

Understanding the Spectral Doppler Waveform of the Hepatic Veins in Health and Disease¹

CME FEATURE

See accompanying test at http://www.rsna.org/education/rg_cme.html

LEARNING OBJECTIVES FOR TEST 5

After reading this article and taking the test, the reader will be able to:

- Describe the components of a normal multiphasic spectral Doppler tracing of the hepatic veins.
- List the physiologic factors that can cause artifacts in the spectral Doppler waveform of the hepatic veins.
- Identify abnormal spectral Doppler waveforms of the hepatic veins and make a specific diagnosis or differential diagnosis.

TEACHING POINTS

See last page

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Duplex Doppler sonography is a fundamental component of the complete ultrasonographic examination of the liver. Accurate interpretation of the spectral Doppler tracing from the hepatic veins is valuable, as it reflects important cardiac and hepatic physiology. Normally, there are four phases: A, S, V, and D; the S and D waves indicate flow in the antegrade direction toward the heart. In hepatic and cardiac disease, these normal waves may be absent, a finding indicative of flow in a nonphysiologic manner. In addition, transient patient factors such as phase of the respiratory cycle may influence the appearance of the spectral tracing. Familiarity with the normal and abnormal spectral Doppler waveforms from the hepatic veins and knowledge of their respective physiology and pathophysiology provide valuable insights. Systematic analysis of the direction, regularity, and phasicity of the spectral tracing and the ratio of the amplitudes of the S and D waves allows one to arrive at the correct differential diagnosis in most situations.

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Abbreviations: ECG = electrocardiography, IVC = inferior vena cava

RadioGraphics 2009; 29:2081–2098 • Published online 10.1148/rg.297095715 • Content Codes: **GI** **US**

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Introduction

Duplex Doppler sonography of the liver plays an important role in evaluation of the liver (1,2). This noninvasive, ionizing radiation-free, portable technique is not only able to quickly assess patency of the hepatic vasculature but is also able to provide additional parameters such as resistivity and direction of flow. Other articles have reviewed imaging evaluation of the liver vasculature (3,4).

In this article, we focus on duplex Doppler evaluation of the hepatic veins by reviewing how these spectral Doppler waveforms are obtained and displayed. We discuss how patient and technical factors may influence their appearance. Because the appearance of the waveforms is profoundly influenced by both cardiac and hepatic factors, we address the normal physiology of flow through the hepatic veins in relation to the cardiac cycle. We provide a unified approach to interpretation of both normal and abnormal spectral patterns, thus allowing the most specific diagnosis or differential diagnosis to be determined. Finally, we discuss the role of spectral Doppler waveform analysis in liver transplant recipients.

Hepatic Vein Anatomy

There are three major hepatic veins—left, middle, and right—which separate the liver along cranio-caudal planes (Fig 1) (5). The plane of the left hepatic vein separates the lateral left lobe (Couinaud system segments II and III) from the medial left lobe (Couinaud segments IVa and IVb). The plane of the middle hepatic vein separates the right and left lobes of the liver, or more precisely, the medial left lobe and the anterior right lobe (Couinaud segments V and VIII). The plane of the right hepatic vein separates the anterior right lobe from the posterior right lobe (Couinaud segments VI and VII). A significant right inferior hepatic vein complements the right hepatic vein in 30%–61% of cases (6–8). The left and middle hepatic veins join to form a single vein before entering the IVC in 60%–86% of people (5,8,9).

In autopsy studies, between three and 50 minor hepatic veins have been found, which drain predominantly Couinaud segments I, VI, and

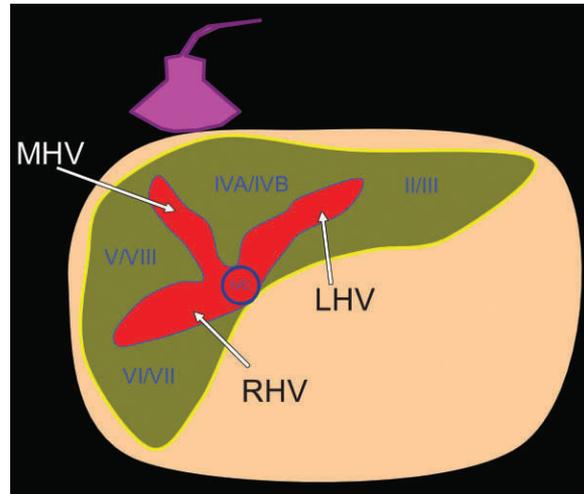


Figure 1. Hepatic veins. Transverse diagram of the liver shows the right hepatic vein (RHV), middle hepatic vein (MHV), and left hepatic vein (LHV) draining into the retrohepatic inferior vena cava (IVC). The hepatic veins divide the liver into Couinaud system segments as indicated. The hepatic veins are interrogated by placing the probe along the anterior or right lateral aspect of the abdomen or lower thorax.

VII and the area just anterior to the retrohepatic IVC (8,9). In one study, one minor vein, two minor veins, or three to four minor veins were found to drain the caudate lobe in 37%, 37%, and 26% of people, respectively (8). These minor hepatic veins are not routinely evaluated with Doppler sonography (2).

Technique

Ultrasonographic (US) images were primarily acquired with a Sequoia 512 (Acuson, Mountain View, Calif) or Philips iU22 (Philips Medical Systems, Andover, Mass) unit. Appropriate acoustic windows were used to image the hepatic veins by using intercostal, subcostal, or transabdominal approaches (10). Gray-scale B-mode evaluation of the liver was first performed, followed by color and spectral Doppler evaluation. Doppler parameters were optimized by following the principles and techniques described in excellent reviews by Kruskal et al (11) and Rubin (12). Concurrent electrocardiographic (ECG) and spectral Doppler recording was performed on the Philips machine by using a standard six-pin, three-lead ECG cable.

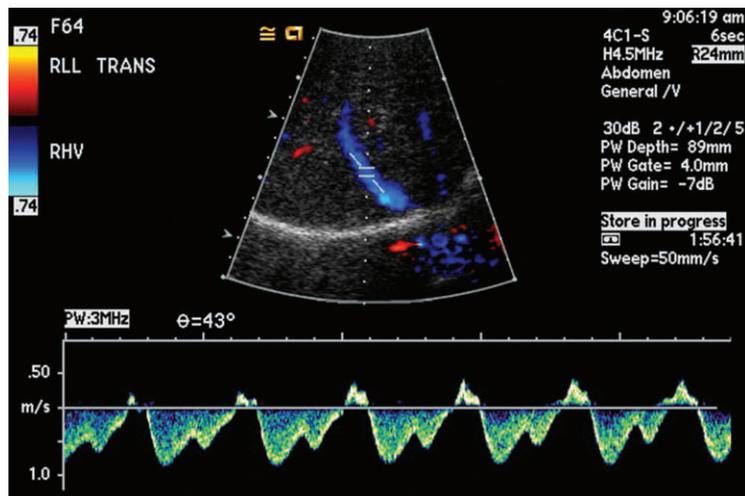


Figure 2. Duplex Doppler display on the Sequoia 512 unit. The left portion of the two-dimensional display has the color scale for the color Doppler image; flow toward the transducer is indicated with shades of red and yellow, whereas flow away from the transducer is indicated with shades of blue and green. The upper right portion of the display lists various technical parameters, including which transducer was used. The central portion of the display has the color Doppler image, with the Doppler gate over the right hepatic vein in this case. Note that flow is appropriately away from the transducer toward the retrohepatic IVC. The bottom portion of the display shows the spectral Doppler waveform, in which blood flow velocity (in meters per second or centimeters per second) within the Doppler gate is plotted versus time.

Duplex Doppler Display of the Hepatic Vein

There are many components found on the duplex Doppler display (Fig 2). The two-dimensional display is composed of a gray-scale B-mode image, usually with color Doppler overlay. Convention dictates that flow toward the transducer is depicted in red and flow away from the transducer is in blue; thus, the normal hepatic veins, which head posteriorly toward the retrohepatic IVC, are depicted in blue. (The color scheme convention should always be confirmed on the color bar of the display, as the color scheme can easily be reversed with an inadvertent push of the “invert” button.) A Doppler gate is then placed over the sample volume within the vessel that the sonographer wants to interrogate, to generate a spectral Doppler tracing.

The bottom portion of the duplex display has the spectral Doppler tracing, where the mag-

nitude of flow velocity is plotted against time (Fig 2). According to convention, flow above the baseline indicates flow toward the transducer and flow below the baseline indicates flow away from the transducer. (This relationship can also be instantly reversed by pressing the invert button, which inverts the tracing and places the word “Inverted” or “Inv” somewhere on the display. Before interpretation of the tracing, it should be clear whether the spectrum has been inverted.)

Physiology of the Normal Hepatic Vein Waveform

The normal hepatic vein waveform, despite commonly being described as triphasic, has four components: a retrograde A wave, an antegrade S wave, a transitional V wave (which may be antegrade, retrograde, or neutral), and an antegrade D wave (13). Let us look at how these waves are

Teaching Point

created by the blood flow through the hepatic veins related to the cardiac cycle (Figs 3, 4) (14).

The A wave corresponds to atrial contraction. With the tricuspid valve open, blood is propelled in two directions: antegrade toward the right ventricle and retrograde toward the IVC and into the hepatic veins. At the end of atrial systole, peak retrograde velocity away from the heart is achieved. As ventricular systole commences, the tricuspid valve closes and the retrograde velocity toward the hepatic veins begins to decrease and approach the baseline. There is conflicting literature as to whether a normal hepatic vein A wave must be retrograde with the spectral tracing peaking above the baseline. Bolondi et al (15) reported that the A wave must be retrograde in normal patients, whereas Pedersen et al (16) showed that an A wave that remains below the baseline may also be found in normal people.

