SEARCH PATTERN

Check indication, hx and priors
- What is the clinical question?
- What were prior PSV and R1 measurements?

Assess adequacy, technique, and limitations
- Relevant anatomy included in the images?
- Color and spectral Doppler images of all major vasculature?

Assess portal veins (MPV, RPV, LPV)
- Grayscale images:
    - Vessels normal in size?
        - Nml diameter < 13 mm
    - Is there echogenic material to suggest occlusive/non-occlusive thrombus?
    - Are there collaterals?
- Color doppler
    - Are the vessels patent?
    - Are there collaterals?
    - Is the direction of flow normal (hepatopedal)?
- Spectral Doppler
    - Is the velocity at least 40 cm/s
        - Nml 16 -40 cm/s
    - Is there respiratory variability?
        - Flow should be consistently antegrade (above baseline) w gentle undulations
    - Is there pulsatile flow, as seen in HF or TR?
- Thrombus??
    - Ideal: low scale / PRF, high gain, small color box, low wall filter, appropriate depth of focal zone
    - Corroborate w power Doppler.
    - Correborate w microflow.
    - Assess w augmentation
        - Hold then release of Valsalva ->blood sent to liver from portal system

Assess the HVs and vena cava (RHV, MHV, LHV, IVC)
- Grayscale
    - Vessels visible and normal in caliber?
    - Echogenic material to suggest occlusive / nonocclusive thrombus?
    - Collaterals?
    - Look for variations in IVC anatomy
- Color doppler

        - Patent vessels?
        - Collaterals?
        - Normal hepatofugal flow?
    - Spectral doppler
        - Respiratory variability?

Assess the hepatic arteries (MHA, RHA, LHA)
- Gray scale
    - Assess the anastomotic site
    - Look for changes in caliber
    - Look for corkscrew appearance (portal HTN)
- Color doppler
    - Patent vessels?
    - Normal direction of flow?
    - Occlusive / non-occlusive thrombus?
- Spectral doppler
    - Velocity of these vessels

Assess splenic vein and portal confluence
- Patent vessels
- Normal direction of flow?
- Occlusive / nonocclusive thrombus?
- Look at SMV if possible

Assess other areas of visualized abd
- Collaterals / splenorenal shunts?
- Cavernous transformation of the PV?
- Ascites, collections, pleural effusions
- Look for biliary ductal dilatation and any liver lesions

Assess Aorta size and patency
- Abd aorta < 3 cm diameter

If evaluating TIPS, do the following:
- Gray scale:
    - Echogenic material to suggest occlusive / nonocclusive thrombus?
- Color doppler
    - Patency? Aliasing?
    - Expected change in flow in the portal system. All flow should be TOWARDS the TIPS
- Spectral
    - Check PSV 2-3 cm prox to the TIPS at the PV. (nml > 30 cm/s
    - Check PSV at portal end, mid portion and iVC end of TIPS (nml 90-190 cm/s)

- Look for significant change in velocities compared to prior or in continuous segments.

Last checks and proofread

- Did you address the clinical question?

LIVER DOPPLER DISCUSSION

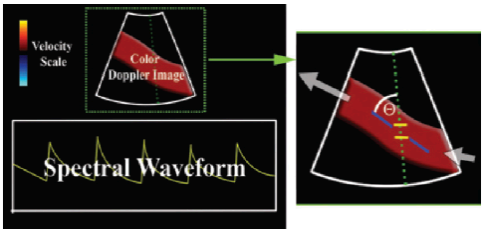


Figure 2. Spectral Doppler examination components. Diagram at left shows the general layout of a spectral Doppler image. The spectral waveform is displayed on the lower half of the image, a color Doppler image is shown above the waveform, and a velocity scale may be shown on either the right or left 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 (Θ), which should be less than 60°; and sample volume or "gate" (yellow). Gray arrows = flow direction.

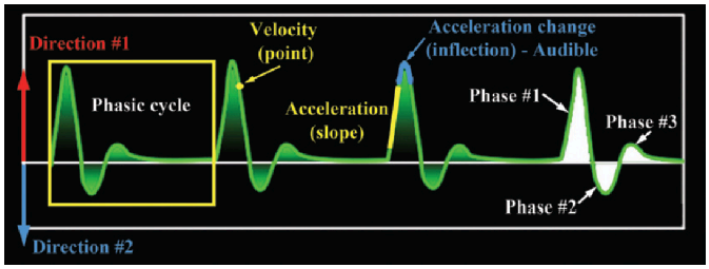


Figure 3. Magnified view of a spectral waveform illustrates its features. Cardiac phasicity creates a phasic cycle, which is composed of phases as determined by the number of times blood flows in each direction. The baseline (x = 0) separates one direction from another. Moving from left to right along 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.

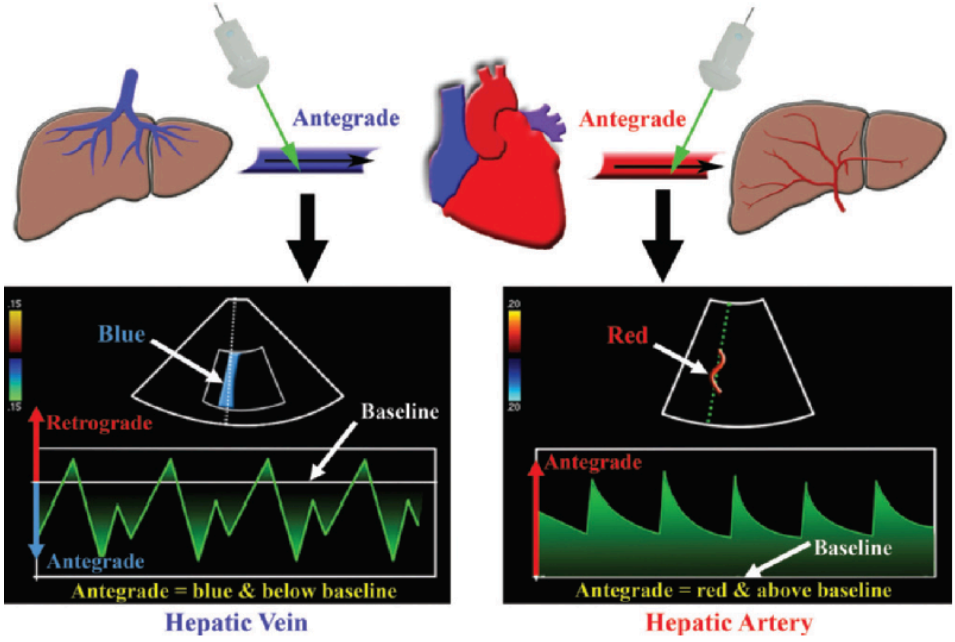


Figure 4. Antegrade versus retrograde flow. Drawings (top) show predominantly antegrade flow from the hepatic veins (blue) to the heart and in the hepatic arteries (red) toward the liver. Retrograde flow would be in the opposite direction. Diagrams (bottom) illustrate typical spectral Doppler waveforms 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).

