CDC 4R051O

Diagnostic Medical Sonography Journeyman

Volume 1. Abdominal Sonography

Air Force Career Development Academy
The Air University
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WELCOME to CDC 4R051O, *Diagnostic Medical Sonography Journeyman*. Entry into this 5-level career development course (CDC) marks a new phase in your continued training as an Air Force diagnostic medical sonographer. During your 3-level training, you worked to obtain basic knowledge and proficiency in this career field. This course is designed to clarify and deepen your training so that you will be able to perform as a fully independent diagnostic medical sonographer.

During Phase I, you were introduced to anatomy and physiology. You also performed simulated examinations designed to develop your hand-eye coordination, as well as to expose you to the intense visual procedures of this field. During your Phase II training, you were introduced to pathological knowledge and performed examinations of the most common abnormalities. In this course, we will clarify these abnormalities and introduce you to a few more in each area.

This course is divided into three volumes, each devoted to a different area of medical sonographic interest. It is not exhaustive, being limited to areas most likely encountered by most Air Force sonographers. This first volume covers information in the area of abdominal sonography with an elaborate discussion on the liver. Unit 1 provides you with a firm understanding and detailed knowledge necessary to perform sonography on the liver. Unit 2 considers the area of the gallbladder and the biliary system. Unit 3 gives you knowledge on the urinary system. The volume ends with Unit 4, which briefly discusses key abdominal structures routinely imaged by Air Force sonographers.

The remaining two volumes examine other areas of the body applicable to routine medical sonography. Volume 2 covers general sonography, with special emphasis placed on the vascular system. The volume also studies sonography of glandular body parts outside of the abdominal cavity, such as thyroid and testes. The final volume, Volume 3, examines obstetrics and gynecology. It is designed to organize and clarify your thinking on the sonographic approach to these challenging subjects.

You will find the information in this course to be a useful addition to information studied from other sources as you prepare to sit for national registry examinations. Close reading and continued study should also set you up for success when the time comes to sit for Air Force promotion testing.

A glossary of abbreviations and acronyms used in this course is included at the end of this volume.

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This volume is valued at 12 hours and 4 points.

Acknowledgment

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DU Figure Title	DU Figure Number	CDC Figure Number
	4–20	F1–12

The figures listed here have been reprinted from *Introduction to Vascular Ultrasonography* (IVU), fourth edition, William J Zwiebel (editor), pages 433, 434, 440, 442, 2000, with permission from Elsevier.

IVU Figure Title	IVU Figure Number	CDC Figure Number
Features of portal hypertension	Figure 29–2	F1–14
Umbilical vein collateral	Figure 29–7	F1–15
Morphological findings associated with cirrhosis	Figure 29–3	F1–13
Major portosystemic collaterals	Figure 29–4	F1–16

The information compiled in this CDC would not be complete without the help of Mr. Robert Magner at the USAF Academy's 10th Medical Group, Technical Sergeant Mabeline Morgan at Luke AFB's 56th Medical Group, and Technical Sergeant Ryan Parnell at Elmendorf AFB's 3rd Medical Group.

NOTE:

In this volume, the subject matter is divided into self-contained units. A unit menu begins each unit, identifying the lesson headings and numbers. After reading the unit menu page and unit introduction, study the section, answer the self-test questions, and compare your answers with those given at the end of the unit. Then complete the unit review exercises.

Please read the menu for Unit 1 and begin. ➔

Unit 1. Liver

THIS UNIT provides information you will need to perform sonographic examinations of the liver. It surveys liver anatomy and clarifies liver functions. It gives standard approaches to obtaining diagnostic sonographic images of the liver and discusses the process of recognizing and documenting liver pathology.

1–1. Liver Anatomy and Physiology

The liver is the largest abdominal organ and, because of its multiple functions, one of the most important. A firm understanding of normal liver anatomy and physiology, or the study of normal function, will increase your sensitivity to sonographically detectable abnormalities.

In this section you will briefly survey liver anatomy significant to sonographers, reinforcing your awareness of normal relationships between liver structures. We will slightly expand and clarify your knowledge of normal liver functions, providing you with a solid background to use when comparing normal processes with abnormalities. We will wrap up this section with a discussion on the relationship between laboratory values and liver functions; thereby increasing the efficiency of your sonographic examinations and aiding radiologists with making potential life-saving diagnosis.

001. Liver anatomy

Anatomy is the description of the form and structure of an organism and its parts. Understanding liver anatomy helps you to accurately locate abnormalities when performing a sonogram of the liver. Anatomy also helps us to understand liver pathology; that is, it can help us to see how diseases affect tissue structure and interfere with liver function. In this lesson, we will briefly examine anatomy with the most significance to sonographers.

Liver structure

Most radiologists expect sonographers to have a working knowledge of liver anatomy. Although there are many anatomic approaches to the liver, a radiologist will likely talk about the liver using lobar anatomy, segmental anatomy, Couinaud's anatomy, or a combination of all three when speaking with a sonographer.

General liver anatomy

The liver has external landmarks that have proven useful for dividing the organ into separate lobes. Sonographers usually divide the liver into three major lobes: right, left, and caudate. The average length of the adult right lobe, as sonographically measured along the mid-clavicular line, a vertical line that passes through the middle of the clavicle parallel to the long axis of the body, is 15 centimeters (cm). However, this length can be considerably increased with the presence of Reidel's lobe, a normal extension of the right lobe as far caudal, the direction toward the feet, as the iliac crest.

Reidel's lobes are usually seen in women. The left lobe and caudate lobe are not measured because of the varying sizes they have from patient to patient (fig. 1–1).

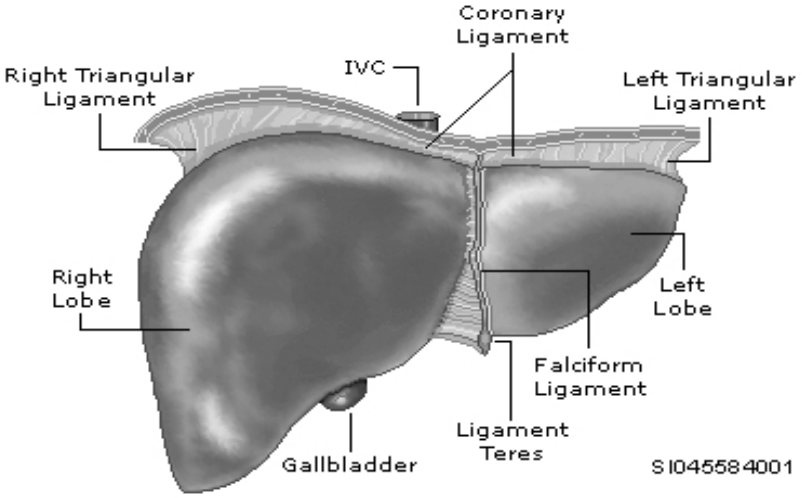

Figure 1–1. Anatomy of the liver, anterior.

The lobes and their subdivisions (segments) are divided by fissures or deep furrows. The left intersegmental fissure separates the lateral segment from the medial segment of the left lobe. Apart from the presence of the falciform ligament on its anterior surface, the fissure contains three structures: the left hepatic vein in the superior portion of the fissure, the left portal vein ascending anteriorly in its middle portion, and the ligamentum teres (round ligament) terminating inferiorly. The ligamentum teres is the obliterated remnant of the fetal umbilical vein. The falciform ligament anchors the liver to the diaphragm superiorly and to the abdominal wall anteriorly.

The fissure *for* ligamentum venosum separates the left lobe from the caudate lobe, located in the posterior part of the liver. This fissure contains the ligamentum venosum, which is the remnant of the fetal ductus venosus. The main lobar fissure is an imaginary line that connects the gallbladder fossa with the inferior vena cava (IVC) in the liver's caudal portion (fig. 1–2). The middle hepatic vein courses in the fissure's cephalic portion. This line is generally used to separate the right lobe from the left lobe. On a longitudinal sonographic image, the main lobar fissure is represented by a hyperechoic line extending from the neck of the gallbladder posterior to the portal vein.

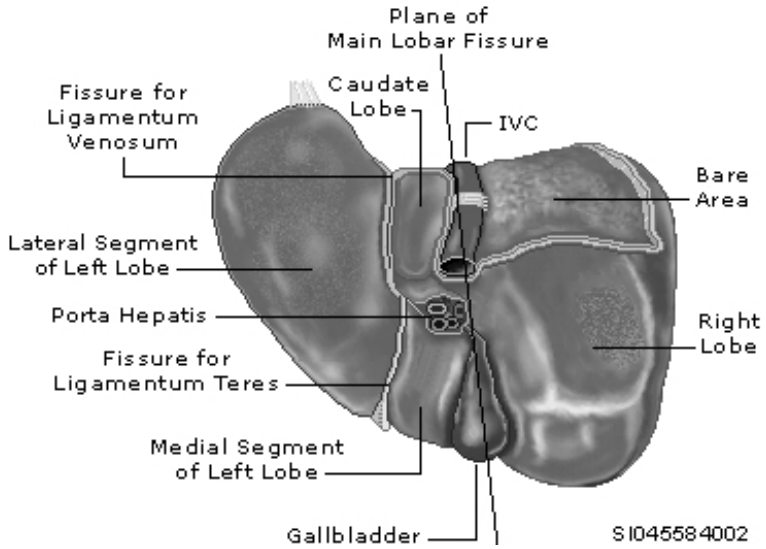

Figure 1–2. Anatomy of the liver, posterior/inferior.

The right lobe is divided into anterior and posterior segments by the right intersegmental fissure. This fissure contains the right hepatic vein.

Sonographers generally do not image the right and left triangular ligaments or other external ligaments of the liver. Without the presence of ascites, an accumulation of serous fluid in the peritoneal cavity, various ligaments attached to the surface of the liver are rarely seen with sonography. Even the fibrous peritoneal covering of the liver, Glisson's capsule, cannot be consistently seen without ascites next to it. Ascites may form in any of the spaces adjacent to the liver with the exception of the bare area, a portion of the posterosuperior right lobe directly attached to the diaphragm. An exception may be the falciform ligament, which is connected to the anterior abdominal wall. With ultrasound, the falciform can sometimes be seen extending into the left intersegmental fissure as a hyperechoic line above and connected to the ligamentum teres

.

Fissures of the Liver		
Fissure	***Location***	***Significance***
Left intersegmental fissure	Boundary between medial and lateral left lobe segments	In most patients, cephalic portion contains left hepatic vein; middle portion contains ascending left portal vein; and caudal portion contains ligamentum teres. Its anterior aspect contains falciform ligament.
Fissure for ligamentum venosum	Boundary between left lobe and caudate lobe	Contains remnant of fetal ductus venosus
Main lobar fissure	Imaginary boundary line extending through inferior vena cava superiorly and gallbladder fossa inferiorly, separating left medial segment from right anterior segment.	Contains middle hepatic vein superiorly and usually the long axis of gall bladder inferiorly
Right intersegmental fissure	Boundary between anterior and posterior segments of right lobe	Contains right hepatic vein

Couinaud's segmental anatomy

General anatomic landmarks have their place in sonography. But to be useful for the radiologist, you need a system that can identify liver location for a surgeon. Therefore, you should be aware of liver division based on internal function rather than surface area and fissures. The method most useful for radiologists who report the location of pathology is Couinaud's segmental anatomy. This method divides the liver into sections based on blood supply and drainage. It allows surgeons to plan the surgical removal of abnormal tissue from a liver segment, called resection, while leaving other segments, blood flow, and biliary drainage intact.

Separating liver segments into Couinaud's scheme depends on either the branching portal or hepatic venous systems. Portal branching is the most useful because the portal veins course through the center of each liver segment (intrasegmental), excluding the caudate lobe, which receives portal blood from branches of the right and left portal veins. The major hepatic veins are more superiorly situated in the liver and do not account for the functional segments found in the caudal liver. They are also situated between the liver's lobes (interlobar) and segments (intersegmental).

Couinaud's system has eight segments. Three imaginary longitudinal planes divide the liver based on hepatic branching, and an imaginary transverse plane cuts across the liver through the left and right branches of the main portal vein (fig. 1–3).

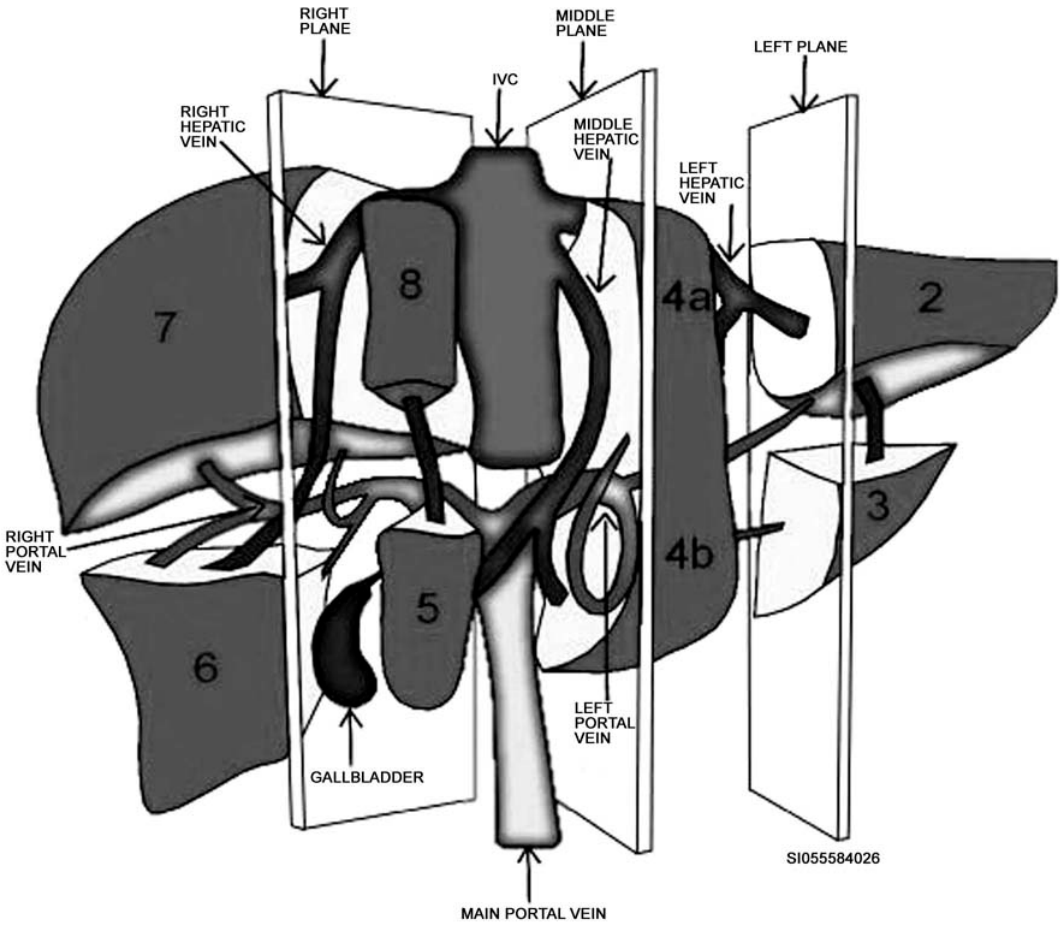

Figure 1–3. Couinaud's segmental anatomy.

Couinaud's Segmental Anatomy		
Segment	***Location***	***Significance***
Segment 1 (not visible from the front)	Caudate lobe	Posterior with vascular supply from both left and middle hepatic veins
Segment 2 Segment 3	Left lateral superior Left lateral inferior	Segments are left of left intersegmental fissure, which contains left hepatic vein, ascending left portal vein, and ligamentum teres
Segment 4 (a) Segment 4 (b)	Left medial superior Left medial inferior	Segments are right of left intersegmental fissure and left of main lobar fissure, which contains gallbladder fossa, middle hepatic vein, and IVC
Segment 5 Segment 6	Right anterior inferior Right posterior inferior	Segment 6 is right of main lobar fissure and Segment 5 left of right hepatic vein.
Segment 7 Segment 8	Right posterior superior Right anterior superior	Segment 8 is left of right hepatic vein.

Vascularity

The liver contains two different circulation systems: portal venous and systemic. The portal veins constitute the portal system and the hepatic veins and arteries constitute the systemic system. These vessels supply the liver with blood (portal veins and hepatic arteries) and drain the processed blood into the inferior vena cava (hepatic veins).

Portal venous system

The union of the splenic vein with the superior mesenteric vein forms the main portal vein. The main portal vein is 13 millimeters (mm) in normal diameter upon expiration of breath. It branches into the right portal vein and left portal vein just after entering the liver through the porta hepatis, a slit in the inferomedial surface of the liver. The right portal vein further divides into anterior and posterior branches that course centrally through the right anterior and right posterior segments of the liver, respectfully. The left portal vein travels horizontally toward the left lobe before ascending anteriorly through the left intersegmental fissure and then splits into superior and inferior branches that course centrally through the superior and inferior segments of the left lobe of the liver. Except for the ascending left portal vein traveling through the left intersegmental fissure, the major branches of the portal system run through the center of each liver segment—a feature that distinguishes the vessels from the hepatic branches. Another feature that distinguishes the portal vein from the hepatic vein is the collagenous tissue surrounding the portal vein, giving the walls an appearance of hyperechoic thickness with sonography.

Later, we will discuss the velocity (direction and speed) of portal blood flow in the unit on vascular sonography. For now, recognize that portal venous blood constitutes the majority of blood flow to the liver. Although this blood is deoxygenated, it is rich in nutrients from the digestive system.

Systemic circulation

As with the rest of the body, oxygenated blood from the heart comes into the organ via arteries and departs in a deoxygenated state through the veins.

Proper hepatic artery

The rest of the liver's blood supply comes through the proper hepatic artery which accompanies the main portal vein anteriorly. The proper hepatic artery provides continuous flow of oxygenated blood. Just after accompanying the main portal vein into the porta hepatis, the proper hepatic artery divides into right and left hepatic arteries. This branching continues (ramifies) throughout the liver with every parallel branching of the portal vein. In fact, hepatic arteries, portal veins (as stated above), and hepatic ducts are all sheathed together in connective tissue and are called portal triads.

Main hepatic veins

Commonly, three major hepatic veins drain blood out of the liver into the inferior vena cava at the superior portion of the liver. Keep in mind that there are variations to this structure from patient to patient. Some patients have four major hepatic veins and some have various branches off each. The average diameter of major hepatic veins is 4 to 7 mm. The left, middle, and right hepatic veins contain venous blood that has been filtered of old red blood cells, bacteria and toxins, but loaded with carbon dioxide. The main hepatic veins are distinguished from the portal system because of their thin walls and branching between the lobes and segments.

002. Liver functions

Normal liver functions are critical for maintaining life. Certain symptoms and laboratory values alert physicians to possible abnormalities that may affect liver function. Usually, but not always, those abnormalities also change the structure of the liver in characteristic ways that are detectable by sonography. Therefore, sonographers should be familiar with the basic functions of the liver.

Functional process

The functions of the liver are closely interrelated with its basic anatomic structure. Most of these functions are carried out through either the liver's vascular system or its exocrine system.

Deoxygenated (but nutrient-rich) blood travels from the intestines, pancreas, and spleen toward the liver through the main portal vein. The portal vein branches throughout the liver and terminates at the hepatic sinusoids. Hepatic arteries, containing oxygenated blood, accompany the portal veins in a close, parallel fashion and also terminate at the hepatic sinusoids. The hepatic sinusoids are spaces

between liver cells (hepatocytes). In the sinusoids, oxygenated blood is mixed with nutrient-rich deoxygenated blood. The hepatocytes are arranged into plates in a wheel-spoke fashion around a branch of a hepatic vein that courses through the center. This arrangement is called a lobule, the functional unit of the liver. The human liver contains tens of thousands of these lobules. After the mixed blood is processed it is sent through the central veins of each lobule to the main hepatic veins.

Production of bile is the excretory, or the elimination of waste, function of the liver. Bile is continuously secreted from the hepatic cells into small channels called bile canaliculi. These channels merge throughout the liver into tubules called hepatic ducts. The ducts eventually form into two main branches, the left and right hepatic ducts, which converge into a common duct as they exit the liver through the porta hepatis. Bile travels through the common duct to empty into the gastrointestinal tract or to be stored in the gallbladder for later use.

Primary functions

Although the liver performs many functions, its primary activities can be divided into four main categories:

1. Bile formation.
2. Metabolism.
3. Detoxification.
4. Storage.

Bile formation

Bile formation and secretion is a function that is unique to the liver. The body uses bile to aid in food digestion. Bile prepares fat in the small intestine to be absorbed into the portal blood through a process called emulsification. In emulsification, fat is formed into small droplets and suspended in the liquid of the small intestine; this action makes it easier for the fat to be absorbed into the bloodstream. Bile is also used to remove the liver's metabolic wastes.

Every liver cell continuously secretes bile into ducts. It is this process that makes the liver an exocrine gland, the largest in the body. Bile itself is composed mostly of water and bile salts (acids). Bile salts are the agents that aid in digestion. Bile is also made of proteins, cholesterol, lecithin, and inorganic substances. Finally, bile carries pigments that are to be excreted or eliminated from the body.

Sonographers pay particular attention to the bile pigment called bilirubin and its levels in the blood. Bilirubin levels have proven useful for signaling the presence of abnormalities in the liver. Bilirubin is formed from the breakup of hemoglobin, a protein that carries oxygen on the surface of red blood cells (erythrocytes). Normal destruction of old or abnormal red blood cells occurs in the spleen and bone marrow, after which hemoglobin is released and broken apart into a heme molecule and four globin molecules. The heme molecule is converted into bilirubin, which the body then seeks to excrete as waste. Bilirubin is joined with a protein in the blood plasma called albumin, which transports it to the liver to be made into a form that can be easily eliminated from the body.

When traveling to the liver, and despite being bound to albumin, bilirubin is said to be unconjugated. A substance that is unconjugated simply means that it has not been combined with another substance. In this case, unconjugated bilirubin is in a form that can pass from liver sinusoids into liver cells, but cannot pass through liver cell membranes into the tiny bile canaliculi connected to the cell walls. Only when bilirubin is combined with another particular substance (conjugated), found within the hepatocyte, will the cell wall allow it to pass through into the canaliculi and on into the intrahepatic ducts.

Metabolism

Metabolism refers to all of the chemical and physical changes that occur in tissue. There are two types: anabolism, or the building of small molecules into large molecule compounds, and catabolism,

which is the reduction of large molecules into small. Metabolism is often used interchangeably with anabolism or catabolism; although the two are strictly different (anabolism uses energy to build molecule compounds and catabolism releases energy during the decomposition of large molecules). Both processes occur inside cells continuously and at the same time. There are three main substances that the liver metabolizes: carbohydrates, fats, and proteins.

Carbohydrates

The liver converts or breaks down complex carbohydrates, or dietary sugars, from digested food into glucose, the simple sugar most used by human cells as a source of energy or fuel. The liver regulates and releases glucose into the blood stream. If there is too much sugar in the blood, liver cells convert glucose into glycogen and store it. When blood glucose levels fall below a certain point, the liver converts glycogen back into glucose for release into the blood stream. Sometimes there is not enough glycogen stored for low blood sugar levels. When this happens, the liver manufactures glucose by breaking down (catabolizing) fats and proteins.

The liver's inability to release glucose or to convert glucose into glycogen may indicate liver disease.

Fats

The liver converts fat into energy and uses it to synthesize, or build, cholesterol, lipoproteins, and phospholipids. Fats stored elsewhere in the body may be transported to the liver and converted into glucose or cholesterol. Cholesterol is an important substance for the body's chemistry and is used by the cells to perform a variety of metabolic functions. Most of the cholesterol found in blood is manufactured in the liver; the rest comes from diet. Cholesterol produced in the liver is primarily converted into bile salts and secreted into bile.

The liver also converts cholesterol and fats obtained from either digested or synthesized food into lipoproteins which are sent to cells throughout the body or stored. In the blood, there are various concentrations of lipoproteins: the high-density lipoproteins (HDL) which transport cholesterol from the body's cells to the liver to be excreted in bile—the so-called "good" cholesterol; the low-density lipoproteins (LDL) which transport cholesterol to locations other than the liver—such as to the walls of arteries, making it the "bad" cholesterol; and the very low-density lipoproteins (VLDL), which transport fats from intestines and the liver to muscle and adipose, or fat cell, tissue.

Fats are also made into phospholipids, which along with cholesterol, are used as components of cell membranes.

Proteins

The liver performs certain important metabolic functions such as synthesizing or breaking down amino acids, removing ammonia through urea formation, and manufacturing plasma proteins.

When liver cells synthesize amino acids, they are primarily building plasma proteins such as albumin, which transports molecules, especially bilirubin, in the blood. By maintaining a certain amount of pressure in capillaries, albumin also prevents plasma from leaking into the tissues. Other proteins manufactured in the liver are substances called factors that are used for blood clotting, or coagulation. For example, the two main factors the liver synthesizes are fibrinogen (Factor I) and, with the help of vitamin K, prothrombin (Factor II).

Detoxification

While going about the function of energy production, the liver will simultaneously filter harmful substances in a process that is commonly referred to as detoxification. Although strictly a metabolic function, it is easier to think of detoxification as a separate process.

The liver converts poisonous or toxic substances, such as drugs and alcohol, into forms that will not harm the body. Detoxification takes place largely within the sinusoids of the liver lobules. Sinusoids

serve as the location for Kupffer cells, which are cells whose purpose is to ingest toxins, bacteria, worn red blood cells, and other particles.

For example, the liver routinely detoxifies ammonia. When liver cells breakdown amino acids, they produce an ammonia byproduct, which is very toxic to the body. The liver itself cannot directly remove ammonia from the body, which is also formed in the intestines and absorbed into the portal blood. However, the liver detoxifies the ammonia by transforming it into urea, which is a nontoxic substance that is eventually removed from the body by the kidneys.

Storage

The liver stores various vitamins and minerals. For example, iron and vitamins A, B_{12}, and D are all stored in the liver for later metabolic use by the body.

Liver function tests

Because of all the metabolic functions that take place within the hepatocytes, damage to the cells may interrupt those functions. Also, many enzymes, which act as catalysts for metabolism, are present in the liver cells and cell damage may release these enzymes into the blood stream. Enzymes and abnormalities in the metabolism offer clues to liver pathology.

To detect and to help diagnose interruption of liver function, physicians order liver function tests (LFT) from laboratories. Liver function tests help physicians determine if the nature of liver disease is hepatocellular or cholestatic (biliary), if the occurrence is acute or chronic, and if the severity is mild or severe. For most of the tests, laboratories take samples of blood from patients for analysis. The following LFTs (there are others) are the most commonly used for detecting, diagnosing, and managing liver disease based on the quantity of certain enzymes, proteins, and other substances in the blood serum.

Aspartate aminotransferase (AST)

This test is the method used to determine the level of aspartate aminotransferase in the serum. AST is a critical enzyme that the liver cell uses to metabolize amino acids. The liver cell, however, is not the only cell that has AST inside of it. The enzyme is present throughout the human body and specifically in tissue known for high metabolic activity, such as the heart, kidneys, and brain.

When liver cells are damaged or destroyed, the AST that was inside of them is released into the bloodstream. This action causes a mild to moderate elevation in the usual quantity found in the blood. The AST test is able to detect this elevation sometimes before the appearance of the signs and symptoms of liver disease.

AST is sensitive for liver disease but not specific for liver disease. By sensitivity, we mean that AST will show mild to moderate elevations with the occurrence of nearly all liver diseases. However, you should remember that not every AST elevation specifically indicates liver disease. Remember that AST is present in areas throughout the body with high rates of metabolism, and damage in those cells will also cause an elevation in the blood serum. For example, damage to heart cells from myocardial infarction may cause a rise in serum AST. For this reason, we say that AST is non-specific for liver disease. Despite its non-specificity, AST will rise to significant levels with the occurrence of acute hepatitis, which is a severe inflammation of the liver, discussed in the lesson on liver pathology.

Alanine aminotransferase (ALT)

Like AST, ALT is an enzyme that metabolizes amino acids and is found throughout the body. However, unlike AST, ALT is concentrated mostly in the liver. Elevations of ALT are rare for abnormal conditions that occur outside the liver. For this reason, ALT is said to be specific for liver disease.

Physicians compare the elevation of ALT with AST to determine if disease is outside or inside the liver. If a liver disease is present, analysis of ALT levels compared to AST may suggest the general

liver disorder. In most liver diseases, ALT will elevate at a level equal to AST, with the exception being cases of alcoholic hepatitis where AST levels are higher. Similar to AST, the highest elevations of ALT are found in acute hepatitis.

Alkaline phosphatase (ALP)

ALP is a group of isoenzymes. Isoenzymes are two or more enzymes with differing chemical structures but similar functions. It is present throughout most of the body, especially in cell membranes. It has a high concentration in the liver, bones, small intestines, and the placenta. In normal or reference range blood serum, most of the ALP present comes from liver and bones.

For sonographers, the liver version of ALP is a useful marker for indicating the presence of liver dysfunction, particularly biliary tract obstruction. Liver ALP is found mostly in the biliary tract in the area of bile canaliculi. Obstruction of the normal flow of bile causes ALP to leak into the blood stream. Intrahepatic obstruction commonly causes high ALP levels and extrahepatic obstruction causes severe levels. Other liver diseases cause moderate or normal elevations.

Bilirubin (total, direct- and indirect-reactive)

Blood serum is tested to determine the source and level of increased bilirubin, or hyperbilirubinemia. There are generally three causes of hyperbilirubinemia:

1. Excessive production of bilirubin.
2. Damaged liver cells preventing uptake, conjugation, or excretion of bilirubin.
3. Damaged liver cells or bile ducts causing spillover of bilirubin into the bloodstream.

In the normal patient, most bilirubin in blood plasma is unconjugated. Recall that unconjugated bilirubin is a by-product of normal red blood cell destruction. The body's natural process is to send this unconjugated bilirubin to the liver to be converted into a form that can be excreted from the body. To be converted into this excretable form, bilirubin is joined, or conjugated, with another substance. The conjugated bilirubin is then excreted through the bile.

When natural bile flow is blocked, conjugated bilirubin, which is still being continuously produced, leaks into the blood stream. Physicians request a specific laboratory method that detects the presence of conjugated bilirubin in the blood. This method takes serum bilirubin and mixes it with a certain acid. The time and nature of the resulting reaction is called direct-reaction, which gives the physician an approximation of the amount, if any, of conjugated bilirubin in the serum. Hence, for simplicity, conjugated bilirubin is often referred to as direct bilirubin.

To determine the amount of unconjugated bilirubin, physicians use an indirect approach to the laboratory method. Alcohol and more time are introduced into the serum bilirubin sample which produces a reaction known as the total bilirubin. The difference between the amount of total bilirubin and the amount of direct bilirubin gives the approximate amount of unconjugated bilirubin. Because of this approach, unconjugated bilirubin is thus referred to as indirect bilirubin.

An increase in direct bilirubin in the blood serum is usually an indication of biliary obstruction which may lead to obstructive jaundice. Jaundice is the presence of yellow bile pigment, the color of conjugated bilirubin, in the skin and mucous membranes of the body. An equal rise in ALP tends to confirm biliary obstruction as the cause of elevated direct bilirubin. Hepatitis, because of its toxic nature, tends to destroy hepatocytes, which also blocks the excretion of conjugated bilirubin. Hence, the flow of direct bilirubin reverses into the blood stream.

Conversely, an increase of indirect bilirubin is usually an indication of excessive production of bilirubin from accelerated red blood cell destruction. This production of bilirubin exceeds the rate of the liver's ability to conjugate it.

There are other liver disorders that may cause rises in both direct and indirect bilirubin, such as hepatitis. Hepatitis not only blocks excretion of conjugated bilirubin as mentioned above, but it also

may, through the same process of liver cell destruction or malfunction, prevent conjugation altogether. This will cause a rise in the continuously produced unconjugated bilirubin in blood serum.

Prothrombin time (PT)

Recall that prothrombin is a protein found in the plasma that is manufactured by the liver. Its purpose is to serve as a coagulation factor. Along with other clotting factors, prothrombin contributes to the process by combining with thromboplastin and calcium ions. This combination yields thrombin, a substance that converts the clotting factor fibrinogen into fibrin, which clots the blood.

PT is the test used to determine if the process has been interrupted. The test measures the time required for clotting to begin after thromboplastin and calcium ions are added to a sample of blood serum containing normal fibrinogen. If the presence of prothrombin is diminished, clotting time is increased.

PT is not specific for liver disease because congenital deficiencies can cause abnormalities in coagulation factors. However, PT serves physicians as a useful value that helps determine the outcome (prognosis) of patients with acute hepatocellular disease. In acute liver disease, liver cells that manufacture prothrombin and that take in the vitamin K needed to make prothrombin are destroyed, increasing blood clotting time.

LIVER FUNCTION TESTS	
aspartate aminotransferase (AST)	Will usually elevate in the presence of most liver diseases; significantly in cases of acute hepatitis. However, not every elevation indicates liver disease.
alanine aminotransferase (ALT)	More specific for hepatocellular damage than AST.
alkaline phosphatase (ALP)	Elevates with biliary obstruction.
Bilirubin	Conjugated bilirubin in blood serum is abnormal and is detected using the direct-reaction testing of serum bilirubin. Total bilirubin testing is done to indirectly determine the level of unconjugated bilirubin in the blood serum. The difference between total bilirubin and direct bilirubin is the indirect-reaction of the serum test, yielding the quantity of unconjugated bilirubin.
prothrombin time (PT)	The time it takes for clotting to occur after the introduction of thromboplastin and calcium ion to blood sample. Indicates the level of prothrombin in the blood.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

001. Liver anatomy

1. When discussing the liver with a sonographer, what three types of anatomy will a radiologist likely use?

2. What is the average length of the adult right lobe of the liver?

3. What liver fissure separates the left lobe from the caudate lobe?

4. What is the significance of the right intersegmental fissure of the liver?

5. Match the Couinaud segment number in column B with each description of its location in the liver in Column A. Each item in column B may be used only once.

Column A	*Column B*
___ 1. Segment is left of left intersegmental fissure.	a. Segment V.
___ 2. Segment is left of right hepatic vein.	b. Segment VI.
___ 3. Segment is right of main lobar fissure.	c. Segment II.
___ 4. Segment is right of left intersegmental fissure.	d. Segment IV.

6. What is the significance of the collagenous tissue surrounding the portal vein?

002. Liver functions

1. What is the purpose of the main portal vein?

2. What is the relationship between bile and digestion?

3. What does the blood plasma protein albumin accomplish for unconjugated bilirubin?

4. What happens to glucose in liver cells when there is too much glucose in the blood?

5. List some lipoproteins manufactured by the liver and their activity in the blood.

6. What does the liver do to detoxify ammonia?

7. What are some of the ways liver function tests help physicians determine the nature of liver disease?

8. Why is AST not considered a useful test for specifying liver disease?

9. What does an increase of direct bilirubin in blood serum indicate?

1–2. Liver Imaging

Producing sonographic images of the liver is a process that involves more than simply moving an ultrasound probe over the abdomen and printing the images that appear on the screen. The process is a spectrum of activity that begins with patient preparation and extends to the final report. You are not alone during the examination; your partner is the radiologist who depends on your knowledge of every facet of the process.

To effectively participate in the process, we will first discuss liver pathology (abnormality) as a way to focus our approach to sonographic imaging. Once we are familiar with some of the things that can go wrong in the liver, we will then move on to imaging both normal and abnormal patients. To do this, we will discuss general procedures to keep in mind when imaging the liver. We will then examine various adjustments to those procedures based on the pathology in the liver to be imaged. The biggest adjustment for each disease is your understanding of a disease's background and its effect on the liver's sonographic appearance. Understanding liver disease and its relationship to its sonographic appearance is part of the process of liver imaging.

