolic flow reversals **(arrows)** on the HV Doppler. This characteristic pattern is evident with every respiratory cycle. **(B)** Restrictive cardiomyopathy. This entity is characterized by increased stiffness of the RV and LV chambers. The augmented venous return that accompanies the onset of inspiration causes a sharp increase in filling pressures in the right heart due to reduced RV compliance and thus leads to flow reversals in the central veins. The corresponding HV Doppler demonstrates a typical pattern with prominent diastolic reversals noted during inspiration **(arrows)**. **(C)** Elevated RV end-diastolic pressure. Disease states such as pulmonary hypertension and those that affect the RV or the pulmonary valve may lead to an increase in RV end-diastolic pressure. The rapid rise in RV diastolic pressure that follows atrial contraction results in an abrupt closure of the TV before RA pressure has begun to decrease. This phenomenon leads to excessive backflow of blood into the HVs. The corresponding HV Doppler shows a prominent A-wave reversal with high peak velocity and prolonged duration **(arrows)**. **(D)** RV systolic dysfunction. The HV Doppler shows an attenuated S-wave, leading to a dominant D-wave flow pattern. Because the S-wave results, in part, from the apical displacement of the tricuspid annulus during systole and thus reflects the longitudinal function of the RV, a reduction in RV systolic function causes blunting of the S-wave. Abbreviations as in Figure 1.

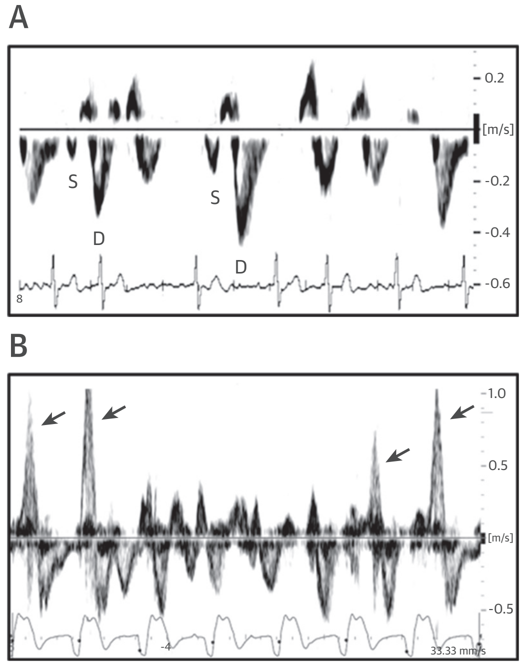


FIGURE 4 Rhythm and Conduction Disorders

(A) Atrial fibrillation. A main hemodynamic feature of atrial fibrillation is the loss of atrial contraction and relaxation. The loss of atrial contraction leads to the absence of the A-wave on the HV Doppler. Because the S-wave results, in part, from RA relaxation, the HV Doppler additionally demonstrates either a blunted or absent S-wave and a dominant D-wave pattern. **(B)** Atrioventricular dissociation. The main finding on the HV Doppler results from the mechanical dissociation between the RA and RV that follows the electrical dissociation. In this patient with asynchronous ventricular pacing and underlying sinus rhythm, the HV Doppler demonstrates intermittent large reversal waves (cannon A waves) **(arrows)**. This phenomenon results from RA contraction occurring during ventricular systole, thus against a closed TV, causing an abrupt rise in RA pressure and backflow into the central veins. The same Doppler pattern occurs in the setting of complete heart block, ventricular tachycardia or premature ventricular beats. Abbreviations as in Figure 1.