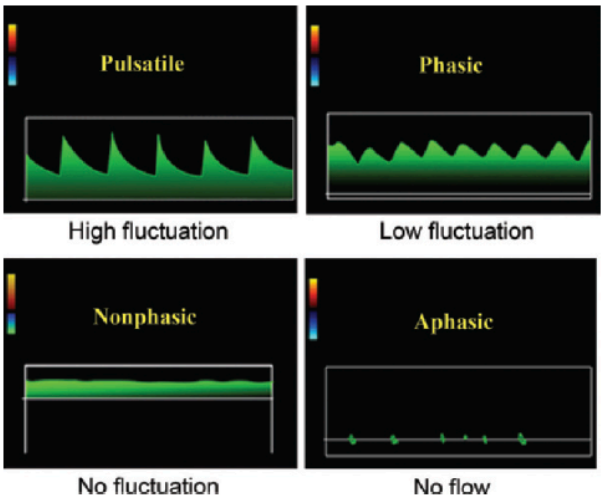


Figure 5. Phasicity. Diagrams illustrate the various waveforms. The terms used to describe the degree of waveform undulation empirically describe the velocity and acceleration features of the waveform. Note that pulsatile, phasic, and nonphasic flow waveforms all have phasicity. Pulsatile flow is exaggerated 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.

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

Internal carotid arteries
Hepatic arteries
Renal arteries
Testicular arteries

Note.—RI = resistive index.

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.

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.

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

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.

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.

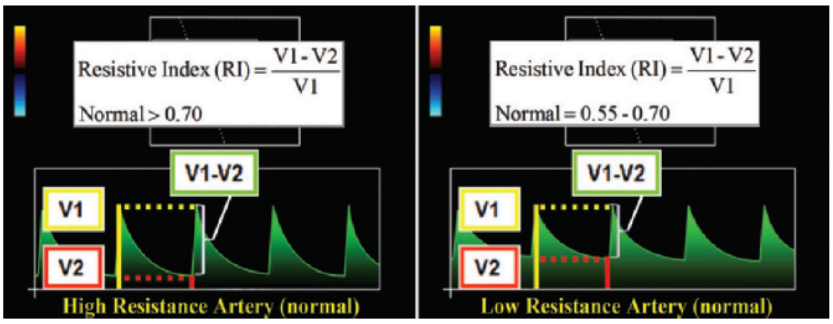


Figure 9. High- versus low-resistance arteries. Schematics illustrate that a high-resistance artery (left) allows less blood flow during end diastole (the trough is lower) than does a low-resistance artery (right). These visual findings are confirmed by calculating an RI. High-resistance arteries normally 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.

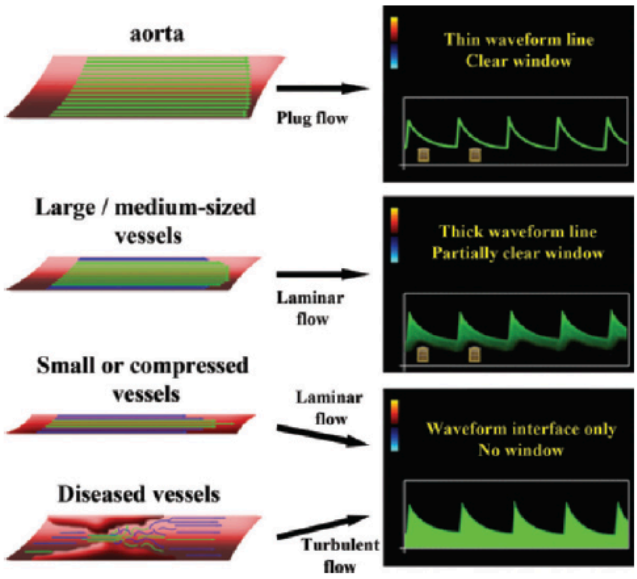


Figure 10. Diagrams illustrate "spectral window" and spectral broadening. In the proximal aorta (top left), plug flow results in a thin waveform and a clear spectral window (top right). Note the actual windows (yellow) superimposed on the first two spectral windows. In vessels smaller than the aorta, 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).

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 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 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.

Artificial broadening is generated 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.

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.

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)

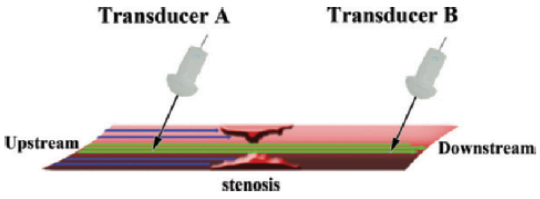
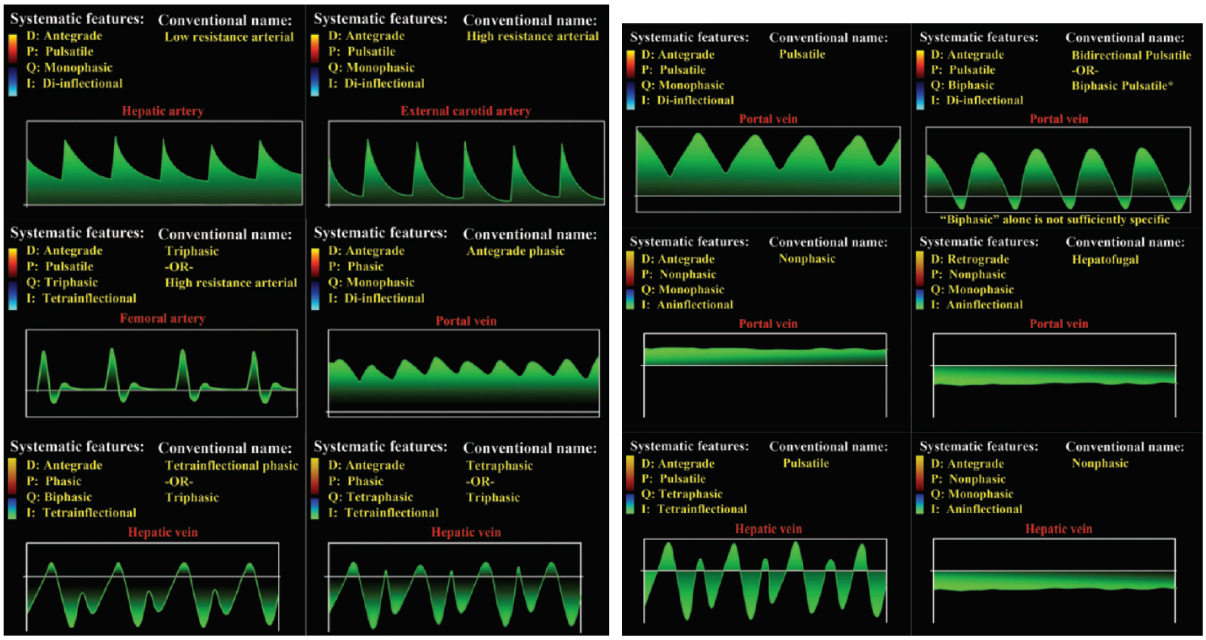


Figure 13. Diagram illustrates how the direction of a "stream" is determined by the direction of flow. *Upstream* refers to blood that has not yet passed a reference point, whereas *downstream* refers to blood that has already passed the reference point. From the perspective of the stenosis, transducer 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.