During ventricular systole, not only do the ventricular walls contract to propel blood into the right ventricular outflow tract, but there is also movement of the tricuspid valve annulus toward the cardiac apex. These actions create a relative negative pressure in the atrium, causing antegrade blood flow out of the liver and into the heart during the S wave. In the normal heart, the largest amount of antegrade blood flow is during this phase (17).

The V wave corresponds to atrial overfilling. As the ventricular contraction becomes less intense and the closed tricuspid valve begins to return to its original position, the atrium fills and blood flow velocity toward the heart decreases. The peak of the V wave may be below, at, or above the baseline, depending on whether there is antegrade flow throughout, transient equilibrium with no flow, or transient retrograde flow, respectively. Note that the term *triphasic* does not include the V wave, perhaps because this wave represents only a transitional phase.

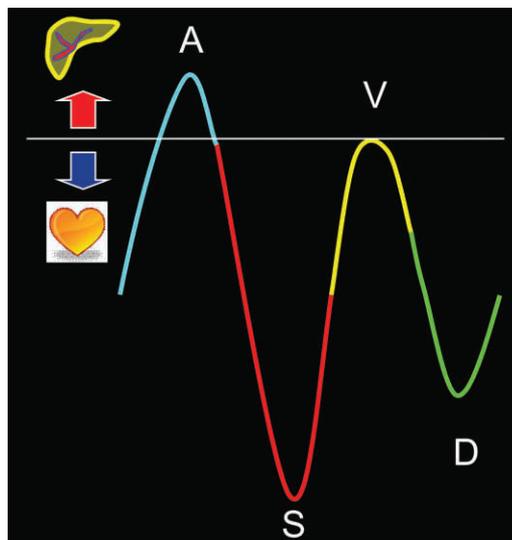


Figure 3. Normal triphasic Doppler waveform. Diagram shows the four waves in the normal spectral Doppler waveform. The A wave (blue), which is above the baseline, has a retrograde component of flow toward the liver. The S (red) and D (green) waves are below the baseline with flow antegrade toward the heart. The V wave (yellow) is a transitional wave, which in the normal patient may peak below, at, or above the baseline.

The D wave begins as the tricuspid valve opens. During cardiac diastole, the right atrium and ventricle fill passively, with antegrade flow of blood from the liver into the heart. In the normal patient, the velocity of this passive flow is almost always lower in magnitude than the velocity during the S wave.

A normal variant, termed the *C wave*, can cause a small retrograde spike following the A wave (Fig 5; also demonstrated in the spectral tracing in Fig 2) (17). As atrial systole ends and ventricular systole commences, the tricuspid valve closes. The tricuspid annulus begins to move toward the cardiac apex and the retrograde velocity of flow toward the liver begins to decrease. However, before the pulmonic valve opens, the pressure in the ventricle increases with

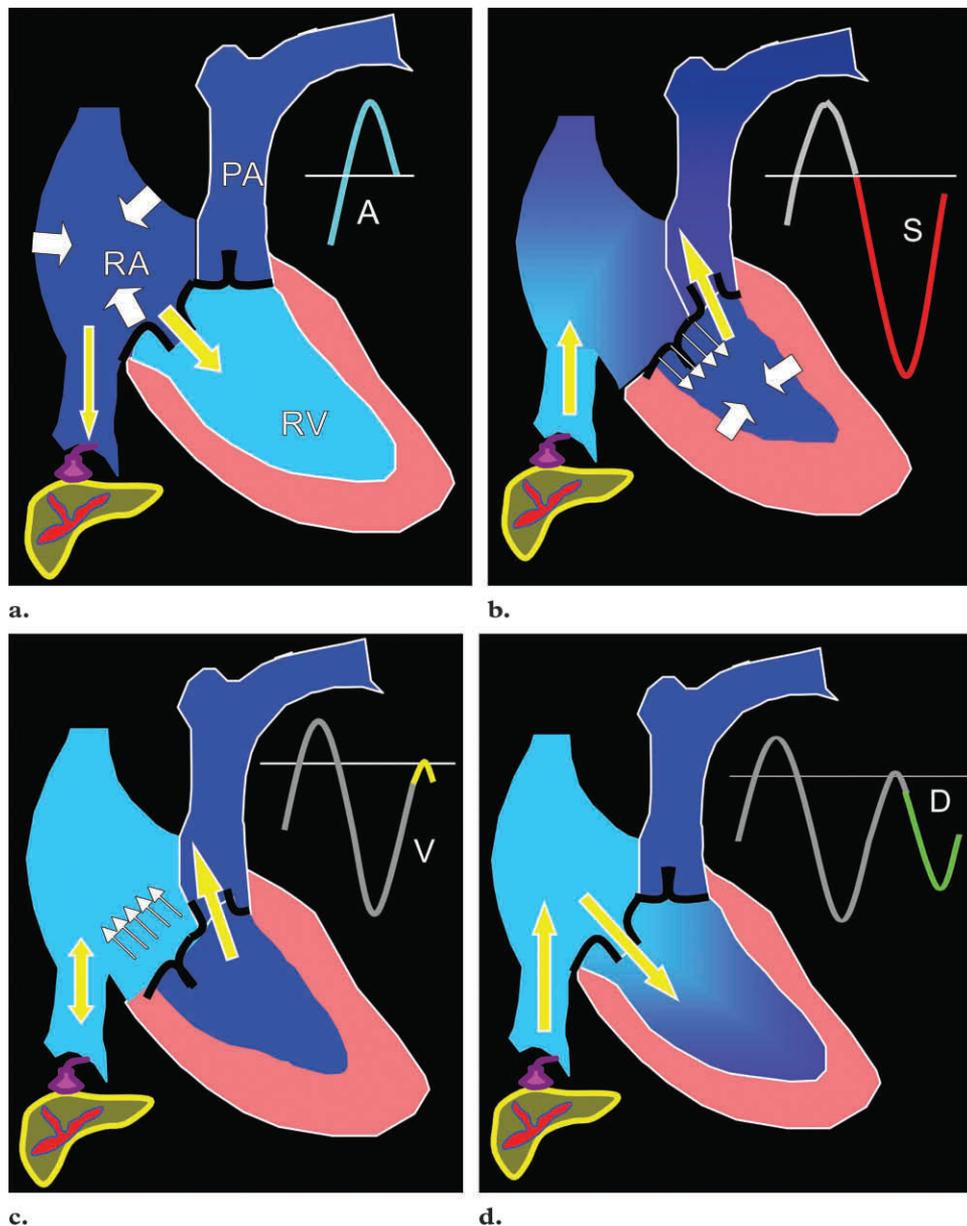


Figure 4. Diagrams of blood flow through the right side of the heart and its influence on the spectral Doppler waveform. The inset shows the part of the waveform formed by each portion of the cardiac cycle. White arrows = wall motion, yellow arrows = blood flow. **(a)** During atrial systole, a portion of the blood flow is toward the liver, yielding a retrograde A wave. *PA* = pulmonary artery, *RA* = right atrium, *RV* = right ventricle. **(b)** During ventricular systole, the tricuspid annulus moves toward the cardiac apex (thin white arrows), causing suction of blood into the right atrium from the liver. The result is the dominant antegrade S wave. **(c)** As the tricuspid valve returns to its resting position, the velocity of blood out of the liver decreases and a transitional equilibrium is reached, yielding the V wave. **(d)** During diastole, with the heart relaxed and the tricuspid valve open, blood flows passively from the liver into the heart, yielding the antegrade D wave.

