SONOGRAPHIC EVALUATION OF THE HEPATOPORTAL SYSTEM

INTRODUCTION

The liver, the body's largest organ, performs more than 500 functions, many of which are related to blood filtration and detoxification, regulation of blood volume, metabolism of products of digestion, and control of body heat. The ability to perform these important functions is ensured by one of the most fascinating and complex circulatory systems accessible to sonographic interrogation. Examination of the vasculature of the liver will reveal the hepatic venous complex which drains the organ and the portal vein and its branches and tributaries which, surprisingly, supply the majority of the liver's blood flow. This extraordinary venous system is complemented by the hepatic artery and vessels that are part of the splanchnic circulation. Together, these arteries and veins ensure adequate blood flow into and from the normal organ. In patients with severe hepatoportal disease, the circulation of the liver is augmented by an extensive network of collateral pathways which move blood from the liver into the systemic circulation.
Duplex examination of the liver has become an important tool in the initial evaluation of patients with suspected hepatoportal disease. High resolution imaging has excellent value for identification of aberrant anatomy, tissue characterization, and confirmation of lesions. When combined with appropriately-optimized spectral and color Doppler, alterations in blood flow patterns and flow direction associated with portal hypertension, portal vein thrombosis, Budd-Chiari syndrome, portosystemic shunts, and arterio-portal fistulas can be detected. As such, ultrasound evaluation of the liver plays a key role not only in the diagnosis of disease but also aids in defining the therapeutic options available for patients with hepatoportal dysfunction.

INSTRUMENTATION

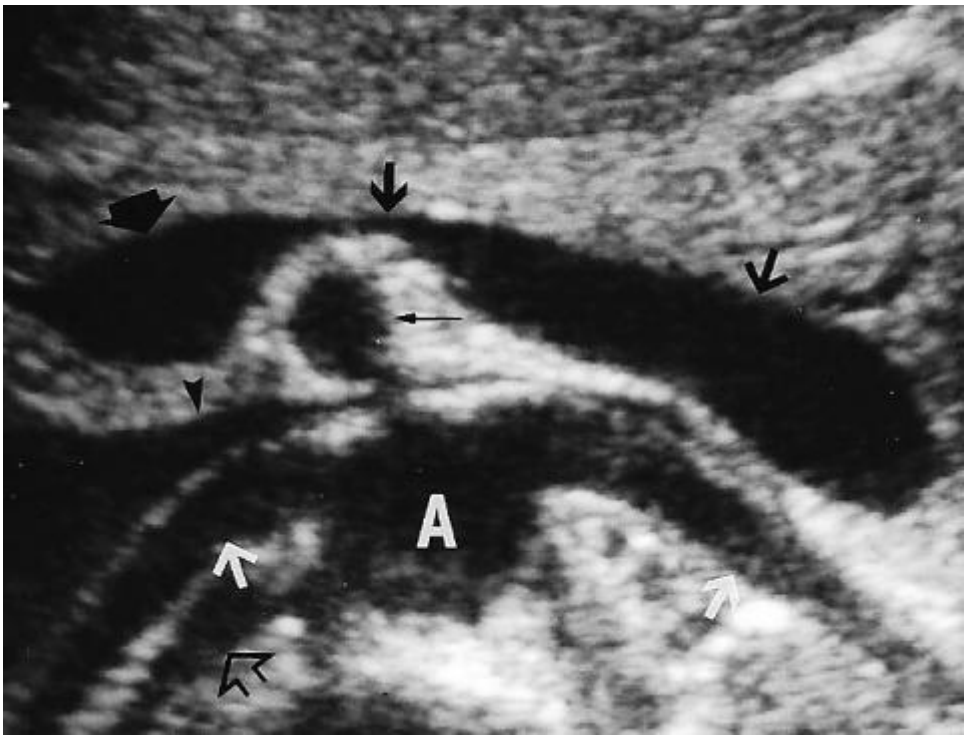
In the absence of hepatic disease, the ultrasound examination will usually be confined to the circulatory system of the liver. When blood flow to the liver is compromised by disease, the sonographic study will be extended to the spleen, kidney, gallbladder, and multiple collateral vessels throughout the upper and lower abdomen that facilitate transport of blood from the liver to the systemic circulation. Evaluation of these organs and vessels requires high resolution grayscale imaging for recognition of variations in anatomy and low-level echoes associated with venous thrombosis, masses, and ascites. Fundamental grayscale imaging can be enhanced with real-time compound and/or harmonic imaging to reduce noise and clutter artifacts. A variety of broad bandwidth transducers operating in a frequency range of 2 MHz to 5 MHz are generally employed to achieve adequate resolution and Doppler sensitivity in the abdomen.

Curvilinear array transducers facilitate imaging in subcostal and intercostal planes while phased array transducers provide excellent tissue characterization in many patients. A combination of grayscale, spectral, color and power Doppler will be required for differentiating dilated conduits in the biliary tract from portal vein collaterals or an enlarged hepatic artery and to highlight the presence and direction of flow in the frequently tortuous arteries and veins of the liver, spleen and collateral network.
Examination of deep abdominal vessels poses technical challenges that are not generally encountered with ultrasound evaluation of peripheral arteries and veins. To ensure that regions of slow flow in small diameter, tortuous arteries and veins are detected, color and power Doppler must be continuously optimized and the width of the color box narrowed to increase frame rate and sensitivity. Spontaneous, slow flow in the hepatic and portal veins can be detected when pulse repetition frequency (PRF, velocity scale) and wall filters are reduced to an appropriate level. While color is a valued tool for hepatoportal evaluations, it is less sensitive to weak echoes from slow-moving blood than spectral or power Doppler. For this reason, it is best used as a “flashlight” to highlight flow direction, the presence of reasonably large flow volumes, regions of disordered flow, and as an additional tool to define the course of tortuous vessels. In patients with hepatoportal disease, flow direction may be altered in the hepatic, splenic, and superior mesenteric veins, the hepatic artery and its branches, and the multiple collateral pathways that move blood away from the liver into the systemic circulation. To ensure recognition of these changes, the examiner must remain constantly aware of the orientation of the sound beam in relation to the expected direction of blood flow. Power Doppler is an excellent tool for demonstrating low velocity, off-axis flow and for highlighting the interface between a vessel lumen and wall. To avoid mirror imaging, an optimal Doppler gain setting and low beam-to-vessel angle should be used. As with other vascular ultrasound examinations, accurate diagnosis of liver dysfunction is in large part dependent on optimized spectral Doppler which is used to confirm the presence, direction, and quality of blood flow in the arteries and veins of the liver and its collateral pathways.

PATIENT PREPARATION, POSITIONING, AND CREATING ACOUSTIC WINDOWS

The arteries and veins that comprise the hepatoportal system lie deep within the right upper quadrant of the abdomen. Examination of these vessels is frequently challenging due to the depth of the vessels, the presence of overlying abdominal gas, and shadowing from ribs when using an intercostal scanning approach on the patient’s right side. To reduce the amount of bowel gas and swallowed air, elective patients are asked to refrain from eating, smoking, or chewing gum after midnight on the evening prior to the examination. Morning medications are to be taken with sips of water only. Diabetic patients are permitted to have dry toast and clear tea as needed to avoid hypoglycemia.
The hepatoportal examination commonly requires inclusion of the vessels within the liver, spleen, and splanchnic circulation. To obtain adequate acoustic windows for interrogation of these vessels, the examination table is placed in a reverse Trendelenburg position (feet 15 degree to 20 degree below heart level) to allow the patient’s visceral contents to descend into the abdomen. The patient may be positioned supine, right or left lateral decubitus, or slightly oblique to allow anterior, coronal, intercostal and subcostal views. Intercostal acoustic windows can often be expanded by having the patient raise their arm upward over their ear and perform deep breath holding. During deep inspiration, a large portion of the liver moves to a position inferior to the costal margin. Superior angulation of the transducer most often yields visualization and Doppler access to many segments of the hepatoportal system.