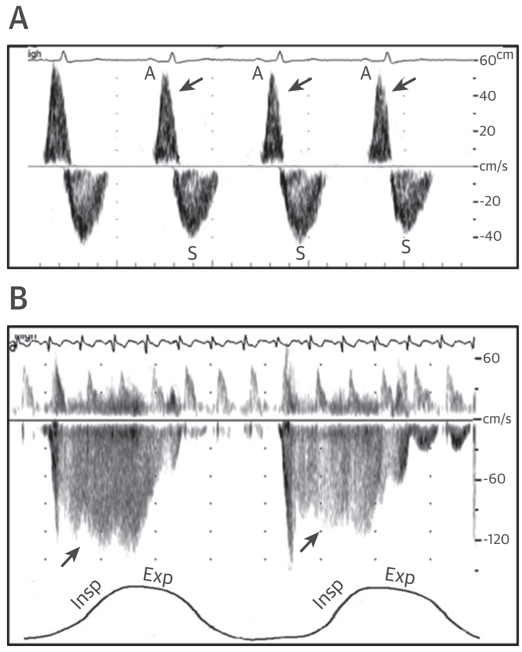


FIGURE 5 Pulmonary Disease

(A) Pulmonary hypertension. In the setting of severe pulmonary hypertension, the HV Doppler demonstrates a prominent A-wave **(arrows)** together with an attenuated or absent D-wave. The augmented A-wave results from elevation in RV end-diastolic pressure that is associated with the increase in RV muscle mass. Blunting of the D-wave reflects an attenuation of the "*y*" descent on RA pressure recording. This phenomenon results from impairment in RV myocardial relaxation and prolongation of the isovolumic relaxation time leading to a delay in TV opening and in the onset of diastole in the right heart. **(B)** Chronic obstructive pulmonary disease. An increase in the respiratory effort and/or the presence of airway obstruction associated with chronic obstructive pulmonary disease results in exaggerated fluctuations in intrathoracic pressure. The more negative pressure that is generated during inspiration and the less negative or even positive pressure that is generated during expiration lead to uncoupling between the HV flow and right heart hemodynamics. Vena caval flow becomes dominated by the changes in intrathoracic pressure irrespective of the phase of the cardiac cycle. The HV Doppler typically demonstrates an increase in forward flow velocities during inspiration with merging of the waveforms and loss of distinct S and D waves **(arrows)**. The resulting signal consists of a high velocity and wide inspiratory wave that encompasses 1 or more cardiac cycles. Abbreviations as in Figure 1.

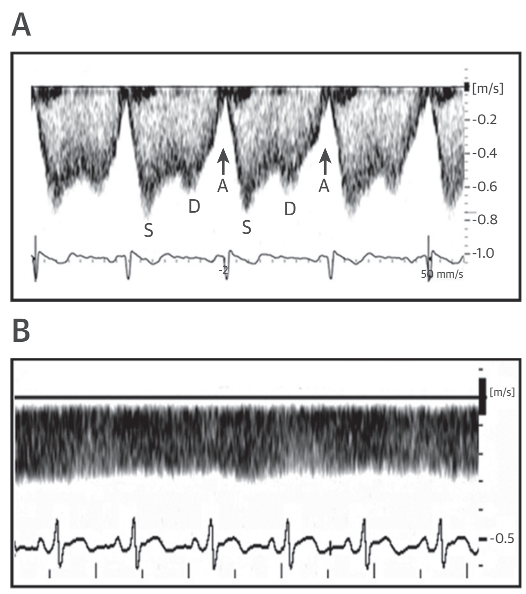


FIGURE 6 Other Conditions

The HVs are thin-walled intrahepatic conduits whose compliance is influenced by the compliance of the surrounding liver parenchyma. Disorders associated with the intrahepatic deposition of fat, fibrosis, or tumor lead to an increased stiffness of liver tissue and thus to a reduction in HV compliance. This phenomenon results in the uncoupling between right heart filling and HV flow, with the latter demonstrating a blunted flow profile and reduced phasicity that is determined by the severity of the underlying liver pathology. **(A)** Obesity. This condition is often associated with fatty infiltration of the liver leading to a reduction in hepatic tissue compliance and partial uncoupling between HV flow and right heart hemodynamics. The resulting HV Doppler demonstrates dampening of waveforms with a biphasic flow pattern showing forward S and D waves with no A- and V-wave reversals. The effect of the A-wave may remain evident on the forward flow velocities **(arrows)**. **(B)** Cirrhosis. Severe liver involvement with fibrosis or tumor leads to complete uncoupling between the HV Doppler and right heart filling with loss of individual waveforms. The resulting HV Doppler demonstrates a characteristic pattern with low velocity monophasic forward flow and no reversals. Abbreviation as in Figure 1.

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APPENDIX For supplemental videos, please see the online version of this paper.