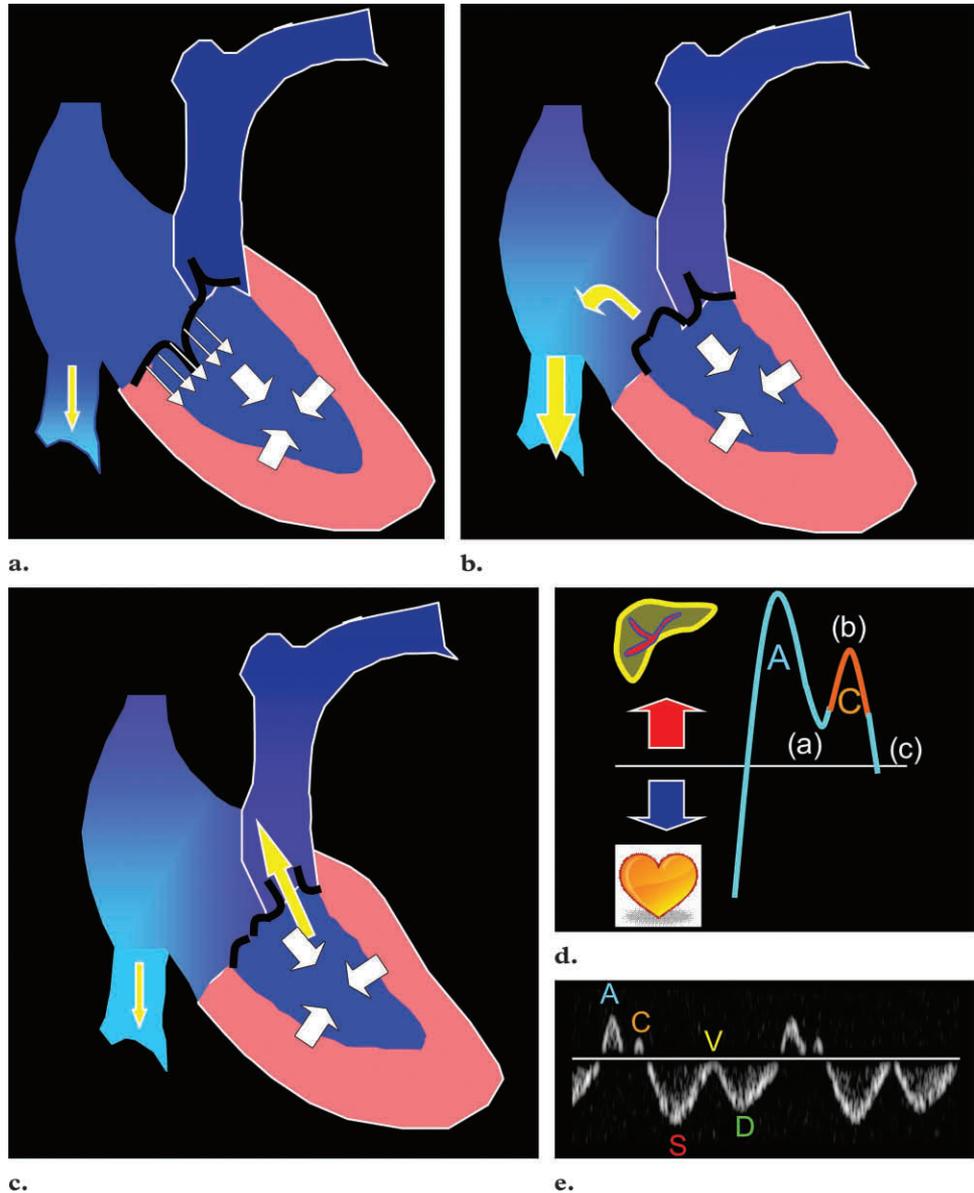
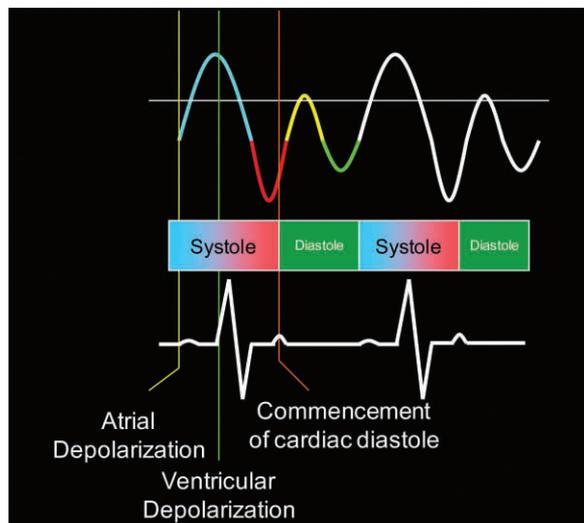
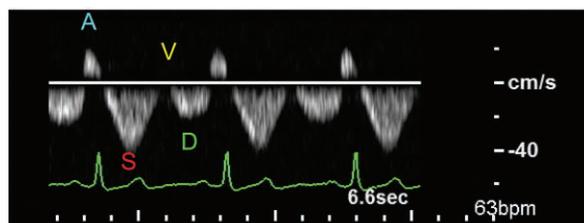


Figure 5. Physiology of the normal variant C wave. White arrows = wall motion. **(a)** As ventricular systole begins, retrograde velocity begins to decrease (small yellow arrow in the IVC), causing the retrograde velocity on the spectral tracing to decrease, as in a regular A wave. **(b)** With the pulmonic valve closed, the ventricular pressure increases before ejection of blood into the pulmonary artery. The tricuspid valve may bulge into the right atrium, causing a transient pulse of increased flow in the retrograde direction (large yellow arrow in the IVC). This pulse is termed the *C wave*. **(c)** When the pulmonic valve opens, blood is ejected from the ventricle (large yellow arrow), and the retrograde velocity progressively decreases (small yellow arrow in the IVC) as part of the normal conclusion of the A wave. **(d)** Diagram shows the A and C waves. Portions of the waveform that correspond to the physiologic events seen in **a**, **b**, and **c** are indicated. **(e)** Spectral tracing shows a C wave in a normal patient. The component waves are labeled.



a.



b.

Figure 6. Spectral Doppler tracing correlated with the cardiac cycle by means of concurrent ECG tracing. (a) Diagram shows the four phases of the spectral Doppler waveform correlated with a concurrently obtained ECG tracing. The A wave begins concurrently with the ECG P wave, whereas the peak of the A wave corresponds to the beginning of the QRS complex. The V and D waves follow the ECG T wave. (b) Spectral Doppler tracing of the hepatic vein with a concurrent ECG tracing obtained in a patient.

continuing contraction of the ventricle, causing a transient bulging of the tricuspid valve into the right atrium. This bulging creates a momentary retrograde pulse toward the liver, causing the C wave. When the pulmonic valve opens and blood is ejected from the right ventricle into the pulmonary outflow tract, the bulge in the tricuspid valve is relieved. Flow into the heart then resumes as usual during the S wave.

Assignment of Waves in Hepatic Vein Spectral Tracing

There are three ways of correctly assigning the various phases of the spectral waveform. First, a component of the D wave is always antegrade. Second, the position of the A wave peak is usually higher than that of the V wave. This observation is not universal and is obviously not helpful when the A and V waves are equal in magnitude. Still, this method is the one used most often. The third and most reliable method of correctly assigning the spectral Doppler waves is obtaining a concurrent ECG tracing with the spectral Doppler tracing (18). Unfortunately, this is usually not available.

Teaching Point

By using a concurrent ECG tracing, the waves in the hepatic vein spectrum can be most reliably correlated with the cardiac cycle (Fig 6). Atrial depolarization, which causes the P wave on the ECG, corresponds to the beginning of the spectral Doppler A wave. The peak of the A wave occurs within 150 msec of the QRS complex, which corresponds to the beginning of ventricular systole. The Doppler V and D waves are seen following the ECG T wave during cardiac diastole.

Effect of Patient Factors on the Appearance of the Spectral Doppler Waveforms

Patient factors play an important role in the appearance of the hepatic vein spectral Doppler waveform (18–20). In Figure 7, note how different spectral Doppler tracings are obtained in the same patient under different respiratory conditions. Triphasic waveforms are produced during end inspiration and with quiet breathing. During these two states, there is sufficient blood return to the heart through the hepatic veins to produce a normal waveform. Nearly monophasic waveforms are produced during end expiration and when the Valsalva maneuver is performed. In these two situations, there is either insufficient blood return to the heart through

Teaching Point

Figure 7. Effect of patient factors on the appearance of the spectral Doppler waveform. Four spectral Doppler waveforms were obtained from the same young healthy volunteer within approximately 5 minutes. The waveforms are markedly blunted when the subject performs the Valsalva maneuver (a) or halts respiration in end expiration (b). The waveforms have a normal appearance during quiet respiration (c) and when the subject halts respiration in end inspiration (d). Note the normal variant C waves in the end inspiration spectrum.

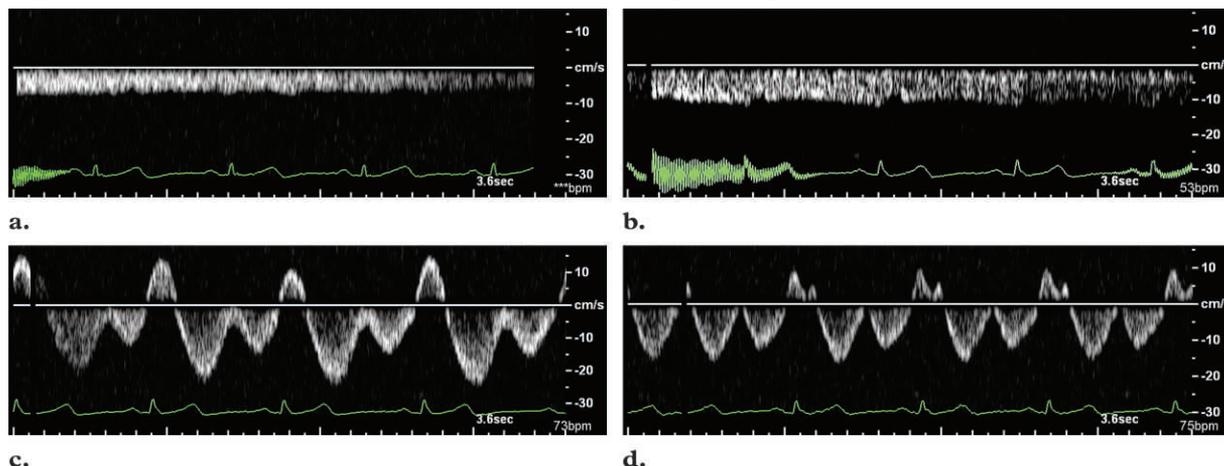
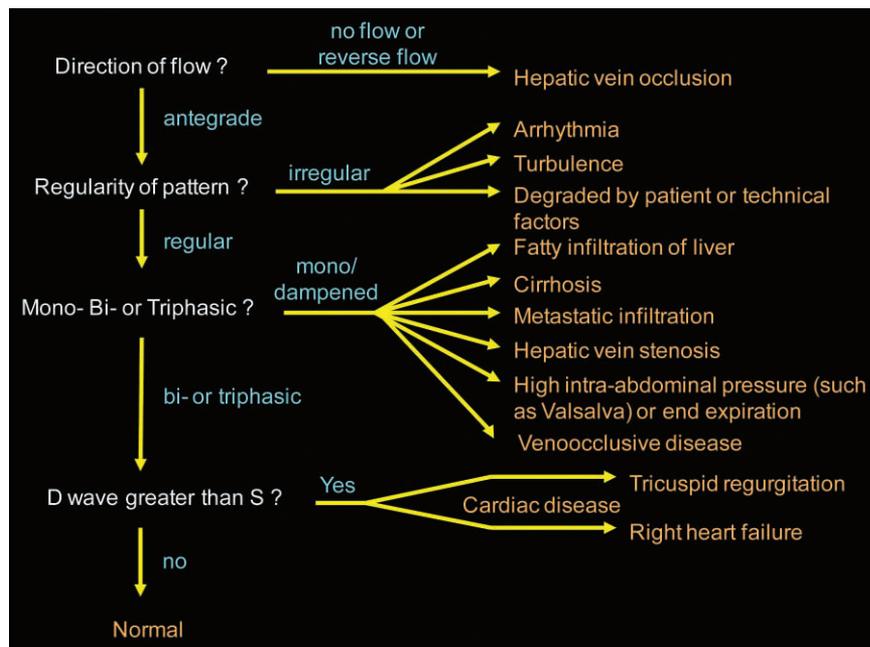


Figure 8. Algorithm for systematic evaluation of hepatic vein spectral waveforms. Systematic evaluation of four factors can lead to determination of a specific diagnosis or differential diagnosis. Note that the listed conditions may coexist, yielding a more complex and perhaps a noninterpretable waveform.