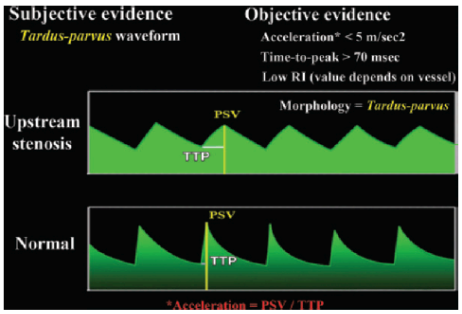
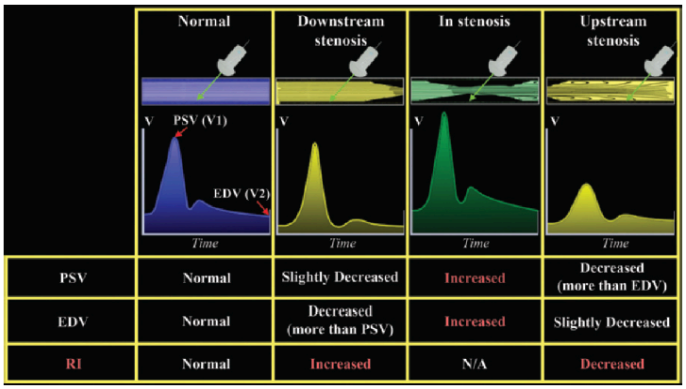


Figure 15. Diagram illustrates upstream stenosis (tardus-parvus waveform). Use of the term *tardus-parvus* requires no measurement or calculation; rather, it is based on subjective observations of the peak of a waveform. When it is apparent that the peak is too late (tardus) and too low (parvus), use of the term is appropriate. This finding occurs only downstream from a stenosis (ie, due to upstream stenosis). It is commonly seen in the setting of renal artery stenosis or aortic stenosis. However, it may also be seen in the setting of hepatic artery stenosis (upstream stenosis). *PSV* = peak systolic velocity, *TTP* = time to peak.



	Normal	Downstream stenosis	In stenosis	Upstream stenosis
PSV	Normal	Slightly Decreased	Increased	Decreased (more than EDV)
EDV	Normal	Decreased (more than PSV)	Increased	Slightly Decreased
RI	Normal	Increased	N/A	Decreased

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.

Transducer distal to stenosis -> RI is low bc PSV decreases disproportionately to EDV
Transducer proximal to stenosis -> RI is high bc EDV decreases more than PSV

Hepatic Artery Waveforms

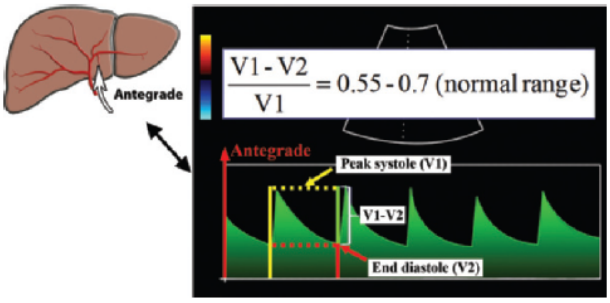


Figure 16. Diagram illustrates normal hepatic arterial flow direction and waveform. The direction of flow in any patent hepatic artery is antegrade (left), which corresponds to a waveform above the baseline at spectral Doppler US (right). The hepatic artery is normally a low-resistance vessel, meaning it should have an RI ranging from 0.55 to 0.7.

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

Pathologic (microvascular compression or disease)
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
Physiologic
Postprandial state
Advanced patient age

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

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

RI is not useful for diagnosing cirrhosis or predicting its severity.

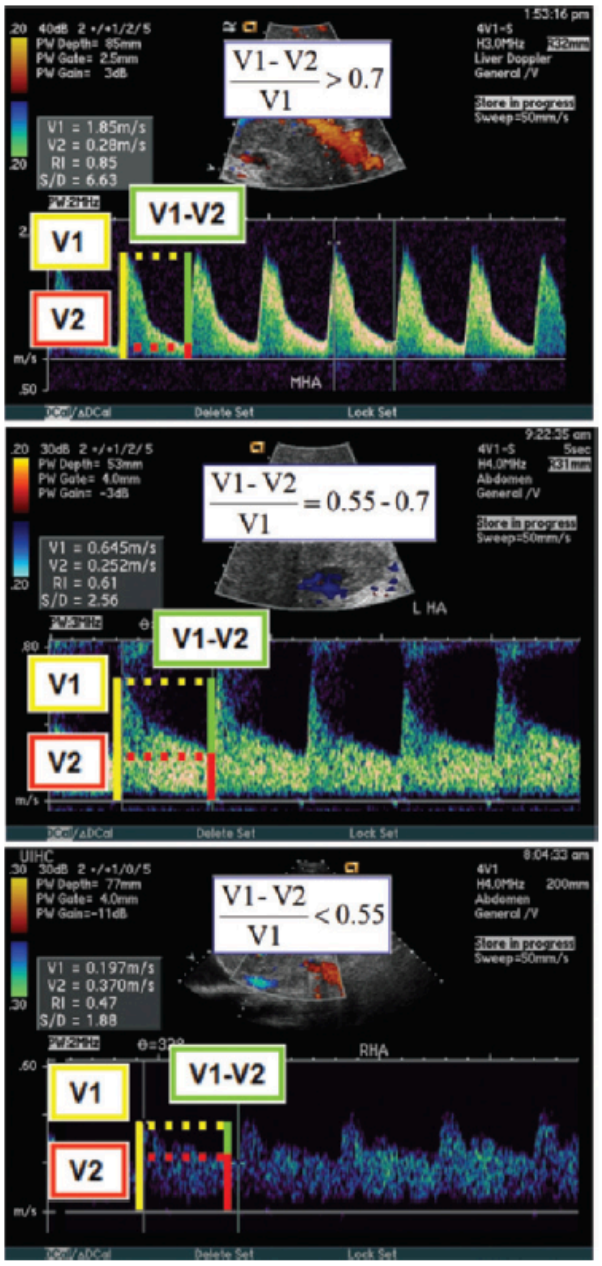


Figure 17. Schematics show a spectrum of increasing hepatic arterial resistance (bottom to top). The hepatic artery normally has low resistance (RI = 0.55–0.7) (middle). Resistance below this range (bottom) is abnormal. Similarly, any resistance above this range (top) may also be abnormal. High resistance is less specific for disease than is low resistance.