From a mid-line approach, with the patient lying supine, the confluence of the main portal, splenic and the superior mesenteric veins can be seen just above the level of the celiac artery bifurcation (FIGURE 1 A, B). The splenic vein can be followed from the hilum of the spleen using a midline transverse oblique image plane; the superior mesenteric vein can be approached most often from a left abdominal sagittal plane as it courses parallel to the inferior vena cava. A right intercostal scan plane is preferred for imaging the main portal vein and its branches within the right lobe of the liver. Occasionally, the superior mesenteric vein and the portal and hepatic vein branches can be viewed from this scan plane but a trial of right and left intercostal windows may be necessary to avoid rib shadowing. A slightly oblique subcostal imaging plane most frequently provides rewarding views of the left portal vein, its major branches, and the hepatic veins. The inferior mesenteric vein follows the course of the inferior mesenteric artery and can be located by imaging in a sagittal plane along the path of the distal superior mesenteric artery and the proximal segments of the inferior mesenteric artery.



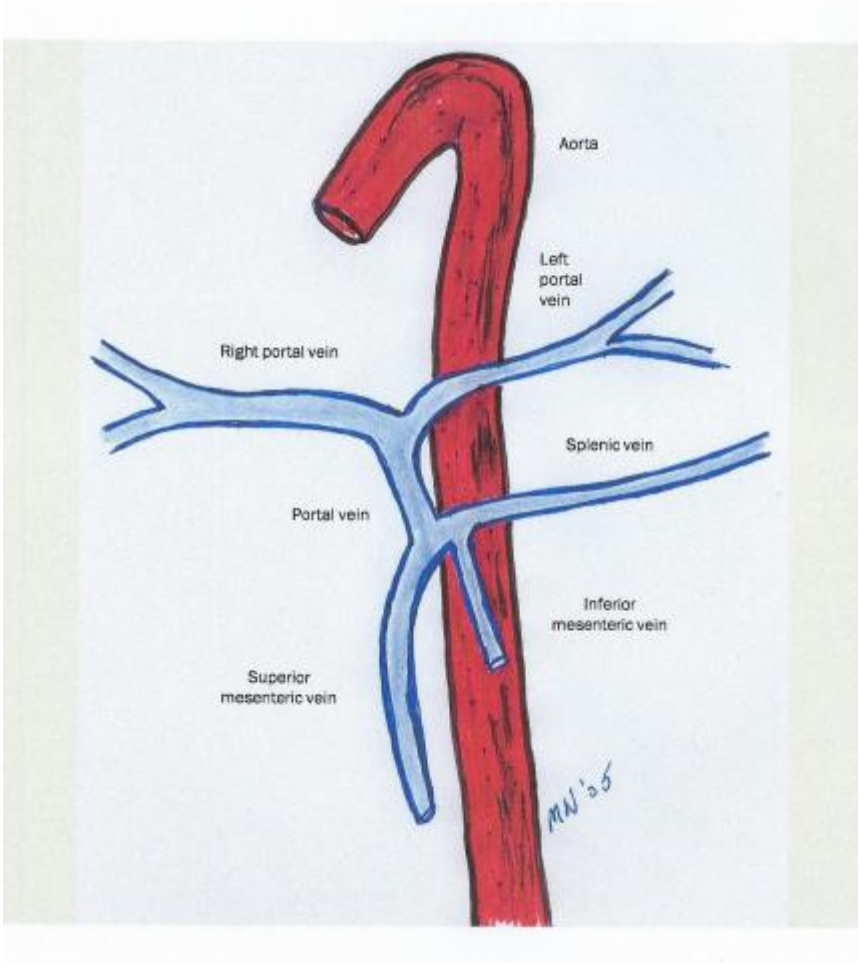


Figure 1. (A). Longitudinal gray scale image of a prominent superior mesenteric vein (SMV). Note the bulbous area posterior to the pancreas where the SMV joins the splenic vein to form the portal splenic confluence. (B). Diagram demonstrating the confluence of the superior mesenteric and splenic veins to form the main portal vein.

THE VASCULAR ANATOMY OF THE LIVER

The liver has three functional lobes: The right hepatic, the left hepatic, and the caudate lobe. Hepatic anatomy is described in a variety of ways. To further confuse the matter, the nomenclature and identification of segments of the liver differ between European and North American countries. Most sonographers prefer the segmental anatomy proposed in 1957 by Claude Couinaud, a French surgeon.(ref 1) This simplified anatomic approach, based on the arterial and venous circulation of the liver, emphasizes two rules: 1) hepatic veins are boundary formers. They define the boundaries of the lobes and segments of the liver, and 2) portal veins are not boundary formers (with one exception, which will be addressed later); rather they are located within the liver's lobes and segments.

The Inferior Vena Cava and Hepatic Veins

The inferior vena cava (IVC), the largest of the vessels that return blood from the extremities to the heart, lies to the right of the abdominal aorta and the spine. The IVC follows a straighter mid-to-distal course than the aorta, curving anteriorly at its proximal end as it enters the atrium of the heart. The role of the IVC is to return oxygenated blood from the abdominal and pelvic organs. A left-sided IVC, duplication of the distal IVC to the level of the renal veins, absence of the intra-hepatic portion with direct drainage of the hepatic veins into the right atrium, and membranous obstruction in the region of the caval confluence of the right hepatic vein may be encountered but these anatomic anomalies are quite uncommon. While the latter anomaly is rare in the United States, it is the most common cause of hepatic

venous outflow obstruction recognized worldwide.(ref 2)

The hepatic veins, the largest visceral branches of the IVC, originate within the liver, receiving blood from the portal veins and the hepatic artery. There are three major hepatic veins, the right, middle, and left (FIGURE 2). These sub-divide into multiple smaller branches which drain the parenchyma of the liver. Hepatic venous anatomy is quite variable. The right hepatic vein drains directly into the inferior vena cava and an additional right inferior hepatic vein is occasionally noted. The middle and left branches may join together to form a single trunk. Remember, hepatic veins are boundary formers. The three main hepatic vein branches define the segments of the liver. The right hepatic vein travels a path between the anterior and posterior segments of the right lobe of the liver. The middle hepatic vein courses between the right and left lobes of the organ, while the left hepatic vein lies along the division of the lateral and medial segments of the left lobe. It is important to note that the left hepatic vein and the ascending segment of the left portal vein (aka, umbilical vein) describe the same boundary between the lateral and medial segments of the left lobe, coursing at slightly different levels. The left hepatic vein is more cephalad and the portal vein is more caudal.

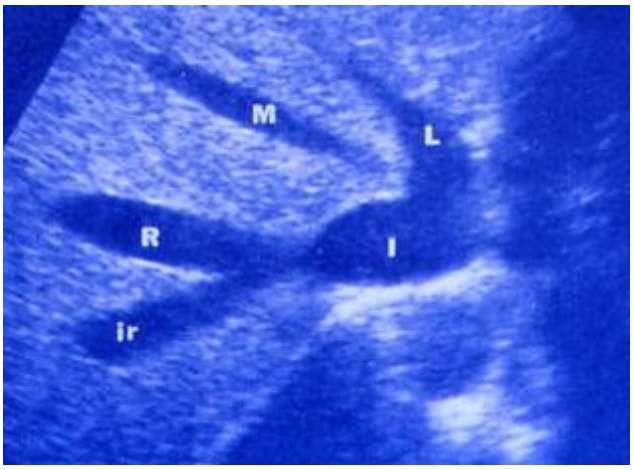


Figure 2. Longitudinal gray scale image of the three major hepatic veins -right, middle, and left – demonstrating their confluence with the inferior vena cava (I). Note the right inferior hepatic vein (ir), an uncommon variant.