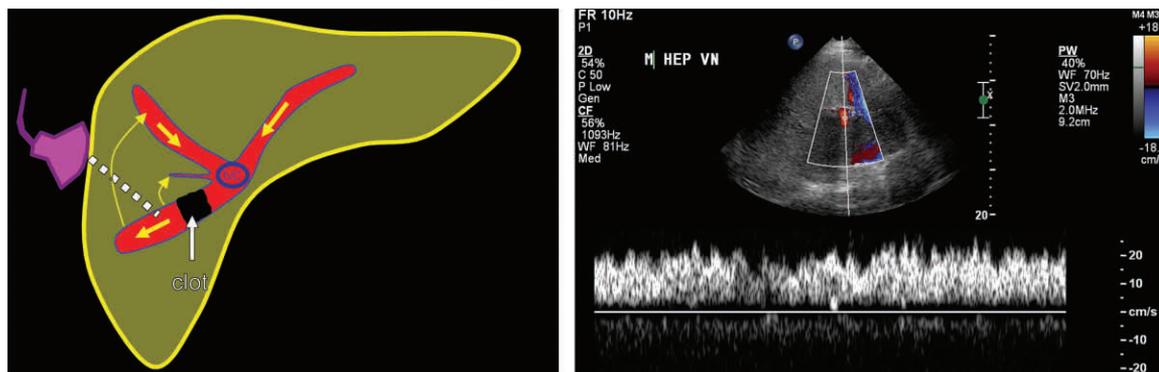


the hepatic veins or a pseudo-obstruction at the level of the diaphragm, giving blunted waveforms in the antegrade direction.

Clinically, peritonitis and tense ascites create a pseudo-Valsalva maneuver state that may blunt

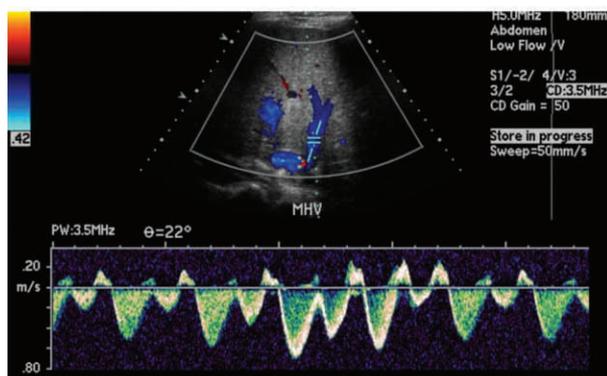
the hepatic vein waveforms, giving a monophasic appearance. Likewise, when patients are asked to hold their breath to obtain the spectral waveform, they may inadvertently perform a Valsalva maneuver, thereby blunting their hepatic vein waveforms (14). Therefore, tracings should always be obtained with the patient in end inspiration or during quiet breathing.

Figure 9. Reversed flow in the hepatic vein due to obstruction. **(a)** Diagram shows a clot in the right hepatic vein. When an area peripheral to the obstruction is interrogated, a monophasic waveform in the reverse direction (hepatopetal) may be present because blood is flowing retrograde toward another route to exit the liver, either via one of the main hepatic veins or via a minor hepatic vein. **(b)** Duplex Doppler image of the middle hepatic vein, obtained 1 day after liver transplantation, shows evidence of reverse flow. A large hematoma in the Morison pouch caused extrinsic compression or kinking of the vein, leading to occlusion. The vein is red on the color Doppler display, a finding indicative of hepatopetal flow (away from the IVC). On the spectral Doppler tracing, there is monophasic turbulent flow above the baseline, a finding also indicative of hepatopetal flow. The extrinsic compression was relieved by surgical evacuation of the hematoma. **(c)** Duplex Doppler image of the middle hepatic vein, obtained in the same patient as in **b** after surgical evacuation of the hematoma, shows normal antegrade flow and a normal triphasic waveform.



a.

b.



c.

After exercise, velocity increases within the hepatic veins, but the waveform morphology remains unchanged (19). Fasting has not been shown to have a significant effect on the hepatic vein waveform morphology (19). Doppler waveforms in the hepatic veins have been shown to become blunted during pregnancy (21). These changes revert to a normal appearance in most patients by 8 weeks after childbirth but persist in a minority of cases (22).

Systematic Analysis of the Hepatic Vein Spectral Tracing

Evaluation of the hepatic vein spectral Doppler tracing involves asking four questions (Fig 8): What is the direction of flow? Is the spectral wave pattern regular or irregular? What is the waveform phasicity? (In other words, is the waveform monophasic or dampened, or is it multiphasic?) What is the relationship of magnitude between the S and D waves?

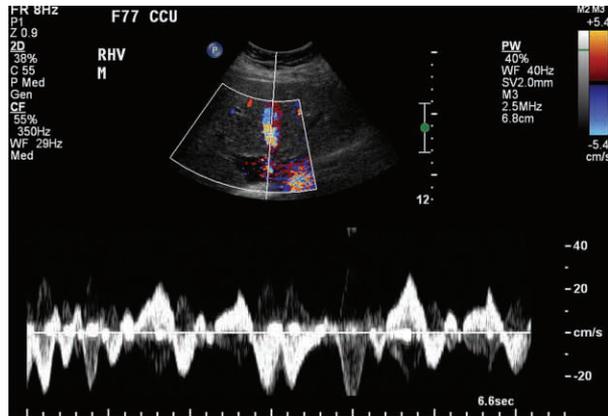
Teaching Point

What Is the Direction of Flow?

If there is no flow or the direction of flow is monophasic in the retrograde direction, the only condition to be considered is hepatic vein obstruction, such as in Budd-Chiari syndrome. Interrogation of the clot will demonstrate no flow; however, interrogation of the region between the periphery of the liver and the clot will show reversed flow, as blood is flowing in the reverse direction to exit the liver via another collateral pathway that leads into the IVC (Fig 9) (23–25). A turbulent waveform will be produced in incomplete obstruction or when interrogating the region between the clot and the IVC (26).

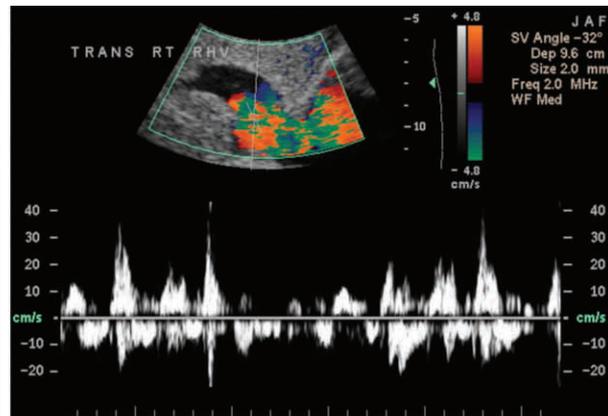
Is the Spectral Wave Pattern Regular or Irregular?

If there is flow in the correct direction, the next question to be asked is whether the wave pattern is regular or irregular. If it is irregular, the possibilities that should be considered are arrhythmias (27), turbulent blood flow, or degradation of the

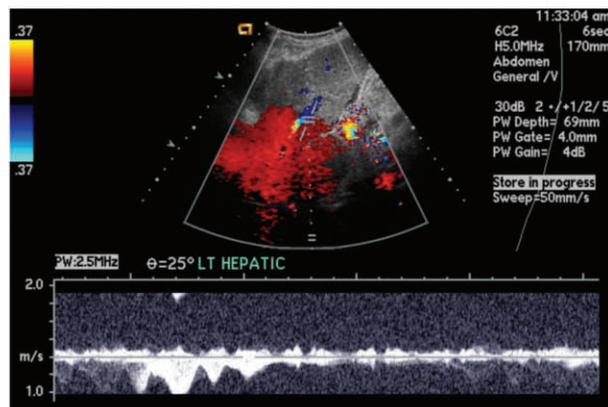


a.

Figure 10. Three causes of irregular spectral Doppler waveforms. (a) Duplex Doppler image of the right hepatic vein, obtained in a coronary care unit patient with atrial fibrillation, shows an irregular waveform. (b) Duplex Doppler image, obtained in a patient with a clot in the right hepatic vein, shows turbulent flow between the clot and the IVC; the turbulent flow causes an irregular waveform. (c) Duplex Doppler image of the left hepatic vein, obtained in a sedated intensive care unit patient receiving mechanical ventilation, shows an irregular waveform.



b.



c.

waveform by patient or technical factors (Fig 10). A degraded waveform is commonly found in sedated patients, patients receiving mechanical ventilation, and patients who are short of breath and cannot cooperate with breathing instructions.

Is the Waveform Monophasic or Dampened, or Is It Multiphasic?

Once we have established that the flow is regular and antegrade, we must evaluate whether the waveform is dampened (Fig 11). Dampening is indicative of relative venous outflow obstruction, which may be due to fatty infiltration of the liver (28), cirrhosis (29,30), or metastatic infiltration (30). Two sources indicate that dampening may also be found in veno-occlusive disease (3,31), which may be seen as a complication of bone marrow transplantation, for example. Other causes include hepatic vein stenosis and physiologic factors, such as high intraabdominal pres-

sure due to a Valsalva maneuver or suspending respiration in end expiration, as discussed earlier.