003. Liver pathology

A wide range of diseases have the capability of profoundly affecting the liver. Some liver abnormalities either partially or completely affect the liver's various functions. Other abnormalities alter the anatomic structure of the liver. Frequently, you will encounter diseases that do both. In this lesson we will take a look at some of the more common pathologies. We will divide our discussion into several areas of liver abnormalities:

- Infectious liver disease.
- Metabolic liver disease.
- Cystic lesions.
- Solid tumors.
- Vascular abnormalities.

Infectious liver disease

A large segment of liver pathology is the result of infection from external organisms and viruses. Introduced into the body through oral routes, bloodborne pathways, and sexual activity, infectious diseases can travel directly to the liver through vascular and lymph channels. Without proper treatment, the liver's function is usually affected first, followed by increasingly severe alteration of anatomy. Of particular concern to physicians are viruses and parasites.

Viral hepatitis

While some viruses, such as Epstein-Barr (human herpes virus 4) and yellow fever, produce liver disease in adults, the most common viral disease with which you should be familiar is viral hepatitis. Hepatitis is an inflammation of the liver caused by sources such as drugs and alcohol, but predominately is a result of infection by viruses. The disease usually follows an acute or chronic course depending on the type of virus. For example, most cases of acute hepatitis that result in complete recovery (uncomplicated) after a few months are due to the *hepatitis A virus* (HAV). While HAV can also worsen, most cases of acute hepatitis that progress rapidly to hepatic failure (fulminant hepatic failure) are due to the *hepatitis B virus* (HBV). Hepatic failure is usually the result of necrotic liver cells. If slightly less than half of liver tissue becomes necrotic, the patient will likely die. HBV is usually found in 20 to 49 year-old patients.

Hepatitis that persists for longer than six months may be considered chronic. Unlike HAV, which is either acutely uncomplicated or acutely fatal, HBV and *hepatitis, type C* (HCV or *non-A, non-B*), can persist as chronic infections. The most common chronic infection is HBV, which has a carrier state

that renders some patients asymptomatic. In the United States, there are more than a million HBV chronically infected Americans. In chronic hepatitis, the disease is usually in an active or persistent state. In the persistent state, the disease does not progress to liver failure. Conversely, the active state can lead to *cirrhosis* (discussed below) and increase the risk for *hepatocellular carcinoma* (liver cell carcinoma, also discussed below) or liver failure.

HAV is transmitted through feces or through anything ingested after contact with fecal material, such as contaminated water. HBV and HCV are contracted through blood and body fluids from sexual activity or through contaminated instrumentation and needle sticks in healthcare workers.

The symptoms that a patient may have when examined by a physician (clinical symptoms) for hepatitis are jaundice, fever, nausea, weakness, and anorexia. Because of the damage that inflammation can cause to liver cells, laboratory values may show an increase in the levels of AST, ALT and, if severe enough, bilirubin.

Pyogenic abscess

An abscess is a collection of pus formed from infection. Most abscesses that develop in the liver are caused by pyogenic (pus-forming) bacteria. The source of the bacteria is usually located elsewhere in the body but has various routes to the liver. For example, the main portal vein is a gateway into the liver for pyogenic bacteria coming from inflammation sites such as appendicitis, diverticulitis, or colitis. Other common routes are the common bile duct and proper hepatic artery.

The bacterium most responsible for liver abscesses is *Escherichia coli*; other common bacteria are *Staphylococci* and *Streptococci*. In approximately half of the cases of pyogenic abscess, the source is unknown.

To the referring clinician, patient symptoms are usually malaise, fever, right upper quadrant (RUQ) pain, vomiting, diarrhea, jaundice, and weight loss. Liver function tests will be elevated. The white blood cell count in these patients will also increase (leukocytosis). Without treatment such as percutaneous drainage and antibiotics, pyogenic abscesses are fatal.

Parasitic liver disease

The liver may be infested with amebae or parasites that create abnormal conditions within the liver. Typically, these organisms enter the body through oral ingestion, but may enter through other avenues. We will look at the more common ones that are detectable by sonography.

Amebic abscess

Amebiasis is an invasive infection by an organism called an ameba, particularly (and usually strictly limited to) the organism *Entamoeba histolytica.* Although the organism may launch from the colon to spread to the lung and brain, its most common occurrence outside the intestines is in the liver. *E. histolytica* enters the liver through the portal vein and may form an abscess of necrotic liver tissue. This cavity is called an amebic liver abscess. The abscess can progress to a point where it may rupture and burst through the liver capsule into surrounding tissues of the body, such as the diaphragm, lung, and peritoneum.

Although less common than pyogenic abscesses, the disease is found in warm climates worldwide, with more common occurrences in areas where water is routinely polluted and food is contaminated. *E. histolytica* is contracted from the organism's cysts, or the early stage in the life-cycle of the parasite. This happens when contaminated food or water is ingested.

Typical symptoms of the amebic abscess are fever and RUQ pain. Other symptoms may occur such as vomiting, diarrhea, anorexia, and nausea. Laboratory values demonstrate an elevation in leukocytes, ALP, and serum bilirubin. A serology test, called an indirect hemagglutination, is positive in most patients with an amebic abscess. This is most useful in areas where the disease is not as prevalent as pyogenic abscesses, such as western countries.

A diagnosis is usually made from a combination of symptoms, sonography, and serologic testing. Treatment is usually limited to amebicidal drugs and, in cases of impending rupture, percutaneous drainage. Surgical drainage usually occurs after a rupture.

Echinococcus (hydatid) cyst

Hydatid disease (echinococcosis) is an infection, usually in the liver, of the larval cystic stage (hydatid cyst) of one of three tapeworms, the most common being *Echinococcus granulosus*. This disease is present worldwide in areas of major sheep and cattle herding, such as the Middle East in particular, the Mediterranean, Australia, and certain regions of North and South America. The organism is found in the intestines of dogs. Humans contract the disease from food and water that has been contaminated by canine feces.

The eggs of *E. granulosus* are swallowed by humans and hatch in the small intestines, where the larvae invade the mucosa. From there, the organisms enter the portal blood system and travel to the liver through the main portal vein. Usually, patients are either asymptomatic or have intense RUQ pain coupled with low-grade fever and an enlarged tender liver. As with amebic abscesses, serologic testing is performed and, if positive, helps to confirm the presence of *E. granulosus*. Also the Casoni intradermal (skin) test may be used, in which hydatid fluid is injected into the skin to induce an inflammatory reaction.

The echinococcus cyst may rupture into the biliary system and cause cholangitis. It may also rupture through the liver capsule and, like ruptured pyogenic abscesses, invade the diaphragm and lung or the peritoneal cavity. This event is usually fatal because of the antigenic nature of the cyst fluid when in contact with body tissue. When it ruptures, the patient may go into anaphylactic shock.

Treatment of asymptomatic echinococcus cyst is usually with an antihelminthic drug, a substance that destroys or expels tapeworms. For symptomatic echinococcus cysts, surgical removal with antihelminthic therapy is used.

Schistosomiasis

Schistosomiasis is an infection from blood flukes (parasitic worms). The major species that mostly affect the liver are *Schistosoma mansoni* and *Schistosoma japonicum*. Although not usually seen in the United States, schistosomiasis is one of the most common worldwide parasite infections in humans. The disease is seen particularly in Africa, Egypt, Arabia, China, Japan, Philippines, and South America.

Almost as soon as humans wash, wade, or swim in waters infested with *S. mansoni* or *S. japonicum*, the parasites penetrate the body through the skin. Once the flukes are in the bloodstream, they hatch eggs that reach the liver through the main portal vein. At the terminal ends of the portal branches, the liver reacts to the organism's presence by forming a granulomatous reaction and fibrosis, particularly in the area of the portal hepatis. The fibrosis blocks or occludes the portal branches which results in increased portal blood pressure, or portal hypertension. The liver and spleen enlarges and ascites may appear in the abdomen.

Patients may present themselves to the physician with transient pruitus (itching and rash) immediately after infection or, later, with fever, chills, fatigue, headache, or GI discomfort. The liver may be tender and enlarged and, in severe cases among portal hypertensive patients, there may be internal bleeding.

Physicians will primarily test for the disease using serologic testing. Laboratory analysis of stool may yield information about the presence of eggs in the patient.

Other infectious diseases

Opportunistic organisms may infest the livers of patients whose immune systems have been reduced (immunocompromised). The suppression may be the result of the human immunodeficiency virus (HIV), which is the agent responsible for causing acquired immunodeficiency syndrome (AIDS).

Compromise may also come from such things as immunosuppressive drugs, chemotherapy, malnutrition, disease, or transplantation.

Mycobacterial infection

In immunocompromised patients, especially those with AIDS, the organism most responsible for liver infection is usually *Mycobacterium avium-intracellulare*, followed by *Mycobacterium tuberculosis*. Both organisms tend to produce an inflammatory reaction in the liver in the form of granulomas, small lesions composed mostly of grain-like particles. These granulomas are occasionally accompanied by calcifications.

Fungal infection

Candidiasis is a fungal infection of the liver. The most responsible organism is *Candida albicans*, which creates small abscesses in the liver after traveling through the blood (hematogenous) from other fungus infected (mycotic) organs. The patients present themselves mostly with persistent fever.

Another organism that may affect the liver of AIDS patients is *Pneumocystis carinii*. It is unclear whether *P. carinii* is a protozoa or a fungus because it possesses characteristics of both and its exact classification is controversial. For our purposes, we will consider it as belonging to the *Fungi* kingdom. The organism is considered a pulmonary disease, yet its manifestation outside the lungs (extrapulmonary) is primarily centered in the liver of AIDS patients.

Infectious Diseases of the Liver			
Viral	***Bacterial***	***Parasitic***	***HIV Patient Infections***
Hepatitis: *Hepatitis virus A, B, C, D, E, & G*	Pyogenic Abscess: *Escherichia coli; Staphylococci; Streptococci*	Amebic Abscess: *Entamoeba histolytica* Echinococcosis: *Echinococcus granulosus* Schistosomiasis: *Schistosoma mansoni; Schistosoma japonicum*	Mycobacterial: *Mycobacterium avium-intracellulare; Mycobacterium tuberculosis* Fungal: *Candida albicans* (Candidiasis)*; Pneumocystis carinii*

Metabolic liver disease

When disease interrupts the function of the liver, the normal metabolism is affected. Usually this is the result of too much or too little activity in the liver cells. This in turn leads to alterations in the structure of liver cells. An example would be excess deposits of glycogen within the hepatocytes, an occurrence that can cause the newly packed liver cells to expand. We will cover some of the more common disorders of liver metabolism.

Fatty liver

A liver that is affected with fatty change, or hepatic steatosis, is called a fatty liver. Fatty change is the presence of abnormal amounts of fat droplets in and between cells of tissue. The term ‘fatty change’ used to be known as ‘fatty infiltration.’ It has numerous causes, mostly those that affect the liver’s normal metabolism of fat. The most common causes are alcohol abuse, diabetes mellitus, obesity, and malnutrition. Fatty liver can be reversed when the cause of the condition is removed. There are other less common causes, such as chemotherapeutic drugs, excessive corticosteroids, and jejunoileal bypass surgery.

Most patients with fatty liver are asymptomatic. As the disease progresses, however, liver function tests begin to elevate, particularly the enzymes AST and ALT. Severe fatty liver patients may have jaundice, an enlarged and painful liver, nausea, vomiting, and hepatitis. Direct serum bilirubin elevates with severe fatty liver.

Cirrhosis

No longer one of the top 10 causes of death in the United States, cirrhosis is still common enough where you may have to perform a sonographic examination on a patient who has the disease. Killing about 26,000 people yearly in the United States, cirrhosis is the end result of many liver diseases. Once contracted, it is irreversible. The disease is chronic and progresses to either liver cell failure or

portal hypertension, of which both are potentially fatal. Its morphological (physical) characteristics throughout the liver are simultaneous parenchymal (tissue) necrosis, regeneration of tissue into small nodules (micronodular) or large nodules (macronodular), and fibrosis from scarring. These characteristics disorganize the structure of the liver.

There are many causes of cirrhosis. The most common cause in the United States is alcohol abuse followed by chronic hepatitis C. Other causes are chronic hepatitis B (world-wide), drugs, toxins, some late-stage parasitic infections such as schistosomiasis, inherited diseases, chronic heart failure, and blocked, inflamed, or destroyed bile ducts.

Many people who have early cirrhosis are asymptomatic. However, as fibrosis and regeneration insults healthy cells, liver functions begin to decline; and the patient may start to show symptoms. For example, a patient may suffer exhaustion, fatigue, appetite loss, nausea, weight loss, and abdominal pain. Many times patients will present themselves to the referring physician only after the point when cirrhosis develops complications. In these cases, the classic symptoms are usually jaundice, hepatomegaly, and ascites. Severe cases of advanced cirrhosis may be accompanied with portal hypertension and its associated esophageal varices (which may result in bleeding) and liver cell failure. Laboratory results will either show normal LFTs or elevated bilirubin and aminotransferases (AST more than ALT), prolonged PT, and normal to three times normal ALP.

Glycogen storage disease

Another liver disease that you may see (mostly in children) is one of 14 glycogen storage diseases (GSD). GSD, type 1, otherwise known as von Gierke's disease, is an inherited disorder of liver metabolism. This genetic defect is a deficiency in glucose–6-phosphatase, an enzyme that abnormally affects glycogen metabolism through excessive storage within the liver cells. Patients usually will have severe hypoglycemia (a spectrum of symptoms resulting from low blood sugar), hepatomegaly (enlarged liver), increased echogenicity, hyperlipidemia (high concentration of lipids in the blood), renal enlargement, and hyperuricemia (increased uric acid, or crystals, in the blood). If treated, children usually survive into adulthood.

Cystic lesions of the liver

Some focal abnormalities found in the liver are cysts. A cyst can be defined as a fluid-filled enclosed space with an epithelial wall. The epithelium, cell layers, in these walls is what distinguishes these lesions from the non-epithelial borders of abscesses, pseudocysts, and parasitic cysts. In the liver the two most common types of cysts you may have to evaluate are simple cysts and polycystic liver disease.

Simple liver cysts

Simple liver cysts can be congenital (present since birth) or acquired; solitary or multiple. They appear increasingly with age, usually after 40 years. Simple liver cysts tend to be found more in females than males. Unless the cysts are very large, hemorrhage, or become infected, there are usually no symptoms, and you will likely discover them incidentally during a sonographic examination.

Complications can arise from liver cysts, such as increased size to as large as 20 cm or more, hemorrhage, and infection. All of these can produce pain in the patient, with infection resulting in fever.

Polycystic liver disease

This is an inherited disease that is either in the adult form (autosomal-dominant) or infantile form (autosomal-recessive). The adult form usually produces multiple cysts in the liver only, the kidneys only, or a combination of the two. At least half of patients with polycystic renal disease will have multiple liver cysts (polycystic liver disease).

As with the clinical situation for simple cysts, patients with polycystic liver disease usually are asymptomatic unless size, hemorrhage, or infection produces pain and fever. If there are enough

multiple cysts or cysts with large sizes, the liver may enlarge. However, the liver functions remain normal and LFTs are usually not affected.

Solid tumors of the liver

What usually concerns physicians about solid masses in the liver is the potential for the tumor to be malignant. A host of focal liver masses can occur within the liver. Normally discovered during computed tomography (CT) studies, liver masses can also appear on sonography. Certain characteristics of liver masses suggest either a benign tumor or a malignancy, and a familiarity with both types helps you to accurately identify these masses. However, absolute confirmation of liver tumors normally involves laboratory examination of the tissue through biopsy.

Benign liver neoplasms

Unless the size of the tumor causes symptoms such as pain, most benign liver tumors are unnoticed by the patient. Indeed, without enormous tumor sizes displacing liver tissue, benign neoplasms rarely affect the normal functioning of the liver. We will take a look at some of the more common benign tumors.

Cavernous hemangioma

Of the benign liver neoplasms, the most common is the cavernous hemangioma. This tumor is composed of tiny vascular channels filled with blood. It occurs at all ages but especially in adult women. Cavernous hemangiomas usually do not produce symptoms in patients, and the tumor is usually discovered incidentally during a sonographic liver examination. Usually, these tumors maintain a stable size; however, if they do grow, they do so at a very gradual rate, sometimes lasting years. Pain from the tumor compressing surrounding liver tissue can occur if the hemangioma is excessively large.

Focal nodular hyperplasia

Although rare, focal nodule hyperplasia (FNH) is the second most common benign tumor of the liver. It is a tumor made up of disorganized, but normal appearing, liver cells, Kupffer cells, and bile ducts with a large artery in the center. Portal venules are absent in the tumor. FNH is usually seen in adult females but can occur in both sexes at any age. Frequently women with FNH that bleed are using oral contraceptives. Most patients are asymptomatic.

Hepatic adenomas

Hepatic or liver cell adenomas are rare and appear mostly in women using oral contraceptives. The tumors are composed of adenomatous (glandular) tissue and normal or slightly abnormal liver cells. They lack bile ducts and Kupffer cells. Hepatic adenomas are associated with glycogen storage disease, type I.

Most patients have RUQ pain, sometimes sudden and severe, due to bleeding within the tumor. If the tumor ruptures, which most do, the patient may go into shock from hemorrhaging into the peritoneum. Generally, the physician will feel a distinct mass and will note an enlarged liver. Hepatic adenomas are usually removed by surgery.

Lipomas

Lipomas are rare, asymptomatic tumors. Most are composed purely of fat; hence the name. However, a small percentage is mixed with various other tissue types. They are associated with kidney angiomyolipomas and tuberous sclerosis.

Malignant liver neoplasms

Of the handful of malignant tumors arising from liver tissue, the most common is hepatocellular carcinoma. Otherwise, malignant tumors usually originate from other parts of the body. We will briefly look at the most common of the primary and secondary malignancies.

Hepatocellular carcinoma

Hepatocellular carcinoma (HCC), or liver cell cancer, is the most common primary malignant tumor of the liver. It is one of the most common malignancies worldwide but less common in the United States. HCC appears more frequently in men. The malignancy is closely associated with cirrhosis, chronic hepatitis B (worldwide), and carcinogenic toxins from fungi in certain foods eaten in developing countries.

Typical symptoms of HCC are hepatomegaly, abdominal pain, and weight loss. By the time a patient is seen in the clinic with these symptoms, the disease has already progressed to an advanced stage. Liver function tests are not as helpful as they can be either normal or have marked elevations. The most useful test for HCC is detection of alpha-fetoprotein (AFP). This is a protein that is usually produced by the liver of a fetus and not an adult. When adult AFP levels are increased, it is suggestive of HCC but only combined with sonographic evaluation of the liver. It remains suggestive because AFP levels can also elevate in cases without HCC such as cirrhosis, chronic hepatitis, and pregnancy. True diagnosis of HCC can only be accomplished with biopsy.

Metastatic disease

Metastasis is the transfer of malignant cells from one organ or part to another. The organ or part of origin is where the original tumor is growing. This original tumor is called the primary tumor or site. After the malignant cells are transferred to another location through the blood or lymph, they will usually grow into new multiple tumors. These metastases are considered secondary tumors.

In the United States, metastatic disease is the most common cause of malignant neoplasms in the liver and occurs much more frequently than HCC. Recall that HCC is a primary liver malignancy; that is; the original tumor is from the liver. Also recall that HCC is only the most common *primary*, malignant liver neoplasm; it is not the most common malignant neoplasm in the liver. That distinction belongs to the secondary or metastatic tumors that originate from the colon, pancreas, stomach, lung, and breast. Clinically, the patient will suffer from hepatomegaly and weight loss. LFTs will usually be abnormal.

Vascular liver abnormalities

Because the liver is a highly vascular organ and functions as a filter for the blood, occasional abnormalities affecting blood vessels within the liver or the circulation of flow through the liver can occur. This is primarily due to obstruction of blood vessels.

Portal venous hypertension

Portal venous hypertension is increased blood pressure in the portal venous system largely as a result of resistance to incoming blood flow. In Western countries, cirrhosis is the primary source of resistance to portal flow. Other causes are hepatic and portal vein obstruction from thrombus or tumor invasion, schistosomiasis, congestive heart failure, and some toxins.

Clinically, patients will have symptoms of cirrhosis, accompanied by complications such as ascites, splenomegaly (enlarged spleen), and, in severe cases, bleeding from gastroesophageal varices (abnormally enlarged and tortuous veins). Because blood from the intestines as well as the spleen cannot find normal routes through the liver to the heart, other vessels are recruited to serve as collateral pathways.

Portal vein thrombosis

Another abnormality of the portal vein detectable by sonography is portal vein thrombosis, which is the partial or complete obstruction of the portal vein. Either a thrombus (blood clot) or an invasive tumor will be the cause of this venous disease. The most common source for thrombosis (clot formation) is extremely slow portal blood flow due to cirrhosis. Other sources that are likely to cause portal venous clotting are pancreatitis, hepatitis, pregnancy, trauma, surgery, portocaval shunts, and portal phlebitis derived from abdominal inflammations such as appendicitis.

The most common invasive tumor that will partially or completely block the portal vein is HCC. Other tumors which may invade the portal system are the many gastrointestinal cancers such as pancreatic carcinoma. Clinically, patients will display the signs of the disease causing the clot or tumor, such as the symptoms of cirrhosis or HCC. Liver function tests are usually normal in thrombus-based portal vein thrombosis.

Budd-Chiari syndrome

The portal veins are not the only vascular structures in the liver that may become occluded. The hepatic veins also may be blocked. When there is a partial or complete obstruction of the hepatic veins, the abnormality is usually referred to as Budd-Chiari syndrome.

Budd-Chiari syndrome is rare and results from various disorders. It is more common in Asia and southern Africa than in Western countries. In non-Western countries the major cause of Budd-Chiari syndrome is congenital membranes or webs blocking the IVC. In the West, the major causes are usually clotting disorders of the blood (hypercoagulation) and other blood disorders such as polycythemia vera (also known as polycythemia rubra vera). Other causes can be from invasion of the hepatic veins by malignant tumors such as hepatocellular carcinoma and renal cell carcinoma. Occasionally, trauma, infections, and the compression of the hepatic veins from cirrhosis may cause Budd-Chiari syndrome.

Acute onset of Budd-Chiari syndrome with complete blockage of the hepatic veins will usually result in liver failure and death in days to weeks. In most cases, the disease is chronic and patients will present with ascites, hepatomegaly, and abdominal pain. Liver function tests are not helpful with this abnormality as they are usually within normal ranges.

004. Considering general procedures for liver imaging

Generally, most sonography departments include the evaluation of the liver among other organs in a comprehensive abdominal sonogram. For our purposes, we will strictly focus on the liver portion of the abdominal examination. Let’s take a look at common approaches to liver imaging before the exam and during the exam.

Process before the liver examination

The first thing you should do to prepare for the examination is read the request written by a physician or an authorized medical provider. Most importantly, a sonographer should ensure the patient scheduled or to be examined is the patient identified on the request. Pay particular attention to the history. The history on a sonogram request does not necessarily include the patient’s medical history. Medical history can be accurately obtained from the patient’s medical records. On the request, physicians may write brief descriptions of a patient’s past medical history as it relates to the patient’s current condition. Otherwise, the written history refers strictly to what physicians may have discovered during examination in the clinic (signs and symptoms) and to their suspicions as to the nature of abnormality. Usually this suspicion is worded in a request to “rule out” a disease process. Laboratory values, or results, may also be included in the request. If it is not, it makes good sense for the sonographer to request liver function tests from the laboratory, the referring provider, or the patient. Lab results help you to focus your exam so that you are not wasting time or effort, but wisely focusing on a particular sonographic appearance from a particular disease.

For most routine examinations of the abdomen, you must prepare the patient in advance. This usually involves instructions about such things as what clothes to wear and any records radiologists may want to review. For most institutions, it is common to instruct the patient to fast approximately six to eight hours prior to the examination. Usually, fasting helps reduce bowel gas that may interfere with the sonographic visualization of abdominal structures, and normally causes the gallbladder and common bile duct to retain enough bile for evaluation. Because the gallbladder and bile ducts are usually examined with the liver, most liver exams benefit from patient fasting even though it is not necessarily needed because of its location in the body.

Review the patient's previous sonographic images, if they exist. This will give you a map of the individual's anatomy and will give you a clue as to the appropriate transducer (probe) to use. You also will find it helpful to review the particular pathology that a requesting clinician wants evaluated. Use textbooks and articles to reinforce your understanding of the suspected disease. Compare the liver function tests to what is known about their levels among various diseases. Don't forget to ask fellow sonographers or the radiologist if you have any questions. This review of pathology should help you to tailor your approach to the exam based on the patient's medical history and the history written by the referring physician. It will also prepare you for any spontaneous questions that a radiologist may ask of you.

Process during the liver examination

Most institutions expect the sonographer to use 3 to 5 megahertz (MHz) sector or curvilinear transducers for liver examinations. Patients generally lay flat on the back or in a slight left lateral decubitus position to make access to the right upper quadrant of the abdomen easier. Keep in mind that you are expected to move or sweep the sonographic probe in such a way that the ultrasound beam covers every part of the liver. This procedure, commonly called "scanning," helps you to detect abnormalities. When you freeze the image on the display monitor and print it, you are "documenting" or "imaging."

Certain images are to be obtained that are considered standard in the field. You should obtain longitudinal images of the left lobe that demonstrate the hyperechoic line of the fissure for ligamentum venosum, anterolateral to the caudate lobe and the left hepatic vein (fig. 1–4). Image longitudinal slices of the medial left lobe and, just to the right, the hyperechoic line of the main lobar fissure that runs from the gallbladder. Longitudinal images of the right lobe that demonstrate the right hepatic vein and the adjacent right kidney should be documented (fig. 1–5). The right kidney should be isoechoic to slightly hypoechoic relative to the liver echotexture.

The size of the right lobe, as measured along the midclavicular line, is commonly known to be averaged at 15 cm or less. Because of the variations from patient to patient in rib cage anatomy, this measurement may be difficult to obtain along the midclavicular line. For this reason, sonographers sometimes use a more coronal approach with the probe aligned along the right lateral side of the patient, an area that usually offers a better sonographic window of the entire right lobe length. However, keep in mind that this approach is not along the midclavicular line, and a radiologist may reject using it for measuring liver length. A few authors consider a size of 20 cm or greater to be hepatomegaly, an enlarged liver. While true, the majority of investigators in the sonographic literature report the threshold to be much lower and consider hepatomegaly to be present with a measurement greater than 15 cm.

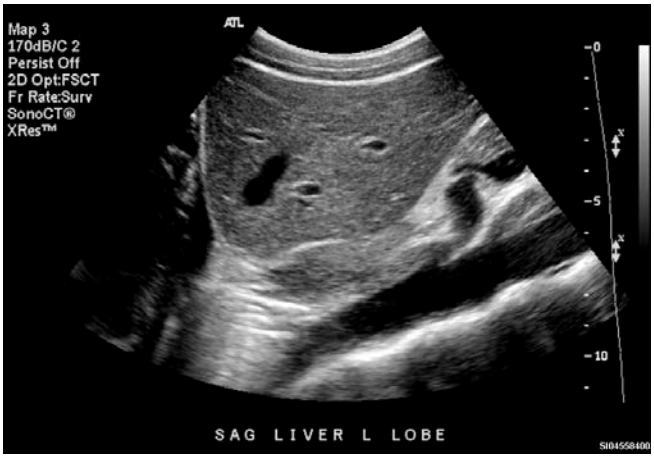

Figure 1–4. Left lobe of the liver, longitudinal.

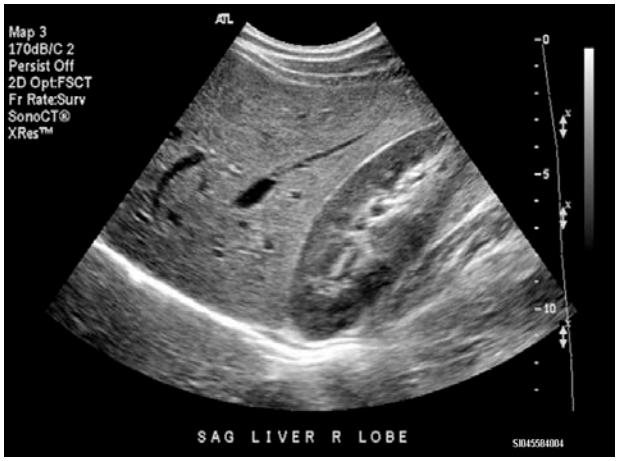

Figure 1–5. Right lobe of the liver, longitudinal.

Obtain transverse images of the left lobe that demonstrate the left portal vein branches as well as the caudate lobe bordering the inferior vena cava (fig. 1–6).

You may see an echogenic focus with slight shadowing in the center of the left lobe (fig. 1–7). This is the cross-section of the ligamentum teres, which many consider to be the falciform ligament. Be aware that the ligamentum teres is contained within the falciform ligament and both ligaments are within the left intersegmental fissure. You may see the falciform ligament as a hyperechoic line that will stretch up from the ligamentum teres to the surface of the liver.

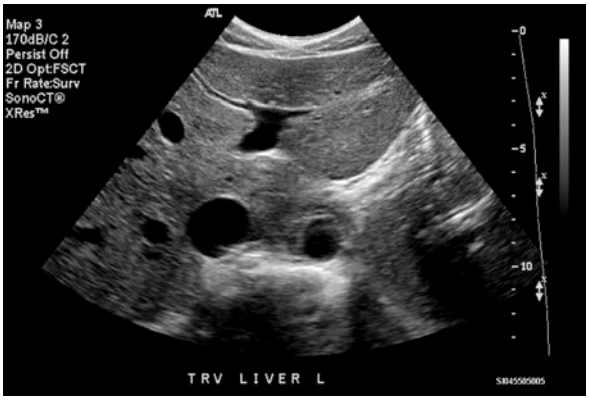

Figure 1–6. Left lobe and caudate lobe of the liver, transverse.

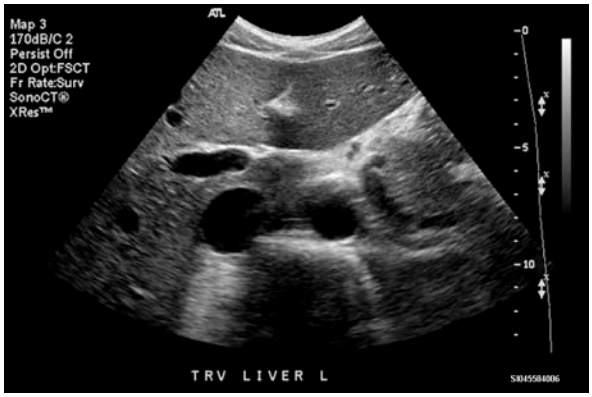

Figure 1–7. Ligamentum teres, left lobe liver, transverse.

Transverse images of the liver that demonstrate the lengths of the main hepatic veins as they course into the IVC—left (fig. 1–8), middle (fig. 1–9), and right (fig. 1–10)—can be documented at the superior level of the liver on one image or, depending on patient anatomy, separately.

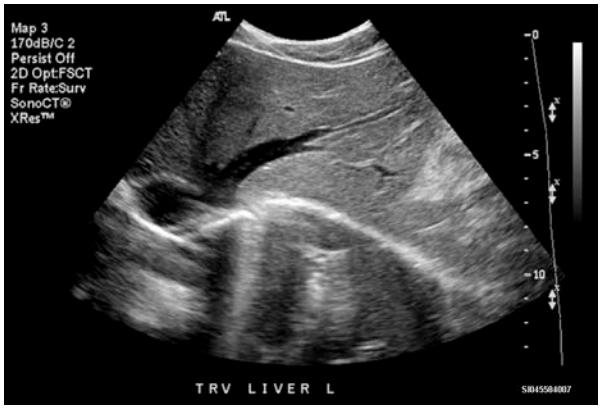

Figure 1–8. Left hepatic vein, transverse.

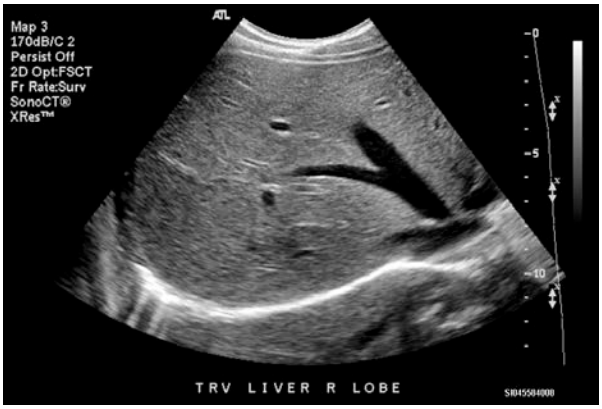

Figure 1–9. Middle hepatic vein, transverse.

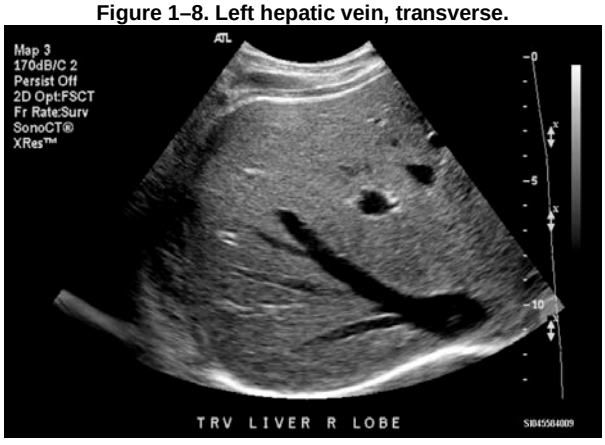

Figure 1–10. Right hepatic vein, transverse.

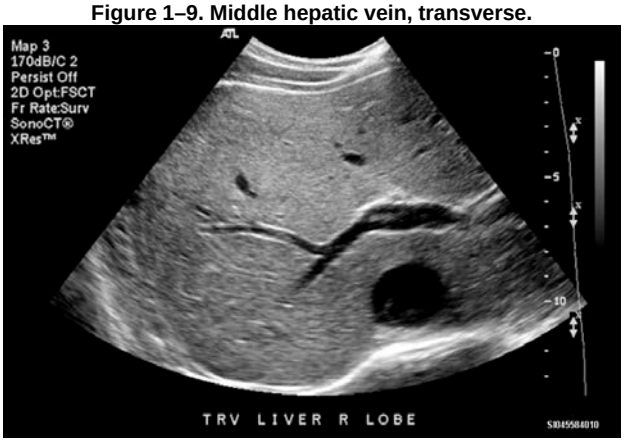

Figure 1–11. Right portal vein, transverse.

Further caudal imaging in the right lobe should demonstrate the anterior and posterior branching of the right portal vein (fig. 1–11).

005. Imaging infectious and metabolic liver disease

You must also be prepared to adjust common sonographic approaches based on suspected abnormalities. Indeed, the approach you use predominately depends on pathology clinicians want to "rule out." Pathologies of the liver are sometimes caused by infectious diseases, which usually inflame the liver to a point where sonographers may detect the physical signs. For the sonographer, commonly encountered infectious diseases that affect the liver are viral, bacterial, and parasitic.

Viral hepatitis

You may have to image a liver that shows signs of viral infection. You should focus on the portal veins when scanning these patients. With acute hepatitis, the sonographer commonly will see either a normal appearing liver or the walls of the portal veins will be more hyperechoic than usual, an appearance called periportal cuffing (fig. 1–12,A, B). There is usually a thick gallbladder wall (fig. 1–12, C, D) with an increase in liver size (hepatomegaly).

