

# Doppler US of the Liver Made Simple<sup>1</sup>

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## CME FEATURE

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## LEARNING OBJECTIVES FOR TEST 4

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

- Discuss the basic concepts and terminology for vascular Doppler US.
- Recognize the characteristic appearances of normal and abnormal liver Doppler waveforms.
- Describe the US manifestations of both normally functioning and malfunctioning TIPS.

## TEACHING POINTS

See last page

Doppler ultrasonography (US) is usually the first-line modality for evaluating flow in native liver vessels and transjugular intrahepatic portosystemic shunts (TIPS). Waveforms, which represent flow in each of the major vessels and in TIPS, have been well described. The appearance of these waveforms should be recognized, and the mechanisms behind their generation should be understood by those interpreting these examinations. Understanding how waveforms are formed—that is, their mechanisms for generation—is predicated on knowing basic vascular Doppler concepts and established nomenclature. This article is a review of these basic concepts and nomenclature as applied to the interpretation of liver Doppler US waveforms.

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**Abbreviations:** CHF = congestive heart failure, IVC = inferior vena cava, PI = pulsatility index, RI = resistive index, TIPS = transjugular intrahepatic portosystemic shunt

RadioGraphics 2011; 31:161–188 • Published online 10.1148/rg.311105093 • Content Codes: **GI** **US**

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## Introduction

The interpretation of liver Doppler ultrasonographic (US) examinations can be a source of anxiety to those unfamiliar with the basic concepts and terminology, and to those with limited experience in reading these studies. Normal and abnormal waveforms for each of the major hepatic vessels (hepatic artery, hepatic vein, and portal vein) have been well described (1–6). The good news is that normal waveforms have characteristic appearances, and the majority of liver diseases cause only a limited number of abnormal waveform patterns. A simple organized approach will help alleviate interpreter anxiety and improve competency.

Competence at liver Doppler US first requires knowledge of the principles of basic flow dynamics and of the terms used in Doppler examinations. Next, it requires familiarity with the unique appearance of each major vessel's waveform, sometimes referred to as its "signature" appearance. To help learn these signature appearances, it is worthwhile to have a conceptualized model of blood flow for each vessel.

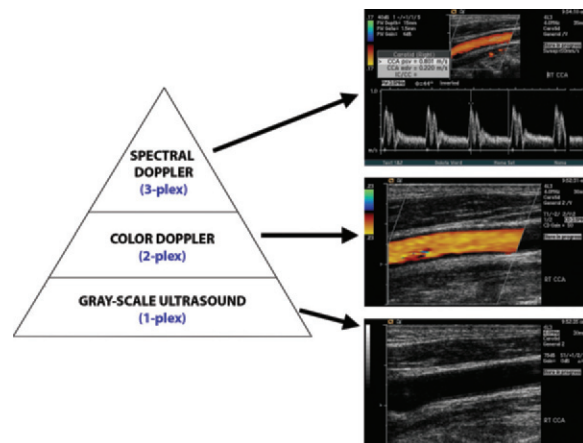
Every disease process that affects the liver has its own characteristic effect on blood flow patterns and, therefore, affects the waveforms for the three major hepatic vessels in a unique way. This fact forms the basis for using spectral Doppler US in diagnostic radiology. Having a conceptual model for the most common disease processes and the most commonly encountered pathologic flow states is the best way to understand and interpret pathologic waveforms. Mastery of liver Doppler US is achieved when one is able to fluently transition between what is expected (physiologically or pathologically) and what is observed at spectral Doppler US.

In this article, we review the terminology used in vascular Doppler US and the basic concepts of flow dynamics in vessels. In addition, we describe the normal waveforms, as well as abnormal waveforms and their causes, for each of the three major hepatic vessels. We also briefly discuss typical US findings at transjugular intrahepatic portosystemic shunt (TIPS) examination.

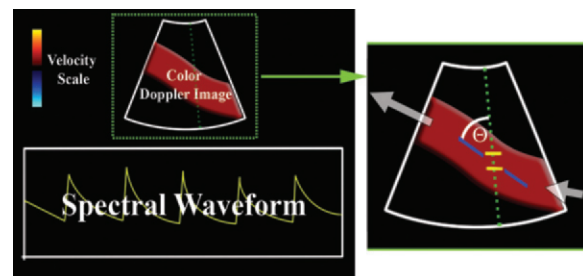
## Terminology and Flow Concepts

### US Modalities

The term *Doppler* should be capitalized because it is an eponym named after Christian Johann Doppler (1803–1853), the Austrian physicist who first described the "effect" (7). Although mentioning this may seem a bit patronizing to the reader, it is not uncommon for the word to be misspelled with a lowercase *d* by both referring physicians and radiologists.

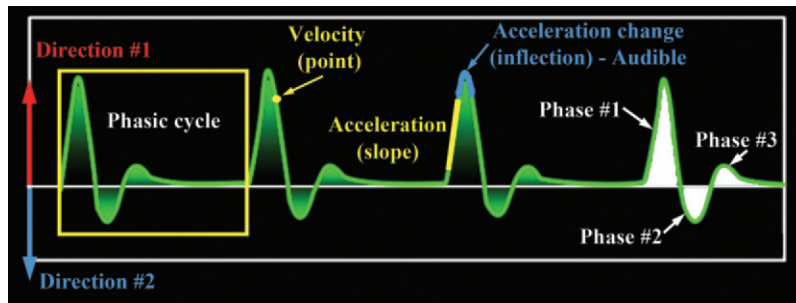


**Figure 1.** Chart illustrates the least ambiguous way to name Doppler examinations. The term *duplex Doppler* can be confusing due to its dual usage. Sometimes, the term is used to refer to color Doppler examinations; at other times, to spectral Doppler examinations. A spectral Doppler examination includes color Doppler US; a color Doppler examination includes gray-scale US (B-mode imaging).



**Figure 2.** Spectral Doppler examination components. Diagram at left shows the general layout of a spectral Doppler image. The spectral waveform is displayed on the lower half of the image, a color Doppler image is shown above the waveform, and a velocity scale may be shown on either the right or left side (top left in this case). Magnified view (right) of the color Doppler interrogation region shows the components used to acquire the waveform: Doppler beam path (green); angle indicator (blue), which is oriented parallel to the long axis of the vessel; Doppler angle ( $\Theta$ ), which should be less than  $60^\circ$ ; and sample volume or "gate" (yellow). Gray arrows = flow direction.

Three basic levels of US can be performed (Fig 1), with each level adding information to the preceding level. At the first level is the traditional standard brightness mode (B-mode) gray-scale examination, in which no Doppler is used. The second level superimposes a color Doppler interrogation region of interest. This level produces an image that shows blood flow in vessels. The third level superimposes a small interrogation region, called a sample volume, over a vessel of interest. Targeted interrogation of the vessel produces a spectral Doppler waveform.



**Figure 3.** Magnified view of a spectral waveform illustrates its features. Cardiac phasicity creates a phasic cycle, which is composed of phases as determined by the number of times blood flows in each direction. The baseline ( $x = 0$ ) separates one direction from another. Moving from left to right along the x-axis corresponds to moving forward in time. Moving away from the baseline vertically along the y-axis in either direction corresponds to increasing velocities. Any given point on the waveform corresponds to a specific velocity. The slope of the curve corresponds to acceleration (ie, a change in velocity per unit time). A bend in the curve, or inflection point, corresponds to a change in acceleration. When these turns are abrupt, they generate audible sounds at Doppler US.

The nomenclature becomes somewhat confusing due to discrepancies between the technically correct terms and the more commonly used terms. For example, strictly speaking, the term *duplex Doppler* refers to an examination consisting of two levels (gray-scale and color Doppler US). However, the term is commonly used by referring physicians when ordering an examination with spectral Doppler, which technically would be more accurately termed *triplex Doppler*. To avoid confusion, it is probably better to use terms that describe the examination more precisely. Such terms include *gray-scale*, *color Doppler*, and *spectral Doppler*.

### Spectral Doppler Components

Spectral Doppler US findings are displayed with the spectral waveform at the bottom of the screen and a color Doppler image at the top (Fig 2). Information for the waveform is obtained from a small (usually 2–4-mm) sample volume that is placed in the center of the vessel by the sonographer (ie, US technologist). Ideally, the sample volume should be placed in the midportion of the lumen, rather than toward the periphery, for optimal estimation of laminar flow. An angle indicator line is subjectively placed parallel to the vessel; however, this placement can introduce error into the final velocity calculation, especially when the Doppler angle ( $\Theta$ )—the angle between the actual Doppler beam and the Doppler interrogation line—is greater than  $60^\circ$ .

To understand why  $\Theta$  must remain less than  $60^\circ$ , one needs to know how the angle is used in

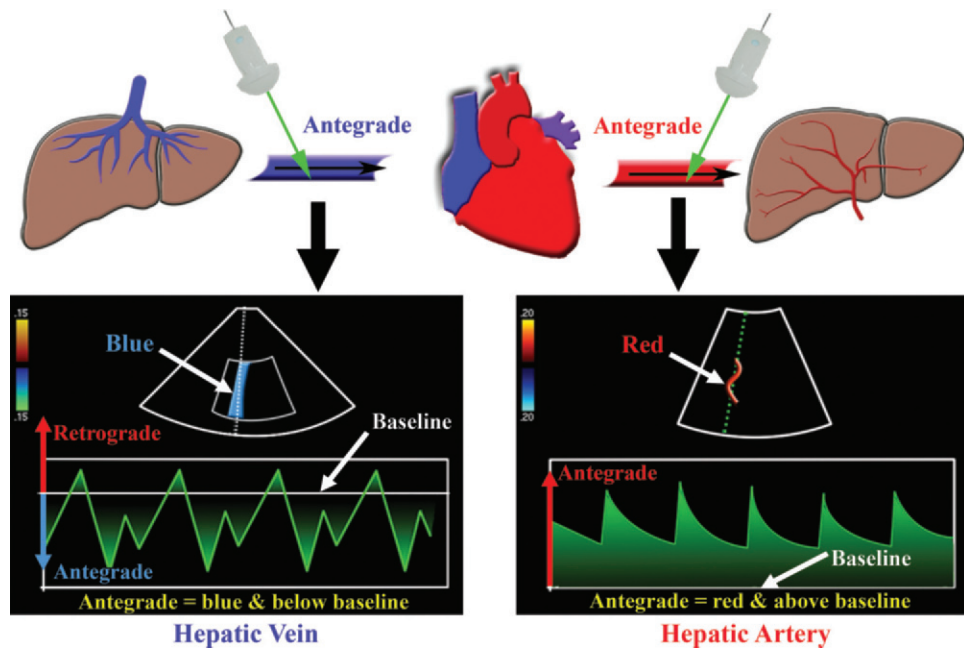
the calculation of velocity. As  $\Theta$  increases from  $0^\circ$  to  $60^\circ$ ,  $\cos\Theta$  decreases from 1 to 0.5; however, as  $\Theta$  increases from  $60^\circ$  to  $90^\circ$ ,  $\cos\Theta$  decreases from 0.5 to 0. Note that there is a larger incremental change in the value of  $\cos\Theta$  as the angle increases; this disproportionate change progressively increases as the angle approaches  $90^\circ$ . Therefore, when  $\Theta$  is greater than  $60^\circ$ , slight variations in the sonographer's placement of the interrogation line are magnified in the velocity equation, such that estimates of velocity are no longer reliable or reproducible (8).

$$V = \frac{(\Delta F)(C)}{2(Ft)(\cos\Theta)},$$

where  $C$  = the speed of sound in tissue (1.54 m/sec),  $\Delta F$  = Doppler frequency shift, and  $Ft$  = transducer frequency.

### Waveform Features and Information

Every spectral waveform has morphologic features that provide information regarding direction, velocity, and acceleration (Fig 3). Directional information is determined on the basis of whether the waveform lies above or below the baseline; velocity information is determined on the basis of distance from the baseline at any given point on the curve; and acceleration information is obtained from the slope of the curve (ie, rate of change in velocity), with changes in acceleration being marked by waveform peaks, or inflection points. These inflection points (acceleration changes) generate characteristic audible sounds at Doppler US.



**Figure 4.** Antegrade versus retrograde flow. Drawings (top) show predominantly antegrade flow from the hepatic veins (blue) to the heart and in the hepatic arteries (red) toward the liver. Retrograde flow would be in the opposite direction. Diagrams (bottom) illustrate typical spectral Doppler waveforms in these vessels. Note that antegrade flow in the hepatic veins is displayed below the baseline, whereas antegrade flow in the hepatic arteries is displayed above the baseline. Antegrade flow may be either toward the transducer (hepatic artery) or away from the transducer (hepatic vein). Similarly, retrograde flow may be either toward the transducer (displayed above the baseline) or away from the transducer (displayed below the baseline).

## Flow Direction

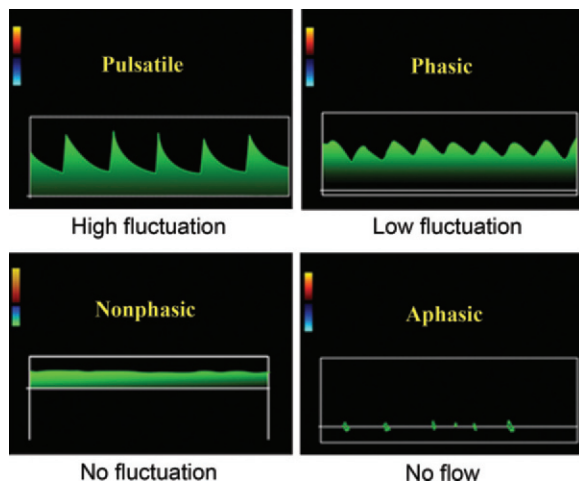
**Antegrade versus Retrograde.**—The direction of blood flow may be described in two fundamentally distinct ways (Fig 4). The first is to describe flow with respect to the circulatory system. The terms *antegrade* and *retrograde* are used to describe flow in this context. The second is to describe flow with respect to the US transducer. In this context, flow is described as moving either toward or away from the transducer. Color Doppler arbitrarily displays blood flow toward the transducer as red and blood flow away from the transducer as blue. At spectral Doppler, blood flow toward the transducer is displayed above the baseline and blood flow away from the transducer is displayed below the baseline.