The Portal Veins

The portal vein is responsible for approximately 70% of the blood flow to the liver. It carries nutrient-rich blood from the digestive system, pancreas, gall bladder, spleen and intestines. These organs are drained by the inferior and superior mesenteric and splenic veins, which unite to form the main portal vein (FIGURE 3). The inferior mesenteric vein joins the splenic vein just posterior to the head of the pancreas and then unites with the superior mesenteric vein to form the portal confluence (aka, the portal-splenic confluence, PSC) (Refer to FIGURE 1B. The portal vein enters the liver through the porta hepatis where it is joined by the common hepatic artery, which courses anteriorly and medially, and the common bile duct which takes a right anterior path (FIGURE 4). These three vessels comprise the portal triad and travel together throughout the liver. Acoustically, the portal vein can be distinguished from the hepatic veins within the liver parenchyma by its thick, fibrous, echogenic wall. Longitudinal images of the three vessels within the porta hepatis form the definitive appearance of “rungs on a ladder”. If the hepatic artery is not identified in this location, spectral Doppler and color flow imaging are mandatory to determine the identity of the visualized structures.

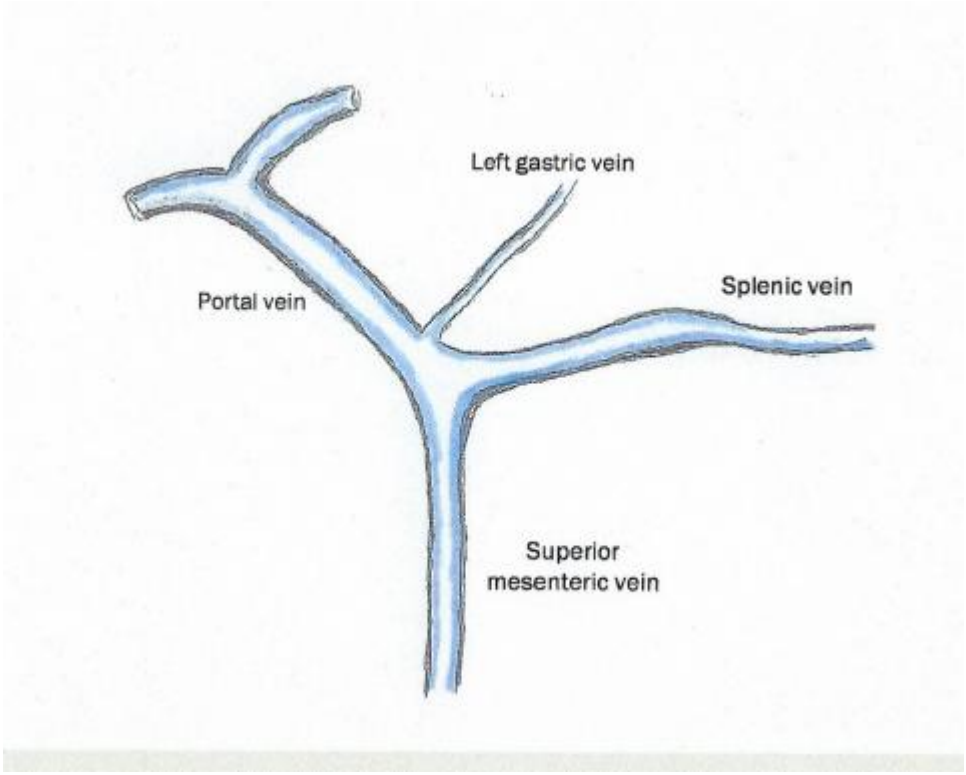


Figure 3. Diagram demonstrating the confluence of the splenic vein and the superior mesenteric vein to form the main portal vein (Refer to Figure 1B). Note the origin of the left gastric vein (aka, coronary vein) from the main portal vein.

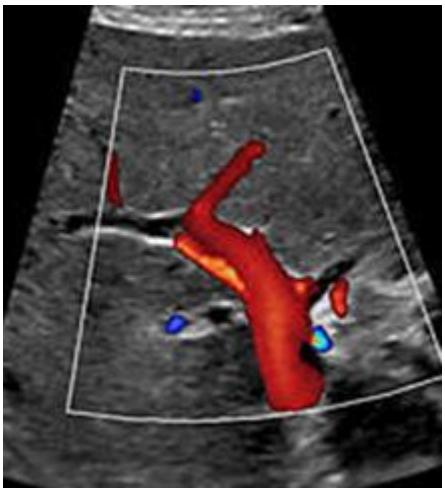


Figure 4. Color flow image of the portal vein and the common hepatic artery within the porta hepatis. Note the bifurcation of the main portal vein into the right and left portal vein branches.

Within the liver, the main portal vein divides into the right and left portal veins. Variations of this anatomy are common. Remember, portal veins are not boundary formers; they supply the hepatic lobes. The right and left hepatic lobes each have two basic segments. The right lobe has an anterior and a posterior segment which are supplied by the anterior and posterior segmental branches of the right portal vein. The left hepatic lobe has a lateral and a medial segment which are supplied by the lateral and medial branches of the left portal vein. The medial segment corresponds with what has been referred to as the quadrate lobe. The right portal vein branches are usually visualized. The left lobe segmental branches appear as small parallel “track-like” lines lying perpendicular to the fissure between the two left lobe segments.

There is more of interest regarding the left portal vein. A short distance beyond its origin from the main

portal vein it courses in a transverse plane and then turns anteriorly in the plane of the fissure between the lateral and medial segments of the left hepatic lobe (FIGURE 5). This segment of the vein is termed the ascending or “umbilical” portion and is important for several reasons. First, it is an exception to our “liver anatomy rules” because unlike the other portal vein branches, this ascending branch of the left portal vein is a boundary former- it courses between the lateral and medial segments. Another reason for its importance is because it serves as a major collateral vein (the recanalized paraumbilical vein) found in many patients with portal hypertension.

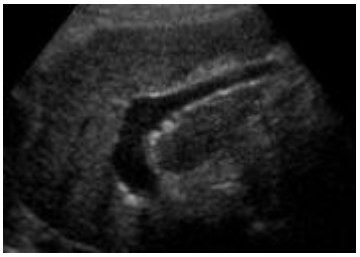


Figure 5. Gray scale image of the ascending (umbilical) portion of the left portal vein. Its anatomic course forms the boundary between the lateral and medial segments of the left hepatic lobe. This segment may serve as an important collateral (the recanalized paraumbilical vein) in patients with portal hypertension.

The Hepatic Artery

The hepatic artery originates from the celiac trunk as the common hepatic artery. As noted above, it enters the porta hepatis as the proper hepatic artery, coursing between the portal vein and the common bile duct (Refer to FIGURE 4). As noted, these three vessels travel together throughout the liver surrounded by Glisson’s capsule, a fibrous membrane which encases the entire organ, including the portal triad, and is responsible for the echogenicity of these vessels. Within the liver, the hepatic artery normally divides into the right and left branches but anatomic variations are common. Approximately 25% of patients will have more than one main hepatic artery.(ref 3) The most common variant is a replaced right hepatic artery. With this anomaly, the artery arises from the proximal segment of the superior mesenteric artery (SMA), and then courses into the liver to supply the right lobe. In approximately 4% of cases, it is the common hepatic artery that is replaced to the SMA.(ref 4) The left hepatic artery may also be replaced. When this variant occurs, the left hepatic artery arises from the left gastric artery (aka, the coronary artery), a small branch of the celiac artery trunk, rather than originating from the celiac artery at its bifurcation. When the left hepatic artery branches from the left gastric artery, it will pass through the ligamentum venosum, a fact that gains importance when considering sectional anatomy of the liver.

The Caudate Circulation

The caudate lobe is the smallest of the three hepatic lobes, lying anterior to the IVC and posterior to the ligamentum venosum. It has two processes: the papillary process, which lies anterior and medial, and the caudate process. The caudate lobe receives its blood supply from both the right and left portal veins and drains into the IVC or a hepatic vein. In patients with hepatoportal disease this small lobe may enlarge as it assumes responsibility for function of the right and left hepatic lobes.