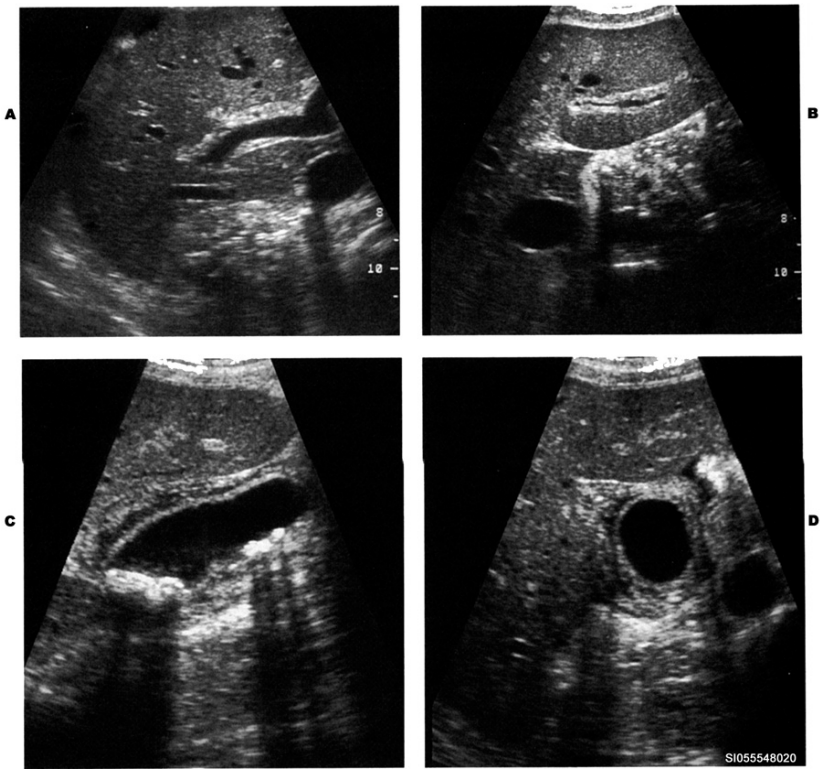

Figure 1–12. Acute hepatitis with thickened gallbladder wall. (Reproduced by permission of Elsevier, Inc.)

In chronic hepatitis, the liver may appear either normal or will take on a slightly coarse, heterogeneous echotexture. In this case, the liver may also appear more echogenic than usual. The portal vein walls are usually not as hyperechoic as in the normal liver. There is no increase in liver size with chronic hepatitis and may even appear slightly smaller than normal.

Pyogenic abscess

On sonography, pyogenic abscesses will be either multiple or solitary. If singular, they are usually located in the right lobe of the liver. The sonographic appearance of a pyogenic abscess depends on its age. For example, an early abscess may be quite small, round, and hypoechoic. Later, the abscess may develop more complex appearances, with internal echoes, internal septations, echogenic foci with posterior reverberations (the "dirty shadowing" of gas), debris, and well-defined or irregular

walls. You should be aware that scanning the liver from the anterior surface of the body may cause difficulty in distinguishing a gas-producing abscess from bowel. With the patient in a supine position, try moving the probe to the right side of the body, posteriorly, and aim the probe up toward the abscess. Thus, you will get behind the dirty shadowing to better see the contents. Another useful method for analyzing an abscess is to turn the patient into a left lateral decubitus position and observe any movement of debris, which should settle by gravity onto any denser fluid portions of the abscess (the so-called "fluid-fluid" level). This varying appearance of the pyogenic abscess is similar to other conditions and presents a differential diagnosis of amebic abscess, echinococcus cyst, and other complex masses such as a hematoma or a cystic neoplasm. You should measure the height or anteroposterior (AP), the length, and the width of the abscess. Finally, use color Doppler to determine the location and character of blood flow in the area of the abscess.

Parasitic liver diseases

Very few parasites can be seen sonographically; most are microscopic in size. However, sonographic evidence of parasitic disease in the liver is readily apparent. Usually, the organisms will form fluid collections in the form of cysts and abscesses.

Amebic abscess

With sonography, an amebic abscess is seen as a well-defined, hypoechoic, oval or round solitary mass predominantly located in the right lobe of the liver. It can very tiny to extremely large. The appearance is similar to pyogenic abscess or neoplasm; however, the walls are not as prominent. Rarely, the disease may appear in multiple locations of the liver. As with pyogenic abscesses, measure the amebic abscess in AP, length, and width dimensions.

Echinococcus (hydatid) cyst

The sonographic appearance of echinococcosis depends upon the organism's larval stage within the liver. Generally, the sequence runs from a well-defined, round or oval cyst, called the echinococcus cyst, to a collapsed calcified focus. The cysts can range anywhere in size from a pinpoint to extremely large. Found mostly in the right lobe of the liver, the early echinococcus cyst is distinguished sonographically from a simple cyst by the presence of fine internal echoes called hydatid sand. The outer layer is an echogenic fibrotic capsule called the pericyst. The inner layer is a thin lining that produces the larvae called the endocyst. The next stage of development seen is the formation of separate endocysts, called "daughter" cysts, within the main pericyst. Sometimes, the echinococcus cyst will show an undulating membrane. This represents the detached endocyst. There may also be debris in the dependent portion of the cyst that may be detected if you role the patient into a decubitus position. In a later stage of larval development, the cyst may resemble a solid mass because of the presence of echogenic material and calcifications. Usually at this stage the cyst will collapse, leaving a hyperechoic focus with distinctive posterior shadowing. At all stages, measure the borders of the pericyst with AP, length, and width measurements. Use color Doppler to evaluate blood flow in the area of the cyst. This may help to distinguish it from similar appearing neoplasms.

Schistosomiasis

The main sonographic identifier for schistosomiasis is widened, portal tracts up to 2 cm in diameter with thickened and echogenic walls. The normal portal vein diameter is approximately 1.3 cm. With this in mind, measure the AP diameter of the inner wall to inner wall of a portal vein. Although there is hepatomegaly, the liver begins to atrophy with the onset of portal hypertension as a result of periportal fibrosis. Granulomas may be seen as hyperechoic foci with shadowing and should be documented. The gallbladder wall may be thickened, in which case it also should be imaged. In portal hypertensive patients, sweep the probe in areas adjacent to the liver to detect and document the presence of ascites.

Other infectious diseases

With mycobaterial infections, the primary appearance to look for is hyperechoic foci representing granulomas, with or without calcifications. The lesions are sonographically similar to that of other infections. As with any other lesion, measure the granuloma (no matter how small) in three dimensions. It may help to magnify the image first before placing the electronic calipers.

Candidiasis infections appear on sonography with a wide range of characteristics. To help you distinguish this disease from metastatic nodules, it helps to group types. Sonographers will generally see one of four appearances:

1. "Wheel-within-a-wheel": A round nodule, hypoechoic on the periphery and echogenic in the center surrounding a hypoechoic nidus. This is the earliest manifestation.
2. "Bull's-eye" or "target": Essentially the same as wheel-within-a-wheel, but without the hypoechoic nidus. Metastatic nodules may have the exact same appearance.
3. "Uniformly hypoechoic focus": This is the most common appearance, which represents fibrosis.
4. "Echogenic focus": This is calcification that represents scar tissue.

Whenever you see any one of these appearances, measure it in three dimensions: AP, width, and length. Usually, the radiologist will have you measure the largest abscess but some may require you to measure every one.

Finally, you may see a range of appearances in cases of *P. carinii*. For example, you may see tiny, non-shadowing calcifications spread throughout the liver, or you may see most of the hepatic parenchyma replaced with clumps of dense, shadowing calcifications. If spread throughout the liver and of similar sizes, measure a representative lesion in three dimensions. Otherwise, measure all of the dense, shadowing types of suspected calcifications from *P. carinii*.

Metabolic liver disorders

Evidence of metabolic disorder is usually spread throughout the liver. We can see this abnormality with sonography as a diffuse change in the echotexture of the liver. We will now discuss imaging three of the most commonly occurring metabolic liver disorders.

Fatty liver

For the sonographer, fatty liver is a fairly common appearance. However, its appearance is also similar to cirrhosis and chronic hepatitis. Generally, you will see a liver texture with increased echogenicity as compared to most normal livers. Ensure that your overall gain is adjusted to a medium setting where a normal liver's appearance would neither be too bright nor too dark. In this way, an echogenic liver will clearly alert you to the possible presence of fatty change. What may help distinguish this brightened appearance from the appearance of cirrhosis and chronic hepatitis is the presence of attenuation in the posterior portion of the right lobe. You may see slight enlargement of the left lobe. Keep in mind that some radiologists may require you to grade the texture of a fatty liver, despite advances in resolution. Liver grading was generally done with a 3.5 MHz transducer and was as followed:

- Grade 1: (mild), slight increase in liver tissue echoes with diaphragm and vessels clearly seen.
- Grade 2: (moderate), moderate increase in liver tissue echoes with decreased ability to see the right hemidiaphragm due to increasing attenuation of sound and difficulty distinguishing vessel borders.
- Grade 3: (severe), significant echogenic liver tissue with extremely poor or no ability to see the right hemidiaphragm and vessels. The right posterior portion of the liver is usually attenuated and cannot be seen. In some cases, more than half of the liver cannot be seen.

Sometimes, you will see cases of fatty liver where there are patches of bright echoes amid a background of normal appearing liver tissue. This situation is commonly called ‘focal fatty infiltration.’ These patches usually have straight edges and are wedge-shaped. They are commonly located in anterior medial segment of the left lobe. On a follow-up ultrasound, you may detect a change in size and shape or even disappearance.

You may also encounter the reverse situation: a diffusely echogenic fatty liver with areas of normal echotexture. This is called focal sparing. These are usually ovoid or round in shape, which can appear to be a mass. The more common locations are the medial segment of the left lobe, the area anterior to the portal vein bifurcation, and the area medial and anterior to the neck and body of the gallbladder.

Both focal sparing and focal fatty infiltration may mimic neoplasms on sonography. Aside from a changed appearance on follow-up, another way that may help you distinguish them from neoplasm is that focal fat or focal sparing does not push aside hepatic vessels (mass effect). Use color Doppler to help determine if the area of focus or sparing is displacing the blood vessels.

Recognizing these varied appearances of fatty liver will help you present your findings, along with clinical history, to a radiologist in a more accurate manner.

Cirrhosis

To help diagnose cirrhosis, you must be aware of its characteristics on the sonographic screen. Early cirrhosis of the liver may enlarge the liver, particularly when alcoholism is the source. As the disease progresses, the right lobe of the liver will shrink and will be small relative to the caudate or left lobe.

Depending on equipment resolution, the echotexture of the cirrhotic liver is usually seen as coarse or grainy. This appearance may not be as apparent on the latest equipment with superior resolution. Compared to a normal liver, the echotexture will at least be heterogeneous. To easier detect these irregular appearances, ensure your equipment is set at ranges that would optimally display a normal liver.

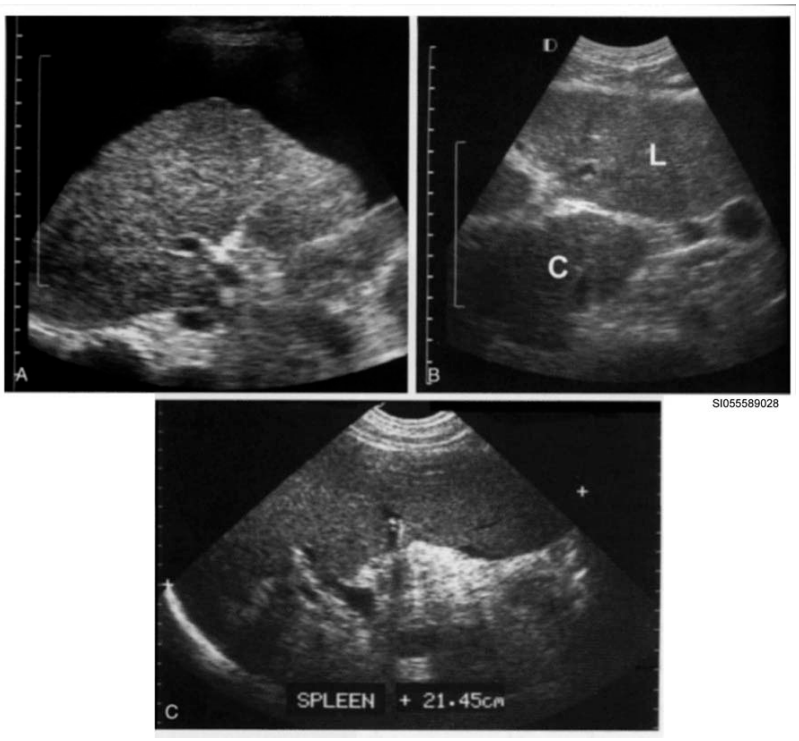

Figure 1–13. Liver cirrhosis. (Reproduced by permission of Elsevier, Inc.)

With fibrosis, the echogenicity of the liver may increase. However, as with echotexture, this characteristic may be affected by the equipment’s overall gain or time gain compensation setting. In some early cirrhotic livers, the echogenicity may resemble that of a normal liver. As the disease continues the echogenicity of the liver resembles that of fatty liver. But what helps distinguish a cirrhotic liver from a fatty liver is the lack of attenuation in cirrhosis. Keep in mind, however, that fatty liver can also be present simultaneously with cirrhosis; in which case, there should be attenuation.

With advanced cirrhosis, it may be possible to detect the presence of nodules. The surface of the liver is usually only seen sonographically in the presence of ascites (fig. 1–13, A). Advanced cirrhosis will also cause the left lobe and caudate lobe to appear enlarged relative to the right lobe (fig. 1–13, B). Finally, you may see evidence of portal hypertension at the severest stage of cirrhosis. We will discuss portal hypertension later in the unit. For now, remember that where there is portal hypertension there is usually an enlarged spleen (fig. 1–13, C).

Glycogen storage disease

Because of GSD's similarity in appearance to fatty liver, the most useful reason for you to produce sonographic images of this liver disease is to monitor a patient known to be suffering from it for complications. Sonography can detect GSD complications, such as the development of hepatic adenomas, hepatomegaly, and renal enlargement.

006. Imaging focal abnormalities of the liver

Now that we have looked at diseases that more or less affect the entire liver, we will look at abnormalities with single locations within the liver; that is, focal abnormalities. From cysts to solid tumors, there are many different focal abnormalities that can occur in the liver. We will begin with cystic lesions.

Cystic liver abnormalities

Depending on patient history, the sonographic appearance of cysts in the liver, if single, generally do not prompt much concern in radiologists. It is when sonography reveals irregular appearing cysts, multiple cysts, or extremely large cysts, that concern increases.

Simple liver cysts

Located mostly in the right lobe, simple liver cysts are anechoic and round with clearly defined walls, which are usually thin. Most are less than 5 cm in diameter. They usually display a posterior acoustic enhancement. Because of this classic description, it is important for you to ensure that your time gain compensation (TGC) and overall gain are set at the medium levels that display the normal liver echotexture. In this way, the distal acoustic enhancement (also called 'through-transmission') will be easily seen compared to the surrounding liver echoes. Also, the inside of the cysts should be as echo-free as possible without sacrificing the medium gray tones of surrounding liver tissue. You should measure liver cysts in three dimensions: AP, length, and width. If there are multiple cysts, and the radiologist prefers measurements of all or a few of the largest, it is helpful to label each cyst that you measure.

Some simple liver cysts do not appear so simple. On the sonogram, you may see the hemorrhagic or infected cyst with fine, low-level echoes, as well as septations and thickened walls. Place a color Doppler box around a large cyst to determine if it is displacing any surrounding blood vessels.

If symptomatic, complicated liver cysts are treated with cyst aspiration followed by injection of alcohol into the collapsed area to prevent reoccurrence. This procedure is sometimes performed under ultrasound guidance.

Polycystic liver disease

You may evaluate a patient with polycystic disease. For this reason, it is a good idea to scan the kidneys if you see multiple cysts in the liver. The sonographic appearances of the cysts in this disease are identical to that of simple liver cysts of varying sizes. Measure only the largest cysts while trying to image as many as possible. Also, you should measure any cysts which appear irregular, as these may turn out to be coexisting malignant tumors with cystic characteristics.

Solid liver tumors

Now we will move into the solid focal abnormalities of the liver. Generally referred to as tumors or neoplasms, we will start with a few that are benign and finish with the most common that are malignant.

Cavernous hemangioma

The sonographic appearance of the cavernous hemangioma varies. The classic appearance you should be familiar with is of a round, hyperechoic, and homogeneous tumor or tumors with clearly seen smooth borders. Some also have posterior acoustic enhancement. You may generally see them as a solitary hyperechoic lesion or multiple lesions in the right posterior lobe of the liver or near the liver periphery. Most are less than 3 cm in diameter and generally do not change size in adults. Depending on the radiologist's preference, you may have to measure larger hemangiomas in three dimensions for comparison with future sonograms. Measurement becomes particularly important in symptomatic patients or patients with a history of malignant disease because change in size helps to confirm or rule out other diseases with similar appearances, such as metastases, hepatocellular carcinoma, hepatomas, or adenomas.

Atypical appearances of hemangiomas can occur, usually in ones larger than 4 cm, and you should be aware that they might be hypoechoic or of a heterogeneous echogenicity. Large cavernous hemangiomas (sometimes called giant hemangiomas when over 4 cm) may occur in pregnant women or women who are using estrogen based oral contraceptives.

Along with measuring the cavernous hemangioma, you can help solidify a diagnosis by placing a power Doppler box over the tumor. This may detect the extremely slow flow of blood within the hemangioma. Color or duplex Doppler will not be helpful in this case.

Focal nodular hyperplasia

The FNH is usually discovered incidentally during a sonographic scan of the liver. You will usually see an FNH as a solitary liver tumor near the subcapsular (just below the surface of the liver) area. FNH size varies from small to large. Although the echogenicity has a variety of appearances, most FNH tumors are isoechoic with the surrounding parenchyma, which makes detecting them difficult. Larger FNH tumors are difficult or impossible to distinguish from hepatic adenomas. Usually, only the presence of bleeding within the tumor resulting in pain may suggest that the tumor is a hepatic adenoma. Sometimes a hypoechoic or hyperechoic linear band in the center can be seen. Place a color Doppler box to display arterial blood flow in this central region that may demonstrate branching arteries radiating out to the edge of the tumor; an arrangement that resembles wheel spokes.

As with any tumor, measure the FNH in three dimensions. It may be helpful to recheck the borders of the tumor by using color Doppler as a guide. You generally do not want to include peripheral vessels in your electronic caliper measurement.

Other benign liver tumors

Although extremely rare, there are other liver tumors that you may have to evaluate such as hepatic adenomas and lipomas. Because many benign liver tumors sonographically resemble malignant tumors, you will likely be required to evaluate any lesion that appears. We will only look at a few of the more common of these rare masses.

Hepatic adenomas

On sonogram, you will see a hepatic adenoma to be nearly identical in appearance to a large FNH tumor, which may have a variety of appearances in echogenicity. Most are 8 to 15 cm in diameter. Measure the tumor's diameter, and use color Doppler to confirm the presence of blood vessels.

Because hepatic adenomas tend to cause death from the shock of internal bleeding and because they sometimes evolve into malignant hepatocellular carcinomas, it is important to combine the adenoma's appearance with clinical symptoms to distinguish it from FNH.

Lipomas

On sonograms, lipomas are difficult to distinguish from hemangiomas, echogenic metastases or focal fat. This is because lipomas are extremely echogenic and well-defined. Just as with any other tumor, measure this one in three dimensions. Check the kidneys for the presence of similar appearing tumors in the cortex (angiomyolipomas). A useful method to help distinguish the liver lipoma from a typical cavernous hemangioma is to examine the diaphragm directly behind or deep to the tumor. If the diaphragm is displaced deeper or discontinuous, it is an indication of the fat content of a lipoma. A cavernous hemangioma does not have this example of propagation speed artifact.

Malignant neoplasms

Next we will briefly cover imaging malignant neoplasms of the liver. Although sonography is limited in its ability to distinguish benign from malignant, it is useful for providing images that suggest characteristics of cancer.

Hepatocellular carcinoma

Your approach to HCC on sonogram should reflect your knowledge of the disease's three basic patterns:

1. Solitary (usually massive), tumor with sharp borders.
2. Nodular (either single or multiple), sharply outlined usually less than 5 cm in diameter.
3. Diffuse, nodules with poorly defined borders spread throughout the liver.

Of these three patterns, nodular HCC is the most common. The echogenicity of HCC on ultrasound is variable and is commonly seen against a background of cirrhosis. Early, small nodules are usually hypoechoic. As the nodules grow larger and age, they become increasingly echogenic with a hypoechoic rim. To compare nodule size over time, you should measure the largest nodules in three dimensions. When the appearance is diffuse, it becomes more difficult to determine borders, particularly in livers with severe cirrhosis.

HCC tends to invade the portal venous system as well as the hepatic veins. In this case, you will usually see a mass of varying echogenicity within the main portal vein or within the main portal branches. Make sure you measure the extent of portal invasion. Keep in mind that peripheral portal branches may also be invaded by HCC but this cannot be demonstrated on ultrasound. Therefore, do not be surprised to learn later in the patient's treatment that there was portal invasion. Also, if HCC has infiltrated the main hepatic veins, make sure to examine the IVC for tumor invasion progressing towards the right atrium of the heart.

Finally, you may find color Doppler to be useful in distinguishing the HCC from other similar appearing tumors. Because HCC nodules may sometimes appear hyperechoic, they can resemble hemangiomas. Place a color Doppler box to determine the presence of blood flow. This will suggest a tumor that requires blood, which is the case with most malignancies. However, this will only suggest HCC. Remember that focal nodular hyperplasia and hepatic adenomas also have distinct blood flows appearing with color Doppler. Therefore, usually the laboratory results describing elevated AFP increases the likelihood of HCC.

Metastatic disease

When you perform a sonogram on a patient whose liver has evidence of metastatic disease, be aware that true diagnosis of the disease can only come from biopsy or surgery. Even though multiple lesions in the liver coupled with clinical symptoms are most commonly metastases, a confirmation cannot come solely from sonography. This is largely due to primary sites displaying different patterns in the liver. However, you can help with the diagnosis by being aware of those various appearances, which can provide you with general clues to the primary locations for further investigation.

Sonographic Patterns of Metastatic Disease		
Pattern	***Description***	***Metastasis***
Hypoechoic	Most have reduced vascularity	*Associated with non-Hodgkin's lymphoma* [Measure the largest nodules and look around the abdominal aorta and near the liver hilum for enlarged lymph nodes.]
Hyperechoic	Likely hypervascular [Use your color Doppler]	*Frequently from the gastrointestinal tract—most common is colon adenocarcinoma* [Other primary sites are the kidneys, pancreas, or reproductive organs.]
Target or "Bull's-Eye"	An echogenic tumor with a hypoechoic rim or halo.	*Any primary source* [Candidiasis may also have an identical appearance.]
Calcified	Extremely hyperechoic with shadowing; calcified completely or partially; rare	*Frequently from GI tract—most common is mucinous adenocarcinoma of the colon* [Other primary sites are the ovaries, stomach, pancreas, retroperitoneum, long bones, adrenal glands, breast, and skin.]
Cystic	Rare; irregular and thick walls, mural nodules, septations, and fluid-fluid levels within the cyst distinguish it from simple hepatic cysts	*Frequently from GI tract—particularly leiomyosarcoma* [Other locations are ovaries, pancreas, skin, and lung.]
Diffuse	Similar to cirrhosis with an inhomogeneous echotexture throughout the liver. No discrete tumors are seen.	*Frequently from breast carcinoma and lung carcinomas.*

Other malignant neoplasms

In adolescents and young adults, male or female, you may see a solid liver tumor in an otherwise healthy liver and without elevated alpha-fetoproteins. This is usually a fibrolamellar carcinoma and it is a variant of HCC. The tumor has varying echogenicities. Look for calcifications and an echogenic scar in the center; as these characteristics are unusual in regular HCC but common with this variant.

In infants and children younger than five years of age, you may have to image rare tumors called hepatoblastomas. Hepatoblastomas are usually solitary and extremely large. The liver is enlarged and alpha-fetoproteins may be elevated. You will likely see a hyperechoic tumor with calcifications. Use the color Doppler to demonstrate the hypervascular nature of this tumor.

007. Imaging vascular abnormalities of the liver

Although we will discuss the mechanics of Doppler sonography in detail later in this course, we will now briefly examine how abnormalities with liver vasculature can be imaged using both Doppler and grayscale imaging.

Portal venous hypertension

Physicians who suspect portal hypertension in patients will request sonographic evaluation. The primary places for you to determine portal hypertension is in the area of the main portal vein and in the splenic vein. After ensuring the patient is supine and quietly breathing, you should measure the diameter of the main portal vein just before it enters the liver (porta hepatis) and where it comes into contact with the IVC. Your ultrasound beam will be in an approximate longitudinal plane. Generally, a diameter greater than 1.3 cm is suggestive of portal hypertension.

If the vein size is normal, do not assume that portal hypertension is not present. Increased diameter of portal veins is due to the liver's resistance to flow, which increases the pressure. This pressure and its effect on vein diameter may be reduced if other vascular channels develop for blood to flow. In other words, if blood cannot get through the liver back to the heart, it will find another way. These other ways are called collaterals or varices, which we will discuss later.

If bowel gas is sufficiently diminished to allow visibility, you should also evaluate the splenic vein and superior mesenteric vein. To do this, start near the confluence of the superior mesenteric vein (SMV) and the splenic vein. Scan along the splenic vein with the transducer transverse to the body. This will place the ultrasound plane roughly parallel with the length of the splenic vein. Examine the SMV by turning the transducer longitudinal and scanning from the confluence along its length. Both vessels will usually double in diameter with deep inspiration in a normal patient. In cases of portal hypertension, however, the diameters of these veins will increase slightly or remain unchanged (fig. 1–14, B–C). Some radiologists will want you to measure the splenic vein diameter, which is usually 1 cm or less in a normal patient.

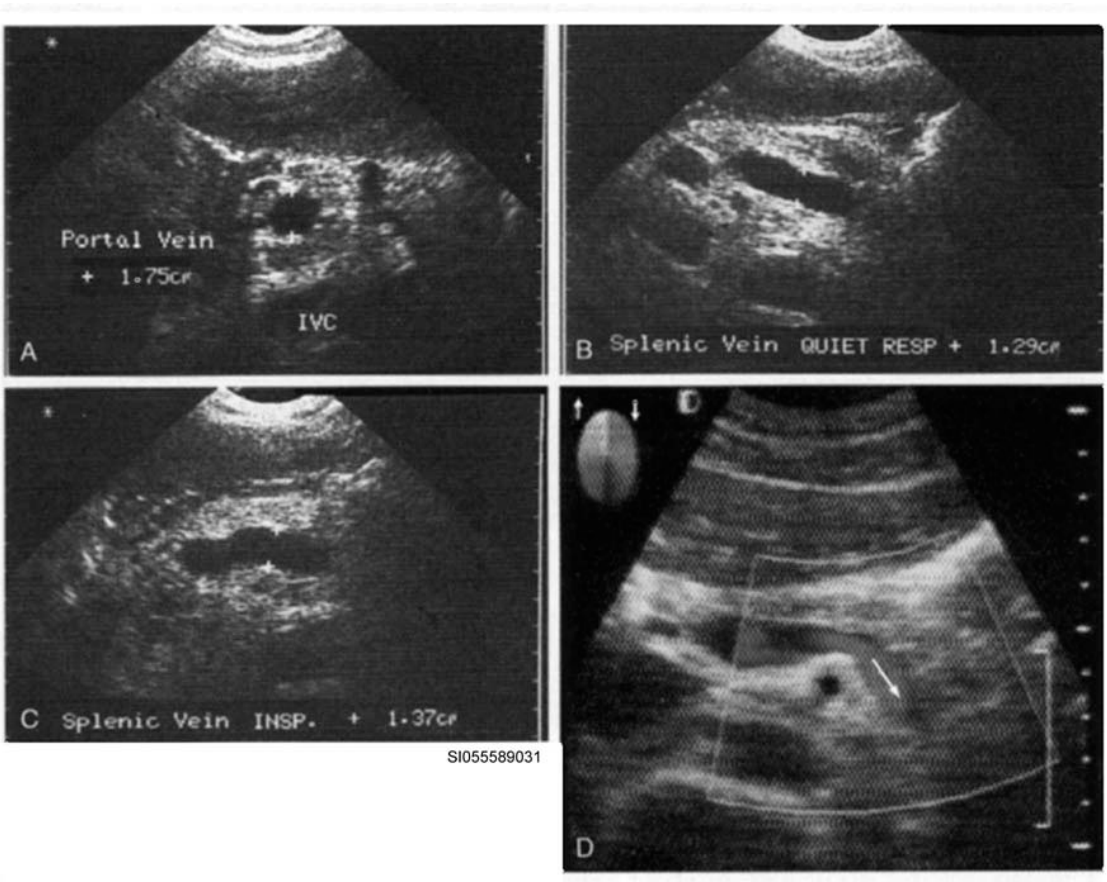

Figure 1–14. Splenic vein. (Reproduced by permission of Elsevier, Inc.)

Along with diameter, you should evaluate and document the presence of portal and splenic blood flow, their velocities, and direction. Usually, this is done with both spectral and color Doppler. Keep in mind that a normal patient's blood should flow hepatopedal or toward the liver and should result in an undulating waveform on spectral Doppler. Using color Doppler, a color (usually red) representing above-baseline flow should be seen. If you see no flow, reverse flow, or a continuous waveform without peaks and valleys (monophasic), you should suspect portal hypertension. Also be aware that there is a wide range of velocities for portal flow. The average flow is 15 to 18 cm per second.

The next most important things to look for are the major portosystemic collaterals. Some collateral vessels that develop tend to hemorrhage. This dangerous situation can cause death; particularly in the gastroesophageal region—one of the most common collaterals, along with coronary collaterals.

However, the first place you should look for collaterals is usually the easiest, along the ligamentum teres, located in the left lobe. You should scan both transversely and longitudinally and attempt to discover if there is a visible blood vessel within the ligament (fig. 1–15, A). This blood vessel is the remnant of the umbilical vein that is usually closed after birth. Portal hypertension will sometimes

cause blood to redirect through this vessel by opening it and its ending around the umbilicus (recanalization), which has venous drainage into the IVC. Use a color Doppler box to determine if there is flow within the umbilical vein (fig. 1–15, B). The direction of the flow will usually be hepatofugal or away from the liver (fig. 1–15, C).

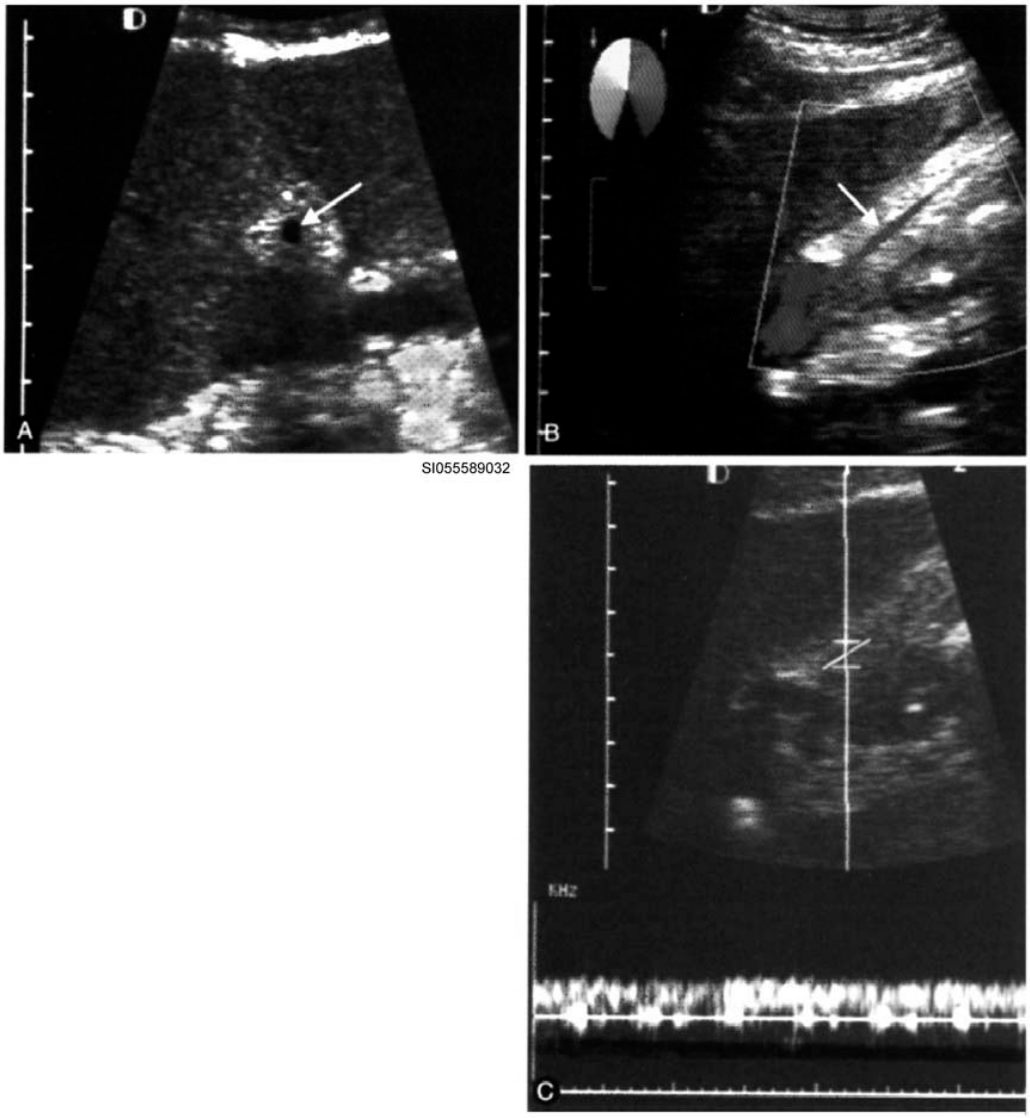

Figure 1–15. Umbilical vein collateral. (Reproduced by permission of Elsevier, Inc.)

The next collaterals to look for, which may be difficult to detect due to bowel gas, are the gastroesophageals at the junction of the esophagus and stomach. Also, look for the gastroepiploics in between the liver and the stomach or in between the stomach and the spleen. These vessels are usually seen posterior to the left lobe of the liver on longitudinal imaging or on coronal images of the upper pole of the spleen and the region where the stomach tapers into the gastroesophageal junction. The primary method to seeing the collaterals in these areas is color Doppler, which should show dilated vessels with turbulent flow.

Other locations are the hilum and upper pole of the spleen to look for splenogastric collaterals. On coronal images of the spleen, use a color Doppler box to see dilated loops of vessels at the hilum. Also, you may see the splenorenal collaterals at the lower pole of the spleen and in between the spleen and the left kidney.

Other major, but less seen, collaterals are the retroperitoneals (sometimes called pancreatic collaterals), and the coronary vein collaterals seen branching off the portal vein just above the junction with the SMV. All of the collaterals are developed as a way for the body to get venous flow back to the IVC in cases of portal hypertensive blockage through the liver (fig. 1–16).