The term *antegrade* refers to flow in the forward direction with respect to its expected direction in the circulatory system. For example,

antegrade flow moves away from the heart in the systemic arteries and toward the heart in the systemic veins. Early learners may mistakenly think that the word *antegrade* describes flow toward the transducer (displayed above the baseline). However, **antegrade flow may be either toward or away from the transducer, depending on the spatial relationship of the transducer to the vessel; therefore, antegrade flow may be displayed above or below the baseline, depending on the vessel being interrogated.** An example of antegrade flow away from the transducer (displayed below the baseline) is seen in the systolic wave (*S* wave) and diastolic wave (*D* wave) of the normal hepatic venous waveform.

The term *retrograde* refers to flow in the reverse direction with respect to its expected direction in the circulatory system. For example, retrograde flow may be seen in severe portal hypertension, in which portal venous flow reverses direction (hepatofugal flow). Early learners may mistakenly think that the word *retrograde* describes flow away

Teaching  
Point



**Figure 5.** Phasicity. Diagrams illustrate the various waveforms. The terms used to describe the degree of waveform undulation empirically describe the velocity and acceleration features of the waveform. Note that pulsatile, phasic, and nonphasic flow waveforms all have phasicity. Pulsatile flow is exaggerated phasicity, which is normally seen in arteries but can also be seen in diseased veins. Nonphasic flow does in fact have a phase (of 1); however, the phase has no velocity variation (*nonphasic* could be thought of as meaning “nonvariation”). The term *aphasic* literally means “without phase,” which is the case when there is no flow.

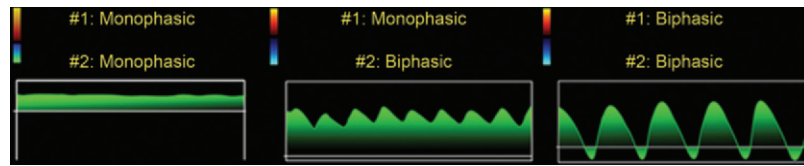
from the transducer (displayed below the baseline). However, retrograde flow may be either toward or away from the transducer. An example of retrograde flow toward the transducer (displayed above the baseline) is seen in the *a* wave of the normal hepatic venous waveform.

**Phasicity versus Phase Quantification.**—Confusion in liver Doppler terminology is related to the subtle differences in meaning between the words *phase* and *phasic*, as well as to differences of opinion as to what constitutes a phase. *Phasic* is another word for *cyclic*; its absence or presence (and degree) may be qualified. *Phasicity* has a similar meaning as *phasic*; any phasic (or cyclic) process can be described as having phasicity. On the other hand, a phase is a stage, or portion, of a phasic process; the number of phases may be quantified.

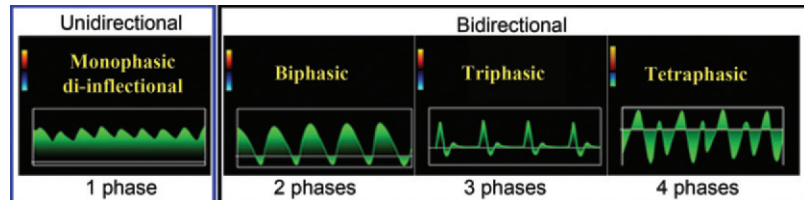
Phasic blood flow has velocity and acceleration fluctuations that are generated by cyclic (phasic) pressure fluctuations, which are in turn generated by the cardiac cycle (cardiac phasicity). When phasic blood flow is sampled at spectral Doppler US, it is displayed as a phasic waveform. In other words, the waveform has phasicity. This finding is either present or absent but is not quantifiable with a number. If there is absolutely no flow (ie, no velocity), there is no phase, and the waveform may be called “aphasic.” As long as there is flow, there is some form of phasicity. When there is flow (ie, velocity) but no velocity or acceleration changes are present, the waveform is flat, and there is no variation in phasicity; this is called a “nonphasic” waveform. If there are changes in

velocity (slopes) and acceleration (inflections), the waveform cannot be described as nonphasic; instead, it is either phasic or pulsatile, depending on the degree of waveform undulation. If there is mild undulation (shallow slopes and a small vertical range between inflections), as in normal veins, the waveform is described as phasic. If there is marked undulation (steep slopes and a wide vertical range between inflections), as in normal arteries, the waveform is described as pulsatile. The concept of phasicity is best learned by reviewing images of each type of flow (pulsatile, phasic, nonphasic, and aphasic) (Fig 5).

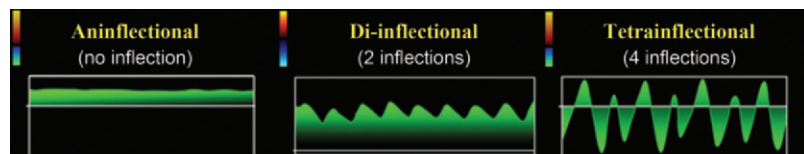
Phase quantification has been controversial ever since the advent of spectral Doppler US (Fig 6). There is a striking lack of consensus, even among sonologists (ie, radiologists who interpret US studies), about what constitutes a phase. Traditionally, sonologists have defined phase in terms of acceleration changes (ie, inflection points) (9,10), based on the observation that inflection points generate audible sounds at Doppler US. According to this approach, the flow pattern is described as “biphasic” if two sounds are heard during each cycle and as “triphasic” if three sounds are heard. More recently, sonologists have held that phase is defined in terms of discrete flow components in either direction (ie, portions on either side of the baseline) (8,11). This definition is based on observations of the spectral Doppler waveform. D.A.M. prefers the latter definition of phase, whereas M.M.A.Y. prefers the former definition.



**Figure 6.** Phase interpretation ambiguity. Schematics illustrate how different interpretations of what constitutes a phase can affect waveform characterization and nomenclature. D.A.M. interprets a phase as a component of the waveform on either side of the baseline; M.M.A.Y. interprets a phase as an inflection.



**Figure 7.** Directionality and phase quantification. When phase is defined as a component of phasic flow direction, waveforms may be described in terms of the number of phases. All monophasic waveforms are unidirectional; bidirectional waveforms may be either biphasic, triphasic, or tetrphasic.



**Figure 8.** Inflection quantification. Schematics illustrate waveforms, which can be characterized on the basis of the number of inflections. Inflections occur in pairs. It is not possible to have an odd number of inflections; otherwise, a cycle would never repeat. Nonetheless, some sonologists (including M.M.A.Y.) may call the waveform on the left monophasic, based on the fact that it has only one flow velocity. M.M.A.Y. calls the waveform in the middle biphasic, based on the number of inflection points (two) per wave.

In the end, what constitutes a phase at spectral Doppler US is arbitrary, as long as the phase is a part, or component, of a phasic waveform. A few points may be adduced in support of the view that phase is determined on the basis of flow direction, not inflection points. First, when one interprets a spectral Doppler US examination, he or she is not “listening” to the findings, but viewing the resulting images. Granted, audio Doppler came first, but spectral Doppler is more advanced. Perhaps when clinicians listen to a patient’s arterial pulse, they should say, “I hear a normal di-inflectional pulse,” rather than “I hear a normal biphasic pulse.” Second, if it were true that the inflection points (acceleration changes) determined the number of phases at spectral Doppler US, so-called monophasic and triphasic waves would not be possible, since inflection points always occur in pairs. Consider triphasic flow, given the assump-

tion that phase is defined in terms of inflection points: No cycle could ever form, since after the third inflection, the wave would simply extend off the top or bottom of the screen, a phenomenon that would indicate infinitely increasing flow velocity, which is physically impossible. Finally, the traditional system describes flat waveforms, without inflection points, as being monophasic. In this case, the system refers to the number of flow velocities (one) to quantify the number of phases. This is an internal inconsistency of the traditional naming system. Because the system describes triphasic waves as having three inflections and biphasic waves as having two inflections, one would expect a monophasic wave to have one inflection. However, these waveforms actually have no inflection (ie, zero inflection points). The definition of a phase has implications for waveform nomenclature that make a difference in terms of consistency. When phase is determined on the basis of direction rather than inflection, systematic character-

**Table 1**  
**Low-Resistance Arteries (Normal RI = 0.55–0.7)**

Internal carotid arteries
Hepatic arteries
Renal arteries
Testicular arteries

Note.—RI = resistive index.

ization of any wave can be achieved such that the words used to describe the wave could be entered into a computer and the waveform recreated.

Regardless, it is good to know that phase quantification descriptors such as *monophasic*, *biphasic*, and *triphasic* can be ambiguous given this difference in the definition of phase. Another point to keep in mind is that nonphasic waves do not actually lack phasicity; rather, they have one phase (ie, are monophasic) without any inflections. It is best to think of the *non-* in *nonphasic* as meaning no fluctuation in velocity. The only wave that truly lacks phase is the aphasic wave, which correlates with absent flow. Similarly, one might think that the pulsatile waveform is not phasic, or that it is the opposite of phasic. In reality, however, pulsatile waveforms are examples of dramatic, or exaggerated, phasicity.

The different meanings of *phasic* and *phase*, as well as the differing opinions as to what constitutes a phase, may be summarized as follows.

Phasicity is defined as the quality of being phasic, just as generosity is the quality of being generous. *Phasic* and *cyclic* are synonyms: both terms refer to processes with recurring cycles. Therefore, waveforms with phasicity have wave components (slopes and inflection points) that repeat at regular intervals. The presence or absence of phasicity can be qualified with various descriptors: pulsatile flow (arteries), phasic flow (veins), nonphasic flow (diseased veins), and aphasic flow (diseased vessels without flow).

Phase is defined as a recurring component, or fraction, of a phasic process. The number of phases can be quantified (monophasic, biphasic, triphasic, or tetraphasic). What constitutes a phase is a matter of interpretation. D.A.M. interprets a phase as each discrete waveform component on either side of the baseline (alternating flow directions) during one cycle. With this interpretation, the number of phases (phase quantification) is equal to the number of waveform components on each side of the baseline during one cycle. M.M.A.Y. interprets a phase as each unique inflection point on the waveform during one cycle, which was originally described in terms of, and

correlates with, the number of sounds heard at audio Doppler US. With this interpretation, the number of phases (phase quantification) is equal to the number of inflection points contained in the waveform during one cycle.

Perhaps in the future, a consensus statement addressing what constitutes a phase and appropriate phase quantification will be forthcoming, especially as it pertains to overall waveform nomenclature. Nonetheless, in this article, D.A.M.'s interpretation of phase will be used for phase quantification and subsequent waveform nomenclature.

**Unidirectional versus Bidirectional.**—The terms *unidirectional* and *bidirectional* can also be used to describe the direction of flow (Fig 7). Vessels with flow in only one direction (whether antegrade or retrograde) can be said to have unidirectional flow, which can only be monophasic (discussed earlier). Vessels that have flow in two directions are said to have bidirectional flow, which may be biphasic, triphasic, or tetraphasic, depending on how many times blood flows in each direction.

**Inflection Quantification.**—As mentioned earlier, any waveform can be characterized according to the number of inflections in each cycle (Fig 8). Inflections must occur in pairs; otherwise, what goes up *doesn't* come down. Waveforms without inflection are aninflectional; those with two inflections are di-inflectional; and those with four inflections—the maximum number of inflections per cardiac cycle—are tetrainflectional. Because sonologists (including M.M.A.Y.) have traditionally considered each inflection point in a cycle (rather than components on each side of the waveform in a cycle) to constitute a phase, there is considerable ambiguity in waveform nomenclature.

**Arterial Resistance.**—In the physiologic state, arteries have the capacity to change their resistance to divert flow toward the organs that need it most. In general, when an organ needs to be “on,” its arteriolar bed relaxes, the waveform takes on low resistance, and the organ is appropriately perfused. When an organ goes to “power save” mode, its arterioles constrict, the waveform switches to high resistance, and flow is diverted to other organs.

During standard Doppler US examinations, arteries have a physiologic tendency to favor either a low- or a high-resistance state. Arteries that normally have low resistance in resting (ie, nonexercising) patients include the internal carotid arteries (brain is always on), hepatic arteries (liver is on), renal arteries (kidneys are on), and testicular arteries (Table 1). The postprandial

(nonfasting) mesenteric vessels (superior and inferior mesenteric arteries) also have low resistance; however, standard spectral Doppler US of these vessels is performed in fasting patients. Arteries that normally have high resistance in resting patients include the external carotid arteries (face and scalp muscles are resting), extremity vessels (muscles are resting), and fasting mesenteric arteries (gastrointestinal tract is resting) (Table 2).

Resistance, or impedance to flow, may be described empirically or quantitatively (Fig 9). Empirical evidence is obtained with visual inspection and characterization of the waveform. If the lowest point (trough) of the waveform at end diastole is high, there is relatively more flow during diastole, a finding that indicates a low-resistance vessel. If the trough is low, there is relatively less flow during diastole, a finding that indicates a high-resistance vessel.