NORMAL BLOOD FLOW PATTERNS

Sonographic evaluation of the normal liver will reveal that each vessel within the hepatoportal system

has a unique Doppler spectral waveform that defines its flow characteristics and the level of vascular resistance in its end organ. Perhaps most importantly, spectral Doppler interrogation will disclose that alterations from normal waveform morphology, such as changes in velocity and/or flow direction, yield clues to the presence or absence of hepatic disorders.

The Inferior Vena Cava

A pulsatile flow pattern is normally exhibited in the proximal IVC as a consequence of changes in right atrial pressure (FIGURE 6). In the distal segment, the IVC normally demonstrates respirophasic flow, similar to the flow pattern noted in the lower extremity veins. Flow velocity should increase with expiration, due to the decrease in intra-abdominal pressure, and diminish with deep inspiration as the diaphragm descends and intra-abdominal pressure rises. The diameter of the normal IVC also varies with respiration and throughout the cardiac cycle; however, it seldom exceeds 2.5 cm.(ref 5, 6) It is important to keep in mind that the diameter will also vary with patient size and position, fluid overload and heart failure.

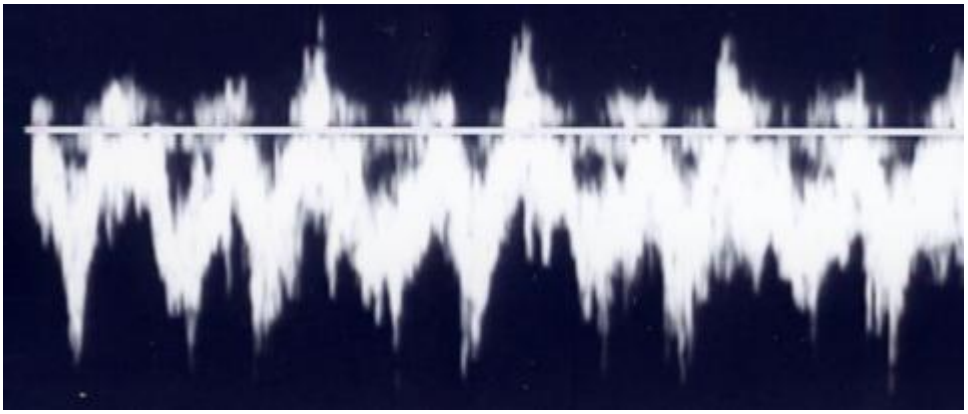


Figure 6. A pulsatile flow pattern is normally exhibited in the proximal inferior vena cava as a consequence of alteration in right atrial pressure.

The Hepatic Veins

The Doppler spectral flow pattern in the hepatic veins is similar to that in the proximal IVC. It is characterized by three phases: two phases are away from the liver and toward the heart and represent right atrial and ventricular diastole. The third phase is characterized by a short spurt of reversed flow, occurring with atrial systole (FIGURE 7). The hepatic venous waveform cycle begins with atrial systolic contraction. During this brief period, pressure in the right atrium increases and blood flows toward the liver (retrograde). This portion of the waveform is termed the A-wave. During ventricular systole, which follows, blood moves from the heart into the main arterial tree and the tricuspid valve moves into the right ventricle. As a consequence of the increased negative atrial pressure, blood in the IVC and hepatic vein is pulled back into the heart. The resultant hepatic vein waveform is characterized by flow toward the IVC. This portion of the cycle is termed the S-wave. The tricuspid valve moves away from the right ventricle and into the right atrium in late systole. Flow velocity toward the heart decreases, or occasionally demonstrates minimal reversal. Although not commonly used, some investigators assign the term “V-wave” to this brief period between ventricular systole and diastole. During cardiac diastole, the tricuspid valve is open, allowing the atria and ventricles to fill with blood. As noted in this portion of the cycle, termed the D-wave, flow is normally antegrade with velocities slightly lower than during systole. Throughout the cardiac cycle, flow is primarily away from the heart, which is termed hepatofugal flow direction.

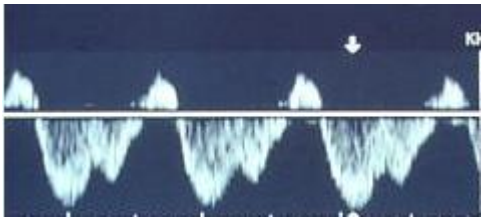


Figure 7. The hepatic veins normally exhibit a Doppler spectral waveform pattern characterized by three phases: two phases of flow away from the liver (toward the heart) represent right atrial and ventricular diastole, and a third phase which is characterized by a very brief period of reversed flow occurring with atrial systole.

The Portal Vein

In contrast to the multiphasic flow patterns exhibited in the hepatic veins, portal venous flow is characterized by a quasi-steady Doppler spectral waveform pattern (FIGURE 8). The minute pulsations noted in the spectral waveforms reflect the subtle cardiac influence on the portal venous system. The splenic and superior mesenteric veins exhibit flow patterns that mimic those of the portal veins. Portal venous flow direction is normally hepatopetal (toward the liver). Portal vein velocity will normally vary with respiration, digestion, and changes in cardiac flow patterns. Given this, a significant range of values for normal portal vein velocity is reported in the literature.(ref 7-12) But, in general, the maximum velocity ranges from 8 – 18 cm/s.(ref 8) It is important to recognize that while very low portal vein velocities suggest portal hypertension, velocities in the normal range do not exclude that diagnosis. Because the portal vein and hepatic artery supply blood flow to the liver, they will exhibit postprandial alterations in velocity. Portal venous flow increases in response to intestinal vasodilation. In contrast, flow in the hepatic artery decreases, most likely as a result of vasoconstriction. Given these fluctuations in velocity, it is important to know the time of the scan in relation to the patient's last meal to ensure an accurate examination.(ref 13)

Figure 8. The Doppler spectral waveform pattern from the portal veins normally exhibits low velocity, quasi-steady flow. Portal venous velocity normally increases postprandially and varies with respiration and alterations in cardiac flow.

Hepatic Artery

The hepatic artery normally supplies approximately 30% of the blood flow to the liver. In the absence of hepatic disease, the liver has a low resistance vascular bed, as evidenced by the need for blood flow throughout the cardiac cycle. This flow demand is reflected as constant forward flow throughout diastole in the Doppler spectral waveform pattern of the normal hepatic artery (FIGURE 9). While velocity normally decreases at end diastole, flow to zero or reversed diastolic flow should alert the sonographer to increased hepatoportal vascular resistance.

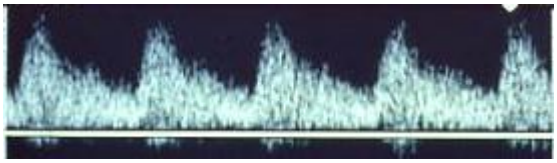


Figure 9. The normal hepatic artery Doppler spectral waveform is characterized by rapid systolic upstroke and constant forward diastolic flow.

COMMON HEPATOPORTAL DISORDERS

Liver disease is among the leading causes of death in the United States with the majority of liver pathologies involving the hepatoportal system. As noted previously, duplex sonography has become the initial procedure of choice for demonstrating the alterations in blood flow that occur with hepatic vascular disorders. The more common of these are discussed below.

Portal Hypertension

Portal hypertension occurs when the pressure gradient between the portal vein and the inferior vena cava exceeds 12 mmHg.(ref 14) Pressure in the portal venous system most often increases as a consequence of alcoholic cirrhosis or chronic active hepatitis. Causes of lesser frequency include, but are not limited to, primary biliary cirrhosis, Budd-Chiari syndrome, portal vein or IVC occlusion, schistosomiasis, sarcoidosis, lymphoma, hepatic fibrosis, and chronic right heart failure.(ref 15, 16) The location of the pathology may be pre-hepatic, post-hepatic, or intra-hepatic. Pre-hepatic pathology such as compression of the main portal vein and post-hepatic obstruction of the hepatic vein or IVC are uncommon causes of portal hypertension. Intra-hepatic pathologies are divided into pre-sinusoidal (before blood enters into the capillaries, or sinusoids, within the liver tissue), sinusoidal, and post-sinusoidal. When pressure increases within the portal venous system, the liver will attempt to decompress by shunting blood flow through collateral pathways which move blood from the liver to the low pressure systemic circulation via the IVC. This allows blood from the gut to return to the heart when flow to the liver is obstructed. The most common portosystemic collateral pathways are illustrated in FIGURE 10.