Hepatic Vein Waveform

Accept the following:

- HV antegrade flow is AWAY from the liver and transducer and TOWARDS the heart
    - The bulk of hepatic venous flow is antegrade
    - Although there are moments of retrograde flow, the majority of blood flow must be antegrade to get back to the heart.
- As pressure changes in the LV are transmitted to systemic arteries, pressure changes in the RA will be transmitted directly to the HVs.
    - Imagine yourself inside of the RA.
    - This model works for physiologic BF and increased pulsatility states (CHF and TR) but not applicable in cases of cirrhosis bc fibrotic parenchyma compresses the veins and limits free transmission of RA pressure changes.
        - Anything that increases RA pressure will cause wave to slope up
            - Atrial contraction toward end diastole, late systolic atrial filling against a closed tricuspid valve) will cause the wave to slope upward.
        - Anything that decreases RA pressure will cause wave to slow down downward
            - Early systolic atrioventricular septal motion, early diastolic right ventricular filling) will cause the wave to slope downward.
        -

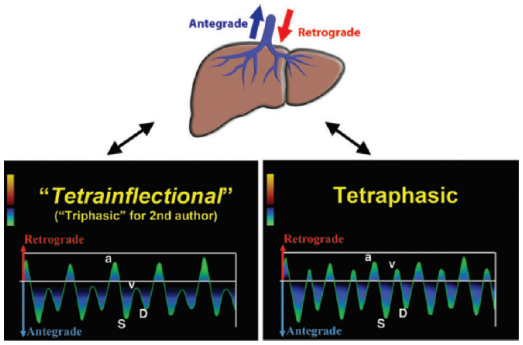


Figure 18. Diagram illustrates normal hepatic venous flow direction and waveform. The direction of normal flow is predominantly antegrade, which corresponds to a waveform that is mostly below the baseline at spectral Doppler US. The term *triphasic*, which refers to the *a*, *S*, and *D* inflection points, 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).

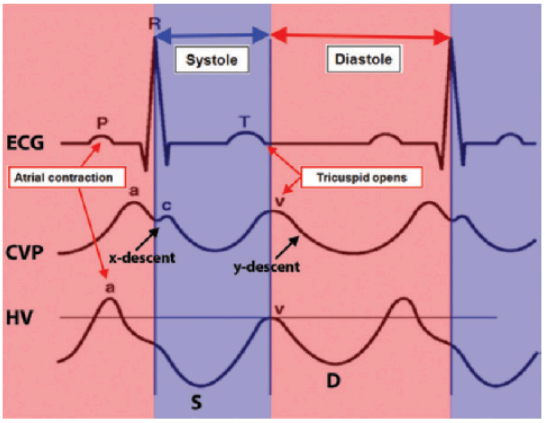


Figure 19. Normal time-correlated electrocardiographic *(ECG)* findings, central venous pressure *(CVP)* tracing, and hepatic venous *(HV)* waveform (4). The peak of the retrograde *a* wave corresponds with *a*trial contraction, which occurs at end diastole. The trough of the antegrade *S* wave correlates with peak negative pressure created by the downward motion of the atrioventricular septum during early to mid*s*ystole. The peak of the upward-facing *v* wave correlates with opening of the tricuspid *v*alve, 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 *d*iastolic 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.

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 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.

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.

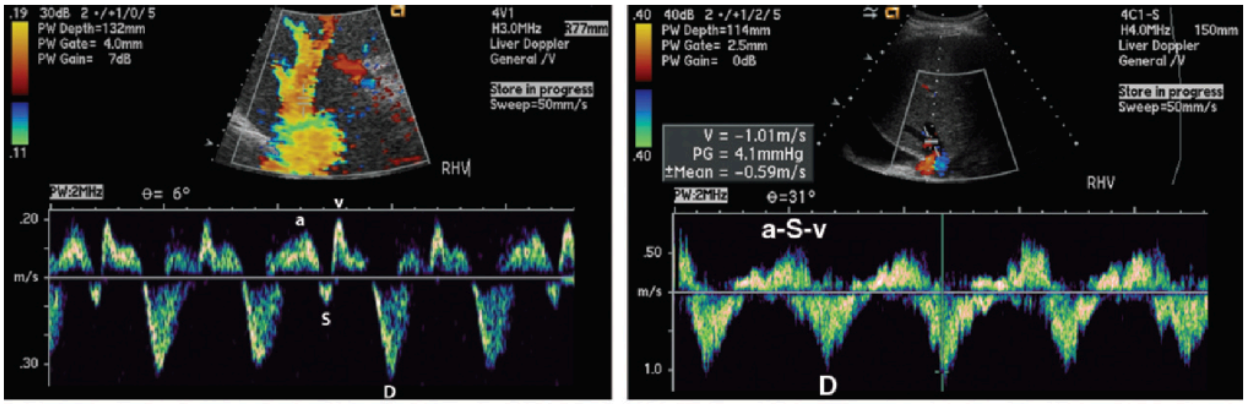
The physiologic flow in the hepatic veins is hepatofugal. The HV waveform is normally phasic and predominantly antegrade.

Abnormal hepatic venous flow manifests in several ways:

- Increased pulsatility; when both antegrade and retrograde velocities are increased relative to physiologic states creating abnormally tall waves.
- Two Conditions can create this waveform and both are associated w a pulsaatile portal venous waveform.
    - TR
    - RHF

Table 6
Causes of Pulsatile Hepatic Venous Waveform

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



a. b.

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.

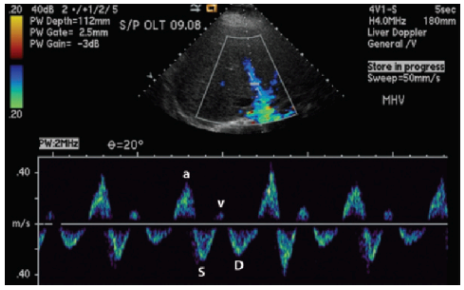


Figure 21. Right-sided CHF without tricuspid regurgitation. Spectral Doppler image clearly shows increased pulsatility. Careful observation shows a pattern that is specific to right-sided CHF without tricuspid regurgitation. The *a* wave is very tall, and the normal relationship between the *S* and *D*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

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

Decreased pulsatility and spectral broadening coexist and represernt the same spectrum of dz/ both result form HV compression. It has been shown that inspiration and expiration both affect the systolic/diastolic ratio, and that the Valsalva maneuver can markedly reduce Pulsatility. 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. 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.

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. 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).

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.