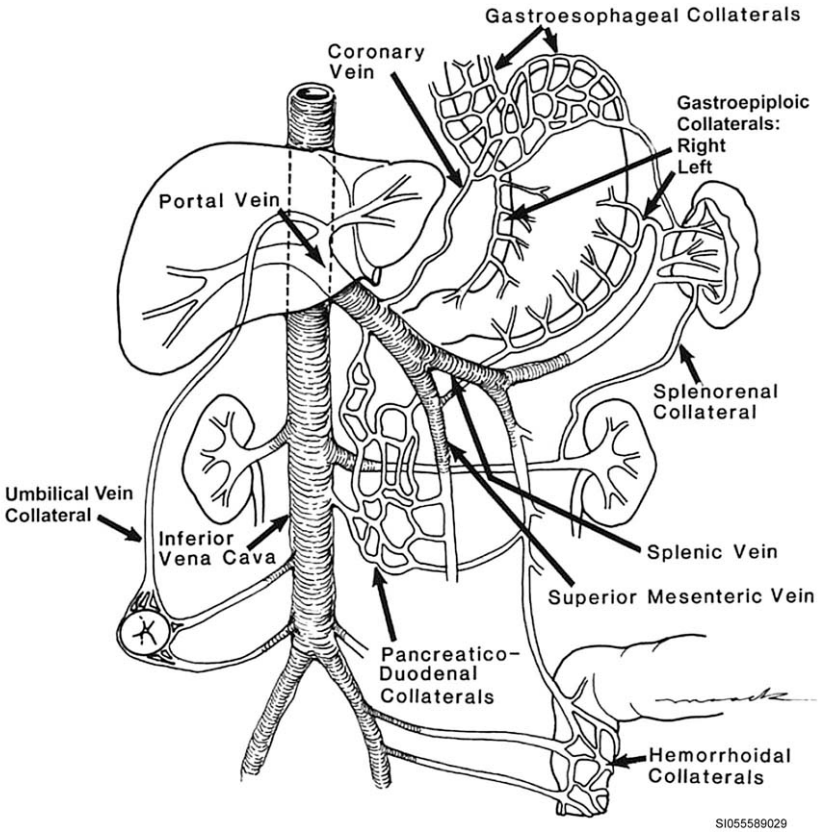

Figure 1–16. Major portosystemic collaterals. (Reproduced by permission of Elsevier, Inc.)

Be aware that the presence of collaterals will reduce the diameters of the splenic and portal veins to a more normal appearing size. Collaterals are sometimes also responsible for the flow reversals or forward and backward direction of flow.

To reduce the risk of pressure causing the gastroesophageal varices to rupture and to bleed, physicians may insert portosystemic shunts surgically between the portal or splenic veins and the IVC. An alternative is for physicians to thread shunts through the jugular vein into a hepatic vein, where a special needle makes a connection between the hepatic vein and a major portal vein, leaving the shunt in place. This shunt is called a Transjugular Intrahepatic Portosystemic Shunt (TIPS), which is metallic and can be seen on sonography. Your job, when evaluating TIPS, is to document blood flow at the portal, SMV, hepatic, and splenic veins. Look for stenosis or obstruction in the shunt from the formation of thrombus.

Portal vein thrombosis

For portal vein thrombosis, you will usually encounter a classic appearance of hypoechoic to echogenic material against the vessel wall or completely filling the entire portal vein. Measure the diameter of the portal vein at the level of thrombosis or invasion. Also place a color box over the main portal vein and portal branches to detect blood flow. The presence of thrombus or tumor in the portal vein and the absence of portal flow are highly suggestive of portal vein thrombosis. Attempt to document or trace the extent of the tumor or thrombus by evaluating the splenic vein and SMV. Portal vein thrombosis will usually cause portal hypertension and the formation of collaterals elsewhere, which may hemorrhage.

Be careful with the use of color flow in the portal vein. Do not assume that lack of color in the portal vein indicates portal vein thrombosis. Increase the sensitivity of your equipment by reducing color flow filters or by using power Doppler to detect low flow portal venous blood. Sometimes, a color box is helpful in identifying a newly formed thrombus, which sometimes appears almost anechoic.

If portal thrombosis is chronic you may see a confusing picture of various vascular channels bundled together in the area where the main portal vein is usually located (porta hepatis). These channels are collateral venous vessels formed around a thrombosed portal vein. This condition, known as *cavernous transformation of the portal vein*, should not be mistaken for dilated biliary ducts.

Budd-Chiari syndrome

While scanning patients with suspected or confirmed Budd-Chiari syndrome, ensure your grayscale images demonstrate the major hepatic veins and the portion of the IVC that passes posterior to the caudate lobe. Look for and document any echogenic material within the hepatic vessels, which may represent thrombus. Also evaluate the walls of the vessels, which are normally smooth and relatively straight; any narrowing would indicate possible stenosis from a tumor compressing the vessel or thrombus within the vessel. Make note of any tortuous vessels branching off of the hepatic veins and coursing either to the surface of the liver or to another main hepatic branch; these are collaterals located in areas where vessels are not usually seen. In Budd-Chiari patients with cirrhotic livers, the hepatic veins are difficult to see or may not be seen at all. The caudate lobe will usually be seen as enlarged compared to an atrophied right lobe in the chronic Budd-Chiari patient. This is due to the caudate lobe's venous blood bypassing the major hepatic veins and directly draining into the IVC via one or two small veins, which are located below the entrance of the major hepatics into the IVC.

It is extremely important that you also use color and pulse-wave Doppler to determine the nature of blood flow in all of the major hepatic veins. Color Doppler may help visualize flow in partially obstructed veins or confirm the lack of flow in completely obstructed veins. In the normal patient, pulse-wave Doppler will usually demonstrate a triphasic flow in the hepatic veins due to the cardiac cycle (the heart is extremely close by) and respiration. With Budd-Chiari, you may see no flow, reversed flow, and even turbulent flow. You may also see a continuous monophasic flow, which represents the presence of partial IVC obstruction.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

003. Liver pathology

1. What is the result of *most* cases of hepatitis type A?

2. What hepatitis virus is responsible for *most* cases of acute hepatitis that result in liver failure?

3. What are some common symptoms of hepatitis?

4. What route will infections from distant inflammation sites take into the liver?

5. What is the bacterium *most* responsible for liver abscesses?

6. What is another name for echinococcus cyst?

7. What are the *most* common causes of fatty liver?

8. List some causes of cirrhosis.

9. What is the *most* common benign liver neoplasm?

10. What clinical symptom may suggest a tumor to be a hepatic adenoma?

11. If a hepatic adenoma ruptures, what may occur to the patient?

12. Liver lipomas are associated with what abnormalities in the kidney?

13. What is the *most* common *primary* malignant tumor of the liver?

14. What is the most useful laboratory test for hepatocellular carcinoma?

15. What is the *most* common cause of malignant neoplasms in the liver?

004. Considering general procedures for liver imaging

1. What is the most important thing a sonographer should do when reading a request for liver examination?

2. What should a sonographer do if laboratory values are not written on a request for liver examination?

3. What is the purpose of having the patient fast for a liver examination?

4. How does the sonographer position the patient to make liver evaluation easier?

5. What are some standard longitudinal views of the liver that a sonographer should obtain?

6. What are some standard transverse views of the liver that a sonographer should document?

005. Imaging infectious and metabolic liver disease

1. After scanning from anterior surface of the body, how should you adjust when attempting to distinguish a gas-producing abscess in the liver from bowel?

2. How should you measure an amebic abscess?

3. What does an undulating membrane within an echinococcus cyst represent?

4. What are some sonographic characteristics of schistosomiasis?

5. What is likely responsible for the sonographic appearance of granulomas in the liver?

6. Match the sonographic description of candidiasis in column B with each characteristic pattern in Column A. Each item in column B may be used only once.

Column A	***Column B***
___ 1. A round nodule, hypoechoic on the periphery and echogenic in the center surrounding a hypoechoic nidus.	a. "Wheel-within-a-wheel."
___ 2. Wheel-within-a-wheel, but without the hypoechoic nidus.	b. "Bull's eye" or target sign.
___ 3. Calcification that represents scar tissue.	c. Uniformly hypoechoic focus.
___ 4. Most common appearance, which represents fibrosis.	d. Echogenic focus.

7. What other conditions have similar sonographic appearances to fatty liver?

8. List and describe the three sonographic grades of fatty liver.

9. What is the echotexture of cirrhosis?

10. What is the sonographic relationship between cirrhosis and fatty liver?

11. What complication of glycogen storage disease can sonography detect?

006. Imaging focal abnormalities of the liver

1. Describe the sonographic appearance of a simple liver cyst.

2. What is the sonographic appearance of a hemorrhagic or infected liver cyst?

3. What is the sonographic appearance of polycystic liver disease?

4. What is the classic sonographic appearance of a cavernous hemangioma?

5. If present, what is the *most* effective way to demonstrate slow blood flow in a cavernous hemangioma?

6. Why is focal nodular hyperplasia difficult to detect?

7. What is the diameter of most hepatic adenomas?

8. How can you determine the difference between a lipoma and a typical cavernous hemangioma?

9. What are three basic sonographic patterns of HCC?

10. Match the sonographic appearance of metastases in column B with its source or association in column A. Items in column B may be used once.

Column A	*Column B*
___ 1. Associated with non–Hodgkin's lymphoma.	a. Hypoechoic.
___ 2. Frequently from breast carcinoma and lung carcinomas.	b. Hyperechoic.
___ 3. Primary site can be from the long bones.	c. Diffuse.
___ 4. Frequently from GI tract—particularly leiomyosarcoma.	d. Cystic.
___ 5. Any primary source.	e. Bulls-eye.
___ 6. Frequently from the gastrointestinal tract—most common is colon adenocarcinoma.	f. Calcified.

11. What is the sonographic appearance of a hepatoblastoma?

007. Imaging vascular abnormalities of the liver

1. What might normal vein size in a case of portal hypertension signify?

2. How do you evaluate the splenic and superior mesenteric veins?

3. What is the significance of collaterals developing in the gastroesophageal region?

4. What is the classic sonographic appearance of portal vein thrombosis?

5. If portal vein thrombosis is chronic, what confusing sonographic picture might you see?

6. In the patient with Budd-Chiari syndrome, what must you look for on sonography?

7. What type of blood flow will you see with Budd-Chiari syndrome?

Answers to Self-Test Questions

001

1. Lobar anatomy, segmental anatomy, and Couinaud's anatomy.
2. 15 cm.

3. Fissure for ligamentum venosum.
4. Divides the right lobe of the liver into anterior and posterior segments.
5. (1) c.
   (2) a.
   (3) b.
   (4) d.
6. A feature that distinguishes the portal vein from the hepatic vein on sonography.

002

1. Transports deoxygenated blood from the intestines, pancreas, and spleen to the liver.
2. The body uses bile to aid in food digestion.
3. Transports unconjugated bilirubin to the liver to be made into a form easily eliminated from the body.
4. The liver cells convert glucose into glycogen and store it.
5. (1) HDL: transports cholesterol from the body's cells to the liver to be excreted in bile.
   (2) LDL: transports cholesterol to locations other than the liver.
   (3) VLDL: transports fats from intestines and liver to muscle or adipose tissue.
6. Transforms it into urea.
7. Liver function tests help determine if liver disease is hepatocellular or cholestatic, acute or chronic, and mild or severe.
8. AST is present in areas throughout the body with high rates of metabolism, and damage to the cells of those areas will cause an elevation in blood serum.
9. Biliary obstruction.

003

1. Complete recovery.
2. Hepatitis B virus.
3. Jaundice, fever, nausea, weakness, and anorexia.
4. The main portal vein, common bile duct, and proper hepatic artery.
5. *Escherichia coli*.
6. Hydatid cyst.
7. Alcohol abuse, diabetes mellitus, obesity, and malnutrition.
8. Alcohol abuse; chronic hepatitis C; chronic hepatitis B; drugs; toxins; schistosomiasis; inherited diseases; chronic heart failure; blocked, inflamed, or destroyed bile ducts.
9. Cavernous hemangioma.
10. RUQ pain.
11. The patient may go into shock from hemorrhaging into the peritoneum.
12. Angiomyolipomas and tuberous sclerosis.
13. Hepatocellular carcinoma.
14. Alpha-fetoprotein.
15. Metastatic disease.

004

1. Ensure the patient scheduled or to be examined is the patient identified on the request.
2. Request liver function tests from the laboratory, the referring provider, or the patient.
3. Fasting helps reduce bowel gas that may interfere with the sonographic visualization of abdominal structures, and normally causes the gallbladder and common bile duct to retain enough bile for evaluation.
4. Have the patient lay flat on the back or in a slight left lateral decubitus position.
5. Left lobe demonstrating the fissure for ligamentum venosum; medial left lobe; main lobar fissure, right lobe demonstrating right hepatic vein and adjacent right kidney.

6. Left lobe demonstrating left portal vein branches and possibly the ligamentum teres; caudate lobe medial to the inferior vena cava; the main, right, and left hepatic veins draining into the IVC; right lobe demonstrating the right portal vein.

005

1. With the patient in the supine position, move the probe to the right side of the body, posteriorly, and aim up toward the abscess.
2. Measure in the AP, length, and width dimensions.
3. The detached endocyst.
4. Widened portal tracts up to 2 cm in diameter; thickened, echogenic walls; hepatomegaly or atrophy if portal hypertension is present; granulomas; thickened gallbladder wall.
5. Mycobacterial infection.
6. (1) a.
   (2) b.
   (3) d.
   (4) c.
7. Cirrhosis and chronic hepatitis.
8. (1) Grade 1: (mild), slight increase in echoes, diaphragm and vessels clearly seen.
   (2) Grade 2: (moderate), moderate increase in echoes, decreased ability to see right hemidiaphragm and vessel borders.
   (3) Grade 3: (severe), poor or no ability to see right hemidiaphragm and vessels. Right posterior portion or more than half of the liver usually cannot be seen.
9. Coarse, grainy, or heterogeneous.
10. Fatty liver can be present simultaneously with cirrhosis, causing the cirrhotic liver to appear with uncharacteristic attenuation.
11. Hepatic adenoma, hepatomegaly, and renal enlargement.

006

1. Anechoic, round, clearly defined thin walls, posterior acoustic enhancement, most less than 5 cm in diameter.
2. Fine, low-level echoes, septations, and thickened walls.
3. Identical to simple liver cysts of varying sizes.
4. Round, hyperechoic, homogeneous, clearly seen smooth borders.
5. By placing a power Doppler box over the tumor.
6. Most focal nodular hyperplasias are isoechoic with surrounding liver parenchyma.
7. 8 to 15 cm.
8. Examine the diaphragm directly behind or deep to the tumor for displacement or discontinuity that indicates the fat content of a lipoma.
9. (1) Solitary: usually massive with sharp borders.
   (2) Nodular: single or multiple, sharp outline, less than 5 cm in diameter.
   (3) Diffuse: nodules, poorly defined borders, spread throughout liver.
10. (1) a.
    (2) c.
    (3) f.
    (4) d.
    (5) e.
    (6) b.
11. Solitary, extremely large, hyperechoic with calcifications. With Doppler, the tumor is hypervascular.

007

1. The development of collaterals or varices.
2. Transverse plane near the confluence of the splenic vein and superior mesenteric vein. Scan along the splenic vein and look for the splenic vein diameter to double with deep inspiration. Scan along the longitudinal plain from the confluence and repeat deep inspiration to evaluate the SMV. Portal hypertension will cause both vessels' diameters to increase only slightly or remain unchanged.
3. The gastroesophageal collaterals may hemorrhage and lead to death.
4. Hypoechoic to echogenic material against the vessel wall or completely filling the entire portal vein.
5. Various vascular channels bundled together in the porta hepatis (cavernous transformation of the portal vein).
6. Any echogenic material within the major hepatic vessels; narrowing of vessel walls; tortuous branches off the hepatic veins traveling to the surface or to another hepatic vein; enlarged caudate lobe relative to right lobe.
7. Absent, reversed, turbulent, or monophasic flow.

Complete the unit review exercises before going to the next unit.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter. When you have completed all unit review exercises, transfer your answers to ECI (AFIADL) Form 34, Field Scoring Answer Sheet.

Do not return your answer sheet to AFIADL.

1. (001) The significance of the liver's external landmarks to sonographers is that they are used to
   a. place surgical markings.
   b. demonstrate the liver's surface.
   c. divide the liver into three major lobes.
   d. determine the liver's relationship to the abdominal cavity.

2. (001) The anatomic relationship between the left hepatic vein, the ascending left portal vein, and the ligamentum teres is that all three are
   a. contained within the main lobar fissure.
   b. contained in the left intrasegmental fissure.
   c. contained within the left intersegmental fissure.
   d. landmarks of the fissure for ligamentum venosum.

3. (001) Based on Couinaud's anatomy, what vessels are most useful for separating the liver into segments?
   a. Portal veins.
   b. Biliary ducts.
   c. Hepatic veins.
   d. Hepatic arteries.

4. (002) What does the liver produce to carry out its excretory function?
   a. Bile.
   b. Glycogen.
   c. Prothrombin.
   d. Aspartate aminotransferase.

5. (002) The purpose of the Kupffer cells found in the liver sinusoids is to
   a. manufacture glucose and glycogen.
   b. transport cholesterol throughout the body.
   c. ingest toxins, bacteria, and worn red blood cells.
   d. convert thromboplastin and calcium ions into fibrinogen.

6. (003) Which type of hepatitis will progress rapidly to hepatic failure?
   a. Hepatitis A virus.
   b. Hepatitis B virus.
   c. Hepatitis, type C.
   d. Hepatitis, non-A, non-B.

7. (003) What is the most common cause of cirrhosis in the United States?
   a. Alcohol abuse.
   b. Blocked ducts.
   c. Chronic hepatitis.
   d. Elevated bilirubin.

8. (003) What is the most useful laboratory test for hepatocellular carcinoma?
   a. Aspartate aminotransferase.
   b. Alanine aminotransferase.
   c. Alpha-fetoprotein.
   d. Prothrombin time.

9. (004) What is the first thing a sonographer should accomplish before performing a liver examination?
   a. Instruct the patient to fast 30 minutes prior to the examination.
   b. Read the request written by an authorized medical provider.
   c. Review the patient's previous computed tomography images.
   d. Bring in textbooks and discuss the case with the radiologist.

10. (005) How may a sonographer distinguish a pyogenic abscess from bowel during a liver exam?
    a. Instruct the patient to sit upright.
    b. Instruct the patient to drink water.
    c. Turn the patient into a left lateral decubitus position.
    d. Ask the patient to lie in a position of his or her choice.

11. (005) How does the sonographer image the portal veins to help diagnose schistosomiasis of the liver?
    a. Turn color Doppler on.
    b. Document portal vein.
    c. Measure the velocities of portal blood flow.
    d. Measure the anteroposterior (AP) diameter of the portal vein.

12. (006) The *best* way to ensure proper documentation of posterior acoustic enhancement of a simple liver cyst is to set
    a. the monitor contrast to a lower setting.
    b. the monitor contrast to a higher setting.
    c. time gain compensation (TGC) and overall gain to a lower setting.
    d. TGC and overall gain to a medium setting.

13. (006) What other organ should you investigate for cysts if you see multiple liver cysts?
    a. Kidney.
    b. Spleen.
    c. Pancreas.
    d. Gallbladder.

14. (006) When evaluating a cavernous hemangioma, power Doppler may be used to
    a. detect slow flow within the tumor.
    b. determine if the tumor is benign or malignant.
    c. distinguish leiomyosarcoma from a cavernous hemangioma.
    d. distinguish a focal nodular hyperplasia from a cavernous hemangioma.

15. (006) What is a proper method to distinguish hemangiomas from hyperechoic nodular hepatocellular carcinoma (HCC)?
    a. Use color Doppler.
    b. Use power Doppler.
    c. Use medium grayscale settings.
    d. Use time gain compensation (TGC) and overall gain.

16. (006) What is a characteristic of metastatic nodules associated with non-Hodgkin's lymphoma?
    a. Hypoechoic.
    b. Hyperechoic.
    c. Isoechoic.
    d. Anechoic.

17. (007) In cases of suspected portal vein thrombosis, what should you do as a precaution if you do *not* detect any flow using color Doppler?
    a. Increase time gain compensation.
    b. Increase color flow filters.
    c. Reduce color flow filters.
    d. Reduce overall gain.

Please read the unit menu for unit 2 and continue. ➔

Student Notes

Unit 2. Gallbladder and Biliary System

THE GALLBLADDER and its connection with the liver's biliary system require a slightly different approach then the one you use for liver sonography. In this unit, we will cover gallbladder and biliary system sonography.

2–1. Gallbladder and Biliary Anatomy and Physiology

There are some considerations you need to keep in mind when approaching the gallbladder and biliary system. In this section, we will briefly examine the anatomy and function of the gallbladder and biliary system you need to know to perform a proper sonographic examination. We will also cover general clinical conditions unique to the gallbladder and biliary physiology.

008. Gallbladder and biliary anatomy

The simple structure of the gallbladder is attached to a slightly more complex arrangement of biliary ducts. While normal variations exist from patient to patient, some quite bizarre, the following basic descriptions cover the majority of people.

The gallbladder is an oval or teardrop-shaped hollow organ attached to the gallbladder fossa or indentation of the liver. The gallbladder has a "top" portion called a fundus, which actually hangs down from the undersurface of the liver distally toward the feet in most patients. The largest portion of the gallbladder is called the body, which tapers down into a narrow tube called the neck. The neck has an inner layer made of mucosal folds called the *spiral valves of Heister*. Although not true valves, these folds sometimes appear on sonography and their spiraling effect can mimic stones in the neck. Otherwise, they are insignificant to sonographic imaging. The neck of the gallbladder is the closest portion to the porta hepatis.

The normal gallbladder size varies. The average range in a fasting adult patient is between 7 and 10 centimeters (cm) in length and approximately 3 to 4 cm in diameter. The normal gallbladder wall is usually no thicker than 3 millimeters (mm).

The neck of the gallbladder empties into a cystic duct, which is rarely seen on sonography. The cystic duct joins the common hepatic duct to form the common bile duct. Because sonography usually cannot detect the exact point where this junction occurs, it is useful to consider the entire duct from the porta hepatis to the head of the pancreas as the common duct. At the proximal end of the common duct, the right and left hepatics merge to form the common hepatic duct in the area of the porta hepatis. The tiny hepatic ducts inside the liver are considered intrahepatic and are third-vessel components of the portal triad. You should consider the portion of the biliary system outside of the liver to be extrahepatic where the diameter or caliber of the duct then becomes measurable on sonography. For example, the common hepatic duct, usually anterior to the portal vein, is normally seen as 4 mm or less in diameter. Keep in mind that the right and left hepatics will sometimes merge within the liver, a situation that would place the common hepatic duct anterior to the right portal vein branch.

In most adult patients, the upper limit of a normal common duct diameter is usually no greater than 7 mm. Sonographers use a rule-of-thumb, which generally has the duct diameter increase 1 mm every decade of life. In patients who have had their gallbladders removed (cholecystectomy), the normal diameter may reach up to 10 mm.

The distal common duct travels through a groove or inside the lateral portion of the pancreatic head. Thereafter, the duct continues a short distance until it terminates at the duodenum, where it normally joins with the pancreatic duct (duct of Wirsung). The juncture of common and pancreatic duct is called the ampulla of Vater, which is surrounded by a smooth muscle layer called the sphincter of Oddi (fig. 2–1).

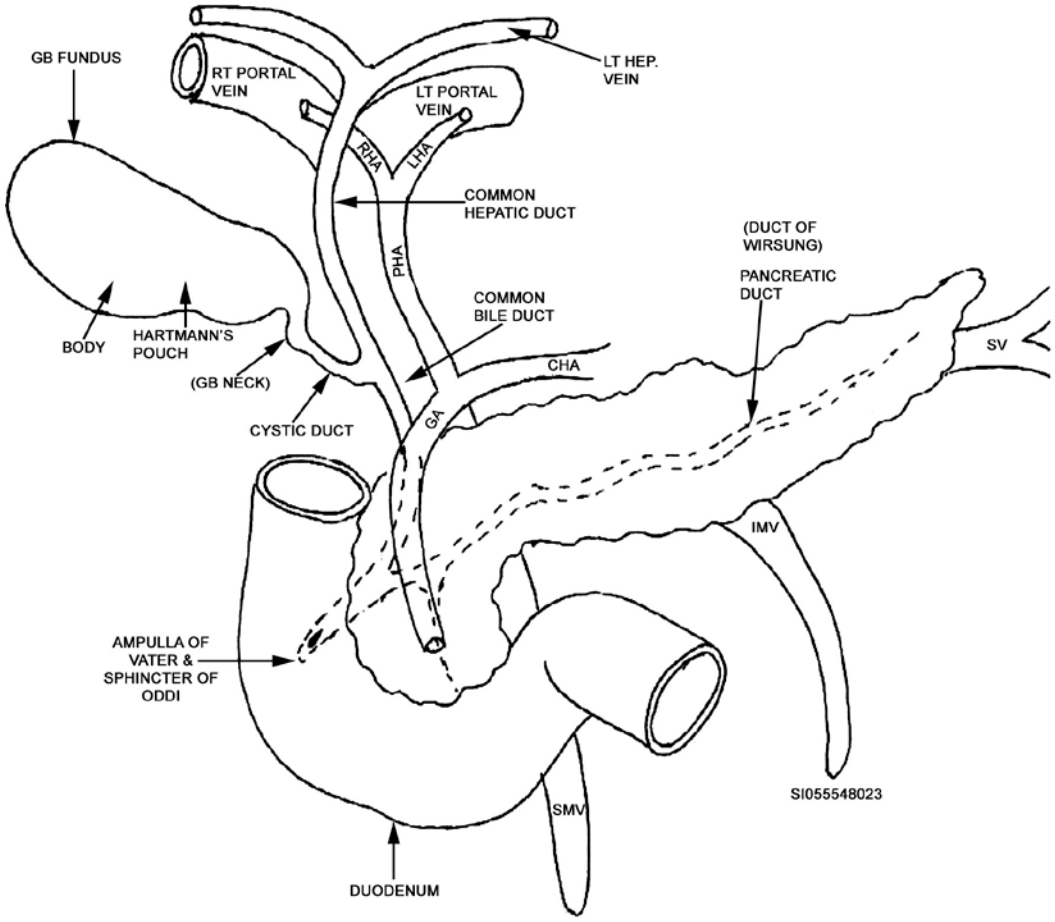

Figure 2–1. Biliary system.

009. Gallbladder and biliary function

The function of the biliary system is to transport bile from the liver to the duodenum and to regulate this flow. The intrahepatic bile ducts gather the bile and secrete it into the right and left bile ducts. The bile is then secreted through the common duct where one of two pathways is open to it. If a person is in a fasting state, the sphincter of Oddi at the distal end of the duct will remain closed, which prevents bile from flowing forward into the duodenum. Because the liver continues to produce bile, pressure will increase within the common bile duct. The only path for the bile to flow is into the lesser pressure of the cystic duct and on into the gallbladder.

It makes sense that eventually the gallbladder will fill to near bursting, and that is precisely what happens in obstructive abnormalities of the common bile duct. In the case of normal bile filling, the gallbladder's function is to store the bile for later release into the duodenum. It stores bile without

bursting at full capacity by concentrating the fluid. The inner lining of the gallbladder absorbs water, sodium, and chloride, which are transported away through the veins in the wall. What is left is concentrated bile made of bile salts, cholesterol, bilirubin, and lecithin. This process allows the gallbladder to store hours of bile filling without straining storage capacity.

A small amount of bile is released through contraction of the gallbladder walls when a person eats food. Conversely, most of the bile stored in the gallbladder will release when fatty foods are eaten. When food, particularly food containing fat, enters the duodenum, the inner lining of the duodenum (mucosa) releases a hormone called cholecystokinin (CCK) into the bloodstream. After traveling through the blood to the gallbladder tissue, CCK causes the walls to contract and to squeeze bile out through the cystic duct. The narrow cystic duct would overdistend and rupture from the pressure without the spiral folds (spiral valves of Heister) inside increasing the surface area. Simultaneously, CCK relaxes the sphincter of Oddi, which relieves pressure in the common bile duct, and allows the increased volume of bile to flow directly into the duodenum. This whole process can be interrupted by obstructive abnormalities in the biliary tract or the gallbladder.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

008. Gallbladder and biliary anatomy

1. What are the three main parts of the gallbladder?

2. Why is it considered useful to refer to the common bile duct as the common duct?

3. What measurement is the upper limit of normal in a common duct diameter?

4. What two ducts make up the ampulla of Vater?

009. Gallbladder and biliary function

1. What is the main purpose of the biliary system?

2. What causes the gallbladder to release its stored bile?

3. Cholecystokinin performs what two functions?

2–2. Imaging the Gallbladder and Biliary System

Our approach to the biliary system must be similar to the liver. We must keep a host of abnormalities and their clinical and sonographic appearances in mind if we are to image properly these structures. Because most of the process of sonographic imaging is a matter of recognition, your knowledge of abnormalities will help to ensure more accurate and effective examinations. As with the liver, gallbladder and biliary system pathology often displays dramatic changes that contrast with normal sonographic appearances. In this section, we will discuss pathology with general approaches to biliary sonography, placing particular emphasis on gallbladder abnormalities.

010. Gallbladder and biliary pathology

The normal function of the biliary system and its attached gallbladder is frequently interrupted by the obstruction of free-flowing bile. Unfortunately, obstruction, for whatever reason, can cause severe pain. However, not all abnormalities of the gallbladder or biliary system result from obstruction. A few occur that do not hinder normal function, some of which may turn out to be quite lethal. We will discuss only the most common.

General gallbladder pathology

There are a number of pathologies associated with the gallbladder. Frequently, the adjacent liver is also involved, as well as other body systems. Most gallbladder abnormalities share a few features that are not specifically present for any single abnormality; that is, the features are non-specific. We will briefly look at some of these non-specific features of the abnormalities that you may have to image.

Wall thickening and sludge

As stated above, the wall of the entire gallbladder should be no more than 3 mm thick in the normal fasting patient. A host of problems can cause the gallbladder wall to thicken, with the most common being acute or chronic cholecystitis, discussed below. Some others are ascites, congestive heart failure, hepatitis, pancreatitis, and renal failure. Wall thickening in one place (focal) may suggest abnormalities such as gallbladder carcinoma, metastatic nodules, complications from cholecystitis, or polyps.

Sludge can be thought of as particles that have solidified from bile, usually as a result of bile stuck in the gallbladder too long (biliary stasis). Biliary stasis is caused by obstruction of the common duct or cystic duct, prolonged fasting, excessive eating (hyperalimentation), and cholecystitis.

Cholelithiasis

Cholelithiasis is the presence of stones in the gallbladder. The condition can occur throughout a lifespan but usually appears after 20 years and more frequently with increasing age. Women are more likely to have gallstones than men are. Obesity, rapid weight loss, excessive eating (hyperalimentation), and family history are common risk factors for the condition.

Gallstones are crystals formed from some of the material composing bile. Most stones are mixtures of cholesterol, calcium bilirubinate, and calcium carbonate. As with sludge, which may be a precursor, gallstones are usually formed when bile has stayed in the gallbladder too long (biliary stasis).

Patients are usually asymptomatic. If there is pain, it is usually due to one or more stones blocking the cystic duct; thus, causing dilation of the gallbladder. With gallstones obstructing the cystic duct or blocking the neck of the gallbladder, a patient’s eating of fatty foods may cause intense pain. However, the pain from an obstructing stone can occur at any time and frequently awakens patients from sleep. The overdilated, or overdistended, gallbladder will cause pain in the right upper quadrant (RUQ) region, with pain sometimes transferring to the tip of the right scapula.

Along with pain, a patient suffering from obstruction due to cholelithiasis may have nausea and vomiting. The laboratory values most useful for suggesting painful cholelithiasis are elevations in total bilirubin and alkaline phosphatase (ALP). Other liver function tests, such as aspartate

aminotransferase (AST) and alanine aminotransferase (ALT) levels will be either normal or mildly increased.

Unless an obstructing stone passes out of a cystic duct, a patient may go on to suffer inflammation of the gallbladder (cholecystitis). Obstructing cholelithiasis, particularly gallbladders filled with stones, will usually prompt surgical removal of the gallbladder.

Cholecystitis

Cholecystitis is the inflammation of the gallbladder, both acute and chronic. The causes, appearance, and progression of cholecystitis are straightforward. However, if the inflammation becomes increasingly severe, complications can occur, such as:

- Emphysematous cholecystitis.
- Gangrenous cholecystitis.
- Empyema.
- Perforation.
- Pericholecystic abscess.

If the inflammation is chronic, other abnormalities may develop, such as milk of calcium bile and porcelain gallbladder.

Acute cholecystitis

Acute cholecystitis is the rapid development of gallbladder inflammation. Most cases, but not all, are due to obstruction of the cystic duct from either a stone or a tumor. Biliary tract infection can also cause cholecystitis.

Patients present clinically with RUQ pain, nausea, vomiting, and fever. Laboratory values reveal increased white cell count (leukocytosis) and, if obstructive, elevations in bilirubin. Elevations in AST, ALP, and ALT are common. If severe enough, the inflammation can progress into one or more of a group of complications. Generally, acute cholecystitis prompts surgical removal before complications develop. Some of the more common complications are briefly described below.

Emphysematous cholecystitis

If acute cholecystitis is severe enough, a complication may occur, such as emphysematous cholecystitis. This rare complication, predominant in males, is caused by gas-forming bacteria such as clostridium and *Escherichia coli*. The organisms invade the gallbladder wall and produce gas and necrosis in the tissue. The disease is considered fatal enough to require emergency surgery, as most cases are accompanied with gangrene and even gallbladder perforation.

Gangrenous cholecystitis

This is an infrequent complication that requires emergency surgical removal of the gallbladder. When acute cholecystitis continues for an extended period of time, the wall tissue, along with its blood and nerve supply, may start to die (necrosis), thus causing gangrene to set in. Bleeding, ulcerations, and tiny abscesses usually will cause an asymmetrically thickened wall. The inner lining of the gallbladder may also shed thin membranes that extend into the lumen. In a small percentage of patients with this complication, tissue and nerves within the gallbladder wall are broken down to a point that leads to eventual perforation.

Empyema of gallbladder

Occasionally, some patients suffer from acute cholecystitis so severe that pus develops in the gallbladder. This condition is known as empyema. The gallbladder itself enlarges as the entire organ is filled with pus.

Perforation and pericholecystic abscess

Perforation of the gallbladder is usually the result of gangrenous cholecystitis. The walls are broken down to the point where it will rupture and gangrenous bile will spill out into the peritoneum (acute perforation), causing general peritoneal inflammation. The gallbladder bed may collect the infected fluid and blood from the wall (subacute perforation). If left untreated the fluid will form into a pericholecystic abscess with a fibrous rind.

Chronic cholecystitis

Resolved acute cholecystitis may return repeatedly. Episodes of inflammation produce a few unique characteristics detectable by sonography. Patients will generally complain of temporary (transient) RUQ pain. This is largely due to stones blocking the cystic duct.

In some patients suffering from chronic cholecystitis, the bile may form sediment of concentrated calcium called *milk of calcium*. The condition is the result of gallstone obstruction of the cystic duct.

An uncommon condition of chronic cholecystitis that usually appears in women older than 50 is a calcified or *porcelain* gallbladder. Associated with stones, this disease represents calcification of either a part or the entire wall of the gallbladder. This disease has a high risk of developing into gallbladder carcinoma and thus will prompt surgical removal.

Focal gallbladder pathology

Focal wall thickening of the gallbladder, particularly if it projects into the lumen, strongly suggests the presence of a benign tumor. Because carcinoma and metastatic tumors of the gallbladder are rare, you will likely encounter either *polyps* or hypertrophic wall disease in the absence of cholecystitis. We will briefly discuss both.

Two types of polyps occur in the gallbladder: *cholesterol* and *adenomatous*, with the cholesterol variety being the most common. They can be single or multiple. Cholesterol polyps are not true neoplasms, being composed mostly of fat compounds (triglyceride) and cholesterol esters (compounds made of an alcohol molecule and certain acids) deposited within the wall, which can project into the lumen and give the sonographic appearance of a tumor. Conversely, adenomatous polyps are true neoplasms and quite rare. However, despite its rarity, the adenomatous polyp is the most common *benign* tumor of the gallbladder. Most other tumors are likely to be malignant.

Adenomyomatosis is not, technically, a neoplasm or tumor. Rather, it is the overgrowth or abnormal proliferation (hyperplasia) of the muscle cells that make up the gallbladder wall. The result is non-inflammatory wall thickening and the formation of spaces within the tissue called *Rokitansky-Aschoff sinuses*. The sinuses are filled with bile and cholesterol crystals. The hyperplasia may be spread throughout the gallbladder or can be focused in one area of the wall, usually the fundus. In particular, the focal variety projects into the lumen, resembling carcinoma on sonography. For that reason, and because there are no obvious symptoms or causes of the disease, the gallbladder is usually treated by removal.