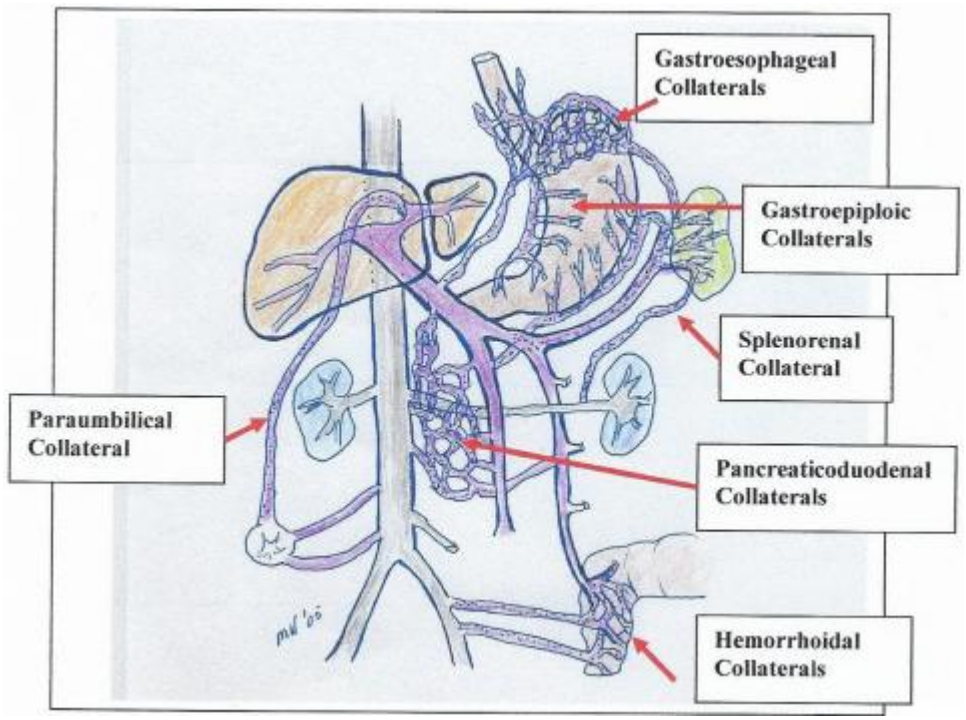


Figure 10. Diagram illustrating the most common portosystemic collateral pathways.

Portal Vein Thrombosis

As the severity of portal hypertension increases, vascular resistance becomes more pronounced in the liver tissues. In response, portal venous flow decreases and the hepatic artery assumes the primary responsibility for blood flow to the liver. As the condition worsens, the portal vein may thrombose (FIGURE 11) or divert blood away from the organ. This decompression of the liver is achieved by reversing flow direction in the portal vein and its branches (hepatofugal flow direction) or by channeling blood through portosystemic collaterals, thereby sparing the patient from life-threatening sequelae (FIGURE 12). Restoration of flow in the thrombosed portal vein and its branches may occur via cavernous transformation. Grayscale and color Doppler imaging will reveal multiple, small diameter, tortuous veins in the porta hepatis and along the usual course of the thrombosed main portal vein (FIGURE 13). Color flow imaging and Doppler spectral waveforms are valued tools for confirmation of hepatofugal flow direction in the portal veins and identification of major collaterals.

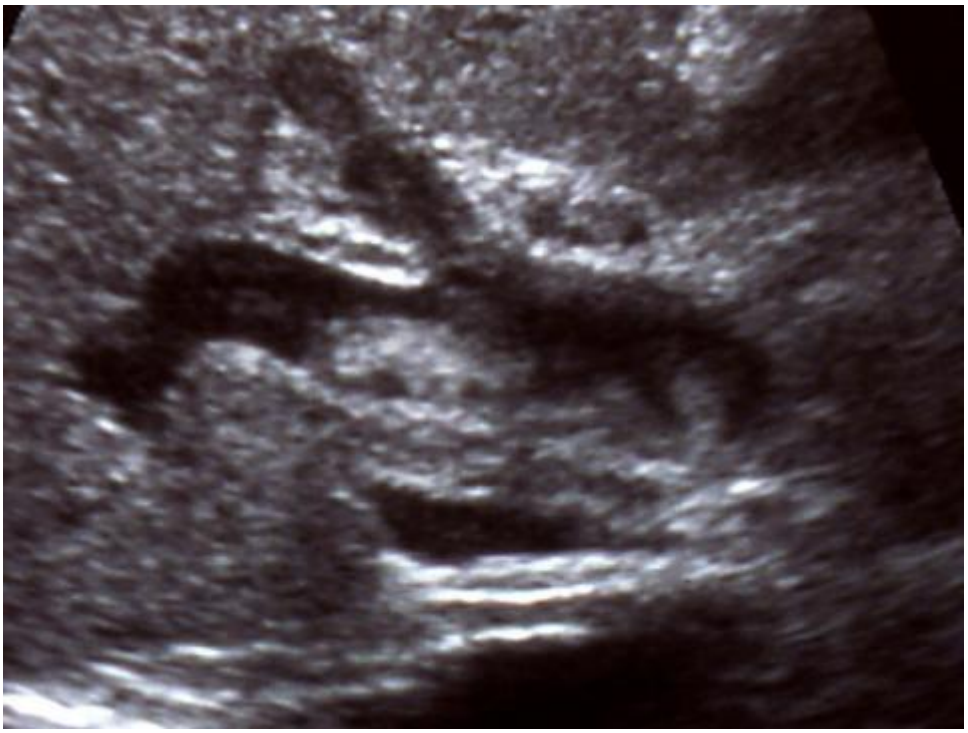


Figure 11. Longitudinal gray scale image of the main portal vein and its branches. Note thrombus within the veins.

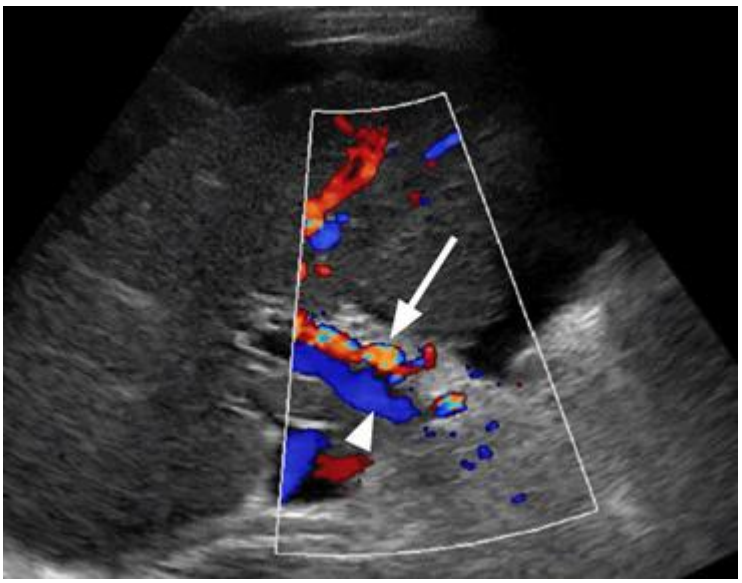


Figure 12. Color flow image of the portal vein and hepatic artery in the porta hepatis. Note hepatofugal flow direction in the portal vein.