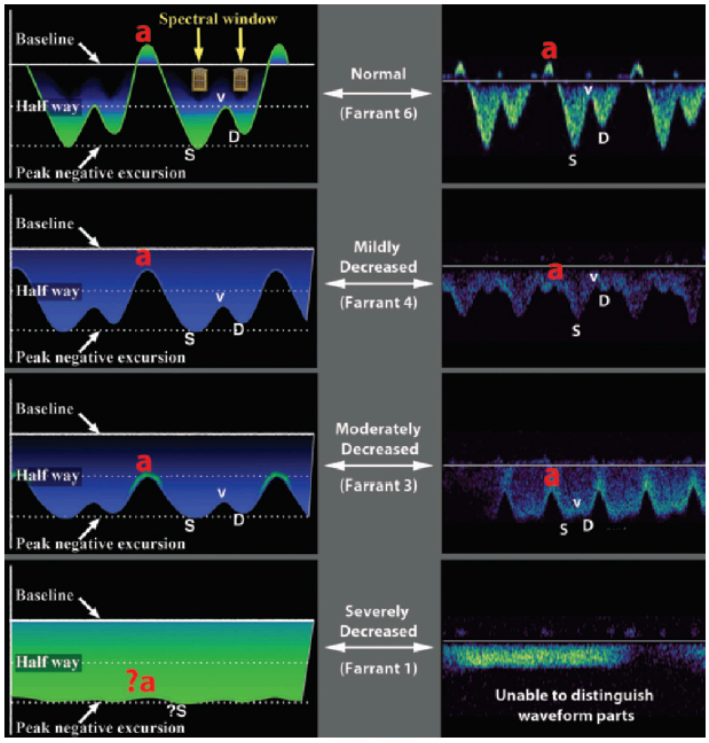


Figure 22. Decreased hepatic venous phasicity. Diagrams illustrate varying degrees of severity of decreased phasicity in the hepatic vein. Farrant and Meire (5) first described a subjective scale for quantifying abnormally decreased phasicity in the hepatic veins, a finding that is most commonly seen 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.

Absent hepatic venous flow (venous outflow obstruction) can manifest as incomplete obstruction with spectral waveform w decreased phasicity or increased flow velocities and turbulence at the level of the stenosis.

Budd chiari syndrome is classified into 1 of 3 types:

- Type 3 / hepatic venoocclusive dz is rare and involves diffuse narrowing at the venule level
- Types 1 and 2 are more common and involve obstruction at the level of the hepatic vein or vena cava. Usually 2/2 bland thrombus from hypercoagulable state.

Hepative vein occlusion is much less common than portal vein thrombosis.
Malignant HV thrombosis is usually from direct invasion from adjacent HCC. RCC, adrenal cortical carcinoma or primary iVC leiomyosarcoma can cause it too.
Both benign and malignant HV thrombosis manifest as gray scale echogenic intraluminal filling defect. LIke portal vein thrombosis, tumor classically enlarges the involved hepativ vein howver acute bland thrombus can also cause enlargmenet.

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 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.

PORTAL VEINS

2 rules

- Physiologic flow should always be antegrade, toward transducer, creating waveform above baseline.
- Hepatic venous pulsatility is partially transmitted to PVs through the hepatic sinusoids, which accounts for cardiac variability in the waveform.

FLow velocity is relatively low (16 - 40 cm/sec) compared to hepatic artery. The normal portal venous waveform should gently undulate and always remain above the baseline.

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).

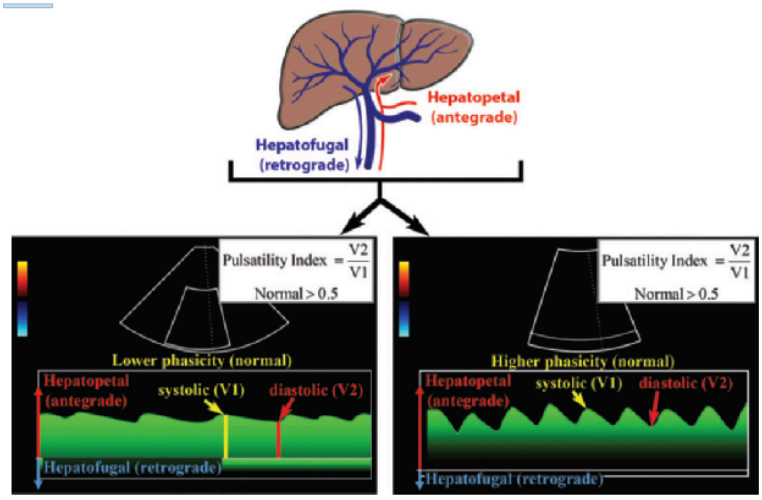


Figure 23. Normal portal venous flow direction and waveform. Drawing at top illustrates that the direction of flow in normal portal veins is antegrade, or hepatopetal, which corresponds to a waveform above the baseline at spectral Doppler US. Normal phasicity may range from low (bottom left) to high (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.

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 = (V1–V2)/Vmean). In the portal veins, the PI is calculated as V2/V1, with V1 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 portal vein is the only vessel in which the terms hepatopetal (physiologic) or hepatofugal (pathologic) are used to describe flow direction.

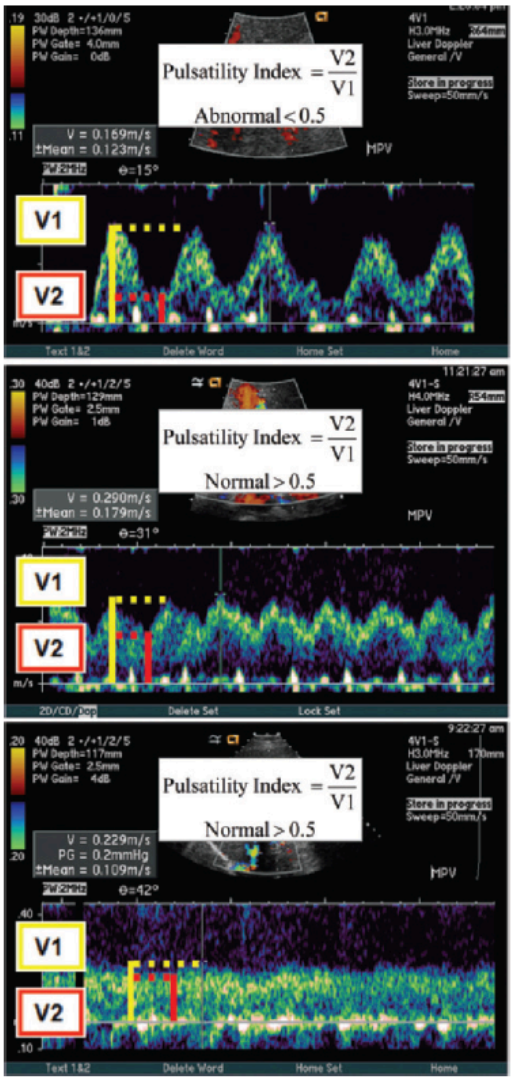


Figure 24. Normal and abnormal portal venous phasicity. Images show a spectrum of increasing pulsatility (bottom to top). Note that increasing pulsatility corresponds to a decrease in the calculated PI. Although normal phasicity ranges widely in the portal veins, the PI should be greater than 0.5 (middle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

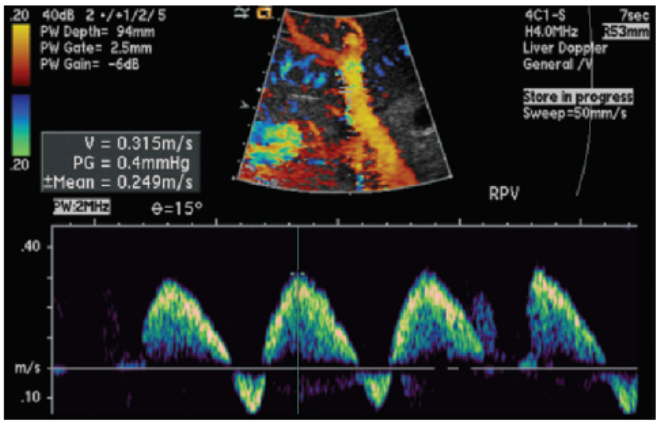


Figure 25. Spectral Doppler US image shows a pulsatile waveform with flow reversal in the right portal vein. The waveform may be systematically characterized as predominantly antegrade, pulsatile, biphasic-bidirectional, and di-inflectional.