Arterial resistance has historically been characterized quantitatively with use of three different indices: the resistive index (RI), systolic/diastolic ratio, and pulsatility index (PI). It should be kept in mind that the PI in arteries ( $[V_1 - V_2]/V_{\text{mean}}$ , where  $V_1$  = peak systolic velocity and  $V_2$  = end-diastolic velocity) is calculated differently than the PI in the portal vein ( $V_2/V_1$ ). The most frequently used index in the hepatic arteries is the RI, which is calculated as

$$\text{RI} = \frac{(\text{PSV} - \text{EDV})}{(\text{PSV})} = \frac{(V_1 - V_2)}{V_1},$$

where PSV = peak systolic velocity and EDV = end diastolic velocity. Calculating the RI is probably the easiest part of measuring and reporting arterial impedance; most US vendors provide software that automatically performs this calculation. What to do with the result is much less clear. This is because the normal range varies from one artery, institution, and published article to the next. Furthermore, the significance of an abnormal result is not always clear. Therefore, it is wise not to rely solely on these measurements; rather, they should be used as supporting data.

In general, low-resistance arteries normally have an RI of 0.55–0.7. The hepatic artery is a low-resistance vessel; however, wider normal ranges of 0.55–0.81 have been reported for this vessel (12–14). Any measured RI above or below the normal range may represent disease.

**Table 2**  
**High-Resistance Arteries (Normal RI >0.7)**

External carotid arteries
Extremity arteries (eg, external iliac arteries, axillary arteries)
Fasting mesenteric arteries (superior and inferior mesenteric arteries)

Note.—RI = resistive index.

High-resistance arteries physiologically have an RI greater than 0.7; any RI lower than this may represent disease.

As mentioned earlier, disease may affect arterial resistance. To determine whether arterial resistance is abnormal (ie, too high or too low), one needs to consider the expected resistance in that particular vessel. For example, an RI of 0.8 in an external carotid artery is perfectly normal, since this artery is a high-resistance vessel; however, the same RI in a low-resistance vessel (eg, the hepatic artery) is abnormal and may represent disease.

In the context of liver Doppler US, the following points provide an oversimplified but nevertheless useful way to understand disease affecting the RI of the hepatic artery.

1. **A high RI is not specific for liver disease; therefore, it is less meaningful as an isolated finding than is a low RI.**

Teaching Point

2. **An RI that is too high may be the result of the postprandial state, advanced patient age, or diffuse distal microvascular disease, which has a wide variety of causes including chronic liver disease due to cirrhosis or chronic hepatitis.**

Teaching Point

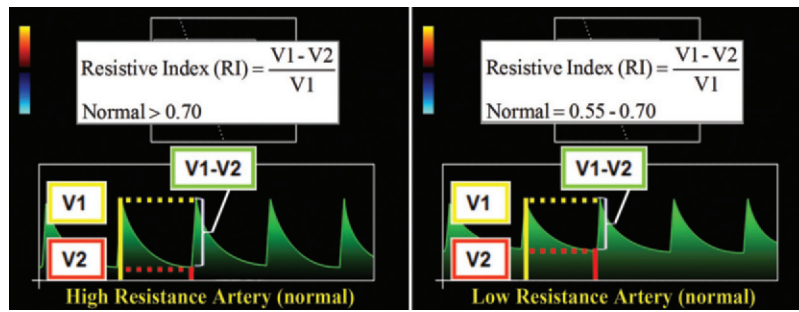
3. **An RI that is too low may be the result of proximal stenosis or distal vascular shunting (arteriovenous or arterioportal fistulas), as seen in severe cirrhosis; trauma (including iatrogenic injury); or Osler-Weber-Rendu syndrome.**

Teaching Point

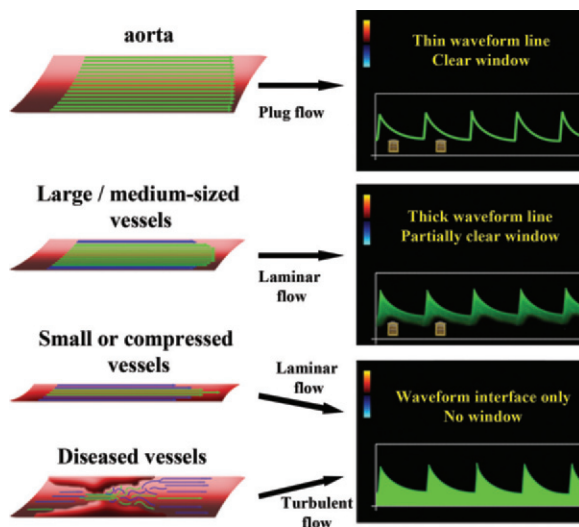
### Flow Patterns and Waveform Findings

There are three basic patterns of blood flow: plug flow (normal), laminar flow (normal), and turbulent flow (normal or abnormal) (Fig 10) (15). The effect of the size of a vessel on its spectral waveform is best understood by considering what happens at the interface between a vascular wall and the blood flowing past it. The wall exerts a “drag” effect on the moving blood, so that the velocity at the periphery of the lumen is lower than at the





**Figure 9.** High- versus low-resistance arteries. Schematics illustrate that a high-resistance artery (left) allows less blood flow during end diastole (the trough is lower) than does a low-resistance artery (right). These visual findings are confirmed by calculating an RI. High-resistance arteries normally have RIs over 0.7, whereas low-resistance arteries have RIs ranging from 0.55 to 0.7. The hepatic artery is a low-resistance artery.



**Figure 10.** Diagrams illustrate “spectral window” and spectral broadening. In the proximal aorta (top left), plug flow results in a thin waveform and a clear spectral window (top right). Note the actual windows (yellow) superimposed on the first two spectral windows. In vessels smaller than the aorta, blood flow is laminar. In large and medium-sized vessels (left, second from top), the waveform is thick, but there is still a spectral window (middle right). In small or compressed vessels (left, second from bottom), there is significant spectral broadening, which obscures the spectral window (bottom right). Diseased vessels with turbulent flow (bottom left) also cause spectral broadening (bottom right).

center. In large vessels, this drag effect is relatively minimal, with the majority of blood moving at a similar velocity and only a small fraction moving more slowly at the periphery. The sample volume is more easily placed in this uniformly moving column of blood. Plug flow is the ultimate large

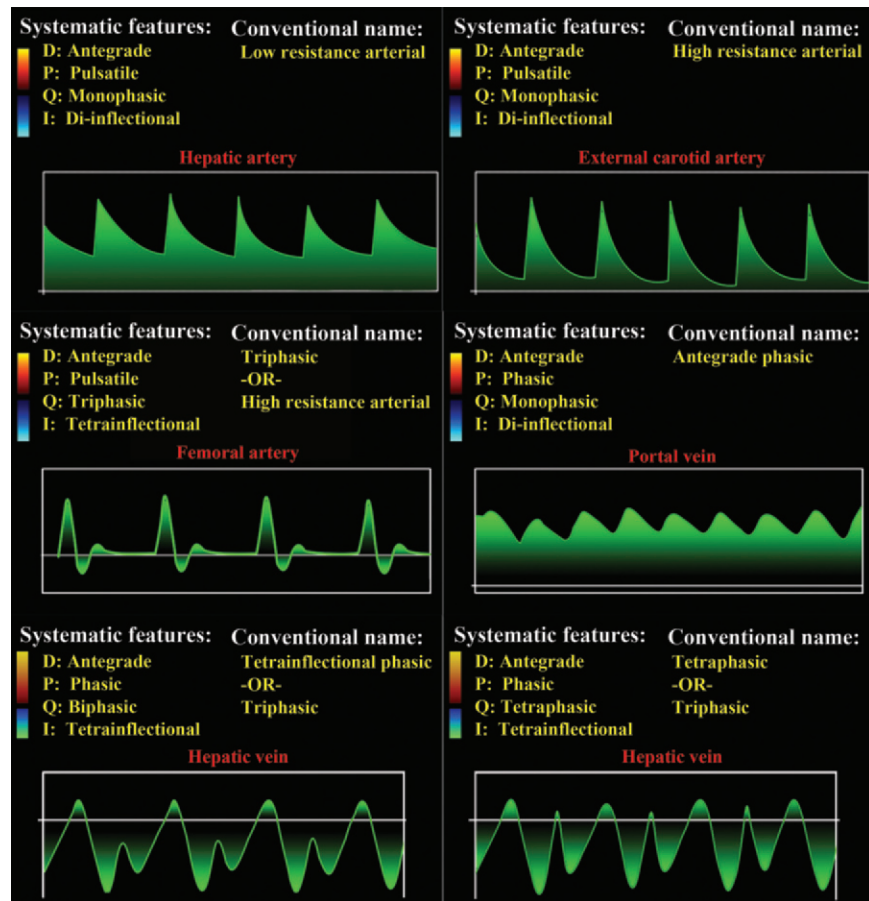
**Table 3**  
**Causes of Spectral Broadening**

Artificial
Large sample volume
High gain
Physiologic
Normal small vessels (hepatic arteries)
Normal turbulence (bifurcations)
Pathologic
Compressed vessels (eg, hepatic veins in cirrhosis)
Turbulent flow (poststenotic flow)

vessel effect, being described only in the thoracic aorta. This pattern of flow produces a crisp spectral waveform that could be drawn with a pencil or marker. In smaller vessels with laminar flow, the drag effect is more significant, with a wider range of velocities from the center to the periphery; this range is often described as having a parabolic distribution. Turbulent flow represents disorganized flow, with pockets of flow moving at different velocities and in different directions. It represents a normal finding at bifurcations and an abnormal finding in the immediately poststenotic portion of a diseased vessel.

Spectral broadening is seen when the waveform is no longer traceable with a pencil or marker. In other words, the spectral window starts to fill in. Spectral broadening can be created artificially, physiologically (in small vessels), or pathologically (Table 3) (16). Artificial broadening is generated

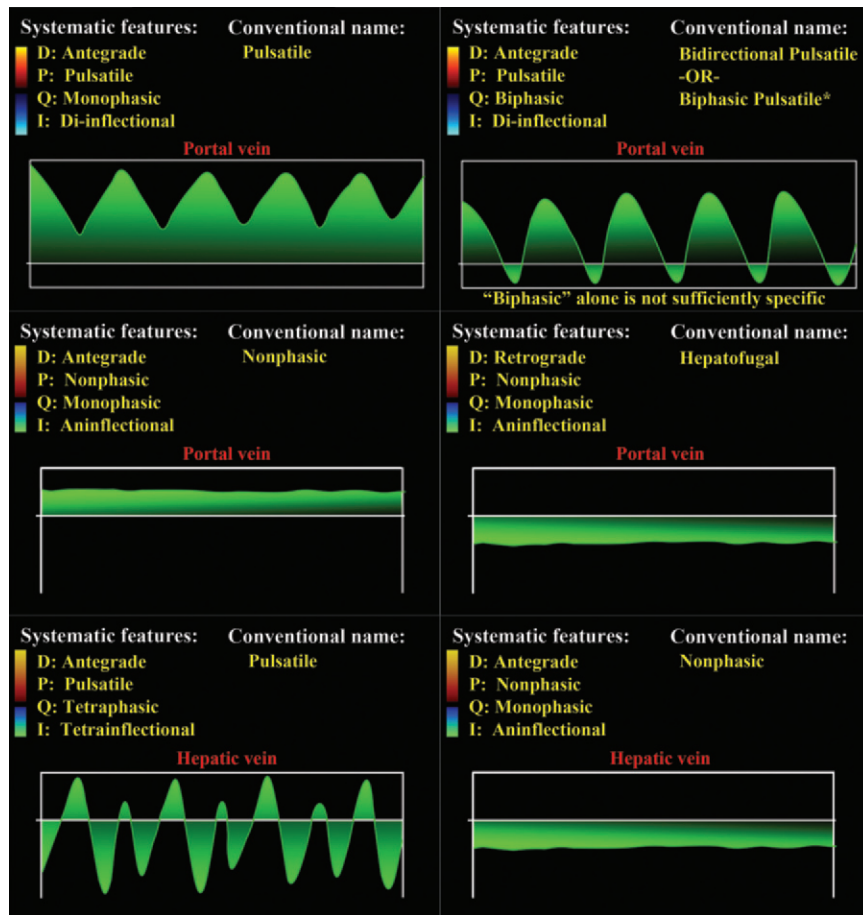
**Figure 11.** Waveform nomenclature (normal waveforms). Diagrams illustrate how normal waveforms can be systematically characterized on the basis of direction (*D*), phasicity (*P*), phase quantification number (*Q*), and inflection quantification (*I*). Arteries can be further characterized on the basis of their level of resistance (high or low). The femoral artery has truly triphasic flow. Normal hepatic venous flow has historically been called triphasic; in reality, however, it is biphasic with predominantly antegrade flow and four inflection points.



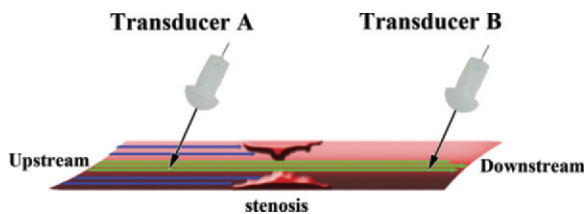
by either (*a*) increasing the size of the sample volume, thereby increasing the range of velocities sampled in the parabolic flow distribution; or (*b*) increasing the Doppler gain. The latter method is analogous to the way any simple cyst can be made to show internal echoes if the gain is high enough. Physiologic spectral broadening occurs in small blood vessels, such as the hepatic or vertebral arteries. In general, the smaller the vessel, the more spectral broadening can be expected, since a wider range of velocities is sampled from the center to the periphery of the vessel. Another cause of physiologic spectral broadening is turbulent flow at bifurcations, such as in the carotid arteries. In such cases, the broadened appearance is due to the wide range of velocities sampled in the disorganized turbulent flow pattern. Pathologic spectral broadening occurs as a result of abnormally compressed (narrowed) vessels, or as a consequence of turbulent flow in the poststenotic portion of a diseased vessel.