An interesting point is that the dampened waveform seen in cirrhosis is reversible with terlipressin (a vasopressin analog) treatment (32). Reversibility was also reported in patients with portal hypertension treated with propranolol (33).

What Is the Relationship of Magnitude between the S and D Waves?

The approach to evaluation of the S and D waves requires some introduction. In the normal hepatic vein waveform, the S wave is larger than or equal to the D wave. This is expected, considering that the powerful systolic movement of the tricuspid annulus toward the cardiac apex causes a large antegrade rush of blood toward the heart.

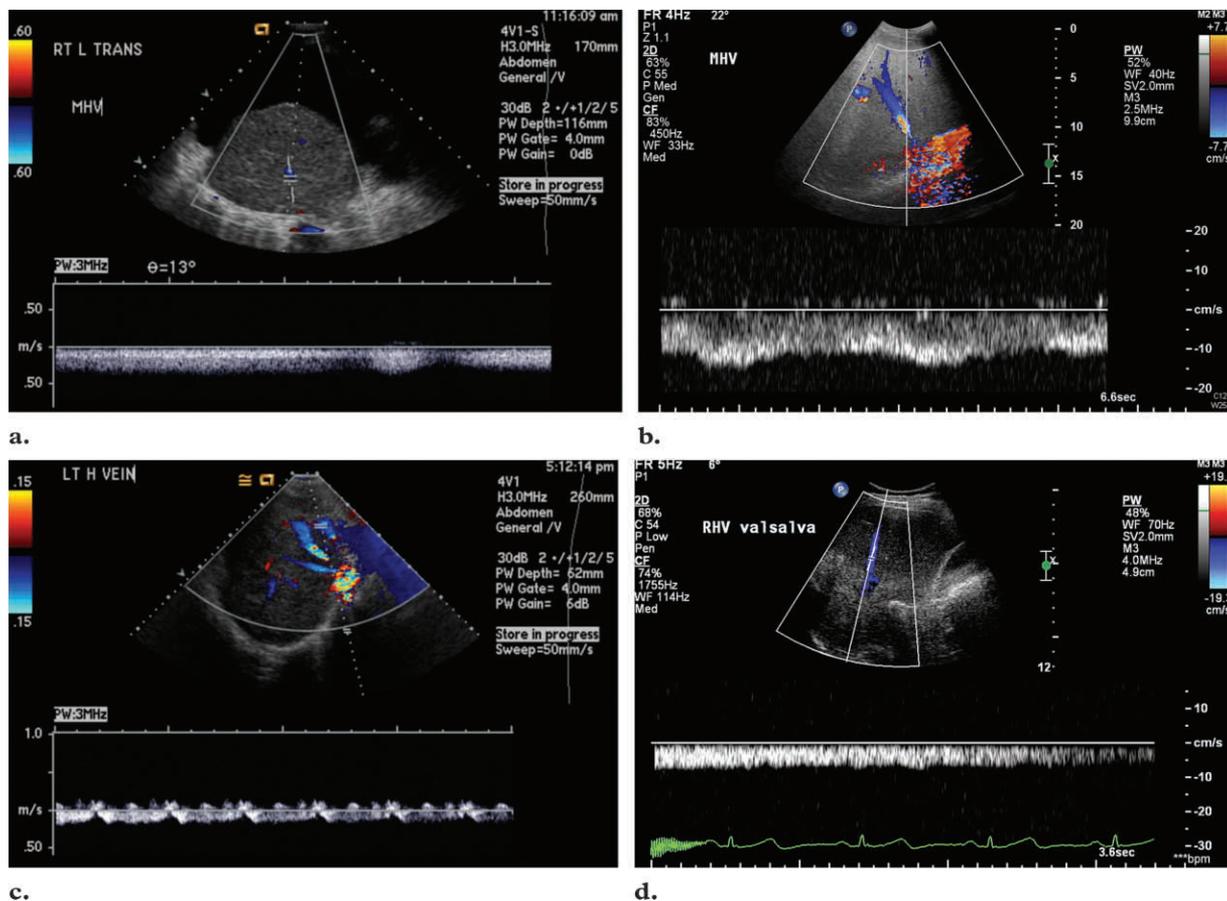


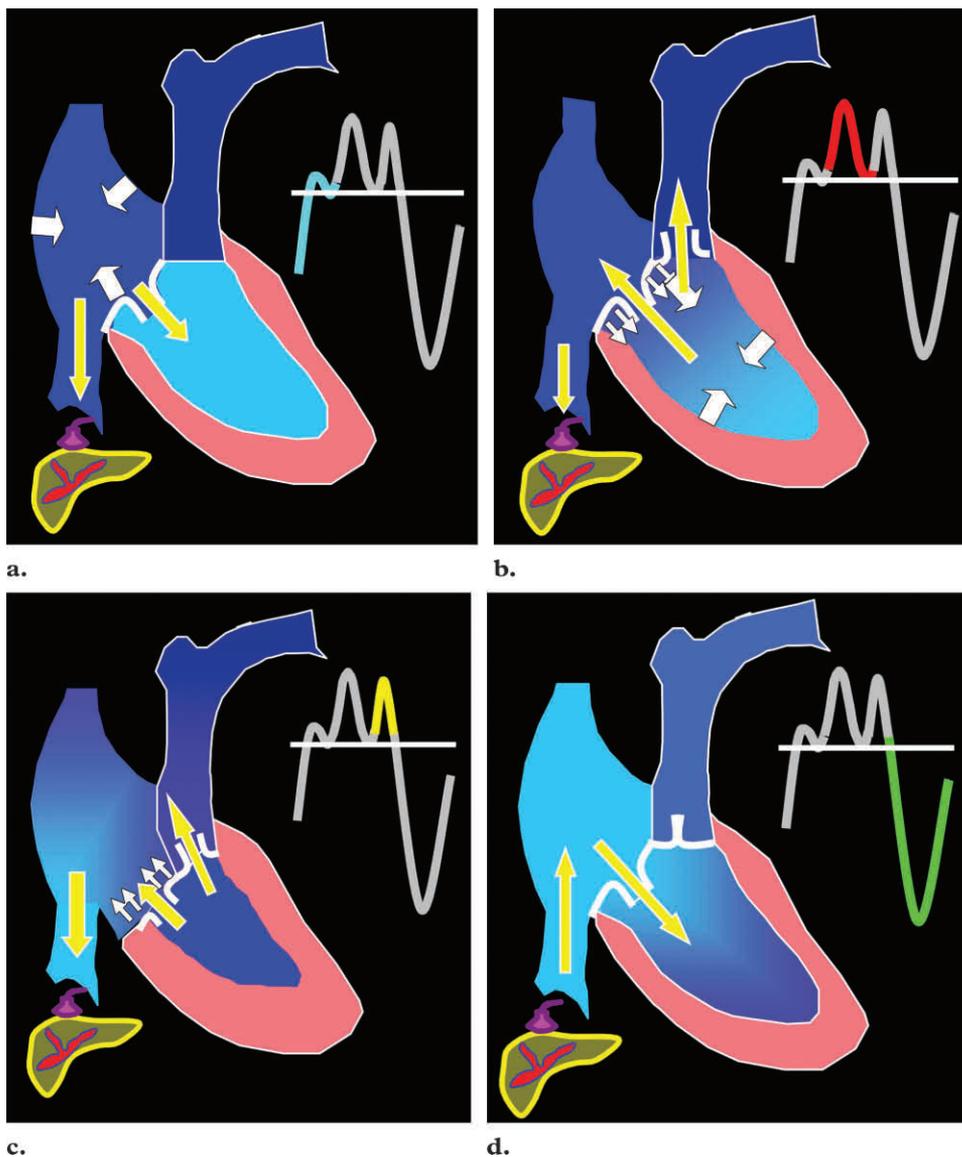
Figure 11. Four causes of monophasic or blunted waveforms. (a) Duplex Doppler image of the middle hepatic vein, obtained in a patient with cirrhosis, shows a monophasic antegrade waveform. On the color Doppler display, there is associated ascites anterior to the liver. (b) Duplex Doppler image of the middle hepatic vein, obtained in a patient with fatty infiltration of the liver, shows a blunted waveform. On the color Doppler display, the liver is hyper-echoic owing to fat infiltration. (c) Duplex Doppler image of the left hepatic vein, obtained in a patient with diffuse large B-cell lymphoma, shows a blunted waveform. (d) Duplex Doppler image of the right hepatic vein, obtained in a volunteer performing the Valsalva maneuver, shows a blunted waveform.

In heart disease, which for our purpose refers to right-sided heart failure or tricuspid regurgitation, the right side of the heart does not function well and greater antegrade blood flow will be seen during diastole (27,34).

Let us first look at severe tricuspid regurgitation as a paradigm to understand the changes that may occur in the various grades of tricuspid regurgitation (17) and right-sided heart failure (Fig 12). As systole begins, the atrium contracts and forces blood antegrade into the ventricle as

well as retrograde toward the liver, producing a small retrograde A wave. During ventricular systole, the ventricle contracts and the tricuspid annulus moves toward the cardiac apex as usual. However, owing to tricuspid regurgitation, blood is forced retrograde into the atrium, IVC, and liver (as well as into the right ventricular outflow tract). This gives a decreased or even a retrograde S wave. As the ventricle begins to relax and the

Figure 12. Diagrams of severe tricuspid regurgitation and the resulting abnormal waveform. White arrows = wall motion, yellow arrows = blood flow. **(a)** The atrium contracts, forcing blood antegrade into the ventricle as well as retrograde toward the liver, producing a retrograde A wave as usual. **(b)** During systole, the ventricle contracts and the tricuspid annulus moves toward the cardiac apex. Blood regurgitates retrograde through the tricuspid valve into the atrium, IVC, and hepatic veins. The result is a retrograde S wave. **(c)** As the ventricle begins to relax and the tricuspid annulus returns to its original position, blood is again forced out of the atrium back into the IVC and liver, producing the retrograde V wave. **(d)** With the atrium and ventricle relaxed and the tricuspid valve open during diastole, blood finally flows passively out of the liver and IVC into the heart, thereby producing the only antegrade wave, the D wave.



tricuspid annulus returns to its original position, blood is forced out of the atrium again back into the IVC and liver, giving a retrograde V wave. Finally, during cardiac diastole, with the tricuspid valve open and the myocardium resting, blood

flows passively from the liver into the right side of the heart, yielding the larger or only antegrade wave, the D wave.