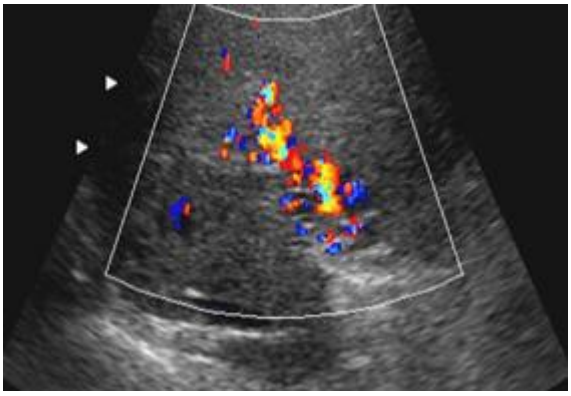


Figure 13. Color flow image demonstrating cavernous transformation of a thrombosed main portal vein. Note the small, tortuous veins in the porta hepatis.

Budd-Chiari Syndrome

Budd-Chiari syndrome refers to acute obstruction of the hepatic veins, a condition that is often a surgical emergency. Most often the major hepatic veins are involved; occasionally, the site of thrombosis is sinusoidal, within the liver parenchyma. The obstruction may result from tumor invasion, or hypercoagulable states (protein S, protein C, or anti-thrombin III deficiency). If outflow from the inferior vena cava is obstructed above the level of the hepato-caval confluence, pressure within the hepatic veins increases with potential for stagnant flow and, ultimately, hepatic venous thrombosis. Evaluation of the caudate lobe for evidence of enlarged veins (>3 mm) should be included in the duplex examination. In the absence of heart failure, enlarged caudate veins have been shown to be present in approximately 50% of cases of Budd Chiari syndrome.(ref 17)

THE DUPLEX EXAMINATION

The role of duplex sonography for assessment of hepatoportal disease has gained increasing importance in parallel with development of sophisticated ultrasound technology. In modern practice, interrogation of the hepatoportal system is successful in a high percentage of patients. As noted previously, detection of low velocity signals in the liver parenchymal vessels and collateral pathways requires optimization of the ultrasound system settings. The following are among the most important considerations: a low frequency, broad bandwidth (2 MHz to 5 MHz) transducer, Doppler angle of insonation less than 60 degrees , Doppler spectral gain adjusted to enhance the waveform envelope without introduction of artifact, a pulse repetition frequency (PRF, velocity scale) appropriate to the velocity of the returned signal, and infrequent use of a Doppler wall filter exceeding 50Hz.

The grayscale examination is an important component of the study. Attention should be given to image depth, transducer frequency, focal points, gain and dynamic range settings appropriate for recognition of acute thrombus which has acoustic properties similar to flowing blood. As the thrombus organizes, it becomes more echogenic and its acoustic features can be more easily differentiated from the vessel wall.

The absence of a Doppler signal may not necessarily indicate venous thrombosis or arterial occlusion. First, ensure that spectral, color and power Doppler settings are adjusted for low velocity flow, the Doppler sample volume size has been enlarged to detect small channels of flow, and the width of the color box has been decreased to enhance color and/or power Doppler sensitivity. Next, seek other clues

that might help to confirm vessel obstruction. Acutely thrombosed veins may be dilated. If the obstruction is chronic, flow in recanalized segments can be identified using low PRF settings for color, power, and/or spectral Doppler.
Throughout the examination, careful attention should be given to changes in diameter of the arteries and veins within the liver, flow response to respiration, alterations in flow direction and Doppler waveform morphology, enlargement of the liver and spleen, and the presence of portosystemic collaterals. Integration of this information will provide clues to the most common causes of hepatoportal dysfunction as outlined below.

PORTAL HYPERTENSION

Portal Vein Diameter.
The diameter of the main portal vein should be measured from a sagittal gray scale image at the porta hepatis where the vein crosses anterior to the inferior vena cava. Normally, the diameter of the main portal vein is < 13 mm during normal respiration with an expected increase in diameter exceeding 70% with deep inspiration. This finding has shown excellent specificity (100%) but low sensitivity (45%-50%).(ref 18, 19) When respiratory alterations in the diameter of the splenic and superior mesenteric veins are taken into consideration, the sensitivity is increased to 80%.(ref 18) In cases of portal hypertension, the portal vein is maximally dilated due to elevated portal venous pressure and the usual diameter increase of the vein will not be seen when the patient takes in a deep breath. It must be noted that portal vein diameter may be increased in patients with severe congestive heart failure due to transmission of right atrial pressure into the hepatic sinusoids with subsequent reflection into the portal venous channels.(ref 15, 16) This finding can be differentiated from portal vein dilation due to portal hypertension by noting the pulsatile flow pattern due to cardiac influence and concomitant dilation of the IVC.
Portal Venous Flow Pattern and Flow Direction
Normally, flow in the portal vein and its branches is characterized by a low velocity, non-phasic pattern. Alterations in the waveform pattern occur in response to elevated portal venous pressure. Deviations from normal may signal the presence of portal or hepatic vein compromise. An “arterialized” flow pattern exhibiting pronounced systolic peaks and high diastolic flow may be demonstrated in patients with advanced cirrhosis or an arterioportal fistula. A helical Doppler spectral and/or color display pattern exhibiting hepatopetal, hepatofugal, or bi-directional flow, may be seen in approximately 20% of patients with severe liver disease. Dependent on where the Doppler sample volume is placed within the helix, this pattern may be confused with flow reversal in the portal vein.(ref 20) Care must be taken to interrogate the portal vein both proximal and distal to the site of helical flow.
Change in normal flow direction may be apparent in one or more of the splanchnic veins or portal vein branches in patients with portal hypertension. Elevated portal venous pressure or right-sided heart failure is suggested by to-and-fro portal venous flow during normal respiration (FIGURE 14). Blood flow direction is hepatopetal during inspiration and hepatofugal during expiration or flow direction may be hepatofugal throughout the cardiac cycle. Flow reversal is in large part dependent on the richness of the collateral compensatory network of vessels, their location, and diameter. For example, if the primary collateral pathway for portal venous decompression is the large diameter umbilical vein which, as you will recall, originates from the left portal vein, hepatopetal flow direction in the main portal and splenic veins may be maintained. Conversely, if flow is shunted from the liver to the systemic circulation via

small diameter spleno-renal pathways, flow reversal may be apparent in the portal vein.(ref 21)

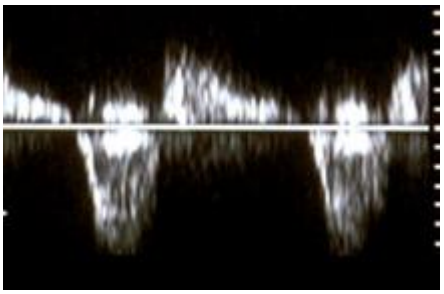


Figure 14. Doppler spectral waveform exhibiting to-and-fro flow pattern in the portal vein. This pattern suggests elevated portal venous pressure or right-sided heart failure.

Hepatic Artery Velocity

As noted, the portal vein and hepatic artery are responsible for blood supply to the liver. When portal venous flow is compromised, hepatic artery flow and diameter normally increase to meet the liver's metabolic demands, a situation that may lead to hepatic ischemia and persistent deterioration of liver function. Normally, peak systolic velocities range from 80 – 100 cm/s in the hepatic artery. The increased flow demand placed on the hepatic artery is evidenced by increased hepatic artery velocity and a somewhat turbulent Doppler spectral waveform pattern which may be mistaken for hepatic or celiac artery stenosis. To differentiate compensatory flow from arterial stenosis, the examination should be extended along the course of the hepatic artery and into the celiac axis. Elevated peak systolic velocities, post-stenotic turbulence, and delayed systolic acceleration distal to the site of interest, suggest flow-reducing stenosis. In contrast, elevated velocities associated with compensatory flow persist along the course of the hepatic artery without evidence of a focal velocity elevation or tardus-parvus waveform distally.