### Waveform Nomenclature

Any waveform can be systematically described or may be named with conventional terms. The systematic characterization of all waveforms includes the features described earlier. Specifically, these features include predominant flow direction (antegrade versus retrograde), phasicity (pulsatile, phasic, nonphasic, or aphasic), phase quantification (monophasic, biphasic, triphasic, or tetraphasic), and inflection quantification (aninflectional, diinflectional, or tetrainflectional). Additional features include the presence or absence of spectral broadening and, in arteries, the level of resistance (high versus low). Pragmatically, work volume and time constraints limit the usefulness of routine systematic characterization for all spectral Doppler examinations. However, conventional naming systems, designed for conciseness, help overcome these limitations. These waveform concepts are best reviewed by perusing multiple normal (Fig 11) and abnormal (Fig 12) waveforms, each with its corresponding identifying characteristics and conventional name.



**Figure 12.** Waveform nomenclature (abnormal waveforms). Diagrams illustrate how abnormal waveforms, like normal waveforms, can be systematically characterized on the basis of direction (*D*), phasicity (*P*), phase quantification number (*Q*), and inflection quantification (*I*).



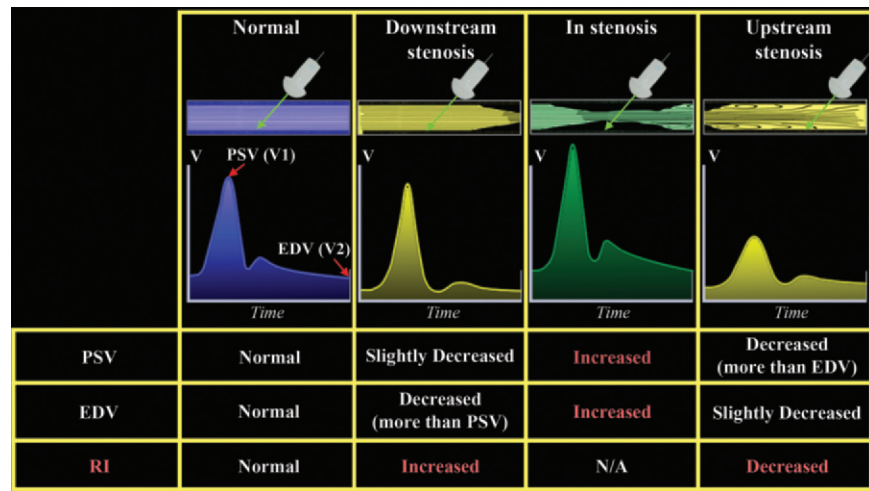
**Figure 13.** Diagram illustrates how the direction of a “stream” is determined by the direction of flow. *Upstream* refers to blood that has not yet passed a reference point, whereas *downstream* refers to blood that has already passed the reference point. From the perspective of the stenosis, transducer A is located upstream. At the position of transducer A, a downstream stenosis is detected. From the perspective of the stenosis, transducer B is located downstream. At the position of transducer B, an upstream stenosis is perceived.

**Stenosis Flow Dynamics**

One should be aware that there is potential for confusion when using the terms *upstream* and *downstream*, depending on whether the US transducer or a point of disease (eg, stenosis)

is chosen as the reference point (Fig 13). For this reason, it is always advisable to qualify the words *upstream* and *downstream* with a descriptor that specifies what is actually upstream or downstream, keeping in mind that flowing blood defines the direction of the “stream.” The phrase *upstream stenosis* means that the transducer is sampling flow velocities from blood that has previously passed through the stenotic portion of a vessel. In such a case, with the stenosis as the reference point, the transducer is actually downstream. Similarly, the phrase *downstream stenosis* means that the transducer is sampling flow velocities from blood that has not yet passed through the stenosis. In such a case, the transducer is actually upstream from the stenosis.

Signs of stenosis may be either direct or indirect (Fig 14). Direct signs are those found at the stenosis, whereas indirect signs are those found upstream or downstream from the stenosis. Direct



**Figure 14.** Flow dynamics in high-grade stenosis. Chart illustrates the effect of stenosis on the contour of spectral waveforms and the measured parameters, such as peak systolic velocity (PSV), end-diastolic velocity (EDV), and RI. Blue = normal vessel and waveform contour, yellow = prestenotic and poststenotic vessels and waveform contours, green = in-stenosis vessel and waveform contour. Note that velocities are increased within a stenotic portion of a vessel, and that the RI is increased when the stenosis is downstream but decreased when the stenosis is upstream. A waveform whose contour is affected by an upstream stenosis is often described as a tardus-parvus waveform.

signs include elevated peak systolic velocity and end-diastolic velocity or spectral broadening due to turbulent flow in the immediately poststenotic portion (8,17). Indirect findings of stenosis can be divided into subjective and objective findings. Subjective evidence of an upstream stenosis is commonly seen as a tardus-parvus waveform. This description is based on empirical observations of the peak of the waveform; specifically, it refers to the late (Latin, *tardus*, “slow” or “late”) and low (Latin, *parvus*, “small”) appearance of the peak (Fig 15). The term *tardus-parvus* is most commonly applied in cases of aortic stenosis and renal artery stenosis; however, this finding may be observed in the poststenotic downstream portion of any vascular territory. A tardus-parvus waveform can be objectively confirmed on the basis of calculations such as acceleration values (normal,  $>5 \text{ m/sec}^2$ ), time to peak (normal,  $<70 \text{ msec}$ ), and RI. The RI is low in the setting of an upstream stenosis because the peak systolic velocity decreases disproportionately more than the end-diastolic velocity. If one measures a downstream stenosis (measuring upstream from the stenosis), the end-diastolic velocity is decreased disproportionately more than the peak systolic velocity; therefore, the RI is higher than normal. It should

be remembered that this finding is not specific for downstream stenosis, since diffuse microvascular disease, among other entities, can also increase resistance.

### Liver Doppler Waveforms

The three major vessels interrogated at liver Doppler US are the hepatic arteries, hepatic veins, and portal veins. **The characteristic waveform signature, which correlates with the unique flow pattern of the vessel in question, is dictated by the anatomic position of the vessel in the circulatory system (systemic arterial side, systemic venous side, or portal venous position) and cyclic pressure variations generated by cardiac activity.** The term *waveform signature* is often used in liver Doppler US because the waveform of each major vessel is so specific that it can be used to identify the vessel, even when the gray-scale or color Doppler US appearance is ambiguous. In fact, sonographers often use the waveform to identify or confirm the imaged vessel.

### Hepatic Arteries

The normal hepatic arterial waveform is the easiest to understand, probably because one can intuitively imagine the wave of pressure generated in the left ventricle and subsequently propagated to the systemic arteries. Simply pal-

Teaching  
Point

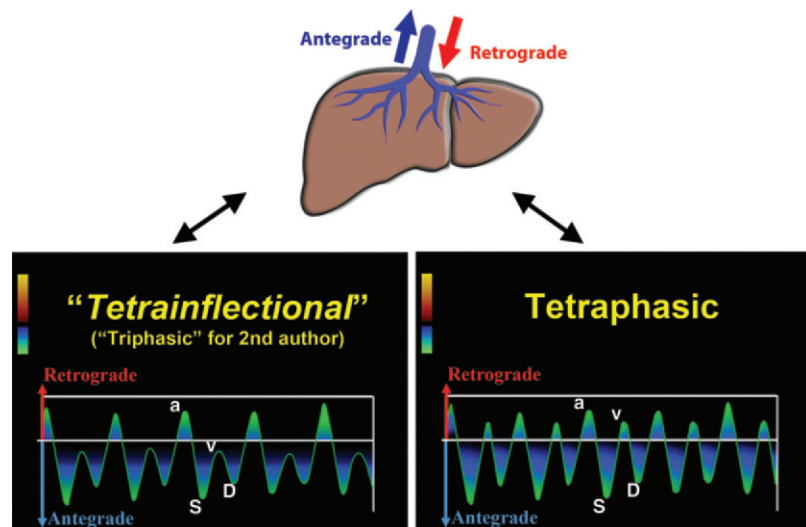


**Table 4**  
**Causes of Elevated Hepatic Arterial Resistance (RI >0.7)**

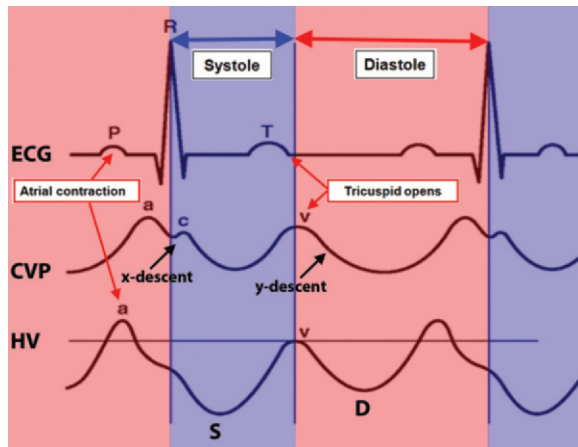
<b>Pathologic (microvascular compression or disease)</b>
Chronic hepatocellular disease (including cirrhosis)
Hepatic venous congestion
Acute congestion → diffuse peripheral vasoconstriction
Chronic congestion → fibrosis with diffuse peripheral compression (cardiac cirrhosis)
Transplant rejection (any stage)
Any other disease that causes diffuse compression or narrowing of peripheral arterioles
<b>Physiologic</b>
Postprandial state
Advanced patient age

**Table 5**  
**Causes of Decreased Hepatic Arterial Resistance (RI <0.55)**

<b>Proximal arterial narrowing</b>
Transplant stenosis (anastomosis)
Atherosclerotic disease (celiac or hepatic)
Arcuate ligament syndrome (relatively less common than transplant stenosis or atherosclerotic disease)
<b>Distal (peripheral) vascular shunts (arteriovenous or arterioportal fistulas)</b>
Cirrhosis with portal hypertension
Posttraumatic or iatrogenic causes
Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)



**Figure 18.** Diagram illustrates normal hepatic venous flow direction and waveform. The direction of normal flow is predominantly antegrade, which corresponds to a waveform that is mostly below the baseline at spectral Doppler US. The term *triphasic*, which refers to the *a*, *S*, and *D* inflection points, is commonly used to describe the shape of this waveform; according to D.A.M., however, this term is a misnomer, and the term *tetrainflectional* is more accurate, since it includes the *v* wave and avoids inaccurate phase quantification. Normal hepatic venous waveforms may be biphasic (bottom left) or tetraphasic (bottom right).



**Figure 19.** Normal time-correlated electrocardiographic (ECG) findings, central venous pressure (CVP) tracing, and hepatic venous (HV) waveform (4). The peak of the retrograde *a* wave corresponds with atrial contraction, which occurs at end diastole. The trough of the antegrade *S* wave correlates with peak negative pressure created by the downward motion of the atrioventricular septum during early to midsystole. The peak of the upward-facing *v* wave correlates with opening of the tricuspid valve, which marks the transition from systole to diastole. The peak of this wave may cross above the baseline (retrograde flow) or may stay below the baseline (ie, remain antegrade). The trough of the antegrade *D* wave correlates with rapid early diastolic right ventricular filling. The cycle then repeats. Note the overall *W* shape of the hepatic venous waveform, which can be remembered by using the word “waveform” as a mnemonic device.

Low hepatic arterial resistance is more specific for disease and has a more limited differential diagnosis, including conditions associated with proximal arterial narrowing (transplant hepatic artery stenosis [anastomosis], atherosclerotic disease [celiac or hepatic], arcuate ligament syndrome) and distal (peripheral) vascular shunts (posttraumatic or iatrogenic arteriovenous fistulas, cirrhosis with portal hypertension and associated arteriovenous or arteriportal shunts, Osler-Weber-Rendu syndrome with arteriovenous fistulas) (Table 5) (18).

The effect of cirrhosis on hepatic arterial microcirculation is complex and variable. Arterial resistance has been shown to be decreased, normal, or increased in cirrhotic patients (19). Some aspects of the disease process, such as inflammatory edema, arterial compression by regenerative nodules, and arterial compression by stiff noncompliant (fibrotic) parenchyma, have been thought to increase resistance (20,21). Other aspects, such as the “hepatic arterial buffer response” (compensatory small artery proliferation and increased numbers of arteriolar beds) and arteriovenous shunting, are thought to decrease

resistance (22). The overall balance of these factors presumably dictates the observed resistance, and it has been shown that hepatic arterial RI is not useful for diagnosing cirrhosis or predicting its severity (9,19).

### Hepatic Veins

The hepatic venous waveform, although well described in the literature, is much more difficult to understand than the hepatic arterial waveform, owing to its many components generated by complex alternating antegrade-retrograde pressure or flow variations, which are in turn created by pressure variations related to the cardiac cycle (1).

A model for understanding this complex waveform requires accepting two pieces of information. First, the bulk of hepatic venous flow is antegrade (Fig 18). Although there are moments of retrograde flow, the majority of blood flow must be antegrade to get back to the heart. Antegrade flow is away from the liver and toward the heart; thus, it will also be away from the transducer and, therefore, displayed below the baseline. Second, just as pressure changes in the left ventricle are transmitted to the systemic arteries, pressure changes in the right atrium will be transmitted directly to the hepatic veins. Viewing oneself as actually sitting inside the right atrium will help predict which way, and how fast, blood flows at each moment of the cardiac cycle. Although this model works for understanding physiologic blood flow and increased pulsatility states (congestive heart failure [CHF] and tricuspid regurgitation), it is not applicable in cases of cirrhosis, since the fibrotic parenchyma compresses the veins and limits free transmission of right atrial pressure changes.