Gallbladder carcinoma, specifically adenocarcinoma, is a rare neoplasm that occurs mostly in elderly women. Clinical symptoms associated with gallbladder carcinoma can occur with non-malignant diseases and, thus, are non-specific; some of these may be RUQ pain (rarely), diminished appetite (anorexia), and weight loss. The neoplasm usually—partially or completely— replaces the gallbladder, and typically will invade the nearby liver. Another form of the disease is a massless thickening of the gallbladder wall, either focal or spread throughout. The least likely form is a polyp-like mass larger than 5 to 10 mm protruding into the gallbladder lumen. Most gallbladder carcinomas also contain gallstones.

Biliary pathology

Most of the abnormalities that can appear within the gallbladder can also appear within the attached biliary system. The bile ducts of the liver (intrahepatics) and the common bile ducts all may contain

stones, sludge, and tumor. In addition, inflammation may spread into the biliary system. Briefly, we will cover the most typical biliary pathologies.

Choledocolithiasis

Choledocolithiasis is the presence of stones in the common bile duct. The condition, caused by gallstones, is responsible for most biliary tree obstruction. When the stones cause severe enough obstruction, complications may occur such as biliary cirrhosis, cholangitis, and pancreatitis. Complications of choledocolithiasis are usually the cause of the classic clinical symptoms of RUQ pain, fever, or jaundice. The laboratory values are elevated bilirubin and ALP.

Mirizzi syndrome

A rare abnormality, which you may, nevertheless, come across, is Mirizzi syndrome. It is the presence of a stone in the cystic duct that cause biliary obstruction in the common hepatic duct and, if prolonged, the intrahepatic ducts are also affected. Normally, a stone in the cystic duct, the narrow tube coming off the gallbladder, will cause only gallbladder pathology, with only slight dilation of the common bile duct. It is the same effect as gallbladder removal and the resulting dilation of the common bile duct in most patients. In Mirizzi syndrome, the common bile duct is at normal diameter while the common hepatic duct is dilated. What causes this is a stone in the cystic duct compressing the nearby common hepatic duct. This parallel course of both common hepatic duct (CHD) and cystic duct directly next to one another allows the CHD to be pinched off by the bulging shape of the cystic duct obstruction. The duct proximal to the compressed area is usually dilated.

Cholangitis

Inflammation of the bile duct wall, cholangitis, can also cause obstruction proximal to the area of infection. The culprit for cholangitis is largely due to bacterial infection (particularly ulcerative colitis—inflammation of the bowels) or wall irritation from stones. Common clinical symptoms are abdominal pain, jaundice, and fever. Along with obstruction causing typical elevations of bilirubin and ALP, there will be an elevated white blood cell count (leukocytosis). There are usually three types:

1. Bacterial.
2. Sclerosing (hardening).
3. AIDS related.

Bacterial cholangitis is usually secondary to infection from the colon. In sclerosing cholangitis, the infection is limited to the bile duct wall itself (primary), the cause of which is unknown. The biliary tree is diffusely inflamed as opposed to the more local inflammation of bacterial cholangitis. This condition is usually found in young males who suffer from fatigue, itching, hepatosplenomegaly, RUQ pain, and jaundice. If prolonged and untreated, the condition may lead to biliary cirrhosis, carcinoma, or hepatic failure.

Choledochal cysts

Another rare biliary abnormality that may appear in adults but is usually limited to children is a choledochal cyst. It is mostly a disease found in females. This is not a true cyst. Rather, it is the dilation of any portion of the biliary tree, mostly in the common bile duct. Patients will usually suffer from RUQ pain and jaundice. Clinicians may be able to detect an abdominal mass via physical examination.

Choledochal cyst, a congenital abnormality, is caused by pancreatic enzymes that have refluxed into the common bile duct and weakened the walls, which causes the dilatation. It may be associated with gallstones, pancreatitis, and cirrhosis. In adults, complications may arise such as infection and rupture of the dilatations, which may spill bile into the peritoneum, causing peritonitis.

Pneumobilia

Pneumobilia is air or gas in the biliary tree. Pneumobilia is usually caused by a malfunctioning sphincter of Oddi (it stays open); post-surgical procedures or gallbladder disease that connect the biliary tree to the gastrointestinal tract (biliary-enteric anastomosis or fistula, respectively); bile duct wall erosion by stone or ulceration.

Biliary atresia

Another congenital abnormality seen shortly after birth is biliary atresia. It is defined as the extreme narrowing or absence of the bile ducts and is extremely rare. It is associated with trisomies 17 and 18. The condition is caused by an infectious, vascular, or immunologic insult before birth. Unless a surgical procedure connecting any remnant of bile duct with the small bowel is performed, the disease is fatal.

Biliary neoplasms

Most intrahepatic biliary tumors are cystadenomas, which are both benign and rare. They will occasionally become malignant cystadenocarcinomas. Most biliary tumors are found in middle-aged women who will suffer from abdominal pain, palpable mass, or jaundice.

The *most* common malignant biliary neoplasm is a *cholangiocarcinoma*. This is a rare tumor that may appear anywhere in the biliary tree but is commonly seen with sonography where the right and left hepatic ducts join to form the common hepatic duct, the hilar, or *Klatskin* tumor. Mostly found in older adults, it has a high association with patients who have suffered from cholangitis and ulcerative colitis. Patients have the usual symptoms of pain and jaundice but may also suffer from weight loss and anorexia.

011. Considering general findings and procedures for gallbladder and biliary imaging

There are a few common procedures and findings that you must either perform or know how to image properly the gallbladder and biliary system. The more knowledge of a patient's condition and history that you know, the more effective will be your evaluation. Patient preparation and positioning are critical steps to taking standard images useful to radiologists.

Procedures and findings before the examination

Because the sonographic examination of the gallbladder and biliary system is normally performed as part of an examination of the entire abdomen, the preparation for the patient is identical to the procedures you learned about in the unit on the liver. However, apart from emergencies, if the focus or suspicion of an abnormality rests on the gallbladder and biliary system, fasting becomes more critical here than for organs such as liver and kidney. When the patient fasts for at least six or eight hours, the gallbladder should fill with enough bile so that its contents and wall can be seen with sonography. Fasting also clears bowel of gas, which often interferes with seeing the gallbladder fundus.

Most patients with RUQ pain, with or without jaundice, will be sent to you to rule out gallstones. Although there are other organs and diseases that can cause RUQ pain, the combination of laboratory analysis of blood samples and the clinical symptoms cause doctors to narrow upon the stones in the gallbladder. Even though most patients with gallstones are asymptomatic, most patients who have RUQ pain are usually suffering from an inflamed gallbladder, which is largely caused by stones. Be aware that a request may want you to rule out stones in the common duct and gallstones will not be mentioned. In addition, some patients will have RUQ pain even if they have had their gallbladders removed, placing you in a situation where you are looking for a gallbladder that does not exist. For this reason and because RUQ pain may come from another source, make sure to ask the patient if he or she has had his or her gallbladder removed (cholecystectomy).

Clues to the source of clinical symptoms can be clarified through liver function tests. For example, elevations in direct bilirubin and ALP will, together, strongly suggest an obstruction somewhere

along the biliary system. Other liver function tests (LFT) such as AST or ALT may be slightly elevated.

For patients who need an emergency sonographic examination of the gallbladder, there is not enough time to prepare. In these cases, remember that the gallbladder may be empty or near empty of bile and difficult to see. A collapsed gallbladder has a naturally thick wall, but it will usually be homogeneous without striations. If you see the collapsed gallbladder, you need to measure and document for abnormalities such as a significantly thickened gallbladder wall. Also, make sure to inform the radiologist of the patient's non-fasting state.

Process during examination

To examine a properly prepared patient suspected of gallbladder disease, you should attempt to answer certain standard questions even if not all of these questions are written on the request. Are there stones? Has the wall of the gallbladder thickened? Is there evidence of a *sonographic Murphy's sign*? Have the bile ducts dilated? Focusing on these basic questions will tailor your examination and make the most effective use of your time. Examine the gallbladder and bile ducts for other possible abnormalities after answering these questions, or if you noticed them during the basic scan.

You should use the highest frequency transducer housed within a probe that is shaped to optimally scan in between the rib cage (intercostals) or below the rib cage (subcostal). For most sonography sections, this means using a 3-megahertz (MHz) transducer for large patients or using a 5-MHz transducer for thin patients. It also usually means using a curvilinear or sector array probe. These instruments will optimize depth penetration of the sound beam without sacrificing resolution.

Position the patient either supine or in a slight left lateral decubitus. Have the patient raise his or her right arm to rest on the examination table over his or her head to allow easier access to the right upper quadrant. Sometimes it becomes necessary for you to have the patient sit or stand in an upright position. In extreme cases, you may have to place the patient in a prone position and scan through the right side. These positions are usually used to demonstrate the mobility of stones or the effect of gravity on sludge.

Standard views for a gallbladder and biliary sonogram should be obtained along with documentation of any other findings during the exam. Before you obtain images, locate and place the transducer directly over the gallbladder and press gently. Hold the pressure for a moment and then remove the transducer. If the patient expresses pain when you press or when you remove the transducer, this is known as a sonographic Murphy's sign. This sign, particularly in the presence of a thickened wall, usually confirms gallbladder disease. If the patient does not respond, have him or her point to the area with the most intense pain and document.

A landmark you can use to locate the gallbladder is the longitudinal view of the main lobar fissure. The cross section of the right portal vein will be at the cephalic end and the gallbladder will be at the caudal end. Another useful landmark is the transverse view of the right kidney, which displays the gallbladder anterior and medial to the kidney.

Most institutions require a longitudinal view of the entire length of the gallbladder (fig. 2–2). The gallbladder wall should be thin and smooth and the inside, or lumen, should be without echoes. Ensure that you sweep the ultrasound beam through the entire gallbladder. Because not every patient will be properly prepared, bowel gas from the duodenum may scatter sound over the fundus of the gallbladder. Have the patient drink water, which helps increase peristalsis and may clear your view of the gallbladder fundus. If a full-length image of the gallbladder is captured, some radiologists may require you to measure the length from the edge of the most proximal portion (where the neck vanishes) to the edge of the most distal (the fundus). Some institutions require sonographic measurement of the anterior wall in the longitudinal view. Some may require this measurement only in the presence of obvious wall thickening. Recall that the normal gallbladder wall thickness does not exceed 3 mm. As far as documentation is concerned, adapt yourself to your institution's protocol;

however, you should always measure the wall during your evaluation. Gallbladder wall thickness above 3 mm has an obvious appearance on a sonogram and once you see it, you should be able to remember its appearance. Also helpful to remember is that thickened gallbladder walls are usually hypoechoic or layered (striated) in the center, unlike the uniform echo of a smooth thin wall. Refer to figure 1–12, D in unit 1 for an image of an obvious gallbladder wall thickening.

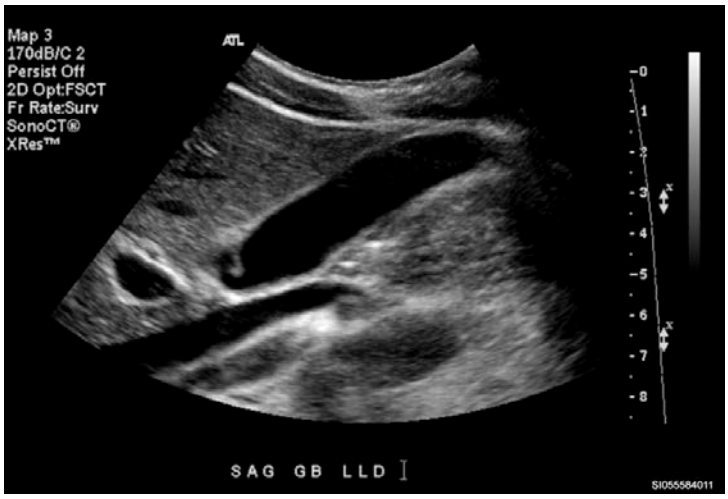

Figure 2–2. Gallbladder, longitudinal.

Another standard image of the gallbladder is the transverse view. With the length of the gallbladder on the screen, turn your probe 90 degrees to the long axis. Again, your institution's protocols will determine if you should document the anterior wall measurement of the gallbladder in this view (fig. 2–3). In addition, your protocol may have you document the diameter of the gallbladder. Most radiologists also find it helpful for you to document transverse views of the fundus, body and neck of the gallbladder.

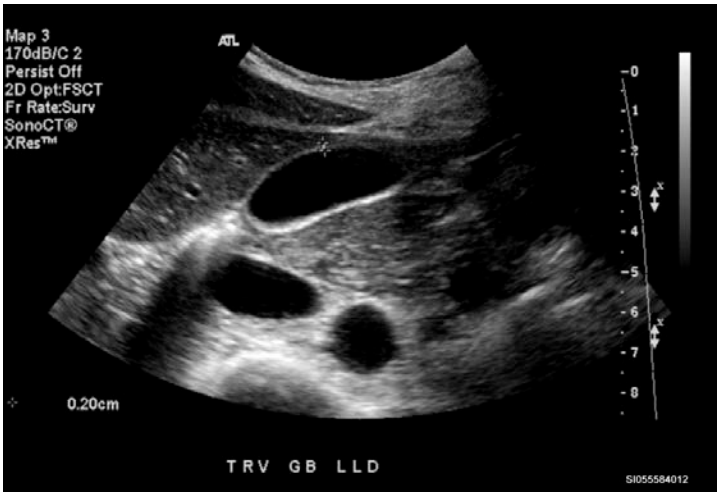

Figure 2–3. Gallbladder, transverse with measurement.

A final view that you should accomplish may aid the radiologist in confirming that there are no stones hidden within the neck of the gallbladder. After obtaining your standard images with the patient in a supine or left lateral decubitus position, you should either roll the patient into a complete left lateral decubitus (from the supine) or sit the patient up. These movements use gravity to cause hidden stones to drift or roll toward the fundus of the gallbladder. Label these positions on your image.

You normally do not see the intrahepatic bile ducts beyond the rarely visible right and left hepatic ducts located at the porta hepatis. If you see the ducts branching from the common duct into the liver,

consider those ducts dilated and document them (fig. 2–4). Place color Doppler over the dilated tubes to make sure they are ducts and not portal venous collaterals. Hepatic ducts should not show any color filling that represents flow.

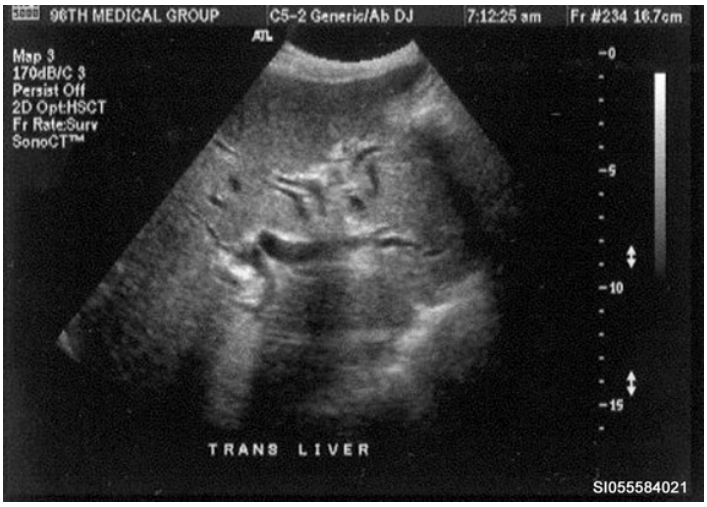

Figure 2–4. Intrahepatic dilatation.

Extrahepatic ducts are usually limited on sonography to imaging of the common duct just above the area where the hepatic artery crosses over the main portal vein (fig. 2–4). Locate the main portal vein as it enters the porta hepatis. Turn transverse and you should see the classic "Mickey Mouse" sign of the portal triad with the common bile duct (CBD) and the right hepatic artery situated anteriorly against the portal vein. Check the lower portion of the pancreatic head and locate the cross section of the distal common bile duct running through it. Measure and turn longitudinal to attempt to follow it both distal and proximal as far as possible.

012. Imaging the gallbladder

When performing a routine abdominal sonogram, you will be required to evaluate and document the condition of the gallbladder. When you see a gallbladder abnormality, it will be helpful for you to be familiar with the particular pathology that may be present. This is because a good deal of the sonographic appearances of gallbladder pathology overlaps. The following sonographic approaches and clues will help you to narrow down potential pathology to present to the radiologist and will increase the accuracy of your exam.

Wall thickening

Measure the anterior gallbladder wall using your department's protocol, in either the longitudinal or the transverse plane. You may hear from radiologists and see in the literature various reasons why one measurement is more accurate than the other. The *best* way to stay above the controversy is to follow the method of your radiologist or department protocols. Ensure that your beam is perpendicular to the gallbladder wall. Again, thickened walls are usually obvious due to the ability to see a hypoechoic zone or striated layers within the center of the wall. The size, easily obtained, of an abnormal wall will likely prove to be above 3 mm in diameter—longitudinal or transverse! Also, in cases of areas along the wall that are irregularly thickened, you should always measure these focal abnormalities in three dimensions: AP, height, and width. We will briefly look at these focal abnormalities below.

Sludge

Occasionally, you will see fine, homogeneous echoes layered in the dependent portion of the gallbladder. With patient movement, these echoes may slowly shift as if through oil. These are sludge echoes. Unlike stones, sludge does not shadow. To better confirm sludge for your radiologist, move your patient into another position, watching as the sludge moves slowly through the bile, and again

document the sludge's new location. You may see sludge collected into a ball within the gallbladder. If the collection slowly shifts with patient positioning but has no posterior shadowing, it is likely to be a sludge ball. Measure the sludge ball.

Always ensure that your overall gain is set so that the gallbladder lumen will *not* reflect echoes. This will prevent your mistaking sludge for artifact echoes within the gallbladder. Sometimes sludge can fill most of a gallbladder, and its echotexture may be isoechoic with the adjacent liver parenchyma. This situation will sometimes make it difficult to identify a gallbladder.

Cholelithiasis

The classic criteria for sonographic diagnosis of gallstones are structures of varying sizes and shapes seen within the gallbladder or cystic duct that are hyperechoic, mobile, and shadowing. You should always use the highest frequency transducer that will adequately penetrate to the level of the gallbladder. Because stones are hyperechoic, you may have to lower your gain settings. Gallstones are defined more sharply on sonography when you place your focal zone directly at or just below the level of the stones themselves. Stones are generally positioned wherever gravity is strongest within the gallbladder—usually the fundus or along the edge that faces bowel.

Occasionally there will be sludge accompanying stones. In this case, and when tiny stones bunch up in the neck of the gallbladder, it is a good idea to reposition the patient to get the stones to move, as described in the previous lesson. The repositioning maneuver will help determine if a shadow from the neck area of the gallbladder is a stone or shadows produced by a fold in the neck (junctional fold) or the spiral valves of Heister.

Be aware that unusual appearances of cholelithiasis may occur. You may not be able to see the gallbladder at all because it may be packed with stones, displaying a sharp-bordered, completely black shadow below an echogenic curving line where the gallbladder should be located. The curving line represents gallbladder wall, which either appears merged with the hyperechoic surfaces of the stones closest against the wall or appears against an anechoic sliver of bile between the stones and the wall. This appearance is called the double arc or the *wall-echo-sign* (WES).

Occasionally you may see gallstones floating. This is usually the result of bile mixed with a heavier substance such as oral contrast from a recent oral cholecystogram study. Rarely, floating stones will be the result of sludge.

Cholecystitis

Inflammation of the gallbladder causes pain. Because of this, most patients with this complaint will be referred by the physicians for right upper quadrant pain. Keep in mind that, while there are two types of cholecystitis, both require measurement of the wall and any stones or masses. Beyond this, various features of complications arising from acute cholecystitis present themselves with which you need to keep in mind as you scan.

Acute cholecystitis

Sonographically, the acutely inflamed gallbladder usually is accompanied with stones, but there are occasional occurrences of it without stones (acalculous cholecystitis) in some severely ill patients. The typical sonographic appearance of acute cholecystitis is a thickened gallbladder wall, stones, and sonographic Murphy's sign. Note that the wall may be hypoechoic and striated, as described in the previous lesson. You should acquire standard views of the gallbladder and measurements of wall and contents.

Emphysematous cholecystitis

On sonography, you will see emphysematous cholecystitis appear as a hyperechoic focus seemingly unaffected by gravity (nondependent), casting a dirty shadow. The focus usually has a ring down or comet-tail artifact due to the presence of gas. The entire wall may be affected and will display a curved hyperechoic line similar to the double-arc or WES appearance. You should be able to

distinguish this abnormality from gallstones because gallstones cast complete shadows. The dirty shadowing of emphysematous cholecystitis is further contrasted with the lack of shadowing found with adenomyomatosis or cholesterol polyps (which also produce ring-down artifacts).

Gangrenous cholecystitis

When the gallbladder wall in acute cholecystitis appears asymmetrically thickened, accompanied with wall striations and fluid around the gallbladder, you should suspect gangrenous cholecystitis. Most cases of gangrenous cholecystitis will not present with a sonographic Murphy's sign because the infection destroys the nerves in the wall. Along with the irregular wall appearance, even for acute cholecystitis, you may see membranes stretching within the gallbladder. These membranes are the separated inner linings or mucosa of the gallbladder.

If you see fluid around a gallbladder with an asymmetric wall, consider perforation and scan in the space between the right kidney and the right posterior lobe of the liver (Morrison's pouch) for fluid. Instead of RUQ pain, the patient may complain of general abdominal pain due to the resulting inflammation of the peritoneum.

Empyema

When you perform sonography in suspected cases of empyema, you will see what appears to be sludge but without the characteristic layering inside a distended gallbladder. Moving the patient will usually cause either a slight shift or no shift in echoes, as the bile has become thickened with pus..

Perforation and pericholecystic abscess

On sonography, you will see evidence of gallbladder perforation by the presence of a pericholecystic abscess, an anechoic to complex fluid collection against a segment of gallbladder. You may see the gallbladder completely surrounded with fluid. If possible measure the length, width, and height of an abscess; or, if the abscess surrounds the gallbladder, measure the width from the wall out.

Chronic cholecystitis

In the absence of a sonographic Murphy's sign and distension of the gallbladder, the presence of stones alone can suggest chronic cholecystitis. However, other abnormal features can be seen through sonography.

For example, you will note the sediment found in milk of calcium bile is usually echogenic, but can resemble the low-level echoes of sludge. To help you distinguish this condition from sludge, remember that sludge does not shadow and milk of calcium has a distinct anechoic shadow.

For suspected porcelain gallbladder, be sure to note that the gallbladder is replaced by an anechoic shadow with sharp borders. The differential diagnosis is the wall-echo-sign. The absence of a double hyperechoic arc will help you to distinguish the porcelain gallbladder from a lumen filled with stones. Be sure to document this clearly for the radiologist.

Focal gallbladder abnormalities

Polyps are commonly seen along the wall of the gallbladder during sonography. Although sonography cannot distinguish between the two types, the one you are likely to see will be the cholesterol polyp. They tend to stick out into the lumen and have the same echogenicity as the wall. They do not move or shadow. Most polyps you see will measure around a few millimeters in diameter. Because polyps that grow more than 5 to 10 millimeters are associated with increased risk for malignancy, you should measure the diameter of any polyp for increased size comparison with potential future studies.

Adenomyomatosis is another focal abnormality that can be difficult to distinguish from the usual wall thickening. If large enough, the *Rokitansky-Aschoff sinuses* can be seen as anechoic spaces in the wall, which resemble necrotic areas seen in some complications of acute cholecystitis. Usually, however, most cases of adenomyomatosis will display hyperechoic spots in the wall, believed to be

cholesterol crystals filling the spaces, which cause comet-tail artifacts to appear. These ultrasound artifacts are usually seen in the gallbladder wall nearest the transducer. Focal areas of wall thickening projecting into the lumen tend to resemble carcinoma.

Apart from benign polyps, rarely will you encounter other focal abnormalities of the gallbladder wall. If you do, the mass will likely be a case of carcinoma of the gallbladder. Except for similarities in appearance with complications that come from severe cases of acute cholecystitis, the appearance of carcinoma of the gallbladder on sonograms will usually suggest a non-inflammatory source. You may also see the tumor invade the adjacent liver or wall calcification. Similar in sonographic appearance to gangrenous cholecystitis, carcinoma of the gallbladder is usually accompanied by gallstones, thickened and significantly irregular gallbladder wall, and heterogeneous echoes that nearly fill or entirely replace the gallbladder. Other appearances may be one or more tumors with heterogeneous echoes larger than 2 cm projecting into the gallbladder lumen.

As with any neoplasm in the body, the tumor should be measured. It may also be helpful to reposition the patient (upright or decubitus) to *prove* that the mass is *not* empyema or sludge trapped in thickened bile, both of which may then show extremely slow moving, heterogeneous echoes in the lumen.

013. Imaging the biliary system

You should scan with a little more care when evaluating the biliary tree. Normally, intrahepatic ducts can only be seen in the presence of abnormalities, specifically pathology that causes the biliary ducts to dilate. Otherwise, evaluation of ducts is usually sonographically limited to the extrahepatic ducts. Many of the complications that occur with gallbladder pathology are also seen in abnormalities of the biliary tree.

Choledocolithiasis

For sonographic imaging of choledocolithiasis, you will have to place the ultrasound beam along the length of the common duct from porta hepatis to as far distal as possible. If the common duct is obstructed, you should easily see it because of dilatation (fig. 2–5). The gallbladder will also probably be distended. You will rarely see stones that cause obstruction in the portion of the duct above the level of the pancreas, even more rarely in the common hepatic duct. Most stones seen will be in the intrapancreatic region of the common duct. This area is thought of as being the distal common duct. If there are stones in the proximal common duct, they will usually cause dilated intrahepatic ducts while keeping the biliary tree distal to the blockage at normal diameter.

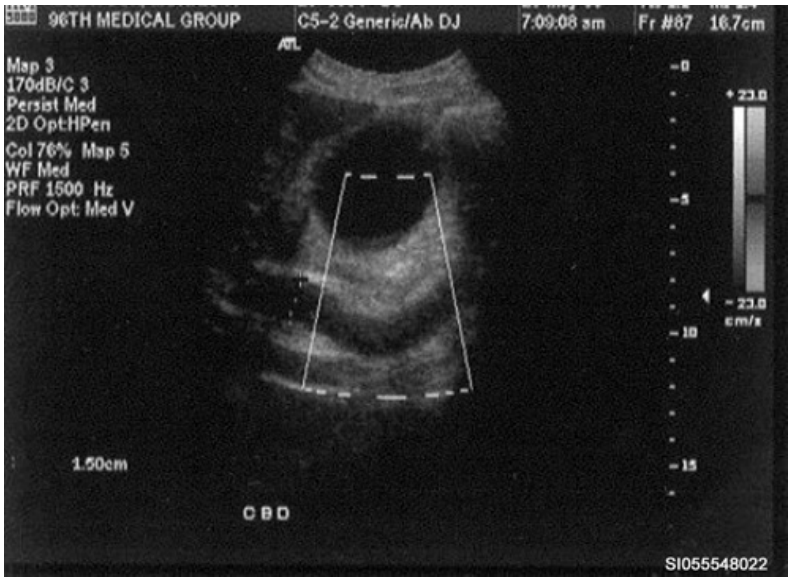

Figure 2–5. Common duct dilatation.

Scanning of the distal common duct can be difficult due to bowel gas overshadowing the pancreatic head region. Shifting of patient position and ingestion of water may clear the duodenum of gas and permit you to see the duct passing through the head of the pancreas. The easiest view for observing the duct at the pancreas is the transverse view, which will show a cross section of the common duct passing through the lateral portion of the pancreatic head. If you notice a hyperechoic focus with sharp distal shadowing, this will probably be a stone. Measure the stone in three dimensions.

As with any abnormality, you should measure any other stones seen in the extrahepatic ducts. This is not always easy because some of the stones can be quite small, giving off subtle acoustic shadowing, particularly with bowel gas surrounding the duct. Be suspicious if you do not see the common duct running distal beyond the porta hepatis. Look for either an abrupt termination of duct or the common duct filled with stones.

Mirizzi syndrome

Mirizzi syndrome may be difficult to clarify by sonography, and the sonographer may see only a large stone in the neck of the gallbladder. Be suspicious of Mirizzi syndrome if you simultaneously see a large stone in the neck of the gallbladder and dilatation of the common duct proximal and adjacent to that level.

Cholangitis

In bacterial cholangitis, you will see significant biliary dilation, wall thickening, and echogenic material within the bile ducts. The echogenic material may represent pus in the bile. The dilatation is proximal to and the result of severe wall thickening, which may narrow the duct to the point of obstruction. Bile stuck in the duct (stasis) may cause additional inflammation of the walls.

As with bacterial cholangitis, the duct walls in sclerosing cholangitis will be unusually thickened, but you may not see dilation if thickening is spread throughout the biliary tree. Some patients will also have gallbladder wall thickening and stones.

In AIDS patients, cholangitis appears sonographically identical to the primary sclerosing type. There are areas along the biliary tree with focal dilation and the gallbladder wall may be thickened. What separates this type from sclerosing cholangitis is the presence of a hypoechoic nodule in the distal common duct, which may represent papillary stenosis or a tumor of the ampulla of Vater.

Measure inflamed bile ducts the same way you would measure normal ducts; AP measurements of the inside walls of the lumen.

Choledochal cysts

During sonography of choledochal cysts, the dilation found within the abnormal portion of duct is so large relative to the normal duct diameter that it resembles a cyst. The “cyst” causes the bile ducts proximal to it to dilate.

You should note and document the appearance, which is often separated into three types:

1. Type I: dilatation of the common bile duct; either cystic (Ia), focal (Ib), or fusiform, which means the entire CBD length (Ic).
2. Type II: diverticulum branching off the common bile duct—extremely rare.
3. Type III: herniation of common bile duct into the lumen of the duodenum at the ampulla of Vater—also extremely rare.

Choledochal cysts resemble hepatic cysts, so be careful. You should see a large cystic structure separate from the gallbladder. Demonstrate both on the same image, if possible. In this way, you will be able to confirm that the choledochal cyst is not the gallbladder. You may also see sludge in the cystic type (Ia) but be careful to scan through the entire dilation. Choledochal cysts also have a high incidence of associated cholangiocarcinoma. In this disease, measure the widest AP diameter of the duct.

Pneumobilia

Occasionally, the biliary tree will show a confusing picture of pneumobilia on sonography. You will see echogenic foci of varying lengths inside the bile ducts. Unlike stones, which have sharp distal shadows, these foci will have dirty shadowing with reverberation (ring-down, or comet-tail) artifact. These unusual appearances represent air or gas in the biliary tree.

Biliary atresia

The picture on sonography will usually be quite confusing. There will be a lack of a visible extrahepatic duct from the porta hepatis to the duodenum. There will also be no intrahepatic evidence of ducts. If there are remnants of ducts in the liver, they will likely resemble hepatic cysts. Some infants with this disease may have a confusing general abdominal appearance with a left liver lobe identical in size and shape to the right, situs inversus (stomach under the right lobe of the liver), multiple spleens (polysplenia), and an interrupted inferior vena. A search for biliary ducts should be made if an infant has any of the above sonographic appearances.

Make sure to use color Doppler over any anechoic tubular shapes within the liver, which might *appear* to be ducts. This will display blood flow if there are blood vessels. Remember to increase your Doppler sensitivity and reduce your Doppler gain.

Biliary neoplasms

Tumors of the biliary tree, when they do occur, are usually seen between the porta hepatis and the region of the pancreas; that is, the common duct. You will see cystic lesions with multiple septations and growths projecting from the walls (papillary excrescences). This appearance can be seen in hemorrhagic or infected cysts, echinococcus cysts, and hematomas. In fact, the sonographic appearances of intrahepatic biliary tumors cannot be distinguished from the sonographic appearances of liver cell (parenchymal) tumors, abscesses, or metastases.

Also, cholangiocarcinoma at the hilum (Klatskin tumor) can be easily overlooked on sonography. The tumor forming at the proximal end of the CHD in the hilum can be quite small and still fill the entire diameter of the duct. Because the tumors are usually isoechoic to the surrounding liver hilum, the only landmark that may be visible to you will be the dilations of the right and left hepatic ducts. These two ducts will be best seen and demonstrated on the transverse view of the porta hepatis, near where you would normally visualize the right and left branching of the main portal vein. Simultaneously, the extrahepatic biliary system will appear normal.

Other locations for the tumor can be difficult or impossible to discern on sonograms. Large cholangiocarcinomas in the intrahepatics cannot usually be distinguished from other tumors and small tumors that fill the tiny intrahepatic ducts will not usually be seen at all. Cholangiocarcinomas in the distal common duct will appear as an abrupt interruption of the course of the bile duct. Care is needed here as sludge and cholangitis can mimic a distal cholangiocarcinoma.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

010. Gallbladder and biliary pathology

1. List causes for gallbladder wall thickening.

2. Match the complications of acute cholecystitis in column B with their most likely description in column A. Items in column B may be used once.

Column A	*Column B*
____1. Pus in the gallbladder.	a. Gangrenous cholecystitis.
____2. Gallbladder wall ruptures.	b. Perforation.
____3. Fluid against gallbladder segment.	c. Empyema.
____4. Wall necrosis.	d. Pericholecystic abscess.
____5. Gas in the gallbladder wall.	e. Emphysematous cholecystitis.

3. What are the three types of cholangitis?

011. Considering general findings and procedures for gallbladder and biliary imaging

1. Why is a physician likely to request that you rule out gallstones in a patient with right upper quadrant pain?

2. What is the relationship between a patient's fasting state and the gallbladder wall?

3. What are some standard questions you should answer to examine properly a patient suspected of gallbladder disease?

4. Why is the transverse view of the right kidney useful as landmark to locate the gallbladder?

5. List the standard views for a gallbladder sonogram.

012. Imaging the gallbladder

1. What can you do to confirm the presence of sludge for the radiologist?

2. How are gallstones more sharply defined on sonography?

3. Describe the gallbladder's appearance in porcelain gallbladder.

4. Why might adenomyomatosis resemble gallbladder carcinoma?

5. Describe the sonographic appearance of gallbladder carcinoma.

013. Imaging the biliary system

1. What should make the common duct easier to see when it is obstructed by stones?

2. What view will allow easier visibility of the distal common duct?

3. What abnormality should you suspect if you see a large stone in the neck of the gallbladder and a dilated common duct, proximal to the level of the stone?

4. Describe the sonographic appearance of the three types of choledochal cysts.

5. List associated abnormalities that may be sonographically seen with biliary atresia.

6. How can you obtain sonographic images of cholangiocarcinoma located in the intrahepatic ducts?

Answers to Self-Test Questions

008

1. Fundus, body, and neck.
2. The neck of the gallbladder empties into a cystic duct, which is rarely seen on sonography. The cystic duct joins the common hepatic duct to form the common bile duct. Because sonography usually cannot detect the exact point where this junction occurs, it is useful to consider the entire duct from the porta hepatis to the head of the pancreas as the common duct.
3. 7 mm.
4. The pancreatic duct and the common bile duct.

009

1. To transport bile from the liver to the duodenum and to regulate this flow.
2. Cholecystokinin.
3. CCK causes the walls to contract and to squeeze bile out through the cystic duct. It also relaxes the sphincter of Oddi, which relieves pressure in the common bile duct, and allows the increased volume of bile to flow directly into the duodenum.

010

1. Acute or chronic cholecystitis, ascites, congestive heart failure, hepatitis, pancreatitis, and renal failure.
2. (1) c.