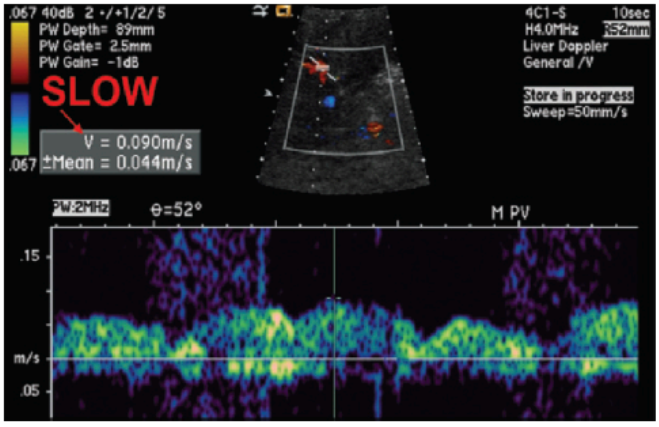


Figure 26. Slow portal venous flow. Spectral Doppler US image shows slow flow in the main portal vein. Slow portal venous flow is a consequence of portal hypertension. In this case, the peak velocity is 9.0 cm/sec, which is well below the lower limit of normal (16–40 cm/sec). Although portal hypertension 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.

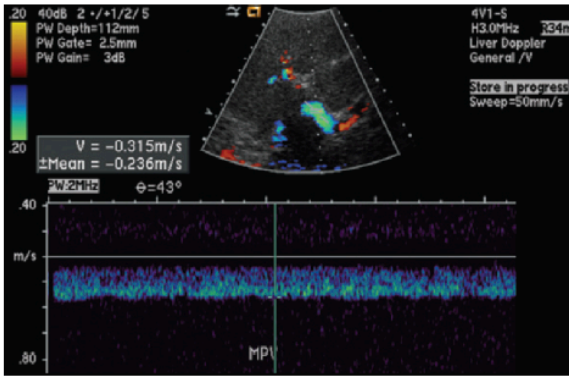


Figure 27. Hepatofugal portal venous flow. Spectral Doppler US image shows retrograde (hepatofugal) flow in the main portal vein, a finding that appears blue on the color Doppler US image and is displayed below the baseline on the spectral waveform. Hepatofugal flow is due to severe portal hypertension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

Abnormal portal venous flow manifests 2/2 one of the following:

- Increased pulsatility
    - Pulsatile is reserved for describing pathologic flow in portal veins. Pulsatile portal venous flow occurs when there is a large difference b/n flow velocity at peak systole and at end diastole.
    - Remember 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
- Slow portal venous flow
    - 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. 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.
    - Cause categories:
        - Prehepatic (eg, portal vein thrombosis)
        - Intrahepatic (eg, cirrhosis from any cause)

    - Posthepatic (right-sided heart failure, tricuspid regurgitation, Budd-Chiari syndrome)
- Hepatofugal / retrograde flow
    - Back pressure exceeds forward pressure, with flow subsequently 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.
- Absent portal venous flow
    - 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. 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.

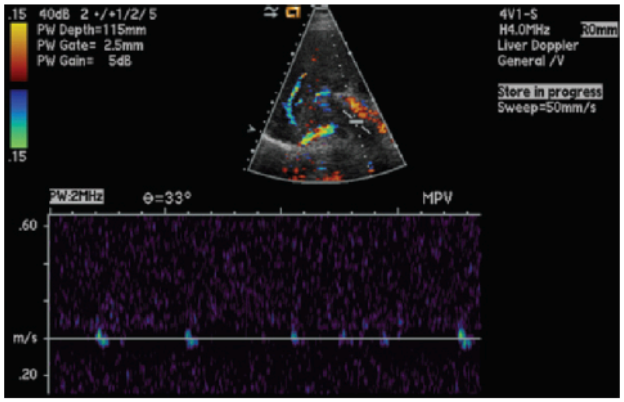


Figure 28. Portal vein thrombosis (acute bland thrombus). On a spectral Doppler US image, the interrogation zone shows no color flow in the main portal vein. The spectral waveform is aphasic, which indicates absence of flow. An aphasic waveform may be produced by either obstructive or nonobstructive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

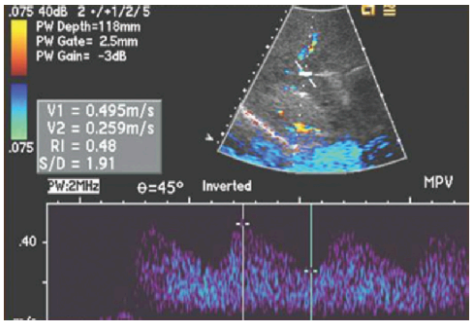


Figure 29. Portal vein thrombosis (malignant tumor thrombus). On a spectral Doppler US image, the color Doppler image shows echogenic material in a distended main portal vein without color flow. Tumor thrombus tends to enlarge veins; however, acute thrombus may do this as well. The spectral waveform 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

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

TIPS

Indications:

- Treatment of severe portal hypertension with refractory variceal bleeding or ascites
- Hepatorenal syndrome
- Hepatic hydrothorax
- Hepatic vein occlusion

The shunt is a relatively low resistance pathway compared w native vasculature which has pathologically high resistance 2/2 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 immediately 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.

Standard TIPS examination involves searching 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.

Table 11
Signs of TIPS Malfunction

Direct evidence
Shunt velocity <90 cm/sec or ≥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)

Sources.—References 34 and 38.

Therefore, a standard TIPS examination is used to sample

- The three parts of the shunt
- Any intervening hepatic vein segment
- The main, right, and left portal veins.