The extent of tricuspid regurgitation will obviously influence the morphology of the spectral waveform, which is graded as types 1, 2, and 3 (Fig 13). Note that there is not a perfect match

Figure 13. Diagrams and tracings of the hepatic vein waveform in the normal state and in tricuspid regurgitation. (a) Normal hepatic vein spectrum shows the normal S wave–to–D wave ratio, with the S wave greater than the D wave. (b) In type 1 tricuspid regurgitation, the relationship between the S wave and the D wave changes, with the S wave smaller than the D wave. However, there is still antegrade flow during ventricular systole. (c) In type 2 tricuspid regurgitation, there is no systolic flow during ventricular systole. (d) In type 3 tricuspid regurgitation, there is retrograde flow during ventricular systole.

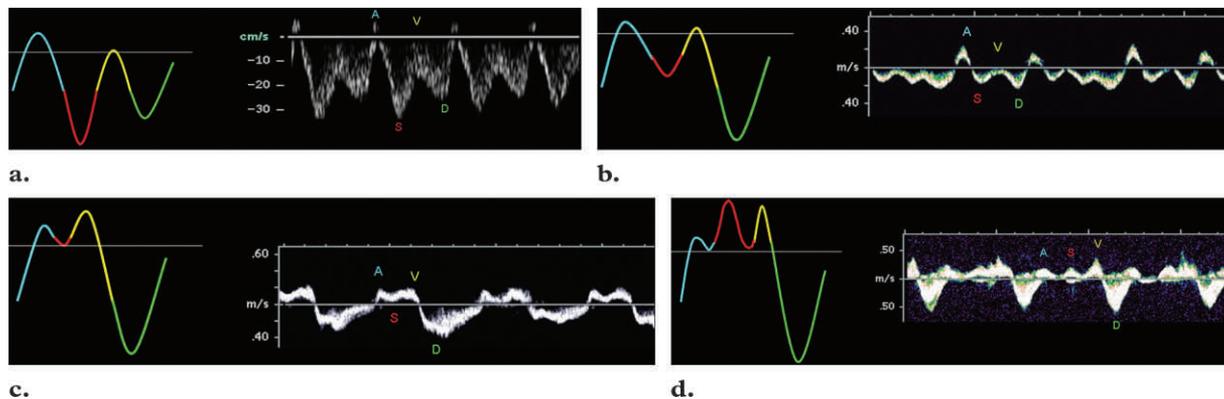
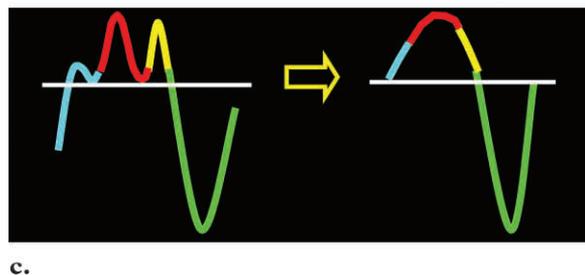
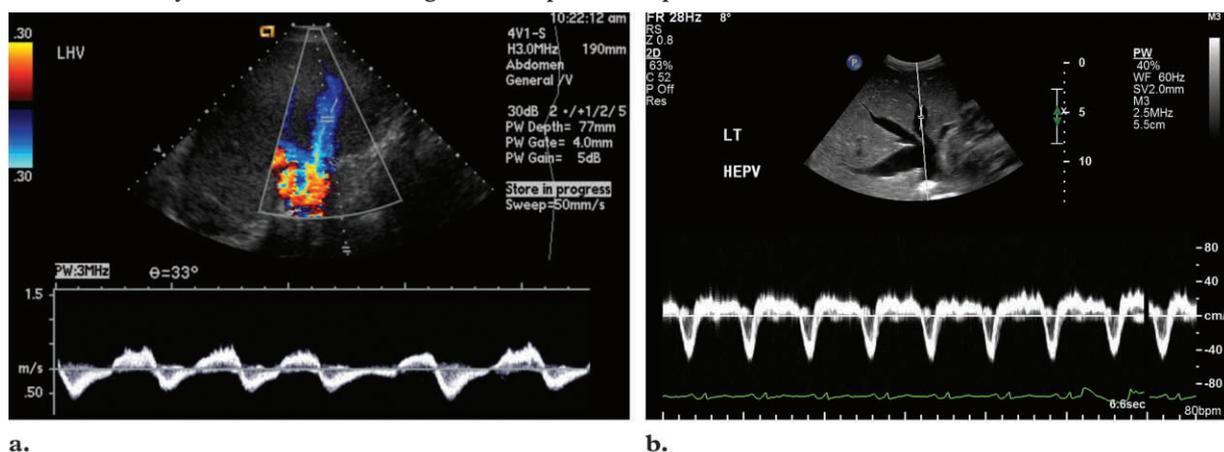
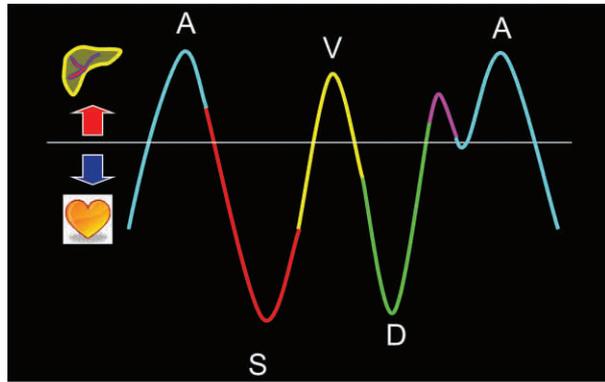


Figure 14. Biphasic waveform. (a) Duplex Doppler image of the left hepatic vein, obtained in a patient with echocardiographically proved moderate right ventricular hypokinesia and severe tricuspid regurgitation, shows a biphasic waveform. (b) Duplex Doppler image of the left hepatic vein, obtained in a patient with the echocardiographically proved combination of tricuspid regurgitation and congestive heart failure, shows a biphasic waveform. Note the dilated hepatic veins on the gray-scale display. (c) Diagram shows how a combination of retrograde A, S, and V waves may combine to form a single wave as part of a biphasic waveform.



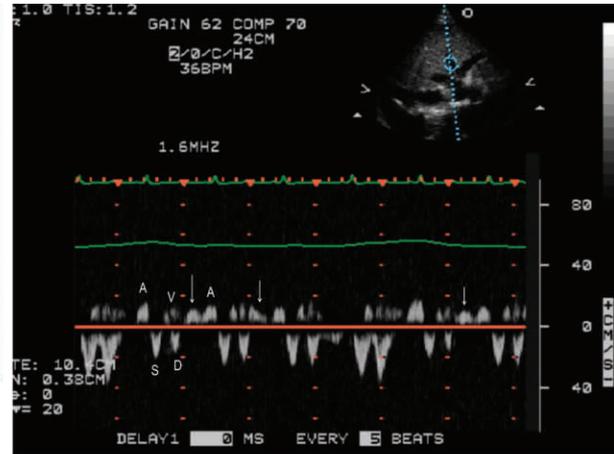
ally corresponds to mild tricuspid regurgitation, whereas types 2 and 3 may correspond to moderate or severe disease. The classically described biphasic waveform contains a single retrograde wave and a single antegrade wave per cardiac cycle (Fig 14). It is usually the result of a combination of tricuspid regurgitation and right-sided heart failure. When the A, S, and V waves are all retrograde, they may fuse into one retrograde pulse that alternates with an antegrade D wave (Fig 14).

between the sonographic “type” and the echocardiographic grade; however, there is rough correspondence. For example, the type 1 pattern usu-

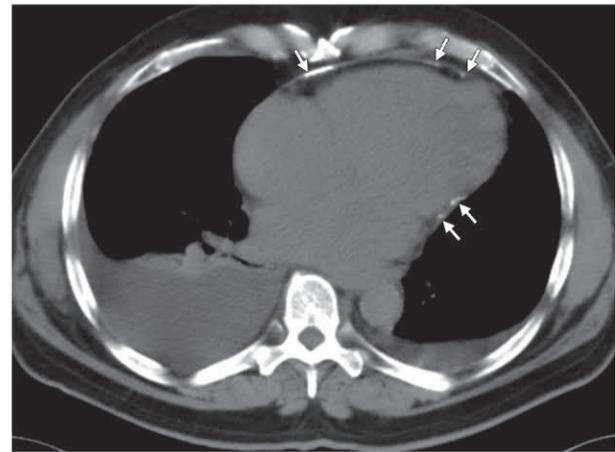


a.

Figure 15. Constrictive pericarditis. (a) Diagram shows the Doppler waveform of the hepatic vein in constrictive pericarditis. Note the retrograde component (purple wave) between the normal-appearing D and A waves. (b) Duplex Doppler image shows the middle hepatic vein in a 61-year-old man. Note the concurrent ECG tracing (top green line); the ECG QRS complex corresponds to the peak of the Doppler A wave, thereby delineating the cardiac cycle. The additional retrograde pulse (arrows) above the baseline between the D and A waves is typical of constrictive pericarditis. (c) Axial computed tomographic image of the thorax, obtained in the same patient as in b, shows pericardial calcification (arrows), which is consistent with the diagnosis of constrictive pericarditis. (Case courtesy of Daniel M. Spevack, MD, Department of Cardiology, Montefiore Medical Center, Bronx, NY.)



b.



c.