(2) b.
(3) d.
(4) a.
(5) e.

3. Bacterial, sclerosing, AIDS-related.

011

1. The combination of laboratory analysis of blood samples and the clinical symptoms cause doctors to narrow upon the stones in the gallbladder.
2. A collapsed gallbladder has a naturally thickened wall.
3. Are there stones? Is the wall of the gallbladder thickened? Is there evidence of a *sonographic Murphy's sign*? Are the bile ducts dilated?
4. The gallbladder will be directly anterior and medial to the right kidney.
5. Longitudinal, longitudinal with measurements, gallbladder wall measurement according to department protocols, transverse views of the fundus, body, neck, possible measurement of the transverse body.

012

1. Move your patient into another position, watching as the sludge moves slowly through the bile, and again document the sludge's new location.
2. When you place your focal zone directly at or just below the level of the stones themselves.
3. The gallbladder is replaced with an anechoic shadow with sharp borders.
4. Adenomyomatosis tends to form in the wall of the fundus and project into the lumen, which may resemble carcinoma.
5. Accompanied by gallstones, thickened and significantly irregular gallbladder wall, heterogeneous echoes that nearly fill or entirely replace the gallbladder, one or more tumors with heterogeneous echoes larger than 2 cm projecting into the lumen.

013

1. Dilatation.
2. Transverse view of the pancreas.
3. Mirizzi syndrome.
4. Type I: dilatation of the CBD; Type II: diverticulum of the CBD; Type III: herniation of CBD into duodenum through ampulla of Vater.
5. Liver lobes identical in size, situs inversus, polysplenia, and interrupted inferior vena cava.
6. Large cholangiocarcinomas in the intrahepatics cannot usually be distinguished from other tumors; and small tumors that fill the tiny intrahepatic ducts will not usually be seen at all.

Complete the unit review exercises before going to the next unit.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter.

18. (008) Which thickness is most likely in a normal gallbladder wall?
    a. 2 mm.
    b. 4 mm.
    c. 6 mm.
    d. 8 mm.

19. (008) What is the upper limit of normal in the diameter of the common duct?
    a. 5 mm.
    b. 6 mm.
    c. 7 mm.
    d. 8 mm.

20. (009) The gallbladder keeps from bursting when filled to capacity by
    a. concentrating bile.
    b. concentrating water.
    c. mixing bile with water.
    d. concentrating cholecystokinin (CCK).

21. (009) Aside from causing gallbladder wall contraction, cholecystokinin (CCK) also relaxes the
    a. cystic duct.
    b. sphincter of Oddi.
    c. intrahepatic ducts.
    d. the portal vein walls.

22. (010) What complication of acute cholecystitis is likely to result in bile spilling out into the peritoneum?
    a. Gangrenous cholecystitis.
    b. Gallbladder collapse.
    c. Cholilithiasis.
    d. Empyema.

23. (010) What gallbladder abnormality involves cholesterol crystals filling Rokitansky-Aschoff sinuses?
    a. Chronic cholecystitis.
    b. Adenomyomatosis.
    c. Cholesterol polyp.
    d. Milk of calcium.

24. (011) What is the purpose for asking patients if they have had their gallbladder removed?
    a. To determine if the patient has properly fasted.
    b. To determine if the patient is able to withstand the Murphy’s sign.
    c. Some patients will mistake a collapsed gallbladder for a removed gallbladder.
    d. Right upper quadrant (RUQ) pain may come from a source other than the gallbladder.

25. (011) How should you confirm gallbladder disease in the presence of a thickened gallbladder wall?
    a. Test for a sonographic Murphy's sign.
    b. Obtain images of a cirrhotic liver.
    c. Obtain laboratory values alone.
    d. Measure the gallbladder wall.

26. (012) What is the *best* view to use for accurately measuring the wall of the gallbladder?
    a. The transverse view.
    b. The longitudinal view.
    c. The view according to your department's protocols.
    d. Only measure when the wall is obviously thickened.

27. (012) Of the following situations, what is the likeliest reason for *not* being able to see a gallbladder on sonography?
    a. Wall-echo-sign.
    b. Choledochal cysts.
    c. Pericholicystic fluid.
    d. Obstruction of the cystic duct.

28. (012) A hyperechoic, non-dependent focus in the gallbladder that casts a dirty shadow likely represents a complication of what condition?
    a. Emphysematous cholecystitis.
    b. Gangrenous cholecystitis.
    c. Chronic cholecystitis.
    d. Acute cholecystitis.

29. (012) When examining patients with chronic cholecystitis, the usefulness in remembering that sludge does *not* shadow is to distinguish
    a. sludge from milk of calcium bile.
    b. sludge from shadowing gallstones.
    c. sludge from emphysematous cholecystitis.
    d. chronic cholecystitis from acute cholecystitis.

30. (012) What should you do if the gallbladder is filled with heterogeneous echoes?
    a. Put pressure directly above the gallbladder and document.
    b. Reposition the patient into a decubitus position.
    c. Examine the portal vein for reflux.
    d. Have the patient drink water.

31. (013) Measuring a stone in the extrahepatic duct is sometimes difficult because the
    a. stones are hidden in the cystic duct.
    b. duct is surrounded by bowel gas.
    c. duct is isoechoic with stones.
    d. stones are masked by sludge.

32. (013) What two features are you likely to encounter in a patient suffering from cholangitis?
    a. Gallstones and sludge in the gallbladder.
    b. Biliary duct wall thickening and dilatation.
    c. Dilated portal veins and gallbladder wall thickening.
    d. Tumor in the ampulla of Vater and the wall-echo-sign (WES).

33. (013) The *best* way to confirm the presence of choledochal cyst is to obtain an image
    a. with the gallbladder and choledochal cyst on one image.
    b. that demonstrates the choledochal cyst and pancreas.
    c. with choledochal cyst wall measurement.
    d. of a measured Type II choledochal cyst.

34. (013) What is a useful way to demonstrate the presence of a Klatskin tumor?
    a. Longitudinal view of the distal common duct.
    b. Longitudinal view of the gallbladder.
    c. Transverse view of the porta hepatis.
    d. Transverse view of the pancreas.

Please read the unit menu for unit 3 and continue. ➔

Unit 3. Urinary System

BEYOND IMPORTANT organs like the liver and gallbladder, organs of the urinary system play an equally critical role in the maintenance of life. In this unit, we will discuss the sonographer's approach to this system. We will look at an overview of kidney and bladder physiology followed by sonographic imaging considerations for the various abnormalities of the system.

3–1. Urinary System Anatomy and Physiology

As with any organ system in the body, a sonographer's familiarity with the basic structure and function of an organ will ensure a more efficient approach to obtaining sonographic images. The urinary system is no exception. In this section, we will briefly give an overview of the anatomy and function of the urinary system.

014. Urinary system anatomy

The urinary system is composed of a closed set of organs placed in the body's retroperitoneum, separate from the abdominal organs contained within the peritoneum. The organs of the system are two kidneys, two ureters, and one bladder. Both kidneys are normally located to either side of the spine against the back muscles at the level between the 12th thoracic vertebra and the fourth lumbar vertebra. Usually the right kidney is slightly lower than the left due to the presence of the liver on the right superiorly and laterally. The ureters travel from both kidneys inferiorly in the retroperitoneal space against the anterior surface of the psoas muscles before diving into the pelvis. Once in the pelvis, the ureters descend to the posterior portion of the bladder.

The kidney is a small oval or bean shaped organ with a slit in its medial, abdomen-facing surface, called the *hilum*. Through the hilum enters the renal artery, which comes directly from the aorta. Also, the cuplike, upper end of the ureter enters the hilum, where it is called the renal pelvis. Renal veins course through the hilum for blood to exit. Lymph vessels and nerves also enter at the hilum. The adult kidney's dimensions are generally 9 to 12 centimeter (cm) in length, 2 to 4 cm in height or thickness, and 4 to 6 cm in width. These sizes have various ranges based on height, sex, and age; and thus, may be smaller in females and older adults, while taking up a relatively larger space in the body of an infant.

The tissue of the kidneys, or its parenchyma, is made of primarily two zones: the center that surrounds the hilum called the renal sinus and the outer cortex, which surrounds the renal sinus. The cortex is made of the beginning portions of the kidney's functional tissue called nephrons. The border between the cortex and the sinus, called the *medulla*, contains the rest of the nephron units. The nephrons are where the blood vessels interface with tubules that filter blood and transfer wastes into urine. This medulla is composed of eight to 18 folds shaped into individual triangular structures

called *medullary pyramids*, with the base of the pyramid facing the surface of the kidney and the apex or point facing the sinus. The pyramids stick out into hollow tubules called *minor calyces*, which are collectively attached to two or three *major calyces*. The widening portions of the major calyces, or *infundibula*, are connected to the renal pelvis at the hilum. The calyces, part of the renal sinus, are surrounded by fat (fig. 3–1).

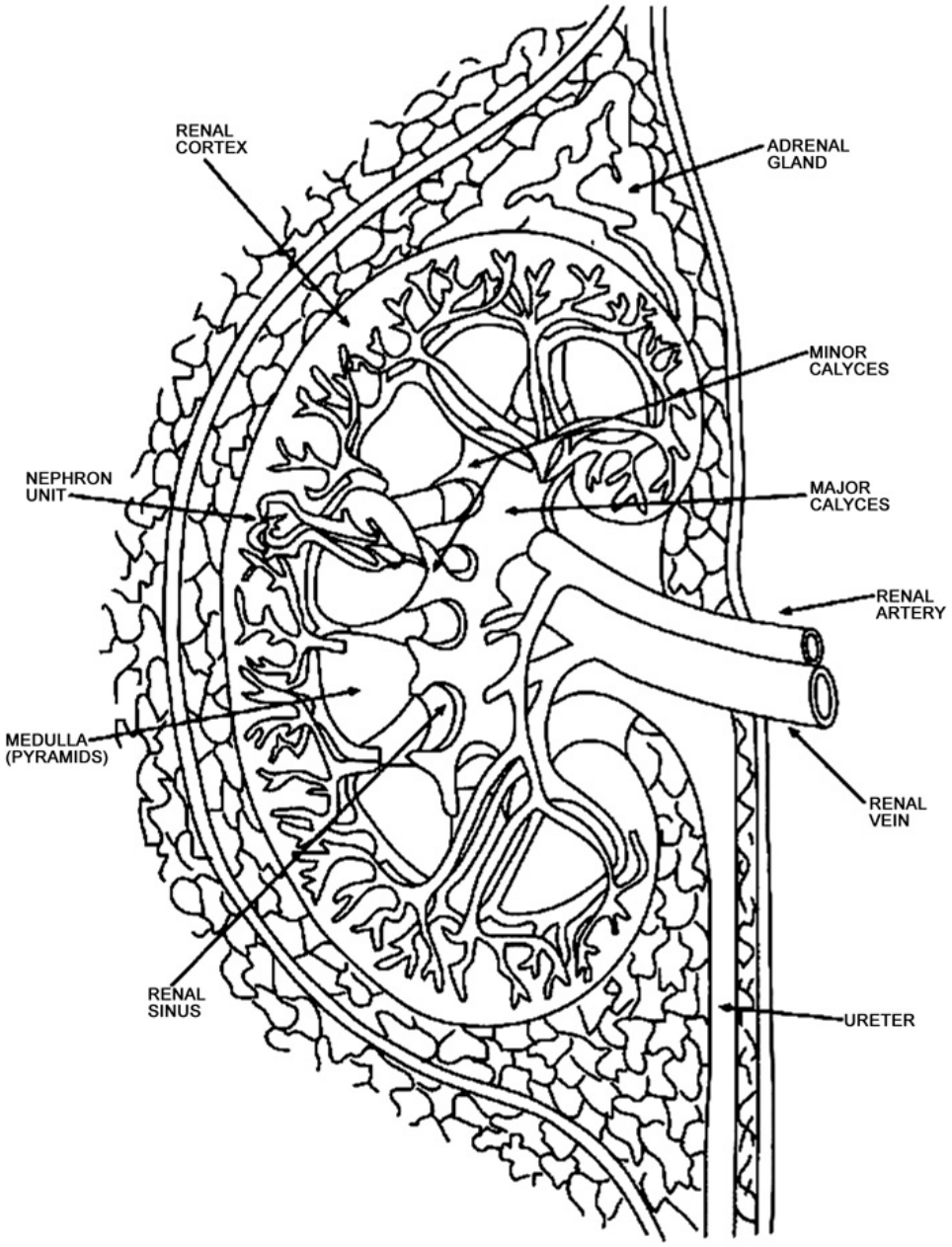

Figure 3–1. Kidney.

Throughout the kidneys are branches of arterial and venous vessels. We will discuss the structure and flow of the kidneys' blood vessels in the vascular volume.

Fat is present outside the kidney (perirenal) but loosely attached to the kidney's tough fibrous capsule. A small organ called the adrenal gland is located at the superior pole of the kidney, measuring 4 to 6 cm in length but 2 to 3 cm in width. The adrenal gland is a thin organ extremely difficult to see with sonography. Both perirenal fat and adrenal gland are surrounded by connective tissue called Gerota's fascia, which anchors the slightly mobile kidney in its place in the

retroperitoneum. Pararenal fat, which allows for movement of the organ in the body during breathing, also surrounds Gerota's fascia.

The ureters are narrow tubules millimeters (mm) in diameter that connect the kidneys to the bladder. The ureters cross into the pelvis anterior to the iliac vessels. The insert into the posterior and inferior floor of the bladder is called the trigone.

The bladder is a hollow sac with a flexible muscular wall. Fluid within the bladder will determine the thickness of the wall, which will normally range from 3 mm for a filled bladder to 6 mm for an emptied bladder.

015. Renal function

The function of the kidney is to excrete urine and to process the contents of blood plasma. These functions are carried out by the nephron. Both functions maintain the body's fluids and chemical components in balance, a state called homeostasis.

The glomerulus, the part of the nephron located in the cortex, filters the blood plasma. The renal tubule, the rest of the nephron that extends into the medulla (pyramids), transforms the concentrated inorganic (salts or electrolytes) and organic waste compounds into urine for excretion. Water is also passed into urine depending upon the amount of water in the body. The tubules will also reabsorb salt and water needed by the body. Thus, the production and excretion of urine maintains the body's salt and water balance. Other balances, such as acid and base in the blood, are also maintained through plasma filtration and urine production. Hormones released by the pituitary gland and adrenal gland cause the kidney to regulate the composition and even the pressure of blood. For example, although we know the liver is the primary organ for regulating blood pressure, the kidneys may release a hormone called renin when blood volume is low and increase blood pressure as compensation. Also, a hormone from the adrenal gland, called aldosterone, will be released when blood volume is low, causing the kidneys to reduce the excretion of salt and water as a way to increase blood volume.

Once urine is produced in the tubules of the nephron units, it is collected in the calyces and excreted out of the liver into the ureters. The urine is propelled down the ureters to the bladder where it is stored until released through the bladder's opening in the anterior portion of the trigone, called the urethra. The urethra excretes the urine out of the body.

Laboratory tests are performed to determine if the kidney is functioning properly. A few of the more critical tests are as follows:

- Creatinine.
- Blood urea nitrogen (BUN).
- Urinalysis.

Creatinine is a waste product of muscle tissue that is constantly and completely filtered through the kidneys into urine. Thus, the amount of creatinine in the blood should remain at a constant level. If the nephron units, specifically the glomerulus or capillary networks, are not properly filtering blood plasma, the creatinine levels in the blood will elevate. Because the clearance of creatinine from the body is normally complete and most causes of creatinine elevation are renal, then any elevation is specific for the kidney's failure to filtrate the blood.

The amount of urea, the waste product from the liver's metabolism of protein, in the blood above normal levels is another indication of interruption in the kidney's filtration and urine production functions. This test, called blood urea nitrogen, may show elevations for reasons other than renal failure, despite renal failure being the most common reason, and thus is not as specific as creatinine.

Urinalysis tests analyze the urine for the presence of hemoglobin, which indicates renal damage; for the presence of pH levels to determine the acidity in the urine; and for the presence of blood (hematuria) or pus (pyuria), a sign of urinary infection.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

014. Urinary system anatomy

1. Where is the normal location of the kidneys in the body?

2. What composes the medulla of the kidney?

3. What surrounds the perirenal fat and adrenal glands?

015. Renal function

1. Explain how kidney function maintains the body's salt and water balance.

2. Why is elevation of creatinine specific for kidney failure?

3. What indicates renal infection on a urinalysis test?

3–2. Imaging the Urinary System

Not every abnormality of the kidney is easily detectable by sonography. This is due to some diseases affecting the kidney's ability to function at a cellular level as opposed to obvious structural distortions seen by sonography; an obvious example would be large tumors. Some of the more subtle, functional diseases, however, may leave clues. In this section we will look at not only the typical approach to urinary sonography, but we will also briefly cover sonography of both common subtle and significant changes in kidney diseases.

016. Urinary system pathology

To recognize the sonographic differences between functional diseases that affect the whole kidney and focal masses that may present problems in certain areas of the kidney, we must first understand the diseases themselves. For our purposes, it is helpful only for you to be familiar with the most common pathology you are likely to encounter when scanning the urinary system. We will focus first on a few cystic abnormalities of the kidneys, followed by a brief survey of solid masses, both benign and malignant. We will then examine the functional diseases that affect the whole kidney, such as infectious and metabolic pathology to include a summary of renal failure.

Renal calcification

Within the kidney collecting system, various forms of calcium sometimes become solid stones. Various conditions cause renal stone formation to include hereditary disorders, metabolic diseases, hyperparathyroidism, ischemia, and renal failure.

Stones can occur anywhere along the collecting system and ureters. Unless they obstruct the flow of urine or cause inflammation from infection, most stones do not cause symptoms. Beyond typical renal collecting system stones, most kidney calcifications are parenchymal (*nephrocalcinosis*), either cortical type or medullary. One particular medullary type is relatively common: *medullary sponge kidney*, characterized by calcifications forming at the periphery of the medullary pyramids. Eventually, the entire pyramid will calcify.

Renal cystic disease

More common in older adults than in children, renal cysts may appear singular or multiple. Most kidney cysts are simple cysts located in the renal cortex (cortical cysts). Simple cysts are not usually significant unless they are large enough to cause pain, hematuria (blood in the urine), or distortion of kidney structures.

Similar to renal cysts are *parapelvic* cysts, located within the renal sinus. These cysts are not connected to the kidney's collecting system, the usual location for fluid trapped by obstruction. Parapelvic cysts do not generate symptoms unless they compress the vessels entering or leaving the renal hilum. They may occur singularly or in groups. As with cortical cysts, the parapelvic cysts are benign.

Polycystic renal disease

An inherited disease, polycystic renal disease comes in two forms: *autosomal dominant* (adult form) and *autosomal recessive* (infantile or juvenile, but also perinatal). The recessive form after birth is lethal and the infant or child will display large, echogenic kidneys as well as evidence of portal hypertension. Patients typically have hypertension and pain in the flanks with laboratory analysis revealing hematuria and infection. As the disease progresses to an advanced stage, the cysts may destroy the remaining tissue and lead to renal failure. In adult polycystic renal disease, cysts will appear elsewhere in the body such as the liver, spleen, and pancreas. There is also an association with brain aneurysms ("berry" or Circle of Willis aneurysms).

Multicystic dysplastic kidney (MDK)

MDK is a fetal developmental abnormality that may carry over after birth. It is the bilateral or unilateral replacement of renal tissue with multiple cysts. However, unilateral disease will be the type you are likely to encounter, as the bilateral type is fatal before birth. In unilateral MDK, there is minimal or no *normal* renal tissue in the kidney and it may be accompanied by ureteropelvic junction (UPJ) obstruction of the opposite kidney. The opposite kidney may also be hypoplastic (underdeveloped). The kidney in MDK is typically smaller than a normal kidney and has no function. Being asymptomatic, the condition usually goes undetected into adulthood because the opposite kidney compensates physiologically. No particular prevalence for male or female exists with MDK.

Renal neoplasms

We will now look at a few of the more common tumors that may arise in the kidneys, most being malignant. Unless the size is large, most of these masses are asymptomatic and typically do not affect the renal function.

Angiomyolipoma

This rare tumor of the renal cortex is usually solitary and small, less than 4 cm, made of fat, muscle, and blood vessels. Angiomyolipomas are hamartomas; that is, malformations composed of disorganized mixtures of tissue, usually tissues from the surrounding parenchyma of an organ. It occurs mostly in middle-aged women and is usually asymptomatic. If the tumor grows, it may bleed and cause pain, blood in the urine, and possible hypertension. In most patients with multiple, bilateral angiomyolipomas, there is an association with tuberous sclerosis, a disease that affects the mental state and produces lesions in the brain, organs, and on the skin.

Renal cell carcinoma (RCC)

The most common tumor of the kidneys, RCCs are also known as hypernephromas, adenocarcinomas, or von Grawitz tumors. Originating from the parenchyma of the kidneys, they appear mostly in elderly males, who suffer from fatigue, fever, weight loss, pain, and hypertension. Clinical evidence that suggests RCC will be a palpable mass with hematuria. RCC is associated with von Hippel-Lindau syndrome (an inherited disease characterized by hamartomas located throughout the body), acquired cystic kidney disease (multiple, small renal cysts caused by prolonged dialysis), adult polycystic kidney disease, and tuberous sclerosis.

Transitional cell carcinoma (TCC)

Another malignant tumor you may see is a transitional cell tumor. This is a tumor that originates from the inner lining of the renal collecting system, ureters, and bladder. It is the most common collecting system malignant tumor, occurring primarily in elderly males. The tumors may cause obstruction of the collecting system, which can yield symptoms such as pain and hematuria. If the tumor becomes invasive and penetrates into the renal parenchyma, symptoms may progress to weight loss, fever, and fatigue. These tumors are frequently multiple and bilateral.

Nephroblastoma

Also called Wilms' tumor, nephroblastoma is a rare tumor, but is the most common renal neoplasm in children. The tumor has slight associations with children who have various syndromes such as Beckwith-Wiedemann, a collection of disorders characterized by the excessive growth of the body or body parts (gigantism), and aniridia, an absence of the iris in the eye. Wilms' tumors may occur at any age but are usually diagnosed in children when they reach three years old. Clinical symptoms are abdominal masses that can be felt by the clinician, hypertension, and hematuria. However, the patient may feel no pain.

Renal infection

Bacterial organisms, usually *Escherichia coli* from the gastrointestinal tract, infect the kidneys and produce a spectrum of inflammation. Renal infection progresses from simple kidney inflammation (acute pyelonephritis) to more severe complications, such as focal bacterial nephritis. Severe inflammation may end with the formation of an abscess.

Most renal infections occur in young women who have clinical symptoms of flank pain and fever. Generally, diagnosis of acute pyelonephritis is accomplished clinically through laboratory analysis, and imaging studies such as sonography are not needed. In severe cases of renal infection, such as acute focal bacterial nephritis, the patient's symptoms may increase to pyuria (pus in the urine), hematuria, and vomiting. Laboratory values may show elevated BUN and leukocytosis. The end result of severe inflammation is a renal abscess.

Aside from abscess, pyonephrosis, pus and debris within the collecting system of the kidneys, may develop. Surgical drainage is needed immediately when pyonephrosis is diagnosed because the patient may develop sepsis, which is a life-threatening infection of the blood by harmful organisms or toxins.

Also keep in mind that not every infection of the kidney is based on bacteria from the gastrointestinal (GI) tract. Other responsible sources for which sonographic detection may not be specifically useful are viruses, parasites, or fungi. For example, one of the more common renal fungal infections is by the organism *Candida albicans*. The fungus produces mycetomas, or fungal balls, in the collecting system.

Acute renal failure

When kidneys are unable to properly function—that is to filter blood plasma or produce urine—they are unable to maintain homeostasis or remove the body's toxins and metabolic wastes. When this occurs the condition is known as renal failure and it can develop suddenly (acute) or over a prolonged

period of time (chronic). Most cases of acute renal failure can be reversed if the cause of insult is removed. General symptoms for renal failure are headaches, nausea, vomiting, and increased or decreased urination (frequency).

Acute renal failure may be thought of as a locational progression of problems that cause eventual failure such as the following:

- Prerenal (hypoperfusion).
- Renal (renal medical disease).
- Postrenal (obstruction).

Prerenal

Recall that kidneys begin their function with the filtration of blood plasma. If blood vessels are blocked, the kidney will eventually fail to remove the body's wastes. Therefore the first stage in renal failure can be thought of as prerenal, which occurs mostly in the renal artery and vein. If these vessels are blocked, the perfusion of needed blood in the kidney will be dangerously reduced (hypoperfusion). Renal artery stenosis or obstruction will prevent blood from getting to the kidney and, if unrelieved, failure is assured. Renal vein obstruction (thrombosis) will prevent blood drainage from the kidney and cause a backup or congestion, which in turn will cause the kidney to resist incoming blood from the renal artery. As with renal artery stenosis, if the obstruction is not removed, the kidney will fail. Also, other causes of reduced or contaminated flow to the kidneys are shock, sepsis, dehydration, and cardiac failure.

Renal medical disease

Once blood has reached the kidneys, it is up to the nephrons, located partly in the cortex and partly in the medulla (pyramids), to filter the blood plasma and to transform the resulting filtrate into urine. If necessary, the nephrons reabsorb water and salt back into the blood stream. Recall that the filtering mechanism within the nephron unit is called the glomerulus (located in the portion of nephron that is mostly in the cortex) and the renal tubules (mostly medullary nephron) produce and collect urine or reabsorb needed water and salt. The malfunctioning of either the glomerulus or the tubules is known as renal medical disease.

The most common case of acute renal failure and, specifically, medical renal disease, is *acute tubular necrosis* (ATN). As the name implies, necrosis and deposits of cell debris exists within the renal tubules, preventing the collection of urine. The damage to the tubules may be from toxins or lack of blood (ischemia).

Obstruction

Once the urine is produced it must be expelled from the body or a backup in the collecting tubules and nephron units, as well as rising toxin levels, will certainly cause the kidneys to fail to remove wastes from the blood plasma. One of the more significant and reversible abnormalities that prevent the removal of urine from the body is obstruction.

Hydronephrosis is the mild to severe dilatation of the renal collecting system caused by total or partial obstruction. Renal stones, called calculi, are the most common cause of hydronephrosis, followed by tumors, infections, and congenital anomalies. Most physicians, based on laboratory results such as hematuria and flank pain, will suspect stones and send the patient to obtain a computed tomography, which is more sensitive for obstructing stones located outside the kidney. Some, however, may use sonography.

Ureter and bladder pathology

One particular abnormality is the result of the distal ureter's inability to force urine into the bladder using normal peristalsis. The ureter, being obstructed, backs up and becomes enormously distended. This condition is called *congenital megaureter*.

Another ureter abnormality, called *ureterocele*, involves the bladder. The ureterocele is a saclike dilation of the end of the ureter, which protrudes into the hollow space of the bladder. Occasionally, the ureterocele will be entirely inside the bladder. The sac interrupts the normal flow of urine and causes dilation of the distal portion of the ureter. This dilation can spread back up the ureter to the kidneys and cause pain. Sometimes an ureterocele will insert in an abnormal location of the bladder. For example, an ectopic ureterocele will locate distal to the bladder's urethra as one-half of a duplicated ureter in a duplex kidney case.

A common disorder that may cause bladder wall thickening is cystitis, an inflammation of the bladder. Usually affecting women, cystitis is typically the result of an infection from *E. coli*. For men, bladder outlet obstruction may be the root cause of bladder wall thickening. Additionally, bladder outlet obstruction can be caused when bladder dysfunction (neurogenic), stones, malignant tumors, and ureteroceles block the exit to the urethra in the floor of the bladder.

TCC tumors or invasion from prostatic cancer in the male and uterine cancer in the female can have a profound effect on the bladder wall and bladder function. TCC in the bladder is particularly common in men.

017. Imaging the normal urinary system and variants

Except for a few minor adjustments, the preparation and patient positioning for the urinary system is identical to the liver and gallbladder. For example, some institutions may require you to include examination of the bladder with the other abdominal organs. Conversely, others may *only* require the bladder during exams that specifically request the urinary system. If your protocols require bladder sonography, you may have to ask the patient to drink approximately 12 to 24 ounces of water one hour prior to the start of the examination. This will allow the bladder to comfortably fill for adequate visibility of its contents and its wall.

Normal urinary system considerations and imaging

You should approach the kidneys with a probe that has the widest field-of-view and the highest frequency transducer possible for the penetration. This will usually be a curvilinear array of at least 5 megahertz (MHz).

Scan the right kidney through the anterior and lateral portion of the right lobe of the liver, just beneath the contour of the ribcage in most patients. You may have to raise the patient's right arm and roll him or her into a slight left lateral decubitus position. The left kidney should be approached more laterally and nearly coronal to the left side of the body. Generally, you will have to scan through the ribcage on the left side, as the left kidney is higher in the body than the right. Roll the patient into a slightly steeper right lateral decubitus position with his or her left arm resting up and out of the way. Have the patient breathe in deeply and hold his or her breath long enough for you to freeze an image of the kidney onto the screen. Deep inspiration shifts the kidneys down and away from the rib cage for clearer viewing. Allow the patient to breathe while you make measurements or manipulate the image. It is helpful to demonstrate to the patient by taking in a deep breath and holding it. Some patients may not be able to hold their breath very long or at all, particularly in an emergency situation. Do not worry about this procedure in such a situation because most ultrasound equipment allows you to freeze the best image you can and roll back through several frames of the same image for a sharp, near motion-free kidney.

On sonography, the normal kidney should be isoechoic or slightly hypoechoic to the echogenicity of the liver and spleen. The medullary pyramids should be seen as hypoechoic areas directly against the echogenic renal sinus. Unless dilated, you will not see the calyces or renal pelvis. Scan through the entire kidney longitudinally and transversely. Be sure to examine the area around the kidney.

Standard kidney images vary from institution to institution and radiologist to radiologist. Commonly, longitudinal views of the entire lengths of the right (fig. 3–2) and left kidney and transverse views of their upper, middle (fig. 3–3), and lower portions are documented.

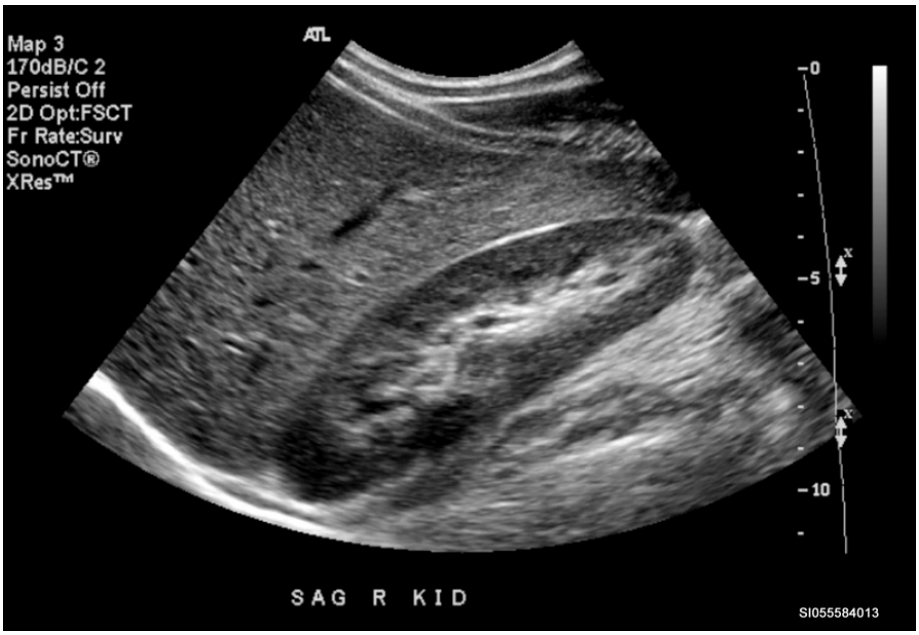

Figure 3–2. Right kidney, longitudinal.

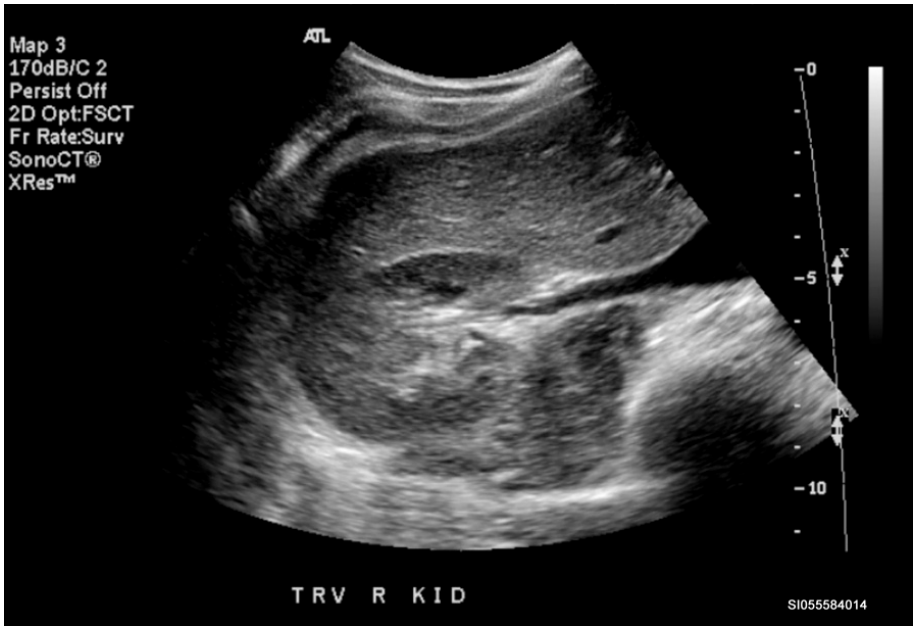

Figure 3–3. Right kidney, transverse.

Most departments require at least a measurement of kidney length (fig. 3–4). Others require length, height, and width. Some require all measurements only when pathology is seen or if the examination is specifically for the urinary system. You may find it useful to combine an image comparing the liver echotexture with the right kidney and measurements of the kidney. Along with longitudinal views of the length, some radiologists may want you to document the longitudinal view of the lateral and medial portions of the kidneys.

The normal ureters are not usually seen with sonography. However, a small length of proximal ureters may be seen by rolling a patient into a decubitus position and scanning laterally through the body, using the kidneys as sonographic windows. In transverse views of the kidneys, the renal pelvis may

be large enough to bulge out of the hilum (extrarenal pelvis) and you may be able to use it as a landmark for following the connected ureter distally. For the distal ureters, use the bladder anteriorly as a sonographic window by sweeping out toward the lateral wall of the pelvis, where you may see a narrow tube in normal patients.

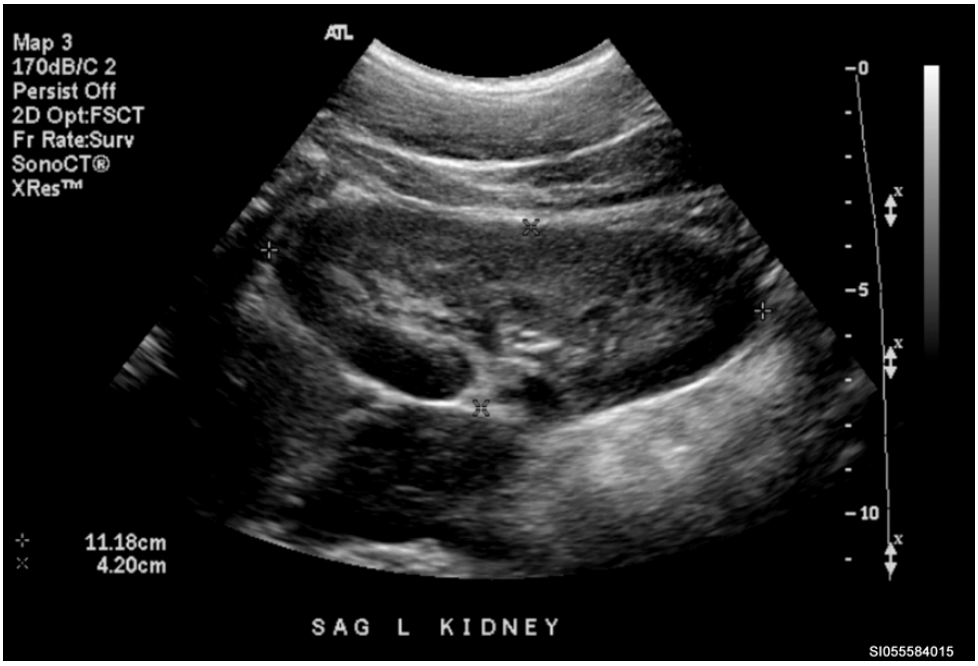

Figure 3–4. Left kidney, longitudinal with measurements.