As mentioned earlier, it is possible to “decode” the hepatic venous waveform by imagining oneself inside the right atrium, “feeling” the pressure changes, and correlating these changes with the cardiac cycle as seen at electrocardiography (Fig 19). Anything that increases right atrial pressure (atrial contraction toward end diastole, late systolic atrial filling against a closed tricuspid valve) will cause the wave to slope upward. Anything that decreases right atrial pressure (downward early systolic atrioventricular septal motion, early diastolic right ventricular filling) will cause the wave to slope downward.

The *a* wave is the first wave encountered on the waveform. It is generated by increased right atrial pressure resulting from atrial contraction, which occurs toward end diastole. The *a* wave is an upward-pointing wave with a peak that

corresponds to maximal retrograde hepatic venous flow. In physiologic states, the peak of the *a* wave is above the baseline, and the *a* wave is wider and taller than the *v* wave (the other potentially retrograde wave). Even in pathologic states, the *a* wave remains wider than the *v* wave, which represents the best way to initially orient oneself on the waveform. The only time this rule breaks down is in cases of severe tricuspid regurgitation, when the *S* wave becomes retrograde and merges with the *a* and *v* waves to form one large retrograde *a-S-v* complex.

The *S* wave is the next wave encountered on the waveform. Its initial downward-sloping portion is generated by decreasing right atrial pressure, as a result of the “sucking” effect created by the downward motion of the atrioventricular septum as it descends toward the cardiac apex during early to midsystole. Note that the tricuspid valve remains closed. If it were open (tricuspid regurgitation), the result would be pathologic retrograde flow. The *S* wave corresponds to antegrade hepatic venous flow and is the largest downward-pointing wave in the cycle. The lowest point occurs in midsystole and is the point at which negative pressure is minimally opposed and antegrade velocity is maximal. After this low point, the wave rises again as pressure in the right atrium builds due to ongoing systemic venous return.

The *v* wave is the third wave encountered on the waveform. The upward-sloping portion is generated by increasing right atrial pressure resulting from continued systemic venous return against the still-closed tricuspid valve, all of which occurs toward the end of systole. The peak of the wave marks the opening of the tricuspid valve and the transition from systole to diastole. Thereafter, the wave slopes downward because right atrial pressure is relieved during rapid early diastolic right ventricular filling. The position of the peak of the *v* wave varies from above to below the baseline in normal states. It should be remembered that if the *v* wave never rises above the baseline, it cannot be called retrograde, since the baseline marks the transition from antegrade to retrograde.

The *D* wave is the fourth and last wave encountered on the waveform. Its initial downward-sloping portion is generated by decreasing right atrial pressure resulting from rapid early diastolic right ventricular filling. The *D* wave corresponds to antegrade hepatic venous flow and is the smaller of the two downward-pointing waves. The lowest point occurs when the ante-

**Table 6**  
**Causes of Pulsatile Hepatic Venous Waveform**

Tricuspid regurgitation
Decreased or reversed <i>S</i> wave
Tall <i>a</i> and <i>v</i> waves
Right-sided CHF
Maintained <i>S</i> wave/ <i>D</i> wave relationship
Tall <i>a</i> and <i>v</i> waves

grade diastolic velocity is maximal. The subsequent rising portion results from increasing right atrial pressure generated by the increasing right ventricular blood volume.

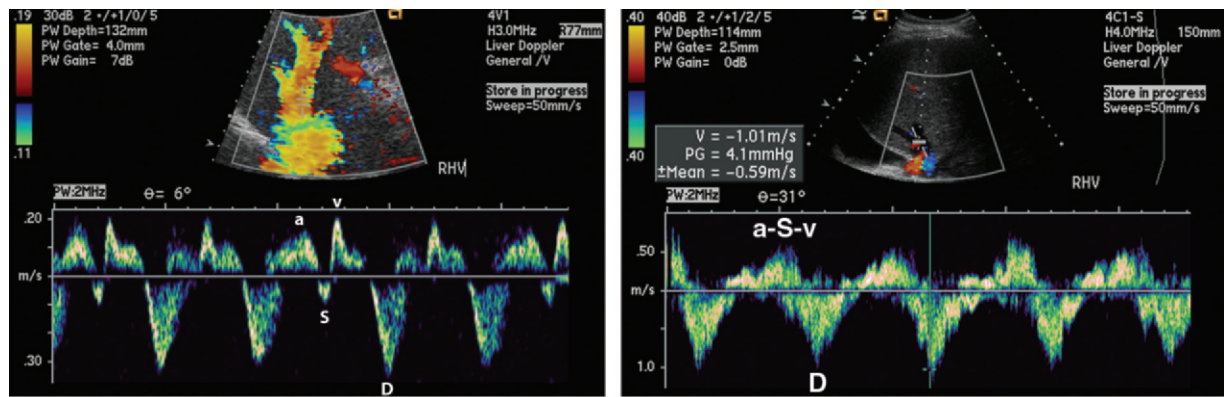
It is almost unheard of to describe flow in the hepatic veins as hepatofugal, since the term is reserved for describing the state of pathologic flow in the portal veins. However, it is important to remember that physiologic flow in the hepatic veins is hepatofugal (ie, away from the liver and toward the heart). In summary, the hepatic venous waveform is normally phasic and predominantly antegrade.

Abnormal (pathologic) hepatic venous flow may manifest in one of several basic ways.

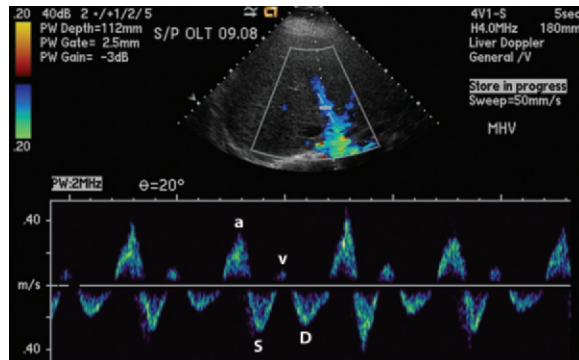
1. Increased pulsatility (pulsatile waveform). Flow in the hepatic veins is described as pulsatile when both the antegrade and retrograde velocities are increased relative to those observed in physiologic states. This creates a waveform with dramatic fluctuations between abnormally tall retrograde waves and abnormally deep antegrade waves. There are two conditions that can create a pulsatile hepatic venous waveform, both of which are also associated with a pulsatile portal venous waveform (3): tricuspid regurgitation and right-sided heart failure without tricuspid regurgitation (2). These two entities can be distinguished by carefully observing the hepatic venous waveform (Table 6).

In tricuspid regurgitation, the incompetent valve limits or even reverses antegrade flow during early systolic contraction and allows retrograde flow during late systole. In early systole, when the atrioventricular septum is descending and would normally create a large burst of negative right atrial pressure, creating the deepest antegrade wave (*S* wave), the incompetent valve instead relieves all or part of the vacuum effect. The result is an *S* wave that is no longer as deep as the *D* wave. This finding has been called the decreased *S* wave (Fig 20a). When tricuspid incompetence is severe enough, flow can switch to retrograde, resulting in an *S* wave that is above the baseline, merging with the *a* and *v* waves to form one large





**Figure 20.** (a) Tricuspid regurgitation. Spectral Doppler image clearly depicts increased pulsatility (ie, wide variation between peaks and troughs). Careful observation shows a pattern that is specific for tricuspid regurgitation. The *v* wave is very tall, and the *S* wave is not as deep as the *D* wave. The latter finding may also be referred to as the “decreased *S* wave” and is specific for tricuspid regurgitation. When tricuspid regurgitation becomes severe, the *S* wave will no longer dip below the baseline, and there will be one large retrograde *a-S-v* complex, or “reversed *S* wave”; when this occurs, the *D* wave is the only manifestation of antegrade flow. (b) Reversed *S* wave. Spectral Doppler image shows a pulsatile waveform with a reversed *S* wave.



**Figure 21.** Right-sided CHF without tricuspid regurgitation. Spectral Doppler image clearly shows increased pulsatility. Careful observation shows a pattern that is specific to right-sided CHF without tricuspid regurgitation. The *a* wave is very tall, and the normal relationship between the *S* and *D* waves is maintained (*S* [systole] is deeper than *D* [diastole]).

retrograde *a-S-v* complex. This finding has been called the reversed *S* wave (Fig 20b) and is seen at clinical examination as a highly pulsatile jugular vein. During late systole, when there should normally be continued systemic venous return against a closed tricuspid valve (rising portion of the *v* wave), the incompetent valve allows large amounts of retrograde flow. This results in the other finding in tricuspid regurgitation, namely, an abnormally tall *v* wave. Toward end diastole, when the right atrium contracts, there is a much higher blood volume (and thus, pressure) than normal, resulting in a tall *a* wave. Therefore, the salient findings in tricuspid regurgitation are a pulsatile waveform with tall *a* and *v* waves and either a decreased or a reversed *S* wave.

In right-sided CHF (Fig 21), as long as the tricuspid valve remains competent, the primary abnormality is too much blood volume on the systemic venous side of the cardiovascular system (including the right atrium). At clinical examination, this pathologic state appears as pitting edema, especially in the lower extremities. At spectral Doppler US, it manifests as abnormally tall *a* and *v* waves. This finding makes sense in the context of our model for understanding these two waves: The tall *a* wave is due to increased right atrial pressure toward end diastole, generated by the larger-than-normal volume contained by the right atrium as it contracts. The tall *v* wave is also due to increased right atrial pressure toward end systole, due to the larger-than-normal volume the right atrium contains while still trying to accommodate continued systemic venous return. Because the tricuspid valve remains competent, the *S* and *D* waves keep their normal configurations relative to each other. The salient findings in right-sided CHF, then, are a pulsatile waveform with tall *a* and *v* waves and a normal relationship between the *S* and *D* waves.

2. Decreased phasicity (decreased pulsatility) and spectral broadening. These two findings go hand in hand; they coexist and represent the same spectrum of disease. Both result from hepatic vein compression.

The waveform of decreased hepatic venous phasicity has been well described in the literature (5). When hepatic venous phasicity is being evaluated, attention to respiratory variation and timing is a critical element of proper US technique.

This is because the waveform is affected not only by the cardiac cycle, but also by respiratory variation. It has been shown that inspiration and expiration both affect the systolic/diastolic ratio, and that the Valsalva maneuver can markedly reduce pulsatility, even to the point of nonphasicity (1). The ideal time to acquire the spectral waveform is during a small (incomplete) inspiratory breath hold. Once proper technique has been confirmed, pathologic causes of nonphasicity may be considered, including cirrhosis, hepatic vein thrombosis (Budd-Chiari syndrome), hepatic veno-occlusive disease, and hepatic venous outflow obstruction from any cause (Table 7). As disease severity progresses and the veins become more compressed by fibrotic constriction or parenchymal edema, they lose their ability to accommodate retrograde flow. This is the one case in which our model for understanding the hepatic venous waveform in terms of right atrial pressure breaks down. Decreased venous compliance is seen as a waveform with a proportional loss of phasicity. A quick and reliable way to grade the severity of decreased phasicity is to visually assess the waveform, focusing on how far the *a* wave drops below the baseline (Fig 22). As long as the *a* wave remains above the baseline, there is normal phasicity; once the *a* wave goes below the baseline, there is at least mildly decreased phasicity, which has been observed in less than 10% of healthy patients (1). Once the peak of the *a* wave is at least halfway between the baseline and the peak negative excursion of the waveform, there is at least moderately decreased phasicity. This degree of decreased phasicity is never normal. When the waveform loses all phasic variation (ie, becomes nonphasic) and no component waves can be distinguished, phasicity is severely decreased.

Spectral broadening is due to the narrowed caliber of compressed hepatic veins, such as occurs in cirrhosis. The hepatic veins are large enough that their waveforms should normally have a thin spectral window. Figure 22 demonstrates spectral broadening in each case of decreased phasicity.

3. Absent (aphasic) hepatic venous flow. Although this finding is diagnostic for venous outflow obstruction (Budd-Chiari syndrome), it should be remembered that this syndrome may also manifest with (*a*) incomplete obstruction, which may have a spectral waveform with decreased phasicity (eg, nonphasicity); or (*b*) increased flow velocities and turbulence at the level of stenosis. Another important observation is the relatively strong association of portal vein thrombosis with Budd-Chiari syndrome. The previous

**Table 7**  
**Causes of Decreased Hepatic Venous Phasicity**

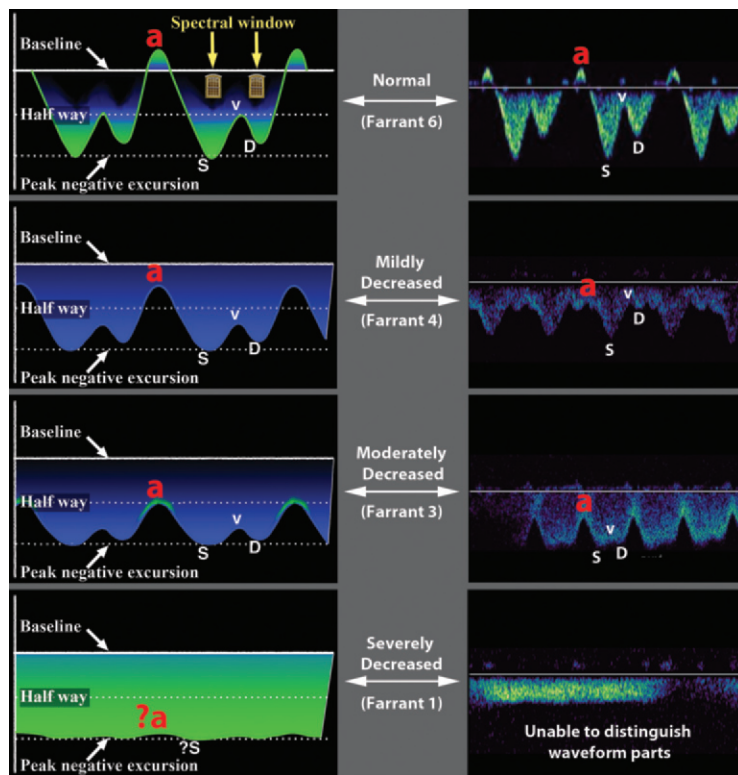
Cirrhosis
Hepatic vein thrombosis (Budd-Chiari syndrome)
Hepatic veno-occlusive disease
Hepatic venous outflow obstruction from any cause

literature indicates that approximately 25% of patients with Budd-Chiari syndrome also have portal vein thrombosis (23).