Portosystemic Collaterals

Sonographic detection of portosystemic collaterals most often serves as confirmation of portal hypertension. Patency of the splenic and superior mesenteric veins should be confirmed prior to arriving at this conclusion because portosystemic collaterals may compensate for thrombosis of these vessels. With portal hypertension, the collaterals divert blood away from the main portal vein and its parenchymal branches into veins that transport the blood directly into the inferior vena cava. The collateral pathways become active when the pressure in the portal venous system equals that in the collateral veins. FIGURE 10 details the major collateral pathways that can be detected with duplex ultrasound in an estimated 65% to 90% of cases.(ref 22-26)

While there are numerous routes that may be used to divert blood away from the liver and into the systemic circulation, several are more common than others. One of the most common collateral pathways is through the left gastric (coronary) and paraesophageal veins. The left gastric vein anastomoses with the paraesophageal veins and then follows a path from the esophagus through the lesser omentum to the splenic or portal vein. When the diameter of the left gastric vein exceeds 5 mm in an adult patient and flow is hepatofugal in direction, portal hypertension should be suspected. Careful attention should also be given to the region surrounding the upper pole of the spleen, the gastroesophageal junction, and the region of the gallbladder for evidence of small dilated collaterals (FIGURE 15). From an image of the left kidney, small, tortuous spleno-renal collaterals may be identified lying between the spleen and the upper pole of the kidney.(ref 23) (FIGURE 16) If this collateral network

is serving as a major shunt, the left renal vein may be dilated. Images detailing the inferior border of the left hepatic lobe may reveal small diameter, tortuous gastroepiploic collaterals.

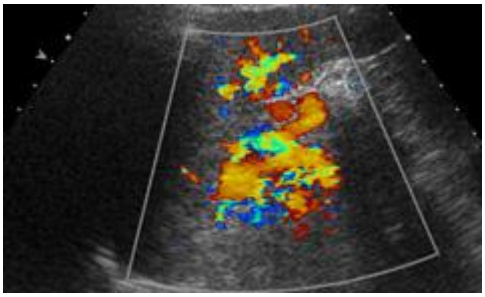


Figure 15. Color flow image demonstrating tortuous collaterals in the splenic hilum.

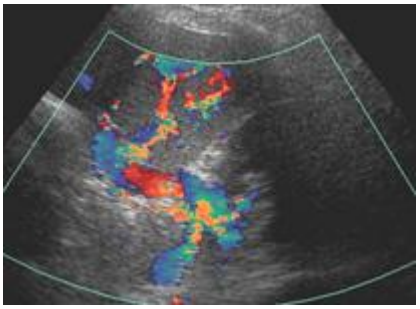


Figure 16. Color flow image demonstrating small diameter, tortuous vessels shunting blood from the splenic hilum to the left renal vein.

The umbilical vein (paraumbilical vein), a branch of the left portal vein, has two distinct features: it serves as a major collateral vessel and, as you will recall, it is a boundary former. After arising as a solitary vein from the left portal vein (FIGURE 17), it winds its way inferiorly through the falciform ligament. It then exits the liver and courses along the anterolateral abdominal wall to enter a periumbilical venous plexus referred to as the *caput Medusae* (head of Medusa) because it resembles the hair of the goddess Medusa and the tentacles of the Medusa jellyfish. If flow direction in the paraumbilical vein is toward the liver, portal venous thrombosis should be suspected whereas flow away from the liver should suggest portal hypertension.

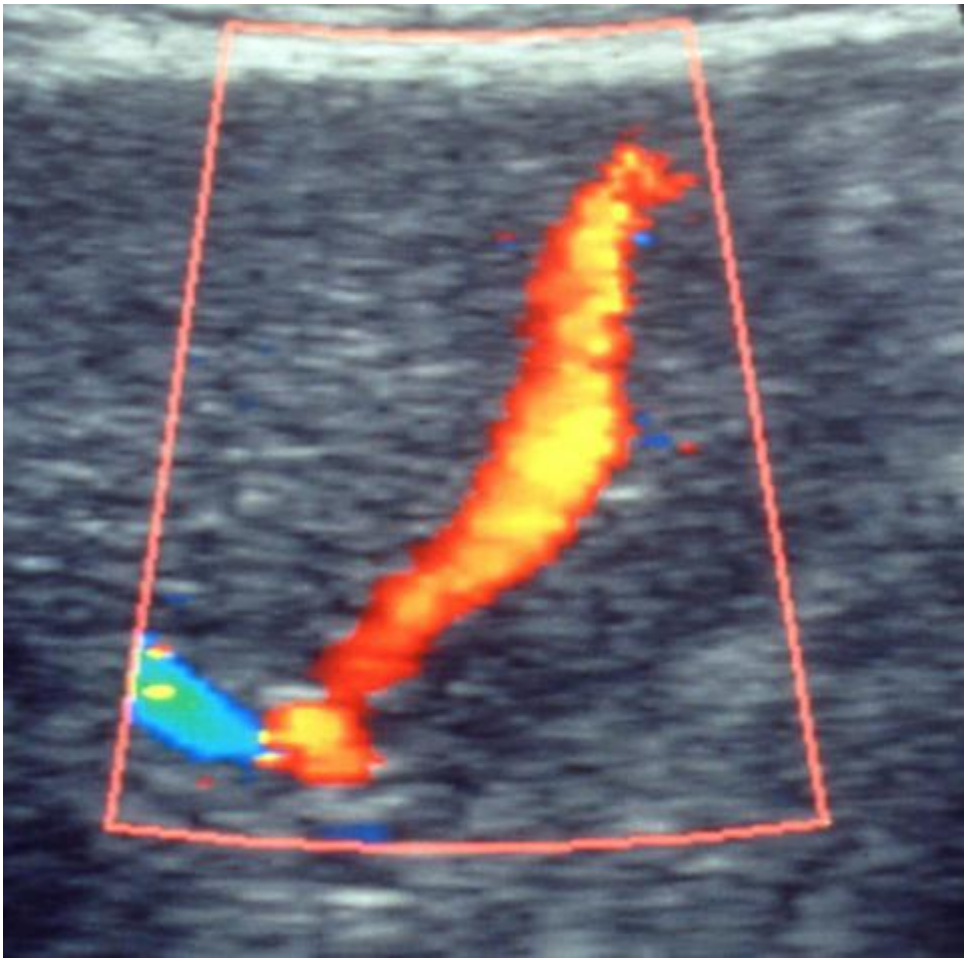


Figure 17. Color flow image of the umbilical vein (recanalized paraumbilical vein) arising from the left portal vein. If flow direction in the paraumbilical vein is toward the liver, portal venous thrombosis should be suspected whereas flow away from the liver should suggest portal hypertension.

It is prudent to proceed systematically in the investigation of the portal and hepatic circulations. Beginning with the main portal vein and its branches, the examiner should determine if flow direction and Doppler waveform morphology are appropriate. If not, determine if the findings are consistent with stenosis, obstruction, or collateralization. Next, follow the left portal vein to the falciform ligament to determine if the umbilical vein collateral is present. Return to the main portal vein and look for a dilated left gastric vein. Next, identify the portal vein confluence. Locate the splenic vein as it courses from the splenic hilum and note flow direction. If flow is reversed, check for spleno-renal or spleno-gastric venous shunts. While on the left side, check for small, tortuous veins along the inferior border of the left lobe of the liver and in the region of the gastroesophageal junction. Finally, look for dilated veins around the gallbladder. Table 1 lists sonographic findings in portal hypertension.

Sonographic Findings in Portal Hypertension
Portal vein diameter > 13 mm
Decreased or absent respiratory variation in portal and splenic veins
To-fro flow or helical flow in the portal veins
Absent portal venous flow
Hepatofugal portal/splenic venous flow
Portosystemic collaterals
Portal vein thrombosis
Increased hepatic artery velocity/diameter

Ascites, splenomegaly

Table 1

PORTOSYSTEMIC SHUNTS

Surgically Created Shunts

If portal hypertension is not reduced through spontaneously-formed pathways that shunt blood from the liver to the systemic circulation, surgically created conduits may be required to carry blood away from the liver through vessels connecting to the inferior vena cava. Several types of portosystemic surgical shunts have been used to prevent recurrent gastroesophageal bleeding.