Longitudinal and transverse views of the bladder should be documented after scanning through and examining the walls. It is helpful to radiologists to prove nonobstruction by placing a color Doppler box over the ureteric orifices, where the ureters enter the bladder. This technique is best seen in the transverse view of the bladder and should show streaks of color projecting periodically into the bladder lumen from both orifices. This represents the free flow of urine into the bladder and indicates open or patent ureters. If required measure the diameter of the bladder wall.

Kidney variants

While scanning the kidney, you should keep in mind a few unusual appearances of the kidney that are normal variations in structure.

Normal Variant	Description
Ectopic kidney	When one or both kidneys are located in the body other than in their usual location on either side of the spine at the level of the thoraco-lumbar vertebrae. *Crossed renal ectopia: Two kidneys on one side of the body* *Crossed fused renal ectopia: Two fused kidneys on one side of the body*
Horseshoe kidney	Both kidneys fused together with the fused portion crossing anteriorly over the aorta at the level of the lower lumbar vertebrae.
Parenchymal junctional defect	A partial fusion of two parenchymal units. Located usually anterior and superior you should see an echogenic line extending from the capsule to sinus. Usually seen on the right kidney.
Dromedary hump	Thickening of the lateral margin of normal left kidney cortex. This may resemble a mass. Look for the medullary pyramids within the thickened portion to rule out a lesion.
Hypertrophied column of Bertin	Sometimes the renal cortex normally between two renal pyramids may thicken and resemble a mass. The echotexture, however, will show no distortion of normal cortex architecture.
Duplex kidney	A kidney with two completely separated sinuses and two ureters.

018. Imaging renal cystic disease

The kidneys may display a range of cystic diseases from obvious simple cysts to a cystic condition found with calcification of the renal pyramids in medullary sponge kidney. Sonographers are generally asked to demonstrate the character or appearance of cystic structures as clues to their nature.

Typical renal cysts

As with cysts that appear elsewhere in the body, the sonographer's role is to demonstrate the sonographic appearance of renal cysts and to characterize them as either simple or complex. Careful attention to basic parameters, such as overall gain and focal zone placements, are critical to remove any artificial echoes appearing within the cyst.

Strict sonographic criteria define a simple cyst:

- Anechoic.
- Smooth wall.
- Posterior acoustic enhancement.
- Round.

Many radiologists will insist on complete measurements of the cysts despite their appearance. Located in the cortex, the cysts can bulge outside the kidney or into the renal sinus. Unless you see them appearing with internal echoes or septations, they are usually of little significance. Despite this, they should at least be measured.

Sonographically identical to simple cyst are parapelvic cysts, however, which are located in the renal sinus. Parapelvic cysts can be difficult to diagnose sonographically because they have an appearance similar to that of other conditions. For instance, hydronephrosis is often confused with multiple parapelvic cysts. What distinguishes two or more parapelvic cyst from hydronephrosis is the lack of communication between each cyst. Conversely, the hydronephrotic kidney will have a dilated renal pelvis connected to dilated infundibula located within the kidney. A single parapelvic cyst also has a similar sonographic appearance to that of an extrarenal pelvis. When you scan and detect these cysts, use caution and try to identify the walls.

Occasionally, renal cysts will have an atypical appearance such as single thin septations or internal echoes. Any appearance other than the strict characteristic criteria for a simple cyst is considered a complex cyst and merit more attention. Complex echoes and septations may be due to infection or hemorrhage.

Polycystic renal disease

The form you will likely encounter is the adult form, which appears in older adults. Adult polycystic kidney disease is the bilateral renal appearance of multiple cysts of varying sizes. The kidneys will usually be enlarged and demonstrate multiple cysts of various sizes. Measure a few of the largest cysts. Some cysts will hemorrhage or become infected, in which case you may see complex echoes within them. Because this appearance is similar to other renal diseases, such as abscess and some malignant tumors, they should always be measured no matter the size. Also use color Doppler to determine if the cyst contains vascular supply, indicating malignancy.

Multicystic dysplastic kidney

On sonography, the affected MDK is small with multiple cysts that do not connect. The cysts may be quite numerous and small or large and lobulated. You should note the absence of both renal cortex as well as the absence of renal sinus. You may also see evidence of calcification. Use Doppler to confirm the typical absence of blood flow within the MDK. Check the opposite kidney for UPJ obstruction, which occurs in a small percentage of MDK patients.

019. Imaging renal neoplasms

As we discuss these masses, be aware that sonography cannot absolutely diagnose malignancy; it can only detect appearances that may suggest malignancy. Because radiologists are suspicious of most renal tumors, treating them all as potential malignancies is the best approach. Thus, you should never dismiss a small tumor by not documenting it.

Be aware that not all focal abnormalities in the kidney are neoplasms. Some may be renal stones. On sonography, you may detect renal stones as hyperechoic foci located anywhere along the urinary pathway. Cortical stones may be diffusely located in the cortex. You may see evidence of medullary sponge kidney by noting the renal pyramids with hyperechoic edges or pyramids filled with shadowing calcifications. If stones are the cause of painful obstruction, look for the hyperechoic shadowing point at the level of the UPJ at the kidney and at the ureterovesical junction (UVJ) at the bladder. You should understand that bowel loops make stones along the ureter extremely difficult to detect.

Benign neoplasms

Most benign neoplasms are situated in the renal cortex. Rarely will you see a large, benign renal tumor. However, do not let small size convince you that a tumor is benign. Again, treat them as potential malignancies and fully document them. That is, measurements and color Doppler should be used.

On sonography, most angiomyolipomas are hyperechoic with an echogenicity equal to or greater than the renal sinus. In large, symptomatic tumors you may see evidence of bleeding by their mixed echogenicity or collection of anechoic fluid around them. Because of this, some angiomyolipomas have a similar sonographic appearance to some adenocarcinomas. Ensure that you measure them in three dimensions. Place color Doppler to determine if there is a blood vessel feeding a possible malignant tumor.

Malignant neoplasms

Certain clues, such as vascular flow already mentioned, will suggest the presence of malignant tumors within the kidney. However, sonography is limited in distinguishing with certainty benign from malignant neoplasms. Physicians will usually choose alternate methods such as computed tomography coupled with biopsies to determine the status of a renal tumor. As with any organ system, malignancy that originates from elsewhere in the body may travel to the kidneys and set up secondary tumors (metastases). For example, metastatic disease from the lungs, colon, skin, and breast may deposit large solitary masses, multiple nodules, or infiltrate and enlarge the entire kidney. Also, the appearance of bilateral, multiple, near anechoic masses that appear to be cysts may represent just one of the types of lymphomas that may travel to the kidney. The lack of posterior acoustic enhancement for the size of the nodule should help you distinguish the renal lymphoma from a cyst. Aside from the relatively rare appearances of metastatic and lymphomatous diseases, you will mostly encounter renal cell carcinomas when imaging malignancies.

Renal cell carcinoma

For RCC, you will see a solid tumor on sonography with a varying range of appearances and sizes. Most are isoechoic with slightly hypoechoic encapsulating rims. Others may be hypoechoic to hyperechoic. The smaller RCCs are more echogenic than larger tumors. Occasionally, the tumors will be cystic with septations, thick walls, debris, and solid nodules inside them. Make sure to measure the tumor and place the color Doppler box over it to display its hypervascular nature. Check as much length of the renal vein and inferior vena cava as possible for infiltration of RCC. This will be seen as low-level thrombosis within the vessels. Also examine the liver for the presence of metastases. Be aware that hypertrophied columns of Bertin may mimic an RCC. To help distinguish between the two, remember that RCC will either destroy or push aside any medullary pyramids (and blood vessels

on Doppler). Conversely, hypertrophied columns of Bertin will contain renal pyramids within them and a more magnified view of this feature should be documented for the radiologist.

Transitional cell carcinoma

On sonography, usually on a transverse view of the renal hilum, you will see either TCC located as a solid mass within the renal pelvis or as a poorly defined tumor in the renal parenchyma. If the tumor spreads to the sinus and cortex, it can be indistinguishable from RCC. The best confirmation will be its appearance within the renal pelvis. TCC appears mostly hypoechoic and will not demonstrate much vascularity on Doppler. Not every TCC can be seen by sonography as some are quite small and may have flattened shapes against the wall of the collecting system. Keep in mind that most TCCs are located in the bladder.

Nephroblastoma

For a sonographic case of suspected nephroblastoma, you should see a large, well-defined, unilateral mass with homogeneous echoes. They are difficult to distinguish from neuroblastomas, tumors that arise from the adrenal glands at the superior pole of the kidney. Clues that you can use to help suggest the difference are to remember that neuroblastomas are significantly more heterogeneous and ill defined than nephroblastomas. Another clue is that Wilms' tumors distort or destroy the renal sinus, cortex, pyramids, and contour while neuroblastomas tend to displace or compress the entire kidney without affecting the internal structures.

Because Wilms' tumors are invasive and spread through the renal vein or directly through the cortex, it is important for you to examine the renal vein and inferior vena cava (IVC) for tumor clot (thrombus). Also, examine the liver for tumor spread as well as the area around the aorta for where the tumor may enlarge the lymph nodes (lymphadenopathy).

020. Imaging renal infection and abnormalities associated with renal failure

Sonography plays more of a role in renal infections that progress to more severe inflammation, reoccur, or develop complications. Otherwise, many renal infections are treated clinically and sonography is used to evaluate the progress of treatment. In this lesson, we will briefly examine the sonographic approach to renal infection and discuss some of the complications that may arise. We will then look at sonographic imaging of renal failure and the pathology that generates it.

Acute pyelonephritis

Most sonography departments will not be sent patients with first time acute pyelonephritis as the disease is diagnosed and managed in the clinic without the need for imaging. Specifically, sonography would not be a useful diagnostic tool because acute pyelonephritis will usually appear normal on sonography. However, occasionally, pyelonephritis will persist after initial treatment, and sonography may be a useful way to determine the state of the disease.

If the kidney does display signs of acute pyelonephritis, the more common changes seen on sonography will be one or more of the following:

- Renal enlargement.
- Compression of the renal sinus.
- Hypoechoic parenchyma from swelling.
- Loss of clear distinction between the cortex and the medulla (corticomedullary differentiation).

In cases of focal nephritis, sonography, which may be increasingly needed, may reveal to you swollen and hypoechoic areas in the kidneys. These focal areas should be measured. Also, focal nephritis may worsen into an abscess. The abscess appears similar to a complex cyst and, like any lesion in the

kidney that does not fit the criteria for simple cysts, is a cause for attention. It should be measured and Doppler should be used to detect potential blood flow.

For pyonephrosis (pus in the collecting system), you will see a dilated collecting system and renal pelvis (hydronephrosis) filled with low-level echoes representing debris. Another infectious abnormality that may affect the collecting system is a mycetoma, or fungal ball. These echogenic masses are mobile and may cause obstruction, but do not cause shadowing. They have a similar appearance to pyonephrosis, blood clots, and TCC. However, remember that pyonephrosis shows low-level fluid-debris echoes that layer after movement, that blood clots do not obstruct the collecting system, and that TCC is not mobile. Be sure to clearly image these distinctions.

Acute renal failure

Nearly all cases of renal failure for which you will be requested to perform sonograms will concern *acute* renal failure or the imminent threat of it, particularly obstruction. However, sonography cannot reliably distinguish between the various causes of acute renal failure.

As stated before, most cases of acute renal failure can be reversed if the cause of insult is removed. Thus, rapid diagnosis by sonography, particularly to detect possible sources of obstruction despite overlapping sonographic features, may help in the eventual treatment of the disease. Conversely, chronic renal failure is usually already clinically known and irreversible, with patients usually on dialysis, and thus diagnosis by sonography is not needed. However, if you should examine such a patient, you will generally see hyperechoic kidneys less than 7 cm in length.

Prerenal

Reduced perfusion of blood through the kidney can be detected through sonography. Color Doppler alone can detect this starting point for renal failure. On sonography, you will have to rely mostly on analyzing the blood flow in the renal artery or in its branches within the kidney, using spectral and color Doppler. The kidneys will usually appear enlarged on sonography, with low-level echoes filling the renal veins and possibly appearing in the IVC. We will discuss briefly sonography of the kidney's blood vessels in the vascular sonography unit in volume 2.

Renal medical disease

With toxic damage, such as from drugs or heavy metals introduced into the blood stream, you will usually only see the ATN kidney enlarged and echogenic on sonography. With the lack of blood, the kidney's parenchyma may be hypoechoic but with normal size.

You may see the same sonographic sign in other cases of medical renal disease such as acute glomerulonephritis, a disease of the glomerulus that also may show normal or hypoechoic kidneys with normal size.

Obstruction

In cases of suspected mild hydronephrosis, you should ensure that you are dealing with true hydronephrosis by having the patient empty his or her bladder. If the dilatation is gone upon reexamination, there was no obstruction. The causes of most renal obstructions, stones, are mostly located at the junction of the ureter and bladder (UVJ) but can sometimes be seen at the level where the ureter joins the renal pelvis called the UPJ.

Sonography of mild hydronephrosis will show the echogenic central renal sinus slightly separated. The more complete or aged the obstruction the more severe the dilation, extending into the major and minor calyces and eventually obliterating the echogenic sinus. Massive dilation of the collecting system may compress the renal cortex into a thin rim surrounding an anechoic space.

Be careful to distinguish small anechoic areas in the renal sinus as hydronephrosis and not parapelvic cysts. The best way to do this is to document any communication between anechoic areas, a feature that parapelvic cysts lack. On transverse views of the kidney, the renal pelvis will be dilated and may

extend out to the source of hydronephrosis in the case of UPJ obstruction. This source may be a stone, which may not show the classic hyperechoic surface with sharp posterior shadowing due to the surrounding tissue.

Always ensure that your patient voids after your examination. Then briefly rescan the kidneys to see if the dilatation of the calyces has resolved. This reevaluation should also include the bladder evaluation and as much of the ureters as can be possibly seen.

021. Imaging ureters and bladder

The ureters and bladder make up the lower tract of the urinary system and can also harbor abnormalities. On sonography, however, the bladder may be difficult to examine, and the ureters nearly impossible.

Ureters

Unless dilated, the ureters cannot be seen by sonography. Even with dilatation, frequently only the proximal or distal portions are seen, usually as a result of a stone or stricture at the level of UPJ or at the level where the ureter joins the bladder at the UVJ.

Rarely, you may see a congenital megaureter on sonography as a ureter massively dilated into tortuous loops. Sometimes, a congenital megaureter may progress to the point of dilating the calyces (hydronephrosis). If detected be sure to measure the diameter of the tube.

Finally, an ureterocele may also be detected on the surface of the bladder trigone. Use color Doppler to determine if the ureterocele is preventing the entrance of urine into the bladder. By using color Doppler in a real-time view of the transverse bladder, you may be able to demonstrate a color jet of urine entering into the bladder in the area of the trigone. Otherwise the uterocele is shown to be an obstruction. Be sure to measure any ureteroceles in three dimensions.

Bladder

Generally, the bladder is sonographically imaged to determine if the wall of the gallbladder is thickened or if there are tumors present.

Recall that the normal thickness of a filled bladder should be no more than 3 mm, and that the wall of an empty bladder is probably abnormal if the wall thickness exceeds 6 mm. Use the AP method, preferably of the wall closest to your transducer. If the wall of the bladder is thickened and the inner lining appears jagged (trabeculated), a search for any of these abnormalities is necessary.

Bladder neoplasms you will mostly see, if at all, are TCC tumors or invasion from prostatic cancer in the male and uterine cancer in the female. Make sure you use color Doppler and measure any focal wall abnormality in three dimensions.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

016. Urinary system pathology

1. What are the two forms of polycystic renal disease?

2. What is the most common renal tumor in children?

3. What is the prerenal phase of acute renal failure?

4. What is the *most* common cause of acute renal failure?

5. What are some causes of hydronephrosis?

6. What is an ureterocele?

7. What are the two conditions that may cause bladder wall thickening?

017. Imaging the normal urinary system and variants

1. When should you see the calyces within the renal sinus?

2. How would you use the kidneys to image the proximal ureters?

3. Match the normal kidney variants in column B with their sonographic descriptions in column A. Items in column B may be used only once.

Column A	***Column B***
____ (1) A kidney with two completely separated sinuses and two ureters.	a. Parenchymal junctional defect.
____ (2) Thickening of the lateral margin of normal left kidney cortex.	b. Hypertrophied column of Bertin.
____ (3) Both kidneys fused together with the fused portion crossing anteriorly over the aorta.	c. Dromedary hump.
____ (4) Thickened renal cortex between two renal pyramids.	d. Horseshoe kidney.
____ (5) An echogenic line extending from the capsule to the sinus.	e. Ectopic kidney.
____ (6) Both kidneys located together on one side of the spine.	f. Duplex kidney.

018. Imaging renal cystic disease

1. What is the sonographic description of a multicystic dysplastic kidney?

2. Describe the sonographic appearance of adult polycystic renal disease?

019. Imaging renal neoplasms

1. What is the sonographic description of most angiomyolipomas?

2. What kidney variant may mimic renal cell carcinoma on sonography?

3. How can you sonographically distinguish a renal lymphoma from a cyst?

4. How can you sonographically distinguish a hypertrophied column of Bertin from an RCC?

5. What is the best sonographic confirmation for TCC?

020. Imaging renal infection and abnormalities associated with renal failure

1. List some of the sonographic signs of acute pyelonephritis?

2. How can you sonographically distinguish between mycetomas and the similar appearances of pyonephrosis, blood clots, and TCC?

3. Describe the sonographic appearance of hydronephrosis.

021. Imaging ureters and bladder

1. What is the sonographic appearance of congenital megaureter?

2. What bladder tumors will you mostly see sonographically, if at all?

Answers to Self-Test Questions

014

1. Both kidneys are normally located to either side of the spine against the back muscles at the level between the 12th thoracic vertebra and the fourth lumbar vertebra.
2. This medulla is composed of 8 to 18 folds called medullary pyramids.
3. Both perirenal fat and adrenal gland are surrounded by connective tissue called Gerota’s fascia.

015

1. The glomerulus filters the blood plasma. The renal tubules transform concentrated inorganic and organic waste compounds into urine for excretion. Water is also passed into urine depending upon the amount of water in the body. The tubules will also reabsorb any salt and water needed by the body.

2. Clearance of creatinine from the body is normally complete and most causes of creatinine elevation are renal.
3. Hematuria or pyuria.

016

1. Autosomal dominant (adult form) and autosomal recessive (infantile or juvenile, but also perinatal).
2. Nephroblastoma or Wilms' tumor.
3. The reduction of blood flow to and from the kidneys (hypoperfusion) that prevent the filtration of blood plasma.
4. Acute tubular necrosis.
5. Renal calculi, tumors, infections, and congenital anomalies.
6. A saclike dilation of the end of the ureter, which protrudes into the hollow space of the bladder.
7. Cystitis and bladder outlet obstruction.

017

1. When they are dilated.
2. Roll a patient into a decubitus position and scan laterally through the body, using the kidneys as sonographic windows. In transverse views of the kidneys, the renal pelvis may be large enough to bulge out of the hilum (extrarenal pelvis) and you may be able to use it as a landmark for following the connected ureter distally.
3. (1) f.
   (2) c.
   (3) d.
   (4) b.
   (5) a.
   (6) e.

018

1. Small kidney with multiple cysts that do not connect. You may also see evidence of calcification.
2. Adult polycystic kidney disease is the bilateral renal appearance of multiple cysts of varying sizes. The kidneys will usually be enlarged and demonstrate multiple cysts of various sizes.

019

1. Hyperechoic with an echogenicity equal to or greater than the renal sinus.
2. Hypertrophied column of Bertin.
3. The lack of posterior acoustic enhancement for the size of the nodule.
4. RCC will either destroy or push aside any medullary pyramids (and blood vessels on Doppler). Conversely, hypertrophied columns of Bertin will contain renal pyramids within them.
5. Its appearance in the renal pelvis.

020

1. (1) Renal enlargement.
   (2) Compression of the renal sinus.
   (3) Hypoechoic parenchyma.
   (4) Loss of clear distinction between the cortex and the medulla (corticomedullary differentiation).
2. Mycetoma, or fungal ball, are echogenic masses which are mobile and may cause obstruction, but do not cause shadowing. They have a similar appearance to pyonephrosis, blood clots, and TCC. However, pyonephrosis shows low-level fluid-debris echoes that layer after movement; blood clots do not obstruct the collecting system; and TCC is not mobile.
3. Sonography of mild hydronephrosis will show the echogenic central renal sinus slightly separated. The more complete or aged the obstruction the more severe the dilation, extending into the major and minor

calyces and eventually obliterating the echogenic sinus. Massive dilation of the collecting system may compress the renal cortex into a thin rim surrounding an anechoic space.

021

1. Tortuous loops of massively dilated ureters.
2. TCC tumors or invasion from prostatic cancer in the male and uterine cancer in the female.

Complete the unit review exercises before going to the next unit.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter.

35. (014) What structures does the kidney's cortex and medulla share?
    a. Renal pelvis.
    b. Renal sinus.
    c. Nephron.
    d. Calyx.

36. (015) The relationship between blood urea nitrogen (BUN) and creatinine is they both are
    a. produced by the adrenal glands.
    b. used to test kidney function.
    c. used to test urine frequency.
    d. produced by the nephrons.

37. (016) What will a sonographer encounter with multicystic dysplastic kidney disease after birth?
    a. Multicystic dysplastic disease after birth.
    b. Bilateral or unilateral kidney involvement.
    c. Unilateral kidney involvement.
    d. Bilateral kidney involvement.

38. (017) The *least* you should do to record renal size with sonography is to document
    a. measurements only when specifically requested.
    b. measurements only when pathology is seen.
    c. length, width, and height.
    d. the kidney length.

39. (017) While evaluating the bladder with sonography, you *best* demonstrate patent ureters by
    a. observing streaks of color from the ureteric orifices.
    b. completely imaging both ureters.
    c. measuring the bladder wall.
    d. measuring bladder width.

40. (018) What distinguishes parapelvic cysts from hydronephrosis of the kidneys?
    a. Size of the cysts.
    b. Size of the hydronephrosis.
    c. The lack of communication between each cyst.
    d. Presence of communication between each cyst.

41. (018) Why measure complex cysts found with polycystic renal disease?
    a. They are the largest polycystic renal disease cysts.
    b. To compare their sizes with corresponding liver cysts.
    c. They can mimic the sonographic appearance of malignant tumors.
    d. To determine if the polycystic disease is autosomal dominant or recessive.

42. (019) After sonographically detecting a solid, isoechoic renal mass with a hypoechoic rim, the purpose for checking the renal vein, inferior vena cava (IVC), and liver is to detect the presence of
    a. kidney.
    b. polycystic renal disease.
    c. Arnold-Chiari syndrome stones.
    d. infiltrating renal cell carcinoma (RCC).

43. (019) What is a sonographic difference between a nephroblastoma and a neuroblastoma?
    a. Nephroblastomas are more homogeneous and well defined than neuroblastomas.
    b. Neuroblastomas are more homogeneous and well defined than nephroblastomas.
    c. Of the two, nephroblastomas do not affect internal renal structures.
    d. Of the two, neuroblastomas distort internal renal structures.

44. (020) When is sonography likely to play a role in renal infections?
    a. Whenever infection occurs.
    b. When infections develop complications.
    c. When patients are recovering from infections.
    d. Whenever women suffer flank pain and fever.

45. (020) The likely significance of the sonographic appearance of hydronephrosis with low-level echoes is the presence of
    a. an abscess.
    b. pyonephrosis.
    c. parapelvic cysts.
    d. an angiomyolipoma.

46. (020) Sonography is useful in diagnosing acute renal failure because early detection
    a. may help stage acute renal failure.
    b. may help reverse acute renal failure.
    c. can help reverse chronic renal failure.
    d. can determine various causes of acute renal failure.

47. (021) What should sonographers do after detecting a trabeculated bladder wall?
    a. Search for the cause of bladder outlet obstruction.
    b. Search kidneys for the presence of stones.
    c. Have the patient stand or lie on the side.
    d. Have the patient empty the bladder.

Please read the unit menu for unit 4 and continue. ➔

Student Notes

Unit 4. General Abdominal Sonography

SONOGRAPHIC EVALUATION of the abdomen requires more than a look at major abdominal organs such as the liver and kidneys. The other smaller organs and even the bowel, which is *not* ideal for sonographic evaluation, are no less important. In this unit, we will briefly discuss these structures.

4–1. Understanding and Imaging the Pancreas

Because problems of the pancreas are normally diagnosed using computed tomography (CT), we will concentrate only on the most common problems of the pancreas for which sonography has played a useful role. We will cover some of the more important features of the pancreatic anatomy. Common pancreatic abnormalities, such as pancreatitis, can cause functional disturbances, which may progress to complications detectable by sonography. Thus, we will also briefly survey pancreatic function.

022. Pancreatic anatomy and function

The location of the pancreas and its internal structure demonstrates its closeness to the liver and gallbladder in the body's work of digestion and metabolism. In this lesson, we will briefly look at the anatomy and physiology of this vital organ.

Anatomy

The pancreas lies either horizontally or obliquely (with the head inferior to the rest of the pancreas) across the body mostly within the retroperitoneum. The pancreas is made up of a head, body, and tail. There are no obvious landmarks on sonography to demonstrate where the head ends and the body begins but it is conventional to think of the neck of the pancreas as being directly above the superior mesenteric vein (SMV) and its confluence with the splenic vein. The pancreas is a long, thin organ measuring 12 to 18 centimeters (cm) in length and no more than 3 cm in thickness at the head. Its body and tail thickness ranges from 1 to 2 cm. The lateral portion of the pancreatic head is in contact with the first loop or curve of the duodenum, and thus is within the peritoneum. The splenic artery superiorly and the splenic vein inferiorly form most of the borders of the pancreas. The stomach lies directly anterior to the pancreas, while posterior to the head and body are the inferior vena cava (IVC) and aorta, respectively. The SMV joins the splenic vein directly posterior to the neck of the pancreas. A portion of the pancreatic head, called the uncinate process, juts posterior to the SMV. Directly posterior to the body of the pancreas is the superior mesenteric artery (SMA).

The pancreas does not have a protective covering and its tissue is directly exposed to the retroperitoneal environment. Most of the cells of the pancreas are functional exocrine cells called acini cells. Acini refer to the arrangement of the cells into clusters resembling grapes. In between the

clusters are tiny ducts that merge eventually into a larger duct running along the central length of the pancreas, called the *duct of Wirsung*. This duct is normally 2.1 millimeters (mm) throughout the pancreatic body. The end of the pancreatic duct is the ampulla of Vater situated in the wall of the duodenum. Branching off the pancreatic duct is a secondary duct running through the upper head of the pancreas and attaching to the wall of the duodenum a few centimeters proximal to the ampulla of Vater. This accessory duct is called the *duct of Santorini* (fig. 4–1).

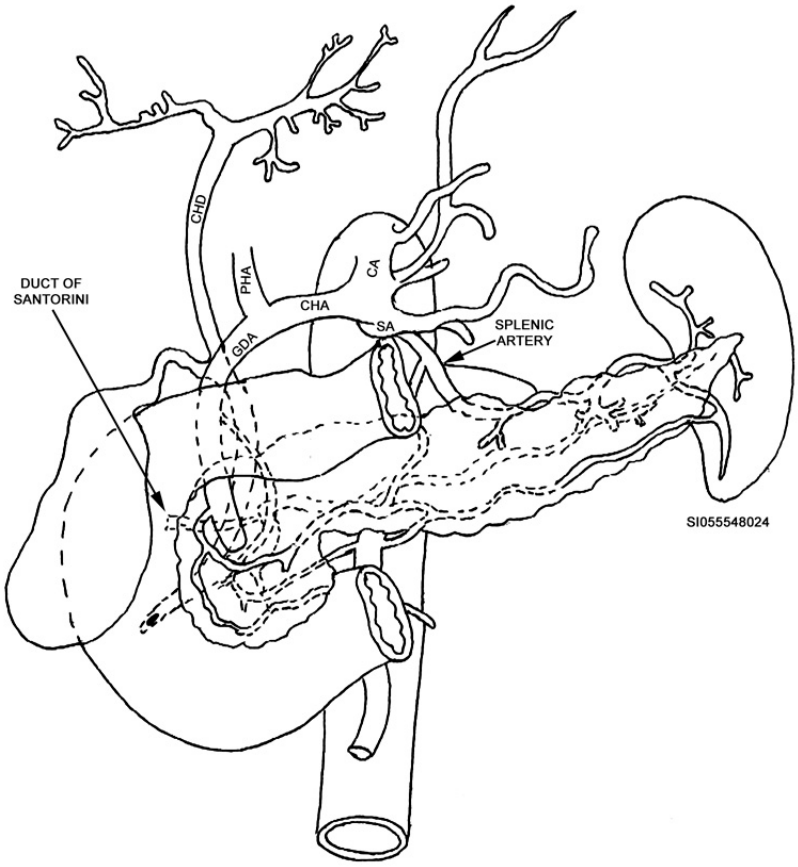

Figure 4–1. Pancreas.

Interspersed throughout the acinar tissue are millions of exocrine cell clusters connected to the venous channels within the pancreas. These exocrine clusters are called pancreatic islets, or the *islet cells of Langerhans*. The clusters are made of primarily beta cells in the central portion and alpha cells on the periphery.

Function

The pancreas has dual endocrine and exocrine functions. Its primary endocrine function is to produce insulin and its primary exocrine function is to produce digestive enzymes.

Insulin

The beta cells of the islets of Langerhans secrete insulin into the body's blood stream. Insulin is a hormone that prompts the liver to convert glucose into glycogen for storage. It also helps move glucose in the bloodstream into the body's tissue; thus reducing the amount of overall blood sugar.

The alpha cells work to reverse this process. They secrete glucagon, a hormone that causes the liver to convert stored glycogen and even fatty acids and some proteins into glucose to be released to the body. This increases the level of sugar in the blood.

Digestive enzymes

The exocrine cells of the pancreas will secrete a pancreatic juice that contains mostly three enzymes: amylase, lipase, and trypsin. Recall from the unit on the gallbladder and biliary system that the small intestine will secrete a hormone called cholecystokinen (CCK) when food particles containing fatty and amino acids enter the duodenum from the stomach. Recall further that CCK has a dual function. First it causes the gallbladder to contract, forcing bile through the common bile duct and into the duodenum to prepare the food (chyme) for digestion. Second, it causes the exocrine cells of the pancreas to secrete the enzymes that will digest food broken up by bile salts.

Amylase digests carbohydrates, lipase digests fats, and trypsin activates all of the other proenzymes in the pancreatic juice such as chymotrypsin, nucleases, and carboxypeptidase. The pancreatic duct's cells secrete sodium bicarbonate into the pancreatic juice on its way to the duodenum. Sodium bicarbonate neutralizes the acid in the food to be digested, which otherwise would prevent the enzymes from working.

023. Pathology of the pancreas

A host of abnormalities affect the pancreas. However, pathology that concerns most physicians is the presence of carcinoma or inflammation. Both abnormalities can seriously disrupt the normal functioning of the pancreas, eventually becoming lethal. So that you may be more familiar with the most significant pathology of the pancreas, we will look at both inflammation and tumor formation (benign and malignant).

Acute pancreatitis

The sudden inflammation of the pancreas is usually caused by biliary tract disease, especially stones, in women and alcohol abuse in men. There are numerous other causes from congenital anomalies to drugs to infectious agents. Most patients will have symptoms of pain directly below the breastbone in the epigastrum, nausea, vomiting, and fever. Laboratory values will show elevations of amylase and lipase because inflammation causes these enzymes to leak out into surrounding tissues, where they are absorbed into the bloodstream. Also, white blood cell counts will be elevated.

Chronic pancreatitis

Chronic pancreatitis is usually the result of alcohol abuse. Recurring episodes of acute pancreatitis, which may be mild or severe, eventually break down the pancreatic tissues into an irreversible destruction. Fibrous scarring from past inflammation replaces much of the acinar tissue. Eventually the fibrosis will break down the pancreatic duct walls and form a series of strictures, causing an irregular beaded appearance on sonography. Fibrosis in the duct may calcify and you will see stones and alternate dilatation among the strictures.

Benign pancreatic neoplasms

Microcystic adenomas (serous cystadenoma) are benign tumors of the pancreas seen mostly in older women. They can appear throughout the pancreas with a third located in the pancreatic head. A microcystic adenoma is composed of multiple small cysts, ranging in sizes of 1 mm to 1 cm. The larger cysts are usually arranged on the periphery of the tumor. Microcystic adenomas are extremely vascular.

The islets of Langerhans will occasionally develop tumors called *Islet cell tumors*, which may be benign or malignant. These tumors usually appear in middle-aged or older patients. Two types exist: functioning and non-functioning. Functional islet cell tumors are actively secreting and increasing the level of pancreatic hormones. They appear throughout the gland.

The most common functioning islet cell tumor is called an *insulinoma* (B-cell tumor), which is typically a benign tumor. Laboratory tests in these patients reveal abnormally high levels of insulin. Most of these tumors are solitary and less than a centimeter in size. A small percentage will transform into malignant neoplasms.

Malignant pancreatic neoplasms

Macrocystic adenomas (mucinous cystadenoma) are usually malignant or have malignant potential. Seen in middle-aged to older women, they are larger than 2 cm and are typically unilocular but can have multiple lobulations. Macrocystic adenomas contain solid elements such as mural nodules or thick septations. The tumors may contain cysts along the outer edge or along the septations.

Adenocarcinoma is a particularly insidious tumor found mostly in older men. Most are caused by chronic alcohol abuse. It is the most common tumor originating from the pancreas. The tumor is usually located in the head of the pancreas, where it may compress the common bile and pancreatic ducts. Clinical signs, especially with bile duct compression, will normally be nausea, vomiting, weight loss, epigastric pain, and jaundice. The serum amylase levels will be normal, distinguishing the tumor from acute focal pancreatitis.

024. Imaging the pancreas

Although CT is heavily used to visualize the pancreas, sonography is the primary modality for its ease of use and its cost effectiveness. Because the pancreas should be imaged as part of a standard sonographic examination of the abdomen, the patient preparation is identical to that of the liver and gallbladder. With sonography, the pancreas is isoechoic or slightly more echogenic than the left lobe of the liver. However, in some emergency situations where the patient has not fasted, interference of ultrasound from the stomach's contents can obscure the pancreas. In those cases, have the patient drink water to give you a clearer sonographic window. The entire pancreas can be seen on one view in most cases. If this is difficult due to duodenal gas or because of the shape of the pancreas, you can easily separate the images into head (fig. 4–2), body (fig. 4–3), and tail (fig. 4–4).