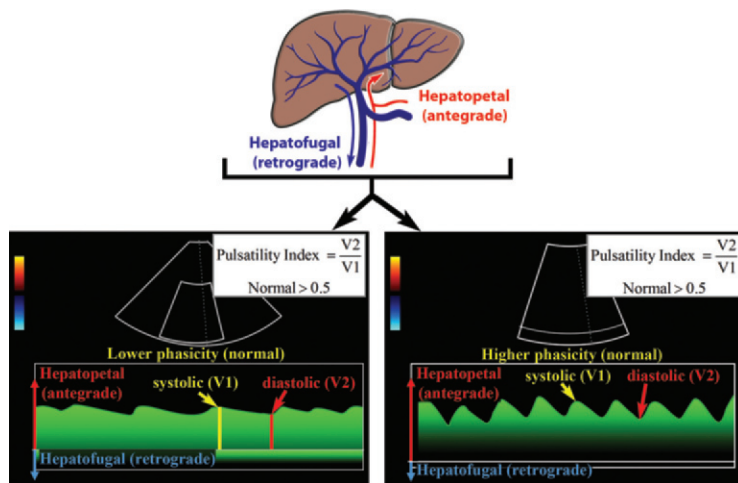
Budd-Chiari syndrome is typically classified into one of three types on the basis of the location of the obstruction. Type 3, also called hepatic veno-occlusive disease, is rare and involves diffuse narrowing at the venule level. Types 1 and 2 are the most common and involve obstruction at the level of the hepatic vein or vena cava. The obstruction is usually secondary to bland thrombus related to a hypercoagulable state; however, the list of causes of hepatic vein occlusion is long and is traditionally divided into primary (eg, congenital webs) and secondary (eg, benign or malignant thrombosis) causes.

Overall, hepatic vein thrombosis is much less common than portal vein thrombosis. Malignant hepatic vein thrombosis (ie, tumor thrombus) is usually the result of direct invasion from an adjacent parenchymal hepatocellular carcinoma; however, any other malignant vena cava thrombosis, such as renal cell carcinoma, adrenal cortical carcinoma, or primary inferior vena cava (IVC) leiomyosarcoma, may also cause Budd-Chiari syndrome. Similar to portal vein thrombosis, both benign and malignant hepatic vein thrombosis may manifest at gray-scale US as an echogenic intraluminal filling defect. In addition, like portal vein thrombosis, tumor thrombus classically enlarges the involved hepatic vein; however, acute bland thrombus can also cause this enlargement. Therefore, vein enlargement is not a reliable discriminating feature.

The characteristic color Doppler US finding in Budd-Chiari syndrome is bicolored, curving hepatic venous collateral vessels. The two colors are generated by the different drainage pathways in these collateral vessels, since they transmit blood to any other patent vein, whether systemic or portal. Potential systemic drainage pathways are intrahepatic (ie, to other hepatic veins, or to the caudate lobe, which usually has its own hepatic venous drainage to the IVC) or extrahepatic (ie, to subcapsular draining veins) (24,25). If there is a malignant thrombus, intratumoral color signals may be appreciated. Spectral Doppler US of bland thrombus will show no appreciable



**Figure 22.** Decreased hepatic venous phasicity. Diagrams illustrate varying degrees of severity of decreased phasicity in the hepatic vein. Farrant and Meire (5) first described a subjective scale for quantifying abnormally decreased phasicity in the hepatic veins, a finding that is most commonly seen in cirrhosis. The key to understanding this scale lies in observing the position of the *a* wave relative to the baseline and peak negative *S* wave excursion. As the distance between the *a* wave and peak negative excursion decreases, phasicity is more severely decreased.



**Figure 23.** Normal portal venous flow direction and waveform. Drawing at top illustrates that the direction of flow in normal portal veins is antegrade, or hepatopetal, which corresponds to a waveform above the baseline at spectral Doppler US. Normal phasicity may range from low (bottom left) to high (bottom right). Abnormally low phasicity results in a nonphasic waveform, whereas abnormally high phasicity results in a pulsatile waveform. The PI is used to quantify pulsatility. Normal phasicity results in a PI greater than 0.5.

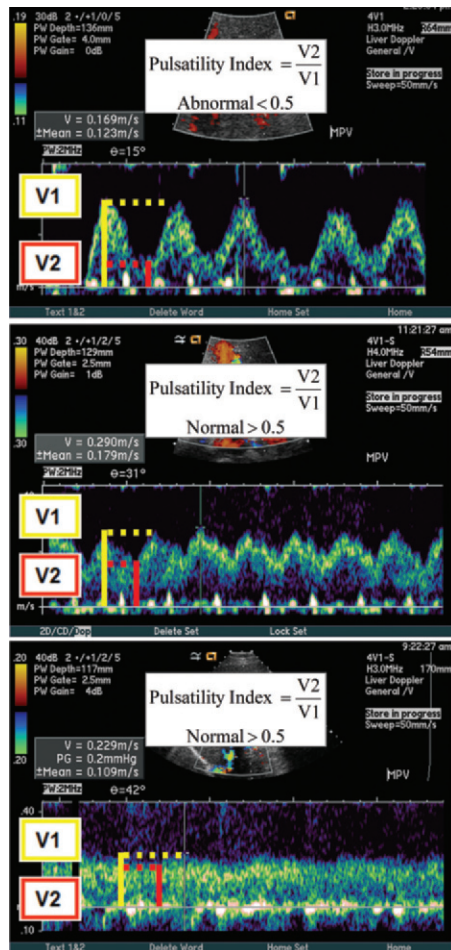
waveform other than noise; however, as in malignant portal vein thrombosis, arterial waveforms may be seen in tumor thrombus. Recent research indicates that contrast material-enhanced US may offer a diagnostic advantage in the detection of malignant hepatic and portal vein thrombosis compared with conventional gray-scale, color Doppler, and spectral Doppler US (26).

**Portal Veins**

In terms of complexity, the portal venous waveform is somewhere between those of the hepatic artery and hepatic veins. A model for understanding portal venous flow requires accepting

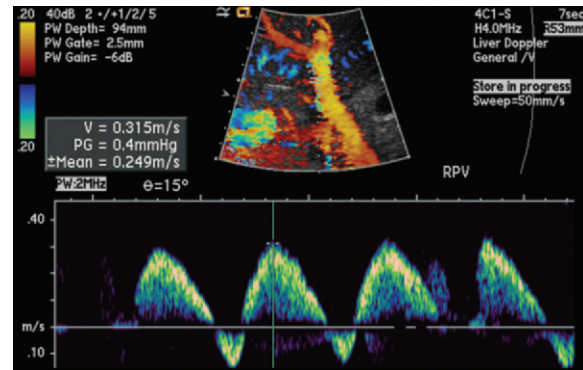
two pieces of information. First, physiologic flow should always be antegrade, which is toward the transducer and therefore creates a waveform that is above the baseline. Second, hepatic venous pulsatility is partially transmitted to the portal veins through the hepatic sinusoids, which accounts for the cardiac variability seen in this waveform. It should also be kept in mind that the flow velocity in this vessel is relatively low (16–40 cm/sec) compared with that in the vessel coursing next to it, namely, the hepatic artery.

The normal portal venous waveform (Fig 23) should gently undulate and always remain above

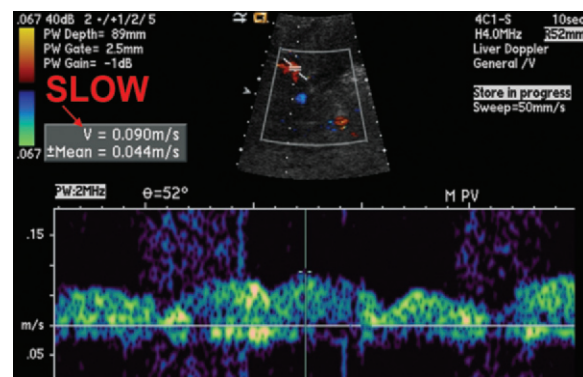


**Figure 24.** Normal and abnormal portal venous phasicity. Images show a spectrum of increasing pulsatility (bottom to top). Note that increasing pulsatility corresponds to a decrease in the calculated PI. Although normal phasicity ranges widely in the portal veins, the PI should be greater than 0.5 (middle and bottom). When the PI is less than 0.5 (top), the waveform may be called pulsatile; this is an abnormal finding.

the baseline. The peak portal velocity ( $V_1$ ) corresponds to systole, and the trough velocity ( $V_2$ ) corresponds to end diastole. At first, one may incorrectly reason that systole should cause back pressure and create the trough; however, such is not the case. The primary influence on variation in portal venous pressure is atrial contraction, which occurs at end diastole. Atrial contraction, toward end diastole, transmits back pressure, first through the hepatic veins, then to the hepatic sinusoids, and ultimately to the portal circulation, where forward portal venous flow (velocity) is consequently decreased (the trough). In fact, prior studies of patients with increased portal venous pulsatility secondary to tricuspid regurgita-



**Figure 25.** Spectral Doppler US image shows a pulsatile waveform with flow reversal in the right portal vein. The waveform may be systematically characterized as predominantly antegrade, pulsatile, biphasic-bidirectional, and di-inflexional.

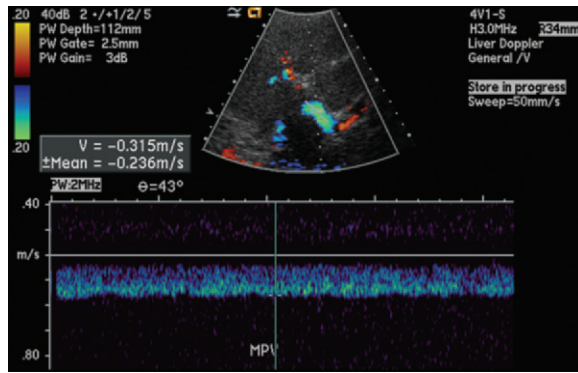


**Figure 26.** Slow portal venous flow. Spectral Doppler US image shows slow flow in the main portal vein. Slow portal venous flow is a consequence of portal hypertension. In this case, the peak velocity is 9.0 cm/sec, which is well below the lower limit of normal (16–40 cm/sec). Although portal hypertension may cause a pulsatile-appearing waveform as seen in this case, the slow flow helps differentiate this condition from hyperpulsatile high-velocity states such as CHF and tricuspid regurgitation.

tion have noted that the portal venous waveform resembles an inverted hepatic venous waveform (1). Therefore, at end diastole, the atrium contracts and the portal venous waveform reaches a low point (trough). The degree of undulation is highly variable but may be quantified with a PI (Fig 24). It is important to note that the PI calculation for the portal vein is different from that for the hepatic arteries (arterial  $PI = (V_1 - V_2) / V_{mean}$ ). In the portal veins, the PI is calculated as  $V_2 / V_1$ , with  $V_1$  normally being greater than 0.5.

Another point worth emphasizing is that lower calculated PIs correspond to higher pulsatility.

Physiologic portal venous flow has been described in many different ways. With regard to flow direction, the terms *antegrade* and *hepatopetal* are synonymous in this vessel. In practice, the



**Figure 27.** Hepatofugal portal venous flow. Spectral Doppler US image shows retrograde (hepatofugal) flow in the main portal vein, a finding that appears blue on the color Doppler US image and is displayed below the baseline on the spectral waveform. Hepatofugal flow is due to severe portal hypertension from any cause.

**Table 8**  
**Causes of Pulsatile Portal Venous Waveform**

Tricuspid regurgitation
Right-sided CHF
Cirrhosis with vascular arterioportal shunting
Hereditary hemorrhagic telangiectasia–arteriovenous fistulas

**Table 9**  
**Findings That Are Diagnostic for Portal Hypertension**

Low portal venous velocity (<16 cm/sec)
Hepatofugal portal venous flow
Portosystemic shunts (including a recanalized umbilical vein)
Dilated portal vein

portal vein is the only vessel in which the terms *hepatopetal* (physiologic) or *hepatofugal* (pathologic) are used to describe flow direction. Early learners often have difficulty keeping these two terms straight, possibly because they incorrectly think that *hepatopetal* means “from the liver and to the feet”; if this were the case, however, the word would be *hepatopedal*, which is not used in the medical literature. The way to keep *hepatopetal* and *hepatofugal* straight is to remember that *-petal* and *-fugal* have Latin origins, with *-petal* describing movement or force toward something and *-fugal* describing movement or force away from something.

With regard to its morphologic features, many have described the portal venous waveform as gently undulating. As previously mentioned, it

is best to avoid the use of confusing descriptors, such as *biphasic*. In summary, the portal venous waveform is normally antegrade and phasic.