The hepatic vein waveform in right-sided heart failure is similar to that in tricuspid regurgitation (34). Early on, there may be mild augmentation of the A wave due to increased backflow, because the failing ventricle is not able to accommodate the entire forward pulse from the atrium (27). Owing to the normal magnitude variability of the A wave, this finding is usually difficult to detect. As the heart failure progresses, there is reversal of the S wave—to-D wave ratio, which may progress to the S wave reaching the baseline (a type 2 tricuspid regurgitation configuration), a finding that signifies no antegrade flow during ventricular systole.

However, an important point is that the S wave does not become retrograde (type 3 tricuspid regurgitation configuration) in the absence of tricuspid regurgitation (17). Differentiating pure

mild tricuspid regurgitation from pure right-sided heart failure is impossible on the basis of spectral tracings alone, as both may have type 1 or type 2 waveforms. Still, it may not be necessary to differentiate these entities because they commonly coexist (27). If there is a retrograde S wave, then at least some tricuspid regurgitation must be present.

Constrictive Pericarditis

The ability to analyze the spectral Doppler waveform of the hepatic veins is particularly important in constrictive pericarditis. Constrictive pericarditis may initially manifest as signs of primary liver disease, including elevated levels of transaminases, ascites, and hepatomegaly, triggering performance

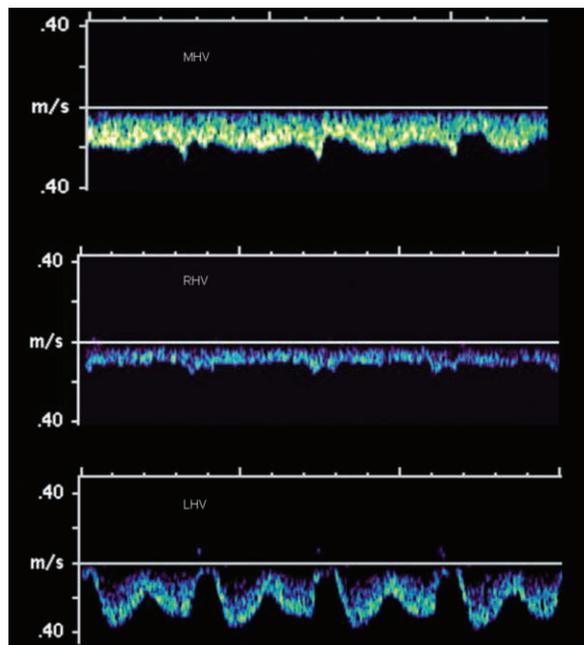


Figure 16. Different waveforms in the three hepatic veins of a patient with no medical history. Doppler spectra show a near-monophasic waveform in the right hepatic vein (middle), a blunted waveform in the middle hepatic vein (top), and a normal waveform in the left hepatic vein (bottom). The patient had no underlying liver disease; we believe that the presence of different patterns was due to technical factors.

of liver sonography (35). The correct recognition of constrictive pericarditis will facilitate proper cardiac work-up and early treatment with pericardiectomy, relieving all symptoms (35).

The waveform found in constrictive pericarditis is a variant of the triphasic waveform that includes an extra retrograde wave at the end of diastole, between the D and A waves (Fig 15) (36,37). Because the right atrium and ventricle fill passively via the superior vena cava and IVC during diastole (the D wave), the right heart capacity is reached prematurely owing to constriction by the pericardium, causing retrograde blood flow to the liver during the end of diastole. The additional retrograde wave is more prominent during the expiratory phase of the respiratory cycle (36,37). This finding can be explained by the pathophysiology of constrictive pericarditis, which is beyond the scope of this review (38).

Teaching Point

Relationship of the Hepatic Veins to Each Other

Although the spectral waveform patterns in the three hepatic veins of a single patient are usually similar, uncommonly they may have different patterns (Fig 16). This appearance may be due to true differences in the different portions of the liver through which the respective veins course or technical in nature. For example, there may be focal or geographic fat infiltration or metastatic involvement such that the milieu of the different hepatic veins are indeed different. Differences in transient factors such as inspiration, expiration, and performing the Valsalva maneuver while holding one's breath may also artificially lead to different spectra in the different hepatic veins. Therefore, these parameters must be monitored carefully by the sonographer.

Evaluation of the Hepatic Veins in Liver Transplant Recipients

US has become the standard modality for evaluating the liver after transplantation to quickly and cost-effectively diagnose complications and prevent graft loss (39,40). Hepatic vein spectral Doppler waveform analysis has also been used in liver transplants, primarily to evaluate for venous stenosis and acute graft rejection. Abnormalities in the spectral Doppler waveform pattern are generally nonspecific. The value of this analysis is that when a normal spectral wave pattern is demonstrated, the clinician can be reassured of a low probability of complications.

In the evaluation of hepatic vein stenosis, Choi et al (41) used intraoperative Doppler US to diagnose hepatic vein stenosis with a sensitivity and specificity of 80% and 82%, respectively. Most important, the presence of a triphasic waveform had a 98% negative predictive value for hepatic vein stenosis (41). In a study that evaluated hepatic vein stenosis after surgery (range, 1–433 days), Ko et al (42) concluded that a persistent triphasic hepatic vein waveform virtually excluded hepatic vein stenosis. On the other hand, a

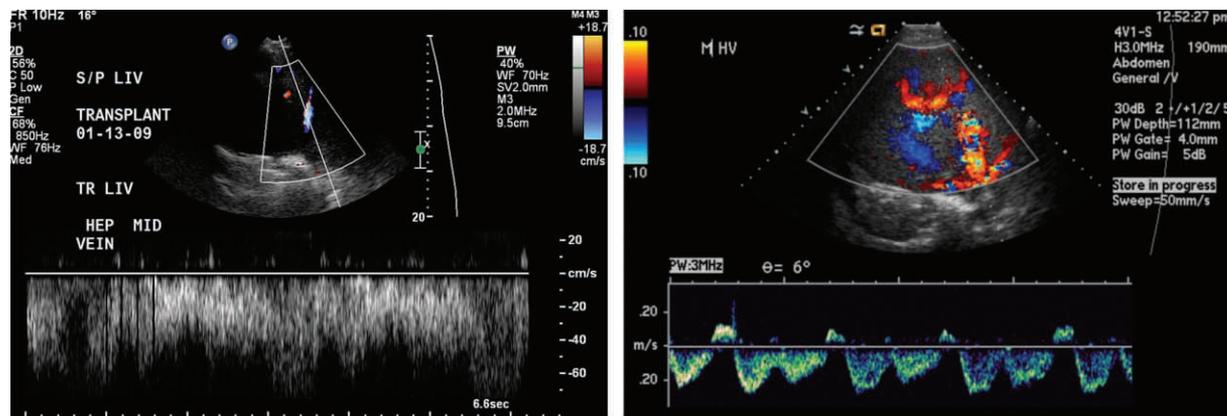


Figure 17. Transient dampening of the hepatic vein waveform in a 51-year-old man after orthotopic liver transplantation. **(a)** Duplex Doppler image obtained 1 day after surgery shows the waveform of the middle hepatic vein. The spectrum is in the correct direction, posteriorly toward the IVC; however, the waveform is blunted. The patient continued to do well clinically. **(b)** Duplex Doppler image obtained 1 day later shows a normal waveform of the middle hepatic vein.

blunted waveform was found to be nonspecific. It may be seen transiently with postoperative graft edema, which resolves spontaneously (Fig 17).

There have been three studies evaluating the usefulness of hepatic vein spectral Doppler waveform analysis to exclude acute rejection. Jéquier et al (43) found that during up to 4 years of follow-up, a persistently triphasic waveform had a 92% negative predictive value for acute rejection. Slightly lower negative predictive values were reported by Britton et al (44) and Zalasins et al (45), who each found that a triphasic waveform had an 84% negative predictive value for acute rejection. Cases of rejection with normal spectral waveforms can perhaps be explained as representing early or mild forms of rejection, where significant graft edema has not set in. Loss of a triphasic waveform was found to be nonspecific for rejection, because conditions such as cholangitis, hepatitis, fibrosis, lymphoproliferative disorder, and juxtahepatic fluid collections were also causes of Doppler waveform blunting (43,45).

Conclusions

We have described the technique of acquiring and displaying duplex Doppler waveforms of the hepatic veins. The normal basic waveform has two antegrade waves, one retrograde wave, and one transitional wave. Wave assignments are usually made by means of pattern recognition; however, when the waveform becomes more complex, such as in arrhythmias and constrictive pericarditis, ECG correlation becomes useful. Care must be taken during acquisition of the Doppler spectra so that artifact is not introduced owing to transient patient factors such as performing the Valsalva maneuver. By using a systematic algorithm for waveform analysis, a specific diagnosis or differential diagnosis can be determined.

Accurate evaluation of hepatic vein spectral Doppler tracings can provide valuable data to supplement gray-scale US and color Doppler information. The appearance of the spectral Doppler tracing can also suggest unsuspected diagnoses, such as constrictive pericarditis or rejection in a liver transplant. The relationship between cardiac physiology and liver physiology is bridged

by the hepatic veins, and Doppler analysis of the hepatic veins can provide valuable insights about the relationship between these organs.