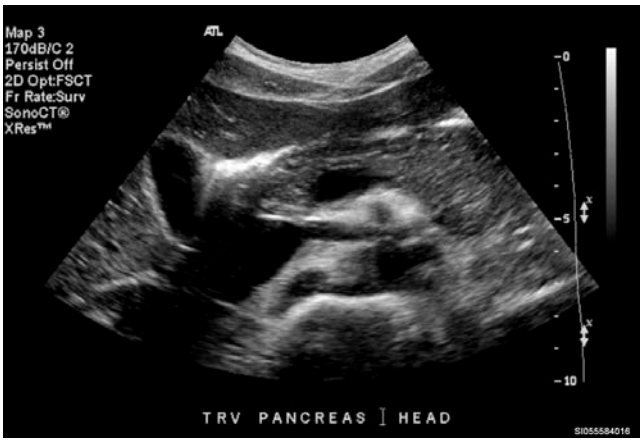

Figure 4–2. Pancreas, head.

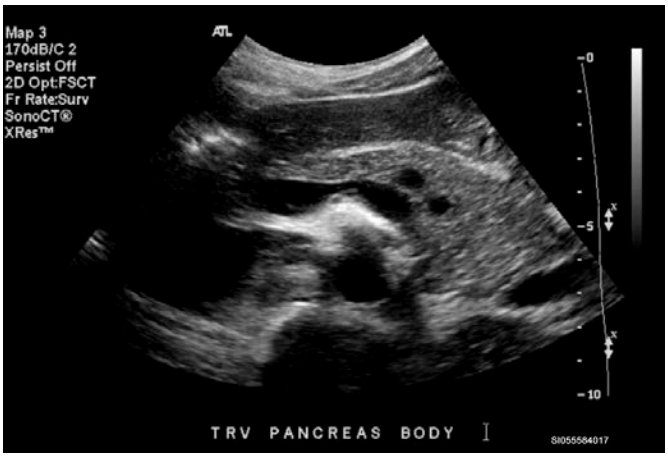

Figure 4–3. Pancreas, body.

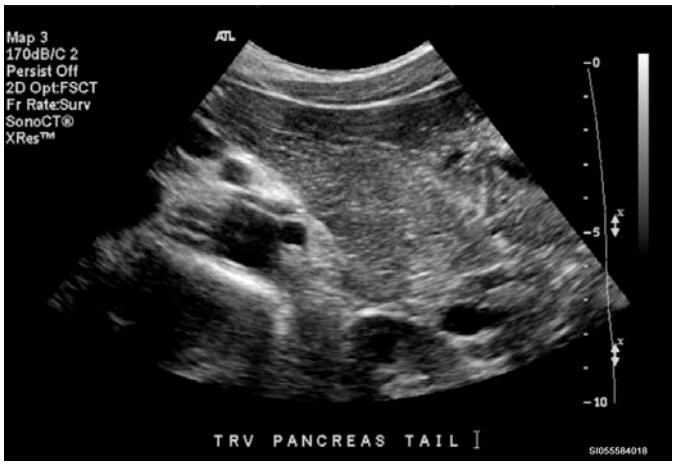

Figure 4–4. Pancreas, tail.

Imaging pancreatitis

Pancreatic inflammation, or pancreatitis, is one of the more common problems afflicting the pancreas. Generally diagnosed clinically and through laboratory analysis, sonography is used primarily to investigate complications or as a follow-up tool to track changes. If CT is unavailable, you may be asked to rule out the presence of pancreatitis.

Acute pancreatitis

The early sonographic appearance of acute pancreatitis will usually display a normal appearing pancreas. However, as swelling increases and enzymes spill out, the organ becomes hypoechoic and enlarged. The head of the pancreas can become so large that it may compress the intrapancreatic portion of the common bile duct. This may cause biliary tree dilatation and jaundice. In these cases, the serum bilirubin and alkaline phosphatase will be elevated.

Complications may produce different sonographic features. Occasionally, acute pancreatitis will cause the organ to hemorrhage and it may appear heterogeneous. In focal pancreatitis, a hypoechoic area resembles a tumor such as adenocarcinoma. However, tumors in the pancreas generally do not cause elevations in amylase; whereas in focal pancreatitis, those levels will increase in the same way as diffuse inflammation. Anechoic or complex areas, representing enzymatic fluid collections, can be seen against the outside of the pancreas. The most common location for fluid collection is between the anterior surface of the pancreas and the stomach. Place a color Doppler box over this area to ensure that you are observing a fluid collection and not the splenic artery; its tortuous appearance can mimic a slender fluid collection. Color Doppler should show a lumen filled with color in a splenic artery while a fluid collection should remain anechoic. If the fluid collection appears to have a thick wall that persists on follow-up sonograms, it is usually called a pseudocyst. The internal echoes of a pseudocyst can range from complex to anechoic. Ensure that the collection is measure in three dimensions.

The pancreatic duct may be dilated in cases of focal pancreatitis that compress a portion of the duct closest to the ampulla of Vater. The duct is dilated if the anteroposterior diameter is greater than 2 to 2.5 mm, depending on your department’s protocol. Otherwise, general acute pancreatitis will compress most of the duct and it will be difficult to see.

Chronic pancreatitis

The sonographic features of chronic pancreatitis range from hyperechoic and hypoechoic patches to diffuse echogenicity. The pancreas tends to become smaller in size and will usually have calcifications. Because the acinar cells are progressively destroyed, enzyme production will be greatly reduced and serum amylase and lipase levels will remain low.

Imaging pancreatic masses

We will briefly examine several common tumors of the pancreas. As with most tumors of the body, sonographic appearance alone cannot distinguish a benign mass from a malignant mass. Most pancreatic tumors prompt radiologists to suspect malignancy. However, there are some clues suggest the presence of benign tumors more than other clues. We will look at the most common masses.

Benign tumors

Microcystic adenomas are well-defined tumors. On sonography, you should either look for an echogenic tumor, possibly with multiple tiny cysts, or search for a mass of separate cysts of 1 to 2 cm arranged around a hyperechoic central scar. The scar will usually have a calcification within it, casting a shadow. Remember that a third of these tumors are found in the head of the pancreas. Use color Doppler over the tumor, as microcystic adenomas should show high vascularity around the periphery and branching from a central scar.

Islet cell tumors are all usually small, hypoechoic tumors. Most are less than 2 cm in size, which makes them extremely difficult to detect with sonography. You will usually see them in any location of the pancreas. Look for solid masses containing cystic areas representing necrosis. Insulinomas are especially difficult to see. Look for these types of islet cell tumors in the body and tail of the hypoglycemic patient.

Malignant neoplasms

Macrocystic adenomas are usually seen in the tail or body. Look for solid components within this mass, as these will sometimes occur. If you see solid elements along the cyst wall projecting into the lumen (papillary excrescences), the mass is probably malignant. Also, you may see a multiloculated tumor that may resemble a collection of cysts; however, you should also see solid elements and possibly debris. This is a good way to distinguish this tumor from a benign microcystic tumor with large cysts. Look for septations as these are suggestive of malignancy.

Adenocarcinoma may compress the common bile and pancreatic ducts. A good way to determine this effect in cases of suspected pancreatic carcinoma is to look for a sonographic sign of dilated ducts and a nontender dilated gallbladder (*Courvoisier's* sign). The mass itself is hypoechoic with irregular borders. Look for small adenocarcinomas to be homogeneous and, as they grow larger, to become increasingly heterogeneous. The serum amylase levels will be normal, distinguishing the tumor from acute focal pancreatitis. Remember that measurements and color Doppler evaluation of the tumor is essential. Also look for ascites and enlarged, hypoechoic lymph nodes around the aorta. The tumor may invade the surrounding tissues in any direction, such as the duodenum, portal vein, and splenic vein. Thus, extremely careful scanning is needed.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

022. Pancreatic anatomy and function

1. Where on the wall of the duodenum does the duct of Santorini attach?

2. What are the primary functions of the pancreas?

3. What are the three main ingredients in pancreatic juice?

023. Pathology of the pancreas

1. Why will amylase and lipase elevate in acute pancreatitis?

2. How does pancreatic adenocarcinoma affect the biliary system?

024. Imaging the pancreas

1. When is a fluid collection considered a pseudocyst?

2. When is the pancreatic duct considered dilated?

3. What are the sonographic differences between microcystic and macrocystic pancreatic tumors?

4. Where else should you look when imaging a pancreatic adenocarcinoma?

4–2. Understanding and Imaging the Spleen

Most of your understanding of spleen abnormality will center on the relationship of its size to disease processes. In this section, we will look briefly at splenic anatomy and function followed by a discussion of splenic abnormalities.

025. Splenic anatomy and function

The spleen is an oval or bean-shaped organ located in the left upper quadrant of the body. It is the largest *reticuloendothelial* tissue in the body; that is, it is the largest example of tissue in the body with the ability to ingest foreign particulate matter and microorganisms. The hilum of the spleen, through which courses the splenic artery and vein, faces the splenic flexure of the colon and the tail of the pancreas. Superior to the spleen is the stomach anteriorly and the diaphragm posteriorly. The inferior border touches the left kidney. The average adult spleen is normally no more than approximately 12 to 14 cm in length. The width of the spleen, from hilum to its lateral side, is approximately 6 to 7 cm. 90 degrees to the width, the thickness is usually no more than 4 cm. As a person ages the spleen may decrease slightly in size.

The spleen is composed primarily of lymphatic tissue called *white pulp*. The white pulp is mostly made of lymph cells and *macrophages*, cells that ingest microbes, particles, and foreign substances in the blood. Along side the white pulp is *red pulp*, composed of venous spaces (sinuses) and splenic cords. The splenic cords contain red blood cells, plasma cells, and some macrophages.

The spleen has the following functions:

- Hemoglobin (red blood cell protein) breakdown and bile pigment formation.
- Filtration of blood through ingestion (*phagocytosis*) of worn blood cells and other particles.

- Formation of antibodies and white blood cells for immunity.
- Blood reservoir.

The spleen can be removed from the body without any danger to survival. Other body structures, such as red bone marrow and the liver, compensate for a missing spleen. However, because it manufactures lymph cells, removing the spleen causes diminished immunity and makes the body particularly vulnerable to blood infection (sepsis).

026. Splenic pathology

Although many abnormalities can affect the spleen, most are usually manifested in two ways: an enlarged spleen or scattered calcifications. Aside from these examples of diffuse pathology, quite a few abnormalities are focally characterized, such as localized infarctions and neoplasms. We will briefly cover the most common splenic pathology.

Splenomegaly

An enlarged spleen is called splenomegaly and it is the most common abnormality of the spleen that you will see while performing sonography. There are many disorders that can cause the volume of the spleen to increase. For example, the spleen will enlarge with infections such as mononucleosis; with malignant blood disorders such as leukemia or lymphoma; with blood anemias such as sickle cell anemia; and even with diabetes. The most common cause of splenomegaly relates to congestion of the spleen found with portal hypertension, usually from cirrhotic livers.

Calcifications

Rarely, the spleen will have *granulomas* or inflammatory nodules; they can be thought of as scars or evidence of past infections. They are typically granular or composed of grain-like particles and quite dense. The most common causes of granulomas in the spleen are *tuberculosis* and *histoplasmosis* infections. Other opportunistic organisms can infect the spleen and cause granulomatous formation, such as the *Candida* fungi also found in the liver, especially in AIDS patients.

Splenic infarction

A particularly common focal abnormality is a splenic infarction, an area in tissue that is dying (necrotic) due to an insufficient supply of arterial blood. The condition has many causes but the most frequent is related to *embolism* of a splenic artery branch. Embolism is the obstruction of a blood vessel caused by an *embolus*, a piece of thrombus or foreign matter. Half of patients with splenic infarct will have multiple sites within the spleen. Patients usually are asymptomatic but can express general discomfort in the area of the left upper quadrant.

Splenic trauma

If the spleen is injured one of two events will occur, each with a corresponding sonographic appearance. Either the capsule or covering of the spleen remains intact or it will rupture. If the capsule remains intact, an injured spleen may bleed, but the blood will remain within the splenic parenchyma. Usually the blood will collect just below the surface forming a subcapsular hematoma. If the capsule ruptures, blood will spill out and may collect around the spleen or spread throughout the abdomen. Most of these intraperitoneal hematomas collect around the spleen and wall themselves off.

Splenic cysts

Occasionally, polycystic disease will place a few cysts in the spleen. All other cases of splenic cysts are rare. Splenic cysts may be congenital in origin or infectious, such as the rare hydatid cyst seen also in the liver. Most splenic cysts you are likely to encounter are due to trauma and are not true cysts, but are hematomas.

Splenic neoplasms

Benign splenic tumors, as with any other tumors of the spleen, are rare. A spleen may harbor a focal mass called a *hemangioma*, which is not a true neoplasm but a congenitally formed mass of proliferating blood vessels. Most focal abnormalities of the spleen will be benign and, as with the hemangioma, non-neoplastic.

Malignant primary tumors that originate within the spleen are extremely rare. However, because the spleen has lymphatic tissue, *primary* lymphoma may occur. If a tumor in the spleen proves to be malignant it will usually be a *secondary* or metastatic disease from lymphoma, skin, breast, or lung cancer. Metastatic spread of *melanoma* (malignant skin neoplasm) to the spleen is the most likely to occur.

027. Imaging the spleen

Spleen imaging is also a part of a routine abdominal examination. The primary focus for most radiologists is the size of the spleen. The presence of splenomegaly can narrow a diagnostic list of possible diseases from infection to malignancy. The sonographic appearance of the spleen can be helpful, but a significant number of diseases have overlapping features. Thus, only a few sonographic clues beyond spleen measurement are particularly useful.

Imaging the normal spleen

Two basic approaches to spleen imaging can be performed. One is an approach with your transducer along the superior and posterior margin of the left side of the body. You will have to use an intercostal approach through the ribs. With the patient lying supine and the left arm raised, this should pose little difficulty. Another approach, particularly in the obese patient, is to raise the patient into a right lateral decubitus and scan from an anterior, subcostal (below the rib margin) approach on the left side.

In your routine examination of the spleen, you should document at least the length from superior to inferior. Frequently, the radiologists will require you to also measure the thickness or width, 90 degrees to the long axis. Tables exist based on age and sex to determine the normal dimensions of the spleen. You will also have opportunity to evaluate the parenchyma for diffuse disease (fig. 4–5).

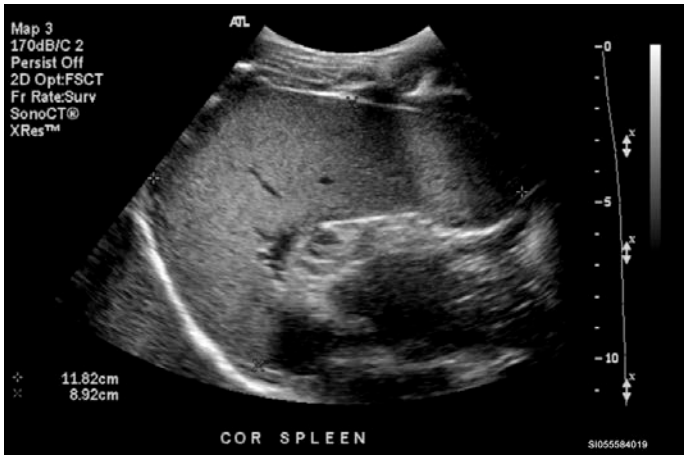

Figure 4–5. Spleen, coronal with measurements.

The echogenicity of the spleen is isoechoic or slightly hyperechoic to the left lobe of the liver, and homogeneously echotextured. You should note the presence of blood vessels entering and exiting the hilum of the spleen. Use color Doppler to note normal flow in this area.

Imaging the abnormal spleen

Diffuse abnormalities, as well as focal abnormalities, occur in the spleen but not as often seen as with the liver. You should keep in mind that most focal abnormalities of the spleen are likely to involve some sort of infectious abnormality, either active or inactive. While cysts can often be seen in the spleen, rarely will you see true neoplasms arising from splenic tissue. Metastatic disease in the spleen is usually a late-stage process and a patient should have manifested metastasis to other locations prior to splenic seeding.

Splenomegaly

Sonography will demonstrate an enlarged spleen filling the left upper quadrant. If your measurement of the splenic length is greater than 12 cm but less than 14 cm, you should combine this finding with laboratory analysis or other patient conditions before suggesting that the spleen is enlarged. Spleens larger than 14 cm, however, are almost always abnormally enlarged. If splenomegaly is suggested, try scanning the area of the hilum. Irregular shaped anechoic areas at the hilum may be dilated vessels. Place a color Doppler box on the hilum and determine if you have a bloom of color. Normally, with color Doppler at medium settings, the splenic artery and vein are the only two vessels seen with a few branches. If the entire hilum seems to light up with color, this is called splenic vein varices and it is an indication of portal hypertension. Also, attempt to follow the splenic vein medially in the body along the posterior border of the pancreas toward the liver for evidence of splenic vein obstruction (thrombosis). Keep in mind that you will not always see varices with splenomegaly caused by portal hypertension.

Calcifications

For spleens with old infections, look for calcifications that may or may not shadow, which should be seen solitary or scattered throughout the organ. Occasionally you will see the calcifications lining the walls of the blood vessels at the hilum. Active infections may not show granulomatous change; instead, you may see a diffuse spread of hypoechoic nodules. Most calcifications are not necessary to measure. However, large solitary ones may need to be measured.

Splenic infarction

With sonography of an early infarct, you should see a classic appearance of a wedge-shaped area hypoechoic to the surrounding splenic tissue. The infarct is almost always in contact with the surface of the spleen. Use color Doppler to confirm the absence of blood flow. Over time an infarct should gradually become more echogenic and decrease in size as a result of healing. Eventually, a healed infarct will either liquefy and become a cyst (which means a regression of sonographic appearance back from hyperechoic to anechoic) or calcify.

Splenic trauma

With sonography, you will mostly see a slightly heterogeneous area, which may resolve into a cyst on follow-up scans. However, in some cases, a subcapsular cyst will be anechoic and may show debris.

Early after rupture, the collection will have an identical echogenicity to the spleen. This may be difficult to see with sonography. With time, the hematoma will become more anechoic and easier to detect on sonography as an irregular collection generally following the contour of the spleen. Most radiologists will require you to attempt to measure any fluid collections of the spleen in three dimensions. Although a massively damaged spleen may appear sonographically to be completely disorganized, do not forget to attempt a measurement of as much of the spleen as possible. Generally, spleen traumas are referred to CT for a more comprehensive overview of the entire organ.

Splenic cysts

If you see a cyst while scanning the spleen of an asymptomatic patient, it is probably the remnant of either an infarct or hematoma from old trauma. The sonographic appearance of a splenic cyst should

fit the criteria for simple cysts elsewhere in the body. All cysts seen should be measured and color Doppler performed to determine vascularity.

Splenic neoplasms

Benign splenic tumors are rare. Usually, benign hemangiomas similar in sonographic appearance to tumors found in the liver will be seen. The splenic hemangiomas are typically hyperechoic but may have a variety of appearances. Ensure that you measure the mass and use color Doppler, particularly with the large heterogeneous types.

If you see hypoechoic nodules spread throughout the spleen, you should be suspicious for the presence of either fungal abscesses (for example, candidiasis) or lymphoma. Otherwise, focal neoplasms are likely to be secondary masses. Rarely will the spleen generate a primary malignancy and so you are not likely to encounter one. Metastases can, like elsewhere in the body, have a range of sonographic appearances. In the spleen, most will be hypoechoic multiple nodules. As with any nodule in the body, measure and use color Doppler.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

025. Splenic anatomy and function

1. What is the significance of the spleen's size in relation to the body?

2. What can be done to the spleen that will *not* have an ill effect on the body?

026. Splenic pathology

1. What are the likely causes of non-shadowing, multiple granulomas seen throughout a spleen?

2. What is the *likeliest* tumor to be seen in the spleen?

027. Imaging the spleen

1. Why follow the splenic vein in cases of splenomegaly?

2. How would you confirm splenic infarct with sonography?

3. What does a cyst seen in the asymptomatic patient represent?

4–3. Gastrointestinal Tract and Transplants

In this section, we will briefly introduce you to sonography of the gastrointestinal (GI) tract. While sonography is not the primary tool for such imaging, it has proven helpful for a number of abnormalities. We will briefly describe how sonographers approach the GI tract in general. We will also provide short introductions to sonography of liver and kidney transplants.

028. Gastrointestinal tract sonography

Most gastrointestinal abnormalities are diagnosed using CT. Occasionally sonograms are helpful for evaluating these problems, particularly appendicitis or various abnormalities in contact with the intestines such as ascites or lymphadenopathy.

The sonographic appearance of the wall of the GI tract corresponds to the layers of the abdominal wall. Alternating layers of wall are echogenic and hypoechoic on sonography, with the hypoechoic areas representing the muscle layers of the wall. The layers have varying thicknesses and, given the contents of the GI tract, may be thinned due to distension. Sonographers recognize that the normal bowel wall is usually no greater than 5 mm without distension. Also the walls are easily collapsible with transducer pressure. Any deviation in thickness and collapsibility raises suspicion for pathology.

One of the more common GI abnormalities sonographers are called upon to scan is *hypertrophic pyloric* stenosis (HPS). This abnormality mostly occurs in male neonates. The terminal end of the stomach, where it connects with the duodenum, is called the pylorus. The muscular walls of the normal pylorus open and close to let food out of the stomach. With HPS, the muscles in the wall grow thicker, permanently lengthening and closing the pylorus and preventing food from leaving the stomach. The patient will vomit forcefully (projectile) and may display an abdominal mass protruding at the epigastrum.

Sonographers look for classic criteria that suggest hypertrophic pyloric stenosis:

- Wall thickness of the pylorus greater than 4 mm.
- Pyloric channel diameter greater than or equal to 1.5 cm.
- Pyloric channel length 1.2 cm or more.

Another abnormality sonographers attempt to see is *acute appendicitis*. An inflamed appendix is an extremely common emergency occurrence, usually diagnosed with CT. However, some physicians prefer the relative quickness and noninvasive nature of sonography to diagnose the condition. Appendicitis is thought to be the result of a fecal calcification within the lumen of the appendix that has migrated from the cecum or bacterial infection. The inflammation can stay strictly within the appendix or spread to the cecum. The danger of appendicitis is the risk that the appendix may perforate and spill toxic inflammatory fluid into the peritoneum.

Patients will usually suffer fever and pain in the right lower quadrant. Sonographers check laboratory values, looking for a signature increase in leukocytes. This information is an important part of the overall diagnostic picture to radiologists.

If a sonographer places transducer pressure directly over a normal appendix, it should collapse into a diameter of a few millimeters. However, with appendicitis, compression will not affect the diameter, which will be greater than 6 mm. Visualization of the appendix in long and transverse views along with diameter measurements is usually required. Calcifications, known as appendicoliths, may also be seen.

In patients with cirrhosis and congestive heart failure, sonographers examine not just the GI tract but also the GI environment for *ascites*, which is an accumulation of serous fluid within the peritoneal cavity. Other diseases, such as neoplasms and infections, are known to cause ascites. Interestingly ascites can be either benign (the majority of cases) or malignant. Sonographers search for ascites within the hepatorenal space or, in women only, a space posterior to the uterus called the pelvic cul-

de-sac. Other less seen locations will be along the flanks, called paracolic (beside the colon) gutters, and anterior to the liver. When ascites comes into contact with the gallbladder, the gallbladder walls tend to thicken.

Other collections of fluid in the abdomen are hematomas and abscesses. Renal abnormalities such as acute pyelonephritis or bleeding angiomyolipoma will cause collections of blood and pus to leak outside the kidneys. For hematomas, sonographic appearance depends on the age of blood, with echogenicity representing clot formation and an echofree collection representing an older collection. Other less common fluid collections are urinomas, found when injured or transplanted kidneys leak urine into the abdomen, or bilomas, collections of bile spilled from a perforated gallbladder. Urinomas and bilomas are generally echofree.

Sonography rarely displays solid masses in the general abdomen. However, careful and slow searching with the transducer may yield evidence suggestive of lymphoma. A common location for sonographers to search for clues of this disease's presence is around the aorta or around the SMA, where hypoechoic masses larger than 1.5 cm represent enlarged lymph nodes. A similar appearance can occur with infections. Other examples are various muscle and nerve tumors both benign and malignant, but largely malignant if seen sonographically.

029. Kidney transplant sonography

Some hospital locations in the Air Force frequently deal with renal transplants, also known as renal allograft. Most of the time, the reason a renal transplant patient would be referred to you is to diagnose the source of kidney failure. To do this, you would have to document the structure and blood perfusion of the transplanted organ.

Generally, sonography is useful for examining transplant complications:

- Vascular (artery stenosis, occlusion, arteriovenous communication or fistula).
- Allograft changes (hydronephrosis, enlargement, and thickened cortex).
- Fluid collections near the allograft (lymphocele is the most common, followed by hematoma, seroma, urinoma, and abscess).

Sonographic evaluation of these complications will involve a combination of Doppler and measurements.

In nearly all cases, you will encounter a kidney superficially located in the right or left iliac fossa. The allograft blood vessels are surgically attached to the recipient's external and internal iliac blood vessels. The point of attachment is called the anastomosis.

030. Liver transplant sonography

A liver transplant is mostly for patients with cirrhosis. Other reasons may include fulminant acute hepatitis, acute liver failure, or sclerosing cholangitis. The recipient may receive a whole liver from a cadaver or a segment from a live relative or cadaver.

Sonography for liver transplant recipients is performed with a different focus both before and after transplantation. Before surgery, sonographers look for main portal vein patency because a narrow or occluded vein will not accommodate attachment to a new liver. Color and spectral Doppler are used to determine flow rates and abnormalities within the portal vein. Because an open portal vein is so critical to the decision to transplant, it is one of the most frequently requested reasons for liver sonography in patients with end stage chronic liver disease. Measurements of the length and diameter of the extrahepatic main portal vein are recorded along with measurements of the liver. Sonographers also check for collaterals and any neoplasms.

The vessels of the donor liver are attached directly end-to-end to the vessels of the recipient. That is portal vein to portal vein, hepatic artery to hepatic artery (or to the recipient aorta if shortened), and

bile duct to bile duct with the help of a stent to allow for monitoring the production of bile into the duodenum.

After surgery, sonography is used primarily to observe flow in the hepatic artery for indications of thrombosis, particularly at the site of anastomosis, which would lead to liver infarction. Sonography also plays a role in evaluating post-transplantation complications such as fluid collections, IVC thrombosis, portal vein thrombosis, rejection, abscesses, surgical hematomas, and biliary obstruction. With the development of complications, patients will show clinical symptoms of fever, pain, and abnormal liver function tests (LFT).

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

028. Gastrointestinal tract sonography

1. List the three classic criteria that suggest hypertrophic pyloric stenosis.

2. What is the diameter of an inflamed appendix?

3. What is *mostly* responsible for ascites?

4. What are some fluid collections found in the abdomen?

029. Kidney transplant sonography

1. List some complications of renal transplants examined by sonography?

2. To what structures are the vessels of transplant kidneys attached?

030. Liver transplant sonography

1. Aside from sonographic portal vein evaluation, what else will sonographers check prior to liver transplantation?

2. List some liver transplant complications.

Answers to Self-Test Questions

022

1. A few centimeters proximal to the ampulla of Vater.
2. To produce insulin and digestive enzymes.
3. Amylase, lipase, and trypsin.

023

1. Inflammation causes amylase and lipase enzymes to leak out into surrounding tissues, where they are absorbed into the bloodstream.
2. Adenocarcinoma, located in the head of the pancreas, may compress the common bile and pancreatic ducts.

024

1. When the collection appears to have a thick wall that persists on follow-up sonograms.
2. Depending on the department protocol, the pancreatic duct is dilated if the anteroposterior diameter is greater than 2 to 2.5 mm.
3. (1) Microcystic tumors are well defined and echogenic with multiple tiny cysts or separate cysts of 1 to 2 cm. The cysts will be arranged around a central hyperechoic scar. The scar will usually have a shadowing calcification. A third of these tumors are found mostly in the pancreatic head.
   (2) Macrocystic tumors are larger than 2 cm, unilocular or multilocular, and found in the body or tail. Some may have papillary excrescences and septations.
4. In the abdomen for ascites and around the aorta.

025

1. The spleen is the largest reticuloendothelial tissue in the body.
2. Removed from the body.

026

1. Tuberculosis and histoplasmosis.
2. Benign hemangiomas.

027

1. For evidence of splenic vein obstruction (thrombosis).
2. Use color Doppler to confirm the absence of blood flow.
3. Remnant of an infarct or hematoma from old trauma.

028

1. (1) Pyloric wall thickness greater than 4 mm.
   (2) Pyloric channel diameter greater than or equal to 1.5 cm.
   (3) Pyloric channel length 1.2 cm or more.
2. Greater than 6 mm.
3. Cirrhosis and congestive heart failure.
4. Hematomas, abscesses, urinomas, and bilomas.

029

1. (1) Vascular.
   (2) Allograft changes.
   (3) Fluid collections near allograft.
2. External and internal iliac blood vessels.

030

1. Collaterals and neoplasms.
2. Fluid collections, IVC and portal vein thrombosis, rejection, and biliary obstruction.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter. When you have completed all unit review exercises, transfer your answers to ECI (AFIADL) Form 34, Field Scoring Answer Sheet.

Do not return your answer sheet to AFIADL.

48. (022) How are the acinar and islet cells arranged within the pancreas?
    a. Islet cells are interspersed throughout acinar tissue.
    b. Acinar cells are interspersed throughout islet tissue.
    c. Islet cells and acinar cells are clustered together throughout beta tissue.
    d. Islet cells and acinar cells are clustered together throughout alpha tissue.

49. (022) The purpose of the islets of Langerhans is to secrete
    a. insulin.
    b. amylase.
    c. cholecystokinen.
    d. sodium bicarbonate.

50. (023) Which condition has the feature of irreversible destruction of tissue?
    a. Fatty liver.
    b. Angiomyolipoma.
    c. Chronic pancreatitis.
    d. Cavernous hemangioma.

51. (024) Which focal abnormality of the pancreas is difficult to detect with sonography?
    a. Macrocystic mucinous cystadenoma.
    b. Microcystic serous cystadenoma.
    c. Adenocarcinoma.
    d. Islet cell tumor.

52. (025) What is the location of the diaphragm in relationship to the spleen?
    a. Inferior.
    b. Anterior and inferior.
    c. Superior and posterior.
    d. Lateral.

53. (026) What allows hematomas to form from splenic trauma?
    a. Splenic artery aneurysm.
    b. Splenic capsule rupture.
    c. Splenic calcifications.
    d. Splenomegaly.

54. (027) What should you see with a splenic infarct using sonography?
    a. Splenomegaly.
    b. Enlarged kidney.
    c. Ascites and jaundice.
    d. A hypoechoic wedge.

55. (028) The walls of the pylorus in hypertrophic pyloric stenosis
    a. grow thicker.
    b. stretch thin.
    c. perforate.
    d. calcify.

56. (028) How will transducer compression affect an inflamed appendix?
    a. It will collapse the appendix.
    b. It will not change the appendix.
    c. The appendix will rebound.
    d. The appendix will perforate.

57. (028) What is the most likely location for sonographers to look for ascites?
    a. Kidney hilum.
    b. Pancreatic duct.
    c. Hepatorenal space
    d. Gallbladder lumen.

58. (029) What is an allograft?
    a. Transplant.
    b. Catheter.
    c. Shunt.
    d. Stent.

59. (029) Where in a recipient's body will a sonographer encounter a transplanted kidney?
    a. Against the gallbladder.
    b. Next to the native kidney.
    c. Within the right or left iliac fossa.
    d. Between the superior mesenteric artery (SMA) and aorta.

60. (030) After liver transplantation sonography is primarily used for evaluation of
    a. hepatic artery.
    b. biliary ducts.
    c. hepatic vein.
    d. gallbladder.

Student Notes

Glossary

Terms

anatomy – Description of the form and structure of an organism and its parts.

aneurysm – A sac formed by the dilatation of the wall of a blood vessel.

anteroposterior – The direction of the ultrasound beam as it travels through the patient from anterior to posterior or front to back.

axis – The central line of the body or any of its parts.

bifurcate – Forked or having two branches.

caudal – Toward the lower part; in the body, toward the feet.

coronal – Frontal plane; a vertical imaginary plane that divides the body into anterior.

Couinaud's anatomy – Segmental anatomy that divides the liver into sections based on blood supply and drainage.

curvilinear array – A collection of active elements within a single transducer housing arranged in an arc that give the imaging plane a natural sector shape.

decubitus – The position of the patient in lying on a side.

distal – Distant part of a limb or organ.

document – In sonography, the act of recording an image.

exocrine – Secretion into ducts.

falciform ligament – Anchors the liver to the diaphragm superiorly and to the abdominal wall anteriorly.

gain – A sonographic technique that allows brightness adjustment of return echoes.

gastrointestinal – Of or relating to the stomach and intestines.

Glisson's capsule – Fibrous peritoneal covering of the liver.

habitus – Physical characteristics of a person.

hiatus – An opening.

hilum – Slit-like opening in an organ through which passes nerves and vessels.

hypoechoic – In sonography, an image echo reduced in brightness relative to surrounding.

iliac arteries – Two arteries resulting from the bifurcation of the abdominal aorta.

ligamentum teres – The obliterated remnant of the fetal umbilical vein.

linear array – A collection of active elements within a single transducer housing.

longitudinal – Running lengthwise along the long axis of the body.

lumen – The potential space in a blood vessel through which blood travels. Also the fluid-filled space or cavity of a cyst or tube.

luminal – The surface of the tunica intima that faces the lumen.

parietal peritoneum – A layer of peritoneum that lines the abdominal cavity.

pathology – Science concerned with the nature, cause, and development of abnormal.

physiology – The study of normal function in living organisms.

porta hepatis – A slit in the inferomedial surface of the liver.

Portal triad – Hepatic arteries, portal veins, and hepatic ducts are all sheathed together in connective tissue.

proximal – Nearest the trunk or point of origin.

ramify – Branching.

rectus abdominis – Anterior muscle of the abdominal wall.

Reidel's lobe – A normal extension of the right lobe as far caudal, the direction toward the feet, as the iliac crest.

renal arteries – Lateral branches off the left and right sides of the abdominal aorta.

retroperitoneal – Area posterior to the peritoneal cavity.

scan – In sonography, the act of sweeping the ultrasound beam through the body.

sector scanner – A transducer that produces a fan-shaped image field.

superior mesenteric artery – Second major branching off the abdominal aorta's anterior surface.

survey – In sonography, the act of examining an entire area of interest before obtaining images.

systemic circulation system – Hepatic veins and arteries.

transducer – The structure of arranged, crystal elements housed inside a probe.

transverse – Lying across the long axis of the body.

ventral – Anterior surface.

Abbreviations and Acronyms

AFP	alpha fetoprotein
AIDS	acquired immunodeficiency syndrome
ALP	alkaline phosphatase
ALT	alanine aminotransferase
AP	anteroposterior
ATN	acute tubular necrosis
AST	aspartate aminotransferase
BUN	blood urea nitrogen
CCK	cholecystokinin
CHD	common hepatic duct
cm	centimeter
CT	computed tomography
FNH	focal nodular hyperplasia
GI	gastrointestinal
GSD	glycogen storage disease

HAV	Hepatitis A virus
HBV	Hepatitis B virus
HCC	hepatocellular carcinoma
HCV	Hepatitis, type C
HDL	high-density lipoproteins
HIV	human immunodeficiency virus
HPS	hypertrophic pyloric stenosis
IVC	inferior vena cava
LDL	low-density lipoproteins
LFT	liver function tests
MHz	megahertz
mm	millimeters
PT	prothrombin time
RCC	renal cell carcinoma
RUQ	right upper quadrant
SMA	superior mesenteric artery
SMV	superior mesenteric vein
TCC	transitional cell carcinoma
TGC	time gain compensation
TIPS	Transjugular Intrahepatic Portosystemic Shunt
UPJ	ureteropelvic junction
UVJ	ureterovesical junction
VLDL	very low-density lipoproteins
WES	wall-echo-sign

Student Notes

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