A thorough sonographic examination of portosystemic shunts includes evaluation of the intra-hepatic portal veins, the shunt site, and the splanchnic and hepatic veins. A patent shunt can be visualized with grayscale imaging; Doppler spectral and color interrogation will define the direction and characteristics of blood flow. Flow velocity may be slightly elevated and somewhat disordered at the shunt origin. Flow-reducing stenosis is suggested when high velocity, turbulent signals are present. Normally, flow direction in the shunt is away from the liver (hepatofugal) and toward the low pressure systemic vein(s).

Shunts are difficult to image when they are occluded or when flow velocity is low. As with the native circulation, it is important to optimize spectral, color, and power Doppler settings for slow flow and to interrogate the shunt site carefully to confirm the absence of flow. Flow direction in the feeding vein and the intra-hepatic portal veins should be toward the liver when the shunt is occluded. Because major collateral pathways may reappear the most common route should be included in the sonographic assessment.

Transjugular Intrahepatic Portosystemic Shunts (TIPS)

In recent years, nonsurgical shunts have become the treatment option of choice for patients with recurrent gastroesophageal bleeding and ascites due to portal hypertension. The shunts are created percutaneously using a guide wire and catheter technique to enter first the jugular vein. A path is created when the wire is moved from the jugular vein to the inferior vena cava and into a hepatic vein (most often the right hepatic vein), through the liver to connect to a portal vein (usually the right portal vein}. A metallic stent is then placed over the wire to create a low pressure pathway for blood to flow from the main portal vein to the right portal vein and into the shunt. The shunt empties into the hepatic vein through which blood then exits the liver emptying into the systemic circulation (IVC). Thus, the acronym “TIPS” (Transjugular Intrahepatic Portosystemic Shunt).

Duplex sonography plays an important role prior to and following shunt placement. Prior to the procedure, duplex can be used to assess patency of the hepatoportal venous system and the internal jugular vein and to rule out space-occupying liver lesions. Following placement of the shunt, duplex imaging has demonstrated excellent value as the preferred method used for surveillance to detect pathology and/or flow abnormalities that may compromise shunt function. Shunt thrombosis occurring in the first weeks or months after placement is most often related to technical problems occurring at the time of shunt placement. Thrombosis or occlusion occurring later is due to neointimal hyperplasia of the shunt wall.

Duplex sonography is an ideal tool for evaluation of the metallic shunt. The TIPS is easily visualized with grayscale imaging (FIGURE 18). The initial post-shunt evaluation should be completed within the first 24 hours to confirm shunt patency and to establish baseline velocities in the portal vein, the shunt, and the

venous outflow segment. Normally, the shunt walls are echogenic and smooth. Intimal hyperplasia and/or thrombotic obstruction are suggested, respectively, by areas of acoustically homogeneous collections along the shunt wall or intraluminally. Doppler spectral interrogation will define velocity and characterize flow patterns and direction which can be confirmed with color flow imaging (FIGURE 19). Power Doppler imaging excels at detailing off-axis, low velocity areas of stenosis. The value of an aggressive surveillance program directed toward early detection of compromised shunt flow can be appreciated when one considers that one-year primary and secondary patency rates of approximately 80% and 99% have been achieved when covered stent grafts are used.(ref 27) A systematic review of velocities and flow direction should be performed on each follow-up evaluation. To ensure detection of shunts in jeopardy of failure, it is important to interrogate the main portal vein; the proximal, mid, and distal shunt; the outflow hepatic vein; the remaining hepatic vein branches; and the proximal IVC. Careful attention should be given to increases or decreases in velocity compared to the previous examinations, changes in flow direction in the hepatic or portal veins, or decreased shunt diameter as these issues suggest shunt stenosis or occlusion (TABLE 2). Maintaining a log which details the examination date, vessels examined, velocity parameters, flow direction, and image characteristics facilitates recognition of significant temporal changes that could impact shunt function and patency.

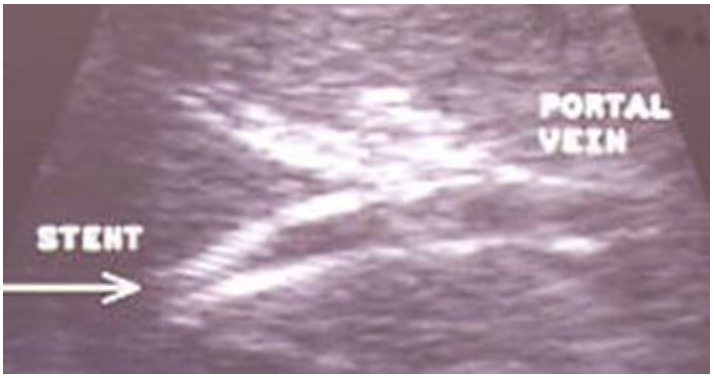


Figure 18. Gray scale image of a Transjugular Intrahepatic Portosystemic Shunt (TIPS) connecting the portal vein to the systemic circulation via a hepatic vein. The echogenic walls of the metallic shunt are well seen, a feature that facilitates duplex evaluation.

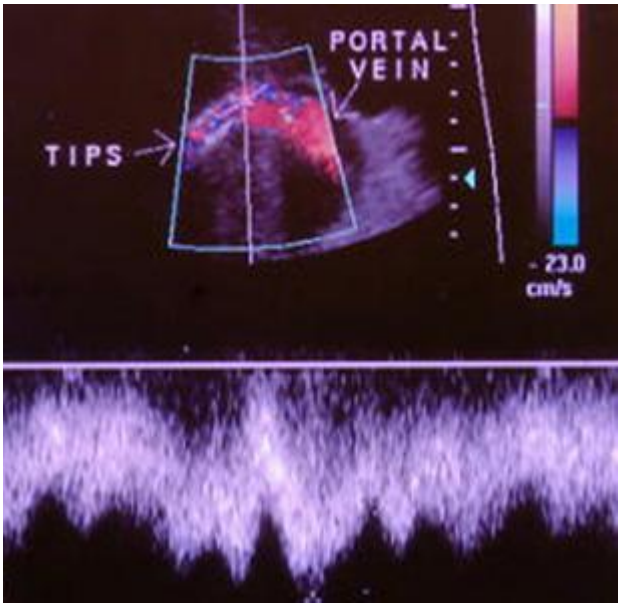


Figure 19. Duplex evaluation of a normal TIPS. The color image demonstrates hepatopetal flow direction (into the liver) in the portal vein as blood enters the shunt and hepatofugal flow direction (away from the

liver) in the shunt as it carries blood toward the hepatic vein and the inferior vena cava. The Doppler spectral waveform demonstrates a normal, slightly pulsatile flow pattern.

Sonographic Findings in TIPS Dysfunction
Portal vein mean flow velocity < 20 cm/s
Increased or decreased velocity > 50 cm/s within any portion of the shunt compared to previous exam
Hepatofugal flow in the main portal vein
Decrease in portal vein respiratory variation
Antegrade flow in the right or left portal veins
TIPS velocity < 90 cm/s or > 190 cm/s
No flow in the TIPS
Retrograde flow in the hepatic outflow vein

Table 2

SUMMARY

The circulatory system of the liver is a fascinating and complex network of arteries and veins accessible to sonographic evaluation in a high percentage of patients. Duplex ultrasound complemented with color and power Doppler imaging provides important information about the anatomy and pathophysiology of hepatoportal disease. As such, sonographic evaluation is most often the procedure of choice for the initial examination of patients with suspected vascular disorders of the liver. Additionally, it is a valued tool for monitoring disease progression and for post-procedure surveillance of interventional conduits used in the treatment of portal hypertension. To ensure accurate sonographic examination, sonographers and interpreting physicians must have knowledge of validated technical applications for abdominal sonography, liver anatomy and common variants, hepatoportal disorders and associated alterations in flow velocity and direction, and the anatomic location of commonly encountered portosystemic collaterals.

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