References

- Zwiebel WJ. Sonographic diagnosis of hepatic vascular disorders. *Semin Ultrasound CT MR* 1995;16(1):34–48.
- Killi RM. Doppler sonography of the native liver. *Eur J Radiol* 1999;32(1):21–35.
- Desser TS, Sze DY, Jeffrey RB. Imaging and intervention in the hepatic veins. *AJR Am J Roentgenol* 2003;180(6):1583–1591.
- Gorg C, Riera-Knorrenschild J, Dietrich J. Colour Doppler ultrasound flow patterns in the portal venous system. *Br J Radiol* 2002;75(899):919–929.
- Skandalakis JE, Skandalakis LJ, Skandalakis PN, Mirilas P. Hepatic surgical anatomy. *Surg Clin North Am* 2004;84(2):413–435.
- Zhuang ZG, Qian LJ, Gong HX, et al. Multidetector computed tomography angiography in the evaluation of potential living donors for liver transplantation: single-center experience in China. *Transplant Proc* 2008;40(8):2466–2477.
- Erbay N, Raptopoulos V, Pomfret EA, Kamel IR, Kruskal JB. Living donor liver transplantation in adults: vascular variants important in surgical planning for donors and recipients. *AJR Am J Roentgenol* 2003;181(1):109–114.
- Nakamura S, Tsuzuki T. Surgical anatomy of the hepatic veins and the inferior vena cava. *Surg Gynecol Obstet* 1981;152(1):43–50.
- Mehran R, Schneider R, Franchebois P. The minor hepatic veins: anatomy and classification. *Clin Anat* 2000;13(6):416–421.
- Hagen-Ansert SL. *Textbook of diagnostic ultrasonography*. 6th ed. St Louis, Mo: Mosby Elsevier, 2006; 197.
- Kruskal JB, Newman PA, Sammons LG, Kane RA. Optimizing Doppler and color flow US: application to hepatic sonography. *RadioGraphics* 2004;24(3):657–675.
- Rubin JM. Spectral Doppler US. *RadioGraphics* 1994;14(1):139–150.
- McGahan JP, Goldberg BB, eds. *Diagnostic ultrasound: a logical approach*. Philadelphia, Pa: Lippincott, 1998; 616.
- Coulden RA, Lomas DJ, Farman P, Britton PD. Doppler ultrasound of the hepatic veins: normal appearances. *Clin Radiol* 1992;45(4):223–227.
- Bolondi L, Li Bassi S, Gaiani S, et al. Liver cirrhosis: changes of Doppler waveform of hepatic veins. *Radiology* 1991;178(2):513–516.
- Pedersen JF, Dakhil AZ, Jensen DB, Søndergaard B, Bytzer P. Abnormal hepatic vein Doppler waveform in patients without liver disease. *Br J Radiol* 2005;78(927):242–244.
- Abu-Yousef MM. Duplex Doppler sonography of the hepatic vein in tricuspid regurgitation. *AJR Am J Roentgenol* 1991;156(1):79–83.
- Abu-Yousef MM. Normal and respiratory variations of the hepatic and portal venous duplex Doppler waveforms with simultaneous electrocardiographic correlation. *J Ultrasound Med* 1992;11(6):263–268.
- Teichgräber UK, Gebel M, Benter T, Manns MP. Effect of respiration, exercise, and food intake on hepatic vein circulation. *J Ultrasound Med* 1997;16(8):549–554.
- Shapiro RS, Winsberg F, Maldjian C, Stancato-Pasik A. Variability of hepatic vein Doppler tracings in normal subjects. *J Ultrasound Med* 1993;12(12):701–703.
- Roobottom CA, Hunter JD, Weston MJ, Dubbins PA. Hepatic venous Doppler waveforms: changes in pregnancy. *J Clin Ultrasound* 1995;23(8):477–482.
- Pekindil G, Varol FG, Yüce MA, Yardim T. Evaluation of hepatic venous pulsatility and portal venous velocity with Doppler ultrasonography during the puerperium. *Eur J Radiol* 1999;29(3):266–269.
- Chawla Y, Kumar S, Dhiman RK, Suri S, Dilawari JB. Duplex Doppler sonography in patients with Budd-Chiari syndrome. *J Gastroenterol Hepatol* 1999;14(9):904–907.
- Millener P, Grant EG, Rose S, et al. Color Doppler imaging findings in patients with Budd-Chiari syndrome: correlation with venographic findings. *AJR Am J Roentgenol* 1993;161(2):307–312.
- Feldstein VA, LaBerge JM. Hepatic vein flow reversal at duplex sonography: a sign of transjugular intrahepatic portosystemic shunt dysfunction. *AJR Am J Roentgenol* 1994;162(4):839–841.
- Ahmed F, Coll D, Jacobson IM. Imaging. In: Schiff ER, Sorrell MF, Schiff L, Maddrey WC, eds. *Schiff's diseases of the liver*. 10th ed. Philadelphia, Pa: Lippincott Williams & Wilkins, 2007; 83–126.
- Allan PL, Dubbins PA, Poznik MA, McDicken WN. *Clinical Doppler ultrasound*. London, England: Churchill Livingstone, 2000; 147–148.

28. Oguzkurt L, Yildirim T, Torun D, Tercan F, Kizilkilic O, Niron EA. Hepatic vein Doppler waveform in patients with diffuse fatty infiltration of the liver. *Eur J Radiol* 2005;54(2):253–257.
29. Colli A, Cocciolo M, Riva C, et al. Abnormalities of Doppler waveform of the hepatic veins in patients with chronic liver disease: correlation with histologic findings. *AJR Am J Roentgenol* 1994;162(4):833–837.
30. von Herbay A, Frieling T, Häussinger D. Association between duplex Doppler sonographic flow pattern in right hepatic vein and various liver diseases. *J Clin Ultrasound* 2001;29(1):25–30.
31. Brown BP, Abu-Yousef M, Farner R, LaBrecque D, Gingrich R. Doppler sonography: a noninvasive method for evaluation of hepatic venocclusive disease. *AJR Am J Roentgenol* 1990;154(4):721–724.
32. Baik SK, Kim JW, Kim HS, et al. Recent variceal bleeding: Doppler US hepatic vein waveform in assessment of severity of portal hypertension and vasoactive drug response. *Radiology* 2006;240(2):574–580.
33. Kim MY, Baik SK, Park DH, et al. Damping index of Doppler hepatic vein waveform to assess the severity of portal hypertension and response to propranolol in liver cirrhosis: a prospective nonrandomized study. *Liver Int* 2007;27(8):1103–1110.
34. Parulekar SG, Bree RL. Liver. In: McGahan JP, Goldberg BB, eds. *Diagnostic ultrasound: a logical approach*. Philadelphia, Pa: Lippincott Williams & Wilkins, 1998; 676–677.
35. Shiffman ML. The liver in circulatory failure. In: Schiff ER, Sorrell MF, Schiff L, Maddrey WC, eds. *Schiff's diseases of the liver*. 10th ed. Philadelphia, Pa: Lippincott Williams & Wilkins, 2007; 1185–1198.
36. Gorka TS, Gorka W. Doppler sonographic diagnosis of severe portal vein pulsatility in constrictive pericarditis: flow normalization after pericardiectomy. *J Clin Ultrasound* 1999;27(2):84–88.
37. von Bibra H, Schober K, Jenni R, Busch R, Sebening H, Blömer H. Diagnosis of constrictive pericarditis by pulsed Doppler echocardiography of the hepatic vein. *Am J Cardiol* 1989;63(7):483–488.
38. Pande AN, Lilly LS. Pericardial disease. In: Solomon SD, ed. *Essential echocardiography: a practical handbook*. Totowa, NJ: Humana, 2007; 191–200.
39. Saad WE, Lin E, Ormanoski M, Darcy MD, Rubens DJ. Noninvasive imaging of liver transplant complications. *Tech Vasc Interv Radiol* 2007;10(3):191–206.
40. Kok T, Slooff MJ, Thijn CJ, et al. Routine Doppler ultrasound for the detection of clinically unsuspected vascular complications in the early postoperative phase after orthotopic liver transplantation. *Transpl Int* 1998;11(4):272–276.
41. Choi JY, Lee JY, Lee JM, et al. Routine intraoperative Doppler sonography in the evaluation of complications after living-related donor liver transplantation. *J Clin Ultrasound* 2007;35(9):483–490.
42. Ko EY, Kim TK, Kim PN, Kim AY, Ha HK, Lee MG. Hepatic vein stenosis after living donor liver transplantation: evaluation with Doppler US. *Radiology* 2003;229(3):806–810.
43. Jéquier S, Jéquier JC, Hanquinet S, Le Coultre C, Belli DC. Orthotopic liver transplants in children: change in hepatic venous Doppler wave pattern as an indicator of acute rejection. *Radiology* 2003;226(1):105–112.
44. Britton PD, Lomas DJ, Coulden RA, Farman P, Revell S. The role of hepatic vein Doppler in diagnosing acute rejection following paediatric liver transplantation. *Clin Radiol* 1992;45(4):228–232.
45. Zalasins S, Shapiro RS, Glajchen N, Stancato-Pasik A. Liver transplant rejection: value of hepatic vein Doppler waveform analysis. *Abdom Imaging* 1998; 23(4):427–430.

Understanding the Spectral Doppler Waveform of the Hepatic Veins in Health and Disease

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RadioGraphics 2009; 29:2081–2098 • Published online 10.1148/rg.297095715 • Content Codes: **GI** **US**

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The normal hepatic vein waveform, despite commonly being described as triphasic, has four components: a retrograde A wave, an antegrade S wave, a transitional V wave (which may be antegrade, retrograde, or neutral), and an antegrade D wave (13).

Page 2087

The third and most reliable method of correctly assigning the spectral Doppler waves is obtaining a concurrent ECG tracing with the spectral Doppler tracing (18).

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Patient factors play an important role in the appearance of the hepatic vein spectral Doppler waveform (18–20).

Page 2089

Evaluation of the hepatic vein spectral Doppler tracing involves asking four questions (Fig 8): What is the direction of flow? Is the spectral wave pattern regular or irregular? What is the waveform phasicity? (In other words, is the waveform monophasic or dampened, or is it multiphasic?) What is the relationship of magnitude between the S and D waves?

Page 2095

The waveform found in constrictive pericarditis is a variant of the triphasic waveform that includes an extra retrograde wave at the end of diastole, between the D and A waves (Fig 15) (36,37).