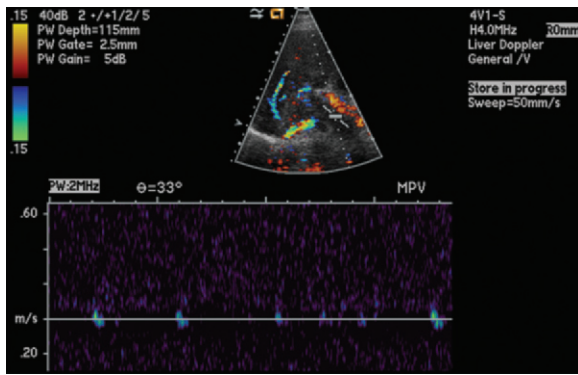
Abnormal (pathologic) portal venous flow usually manifests in one of four ways.

1. Increased pulsatility (pulsatile waveform) (Fig 25). As mentioned earlier, the normal portal venous waveform is described as phasic; therefore, the word *pulsatile* is reserved for describing pathologic flow in portal veins. Pulsatile portal venous flow occurs when there is a large difference between flow velocity at peak systole and at end diastole. The model for understanding processes that can increase pulsatility involves remembering that the hepatic sinusoids connect the portal veins with the hepatic arteries and veins. In the normal state, the arteries do not contribute significantly to pulsatility, whereas the hepatic veins contribute as described earlier. Anything that abnormally transmits pressure to the sinusoids will result in a pulsatile portal venous waveform. On the hepatic venous side, tricuspid regurgitation and right-sided CHF transmit pressure and increase pulsatility. On the arterial side, arteriovenous shunting (as seen in severe cirrhosis) or arteriovenous fistulas (as seen in hereditary hemorrhagic telangiectasia) may have this effect (Table 8).

In practice, it is not difficult to discriminate among the causes of increased pulsatility. Hereditary hemorrhagic telangiectasia is rare and is usually clinically apparent. Tricuspid regurgitation and right-sided CHF can be differentiated by analyzing the hepatic venous waveform and are easily distinguished from cirrhosis, since they manifest as dilated hepatic veins at gray-scale US, whereas cirrhosis manifests as compressed hepatic veins.

2. Slow portal venous flow (Fig 26). Abnormally slow flow occurs when back pressure limits forward velocity. Slow flow is diagnostic for portal hypertension, which is diagnosed when peak velocity is less than 16 cm/sec (Table 9). Portal hypertension is caused by cirrhosis in the vast majority of cases; however, the exhaustive list of causes is generally divided into prehepatic (eg, portal vein thrombosis), intrahepatic (eg, cirrhosis from any cause), and posthepatic (right-sided heart failure, tricuspid regurgitation, Budd-Chiari syndrome) causes (6). The most specific findings for portal hypertension are development of portosystemic shunts (eg, a recanalized umbilical vein) and slow or reversed (hepatofugal) flow. Splenomegaly and ascites are nonspecific and may be seen in other pathologic conditions.

3. Hepatofugal (retrograde) flow (Fig 27). Hepatofugal flow occurs when back pressure exceeds forward pressure, with flow subsequently

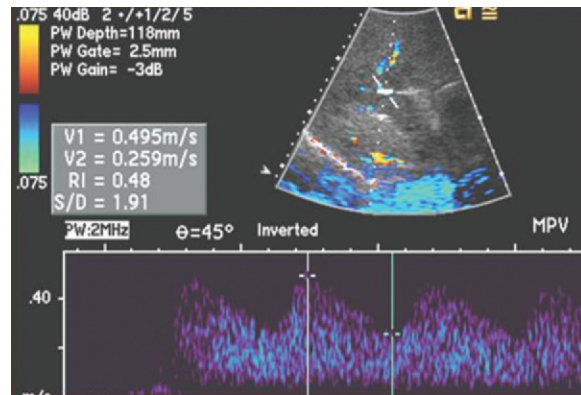


**Figure 28.** Portal vein thrombosis (acute bland thrombus). On a spectral Doppler US image, the interrogation zone shows no color flow in the main portal vein. The spectral waveform is aflat, which indicates absence of flow. An aflat waveform may be produced by either obstructive or nonobstructive disease.

reversing direction. This results in a waveform that is below the baseline. As with slow flow, this finding is diagnostic for portal hypertension from whatever cause.

4. Absent (aflat) portal venous flow (Fig 28, Table 10). Absent flow in the portal vein may be due to stagnant flow (portal hypertension) or occlusive disease, usually caused by bland or malignant thrombosis. Although absent portal venous flow is the sine qua non of occlusive portal vein thrombosis, it must be remembered that intraluminal filling defects may also be nonocclusive if they fail to occupy the entire lumen. In such cases, there will be some degree of flow, which may be increased at the stenosis, turbulent immediately beyond the stenosis, or decreased farther downstream in the poststenotic portion of the vessel.

As mentioned earlier, not all cases of absent flow represent occlusive disease. In severe portal hypertension, there is a period of time during the disease course when flow is neither hepatopetal nor hepatofugal, but stagnant. This results in absent portal venous flow (appreciable at Doppler US) and puts the patient at increased risk for portal vein thrombosis. These same patients (ie, those with cirrhosis) are also at risk for hepatocellular carcinoma and tumor thrombus. Hepatocellular carcinoma is the most common cause



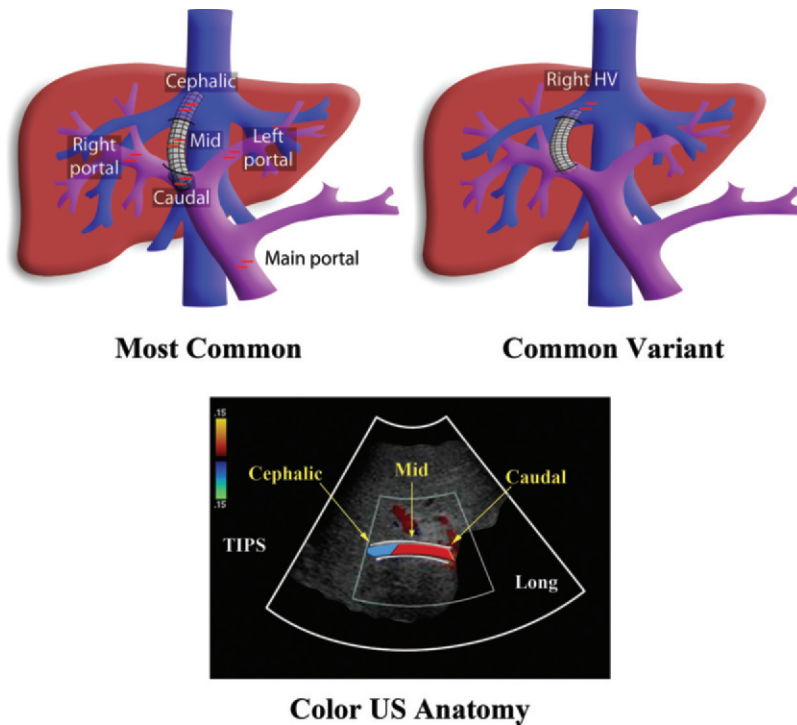
**Figure 29.** Portal vein thrombosis (malignant tumor thrombus). On a spectral Doppler US image, the color Doppler image shows echogenic material in a distended main portal vein without color flow. Tumor thrombus tends to enlarge veins; however, acute thrombus may do this as well. The spectral waveform is pulsatile, a finding that is abnormal in the portal vein. In fact, the pulsatility of this waveform resembles that seen in arteries; hence the term *arterialization* (of the portal venous waveform). This finding is specific for malignant tumor thrombus.

**Table 10**  
**Causes of Absent Portal Venous Flow**

Stagnant flow (severe portal hypertension)
Portal vein thrombosis (bland thrombus)
Tumor invasion

of malignant thrombosis (tumor thrombus), although other possible causes include pancreatic carcinoma, cholangiocarcinoma, metastatic disease, and primary portal venous leiomyosarcoma.

Separating benign from malignant portal vein thrombosis is a regular task for radiologists. The literature is full of tips and techniques for making this distinction at US, as well as at computed tomography (CT) and magnetic resonance imaging. At gray-scale US, both benign and malignant forms typically manifest as an echogenic intraluminal filling defect. The echogenicity of the filling defect cannot be used to distinguish benign from malignant thrombosis, since an echogenic filling defect is seen with equal frequency in both conditions (27). Another weakness of relying on an echogenic filling defect to detect portal vein thrombosis is the fact that the echogenicity of a clot may vary, depending on its age: New clots



**Figure 30.** TIPS anatomy. Drawings at top illustrate the most common positions of a TIPS relative to the native vascular anatomy. Color Doppler US image at bottom shows the appearance of a TIPS. Note that the shunt is best seen in the long view, and that normal flow starts toward the transducer (red, above the baseline) in the caudal portion of the shunt and then moves away from the transducer (blue, below the baseline) in the cephalic part. *HV* = hepatic vein.

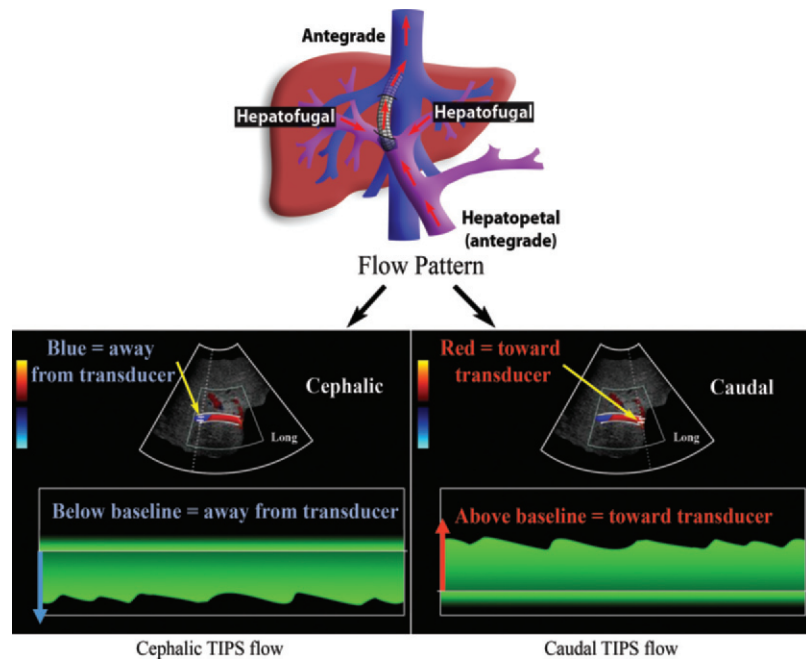
may be hypoechoic or even anechoic. This latter point highlights the usefulness of the color and spectral Doppler portions of a US examination (28). In addition to thrombus echogenicity, portal vein enlargement has been described as a gray-scale US feature of malignant thrombosis. However, tumor thrombus can be seen in the setting of a normal-sized portal vein, and bland thrombus, when acute, can sometimes enlarge the portal vein; therefore, portal vein diameter is not considered a reliable distinguishing feature (27). The most reliable distinguishing gray-scale US feature of malignant thrombus is the combination of an echogenic filling defect with an adjacent liver mass.

As mentioned earlier, in occlusive thrombosis, color Doppler US will demonstrate absent flow. As expected, there will be no normal portal venous waveform in the spectral Doppler portion of the examination. In some cases of malignant thrombosis, there may be color signals within the thrombus; this finding has been referred to as the “thread and streak sign” at both CT angiography and color Doppler US (29). When sampled for spectral evaluation, these color signals show arterial (pulsatile) waveforms, which is a specific sign of tumor thrombus (Fig 29) (26). Another feature of occlusive portal vein thrombosis (especially the nonacute variety) is the development of collateral

vessels in or around the occluded portal vein; this condition is referred to as *cavernous transformation* (30). Cavernous transformation tends to be a marker for bland thrombus, since these collateral vessels usually take a long time (months to years) to develop, and when patients have tumor thrombus, they usually do not live long enough for this development to occur. Nonetheless, cavernous transformation has been documented as occurring within a matter of weeks in occlusive malignant portal vein thrombosis (31).

### Transjugular Intrahepatic Portosystemic Shunts

TIPS are most commonly used for the treatment of severe portal hypertension with refractory variceal bleeding or ascites. Other indications include hepatorenal syndrome, hepatic hydrothorax, and hepatic vein occlusion (Budd-Chiari syndrome) (8). Doppler US has a long track record of reliably helping detect TIPS malfunction (32–40). The key to successful TIPS evaluation lies in knowing the most common anatomic positions of these shunts and how shunt placement affects blood flow (Figs 30, 31). The anatomy may vary, but the fundamental principles of plumbing do not. The basic



**Figure 31.** TIPS flow pattern. Drawing at top illustrates the expected flow pattern within a TIPS and the surrounding vessels when the TIPS is in the most common position. Note that any segment of the portal vein between the caudal portion of the TIPS and the portal bifurcation will have hepatopetal flow. Diagrams at bottom illustrate the appearance of normal flow in the cephalic (left) and caudal (right) parts of the TIPS.