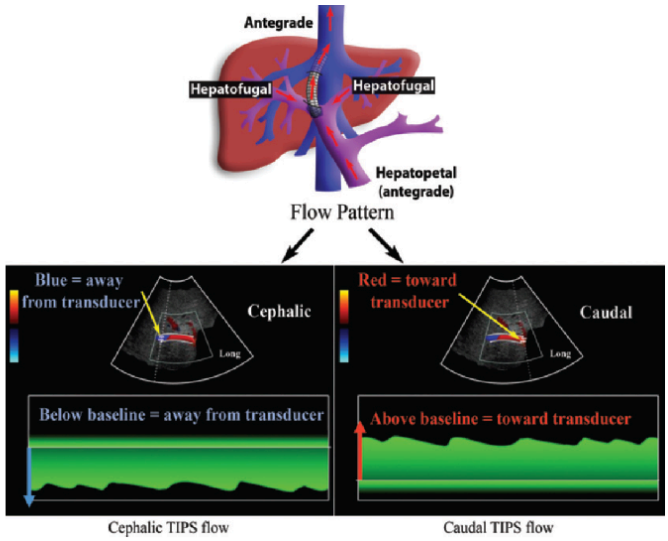


Figure 31. TIPS flow pattern. Drawing at top illustrates the expected flow pattern within a TIPS and the surrounding vessels when the TIPS is in the most common position. Note that any segment of the portal vein between the caudal portion of the TIPS and the portal bifurcation will have hepatopetal flow.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

Must also compare velocities and flow directions to those from prior exams.

- First baseline: perform w/n 1 week of initial placement for Wallstents (Boston Scientific, Natick, Mass) and 1 month after initial placement for covered stents.
    - 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
- Surveillance schedules consist of exams 3 months after baseline with additional exams every 6 months thereafter.
- 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. 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 malfunction.
    - Abnormally high (>190 cm/sec) or low (<90 cm/sec) velocity within the shunt
    - Abnormal change in velocity (increase or decrease >50 cm/sec) compared with the prior examination.
    - Intrahepatic portal venous flow previously hepatofugal now hepatopetal flow
    - 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.

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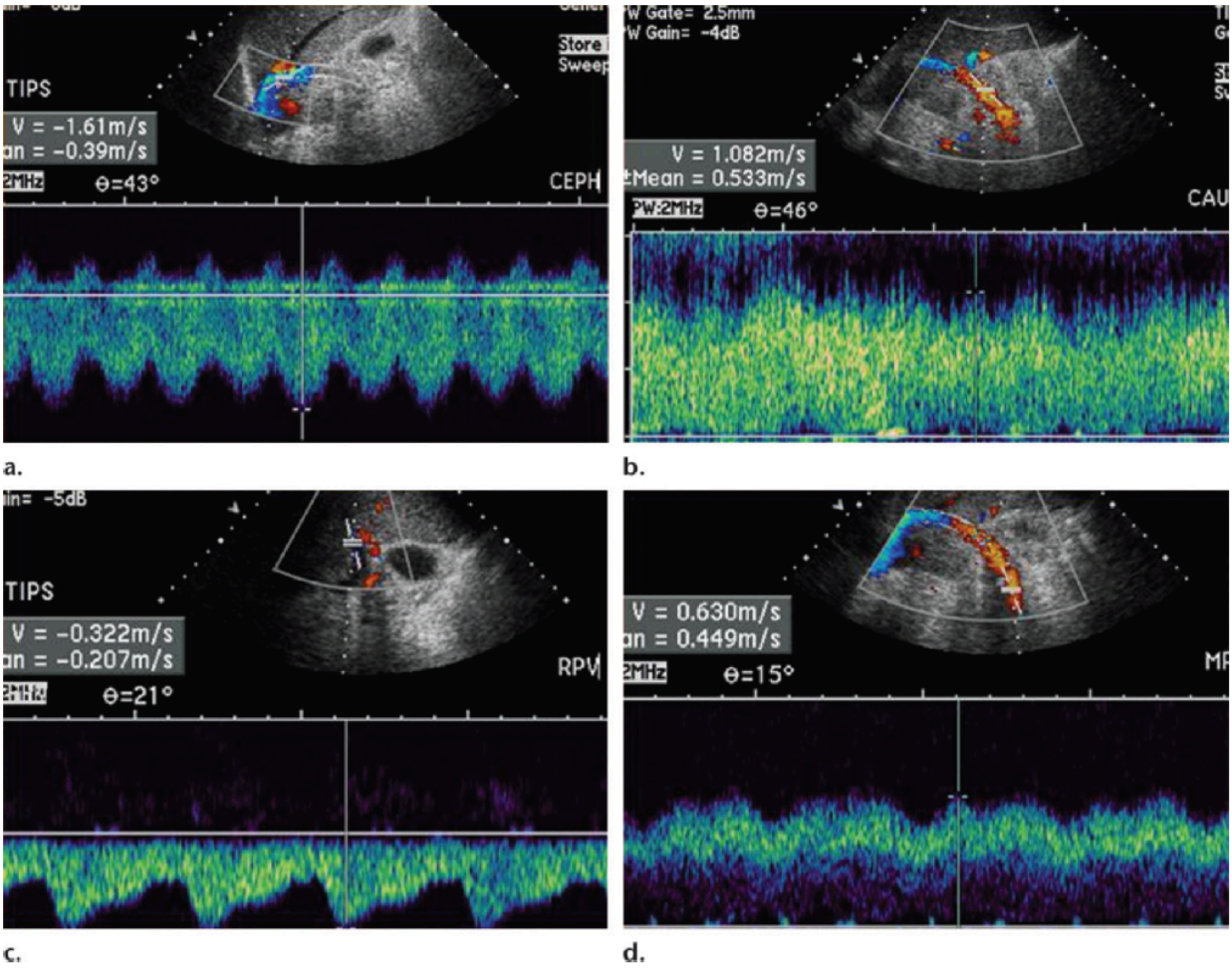
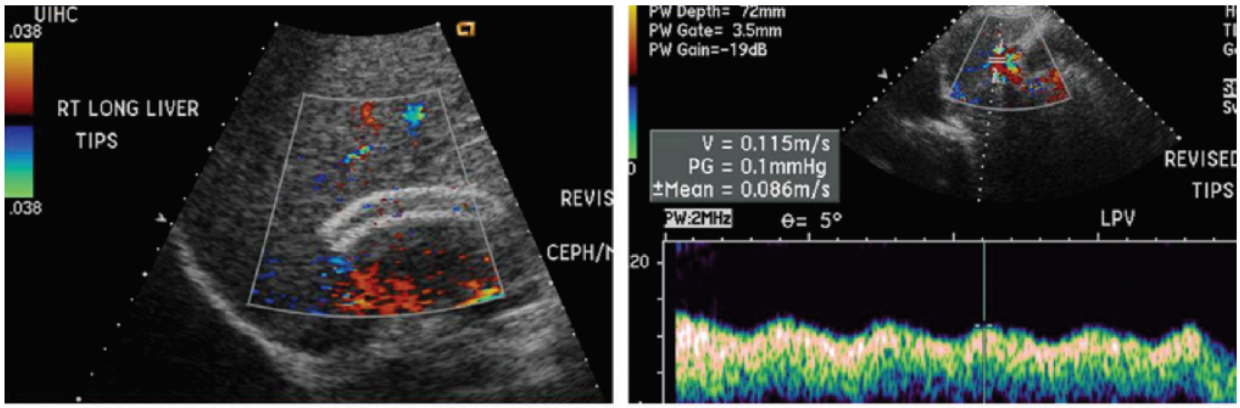


Figure 32. Normally functioning TIPS. **(a)** On a spectral Doppler US image, the color Doppler image shows the cephalic end of a TIPS in blue. The waveform is below the baseline, a finding that corresponds to antegrade flow. **(b)** Spectral Doppler image shows the caudal end of the TIPS in red. The 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.



a. b.

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.

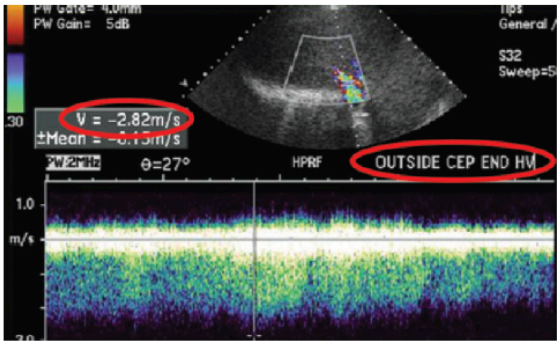


Figure 34. TIPS malfunction (hepatic vein stenosis). Spectral Doppler US image shows high-velocity flow (282 cm/sec), which is evidence of hepatic vein stenosis. Visually perceptible narrowing was also apparent in the color Doppler image.

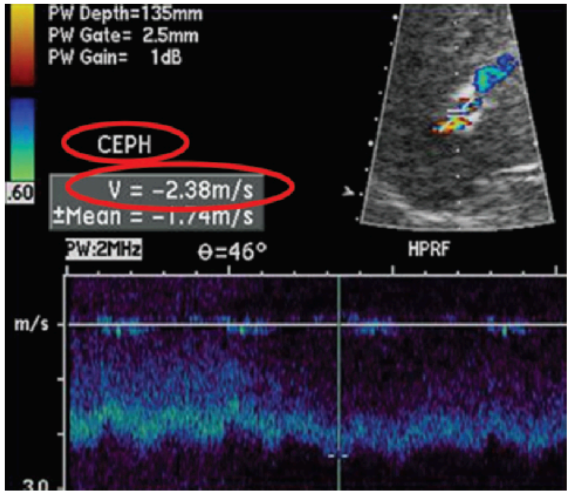


Figure 35. TIPS malfunction (cephalic stenosis). In a spectral Doppler US image obtained in the cephalic portion of a TIPS, the waveform shows a markedly increased flow velocity of 238 cm/sec. This location was the site of the highest flow velocity. Just upstream, in the middle portion of the TIPS, the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

Pathology

Portal vein thrombosis

[Portal Vein Occlusion]

Imaging Recommendations

- Best imaging tool
  - Color Doppler US initially: Highly accurate and cost effective
    - CEUS: Helps distinguish malignant from bland thrombus
  - CECT or MR for comprehensive evaluation
- Protocol advice
  - Color and spectral Doppler US, CEUS
  - Contrast-enhanced CT or MR

General Features

- Best diagnostic clue
  - Low-attenuation thrombus in PV on CECT
  - On MR and power Doppler US
    - Absence of blood flow or flow void in PV
      - May be caused by slow flow in portal hypertension
    - Nonvisualization of PV (chronic occlusion)
    - Cavernous transformation of PV (collateralization in porta hepatis)
- Location
  - May involve any portion of intra- or extrahepatic PV

Ultrasonographic Findings

- Grayscale ultrasound
  - Acute
    - Echogenic or anechoic clot
  - Subacute
    - Isoechoic clot
- Color Doppler
  - Lack of flow within PV more evident on color Doppler
  - Tumor vessels usually visible within tumor thrombus
  - Partial thrombosis
    - Filling defect within PV
  - Cavernous transformation
    - Numerous venous collaterals in porta hepatis
    - Large collaterals may be mistaken for patent PV
  - Neoplastic invasion of PV
    - Pulsatile arterial waveforms with reversed flow
- Contrast-enhanced US (CEUS)
  - Helps distinguish malignant from bland thrombus

Treatment

- Anticoagulation for acute bland thrombosis (if liver function permits)
- Systemic antibiotics for septic thrombophlebitis

DIAGNOSTIC CHECKLIST

Consider

- Distinguish between tumor and bland thrombus in setting of cirrhosis

Image Interpretation Pearls

- Enhancing thrombus contiguous with parenchymal tumor = tumor thrombus

Reporting Tips

- Thrombosis or fibrosis of extrahepatic PV may complicate or preclude liver transplantation

DIFFERENTIAL DIAGNOSIS

Streaming Artifact

- Low-attenuation pseudothrombus due to uneven mixing of blood during PV inflow
- PV fills during later scans (equilibrium)

Extrinsic Compression

- Mass effect on PV from porta hepatis nodes, hepatic or pancreatic mass

Budd-Chiari Syndrome

- **Thrombosis of hepatic (not portal) veins ± inferior vena cava**
- Central hepatic sparing and hypertrophy, peripheral atrophy and necrosis

- Near-occlusive portal vein thrombosis extending from the portosplenic confluence through the main portal vein into right and left intrahepatic portal branches
- Some hepatopetal flow is documented in the portal vein with documented velocities of 13 cm/s in the main portal vein, 13 cm/s in the right portal vein, and 10 cm/s in the left portal vein
- Hepatic veins are patent
- Coarsened hepatic echotexture with nodular surface contour
- Normal appearance of the spleen
- Moderate volume ascites

Diagnosis

Portal vein thrombosis

Sample Report

Near-occlusive portal vein thrombosis extending from the portosplenic confluence through the main portal vein into right and left intrahepatic portal branches.

Coarsened hepatic echotexture with nodular surface contour, suggestive of cirrhosis.

Moderate volume ascites.

- Portal vein thrombosis (aka pylethrombosis) is a rare cause for acute abdominal pain
- Patients at increased risk for this condition include those with hypercoagulability disorders, cirrhosis, and upper abdominal tumors
- If thrombus results in chronic occlusion, numerous collaterals will develop resulting in cavernous transformation of the portal vein
- Pylephlebitis is thrombophlebitis of the portal vein, which rarely occurs as a complication of gastrointestinal infections. Worry about this if you see portal vein thrombosis in patients with gastrointestinal infections, if you see portal venous gas coexisting with thrombus, or if you see abscesses adjacent to thrombosed portal vein branches
- The most reliable discriminator between bland and tumor thrombus is the presence (tumor) or absence (bland) of postcontrast enhancement
- Normal portal vein findings on Doppler ultrasound:
    - Flow should normally be toward the liver (hepatopetal, not hepatofugal)
    - Flow should be consistently antegrade (always above the baseline) with gentle undulations
    - Normal flow velocity: 16-40 cm/s
    - Normal diameter: < 13 mm