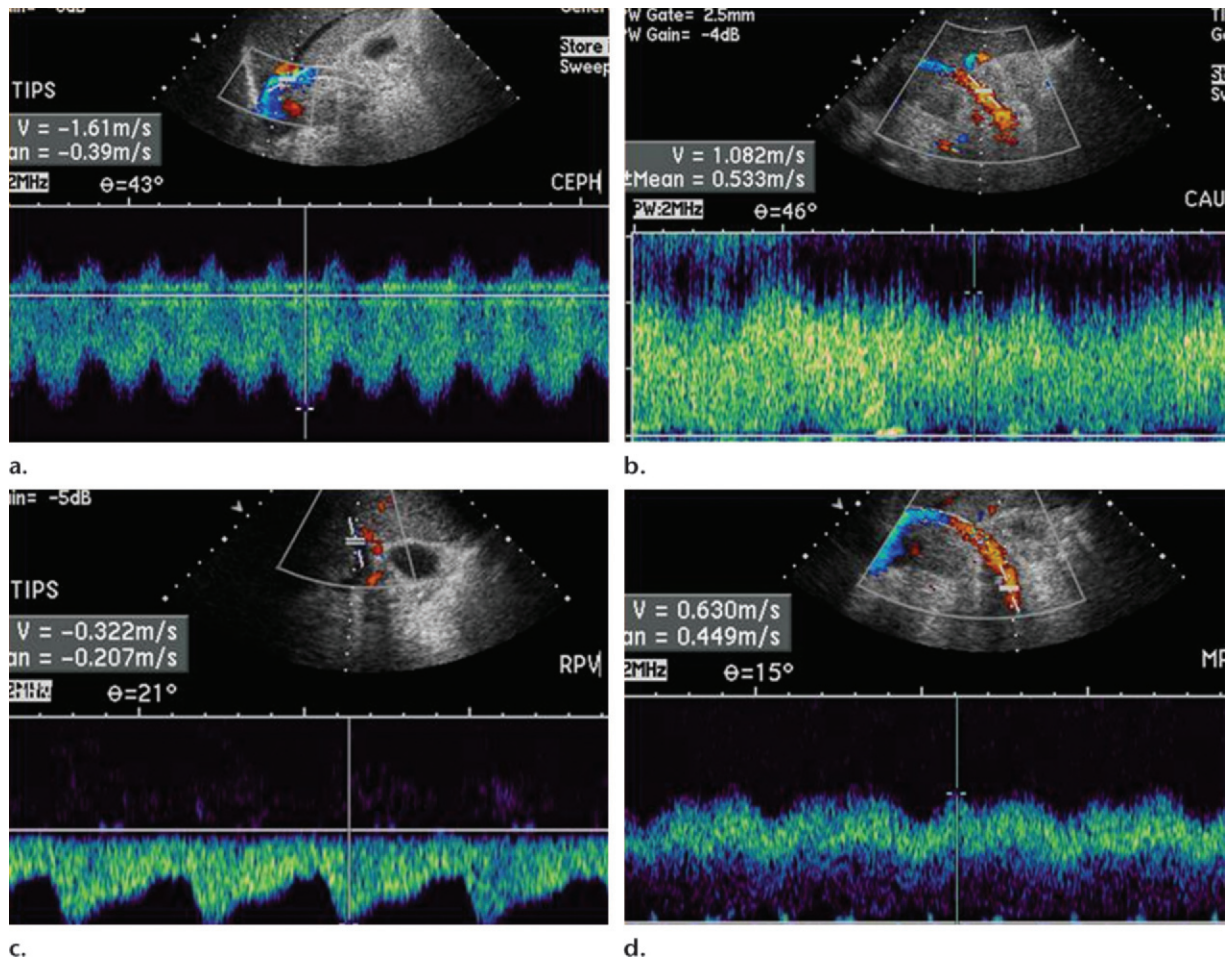
idea is that the shunt is a relatively low-resistance pathway compared with the native vasculature, which has pathologically high resistance, most commonly from cirrhosis (compression of small vessels). Blood preferentially flows into the newly placed low-resistance shunt in approximately two-thirds of patients (41). In terms of anatomy, the cephalic end of the shunt is most commonly located immediate to the connection of the right hepatic vein with the IVC, and the caudal end is located in the right portal vein. However, the cephalic portion may connect with a variable length, or segment, of the right hepatic vein between the shunt and the IVC. Alternatively, the shunt may connect the left hepatic and left portal veins.

At a standard TIPS examination (Fig 32), the examiner searches for direct and indirect evidence of failure. Direct evidence is obtained by imaging the consequences of failure at the site of disease, which may be within the shunt (cephalic, middle, or caudal portion) or in any hepatic vein segment between the cephalic portion and the IVC. On the other hand, indirect evidence of failure is obtained by imaging the consequences of failure in other vessels, such as the main, right, or left portal vein. Therefore, a standard TIPS examination is used to sample (a) the three parts of the shunt; (b) any intervening hepatic vein segment; and (c) the main, right, and left portal veins.

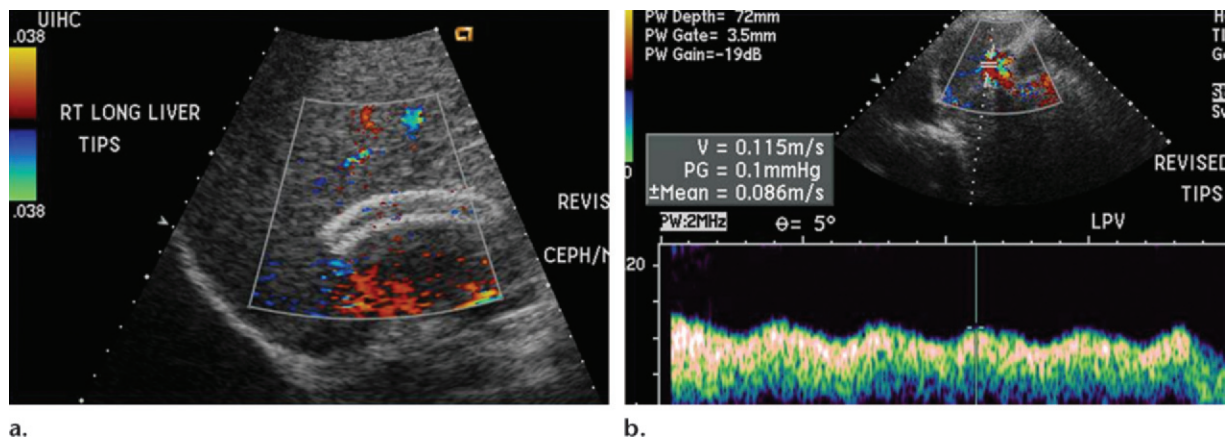
It is impossible to draw a meaningful conclusion from a TIPS examination without comparing velocities and flow directions to those from prior examinations. The first baseline examination should be performed within 1 week of initial placement for Wallstents (Boston Scientific, Natick, Mass), and 1 month after initial placement for covered stents. An acceptable surveillance schedule consists of an examination 3 months after the baseline examination, with an additional examination every 6 months thereafter. The reason why the baseline examination for covered stents is performed 1 month after stent placement is because the polytetrafluoroethylene graft lining contains a small amount of air, which eventually reabsorbs but also generates US artifact soon after placement. Because baseline and prior surveillance examinations are always required for comparison, records must be safely stored and immediately accessible.

Shunt malfunction is the result of narrowing or occlusion caused by intimal hyperplasia or in situ thrombosis. Stenosis, or occlusion, can occur anywhere within the stent; however, it most commonly occurs in the cephalic portion. In addition, stenosis may occur in the variable length of hepatic vein between the stent and the IVC. Occlusion is the easiest type of failure to detect, since it manifests as absent flow at color Doppler US and has an aphasic spectral waveform (Fig 33a). If the type of disease is nonocclusive (ie, stenosis), signs of stenosis indicate TIPS

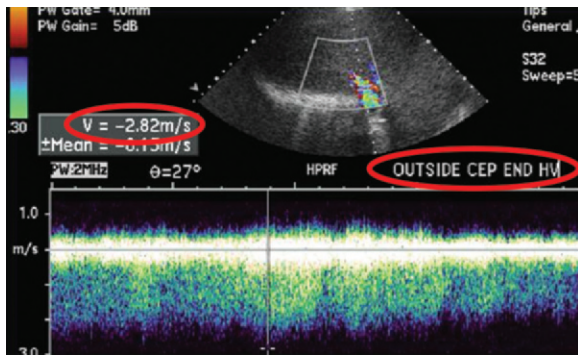




**Figure 32.** Normally functioning TIPS. **(a)** On a spectral Doppler US image, the color Doppler image shows the cephalic end of a TIPS in blue. The waveform is below the baseline, a finding that corresponds to antegrade flow. **(b)** Spectral Doppler image shows the caudal end of the TIPS in red. The waveform is above the baseline (antegrade flow). **(c)** On a spectral Doppler US image of the right portal vein, the waveform is below the baseline. Flow within the vein is hepatofugal, as would be expected in a functioning TIPS. Left portal venous flow was also hepatofugal. **(d)** Spectral Doppler US image shows the main portal vein in red and a waveform above the baseline, both of which findings indicate the expected hepatopetal flow. The velocity (>16 cm/sec) is not low, a finding that supports the patency of the TIPS.



**Figure 33.** TIPS malfunction (occlusion). **(a)** Color Doppler US image obtained in the longitudinal plane shows a TIPS with no color flow, a finding that represents direct evidence of TIPS malfunction. **(b)** Spectral Doppler US image shows hepatopetal flow in the left portal vein. Flow in the right portal vein was also hepatopetal. The prior examination, performed when the TIPS was patent, showed flow in these veins to be hepatofugal; thus, the now hepatopetal flow is indirect evidence of malfunction.



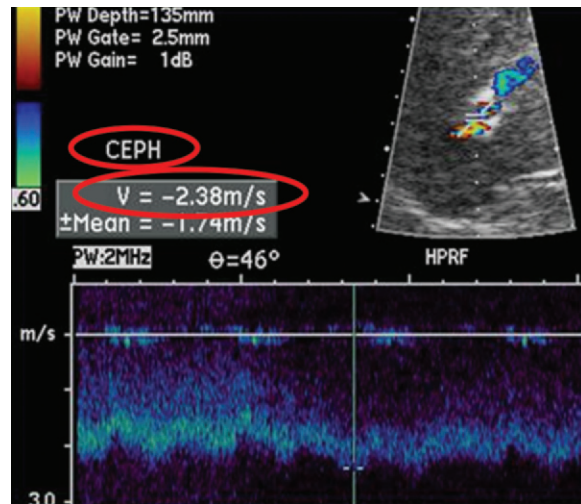
**Figure 34.** TIPS malfunction (hepatic vein stenosis). Spectral Doppler US image shows high-velocity flow (282 cm/sec), which is evidence of hepatic vein stenosis. Visually perceptible narrowing was also apparent in the color Doppler image.

malfunction (Figs 34, 35; Table 11). These signs include an abnormally high (>190 cm/sec) or abnormally low (<90 cm/sec) velocity within the shunt, reminiscent of the criteria used to detect stenosis in a carotid artery. Other evidence includes an abnormal change in velocity (increase or decrease >50 cm/sec) compared with the prior examination. Intrahepatic portal venous flow that was hepatofugal on the prior examination and has changed to hepatopetal flow (Fig 33b) is also evidence of failure. Low velocity (<30 cm/sec) in the main portal vein, or the development or recurrence of collateral vessels such as a recanalized umbilical vein, also suggest failure. Evidence of failure at gray-scale US includes new, recurrent, or worsening ascites.

### Conclusions

An understanding of the basic principles of vascular Doppler US is required to successfully perform liver Doppler US. The conventional nomenclature used to describe the waveforms encountered at liver Doppler US remains inconsistent; however, systematic characterization can be achieved in a reproducible manner.

Fluent interpretation of liver Doppler waveforms is achieved when one is able to comfortably transition between conceptual models of blood flow and the observed waveforms. Pathologic conditions such as portal hypertension,



**Figure 35.** TIPS malfunction (cephalic stenosis). In a spectral Doppler US image obtained in the cephalic portion of a TIPS, the waveform shows a markedly increased flow velocity of 238 cm/sec. This location was the site of the highest flow velocity. Just upstream, in the middle portion of the TIPS, the velocity was 154 cm/sec; just downstream, in the right hepatic vein, the velocity was 126 cm/sec.

right-sided heart failure, and tricuspid regurgitation have characteristic effects on Doppler waveforms.

Doppler US remains the “workhorse” modality for the evaluation of TIPS patency. Competency in interpreting these examinations requires an understanding of TIPS anatomy and expected flow patterns, the availability of prior examination records, and a knowledge of established criteria for shunt failure.

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**Table 11**  
**Signs of TIPS Malfunction**

Direct evidence

- Shunt velocity <90 cm/sec or  $\geq 190$  cm/sec
- Temporal increase or decrease in shunt velocity >50 cm/sec

Indirect evidence

- Main portal venous velocity <30 cm/sec
- Collateral vessels (recurrent, new, or increased)
- Ascites (recurrent, new, or increased)
- Right-left portal venous flow reversal (ie, hepatofugal to hepatopetal)

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## Doppler US of the Liver Made Simple

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RadioGraphics 2011; 31:161–188 • Published online 10.1148/rg.311105093 • Content Codes: GI US

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antegrade flow may be either toward or away from the transducer, depending on the spatial relationship of the transducer to the vessel; therefore, antegrade flow may be displayed above or below the baseline, depending on the vessel being interrogated.

### Page 168

A high RI is not specific for liver disease; therefore, it is less meaningful as an isolated finding than is a low RI.

### Page 168

An RI that is too high may be the result of the postprandial state, advanced patient age, or diffuse distal microvascular disease, which has a wide variety of causes including chronic liver disease due to cirrhosis or chronic hepatitis.

### Page 168

An RI that is too low may be the result of proximal stenosis or distal vascular shunting (arteriovenous or arterioportal fistulas), as seen in severe cirrhosis; trauma (including iatrogenic injury); or Osler-Weber-Rendu syndrome.

### Page 172

The characteristic waveform signature, which correlates with the unique flow pattern of the vessel in question, is dictated by the anatomic position of the vessel in the circulatory system (systemic arterial side, systemic venous side, or portal venous position) and cyclic pressure variations generated by cardiac activity.