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BEYOND THE sonographic examination of the structures within the abdomen, sonographers are often called on to evaluate the rest of the body's parts. Sonography has proven extremely useful for helping physicians diagnose the cardiovascular system, the male reproductive system, and superficial glandular structures like the thyroid and breast. This volume will cover these general topics in sonography.

Unit 1 covers the basic characteristics of moving blood and cerebrovascular imaging. Unit 2 provides the basic knowledge you will need to perform sonographic examinations of the abdominal aorta, the aortic branches, the venous vessels of the abdomen, and the blood vessels in the upper and lower extremities. Unit 3 covers the major glandular small parts of the body such as thyroid, testicle, and breast. Unit 4 briefly describes some of the specialized roles of sonography.

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This volume is valued at 18 hours and 6 points.

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.

Page

Please read the menu for Unit 1 and begin. ➔

Unit 1. Cerebrovascular

MEDICAL sonographers are frequently called upon to examine the flow of blood in the body. They evaluate blood vessels directly using the same instrumentation they use for organs such as the liver. The principles of ultrasound physics also remain the same except for one significant difference: blood is imaged and measured while in motion. Therefore, a slightly different approach must be used. While routine sonography is still the method for examining abnormalities that affect the structure of blood vessels and surrounding tissue, vascular sonography that detect abnormalities of blood flow uses different instrumentation. For this reason, you should be familiar with the basic characteristics of moving blood.

1–1. Vascular Physics

This section will briefly examine basic characteristics of arterial and venous blood flow and their relationships in terms of pressure and resistance. By familiarizing yourself with these principles, you will improve your understanding of normal and abnormal blood flow throughout the body.

201. Arterial hemodynamics

Blood is composed of plasma (fluid), red blood cells (erythrocytes), white blood cells (leukocytes), and platelets (thrombocytes). Blood transports oxygen, nutrients, and wastes throughout the body; as well as other substances, hormones, and enzymes that perform various functions. For blood to carry out its transportation function, it must be able to move or circulate through the body. Because of the complexity of blood flow in the human body, we will only briefly cover the most basic topics. As with all fluids, three factors affect blood flow:

1. Pressure.
2. Resistance.
3. Volume.

All three factors influence blood flow *simultaneously*. However, for convenience, this lesson will cover a few things about each one separately.

Pressure

Pressure is the amount of force that will raise a column of mercury (Hg) 120 millimeters (mm) against gravity within a glass tube called a manometer. Because fluid flows from high pressure areas to low pressure areas, or the path of least resistance, we can say that pressure provides the drive for fluid motion. For example, in a tube with high pressure on one end and low pressure at the opposite end, fluid will move from high to low because of the difference in pressure.

In the human body, the high pressure areas are the left ventricle of the heart, the aorta, and the large arteries. As the heart contracts, the left ventricle pumps blood into the aorta and its large branches. The arterial walls expand to accompany the increased fluid volume. The normal peak amount of force

or pressure generated by the heart contraction is 120 millimeters of mercury (mm Hg). Another term for this contraction is *systole*. When the contraction is relaxed, it is termed *diastole*.

The high pressure end of left ventricle, aorta, and large arteries is in direct contrast with the low pressure end of the inferior vena cava and right atrium of the heart. Because the circulation system is a closed, systemic circuit, blood flows forward continuously throughout its arterial and venous branches, from high pressure to low pressure. That is, a pressure difference continues to exist in systole and diastole, which causes continued forward flow (fig. 1–1).

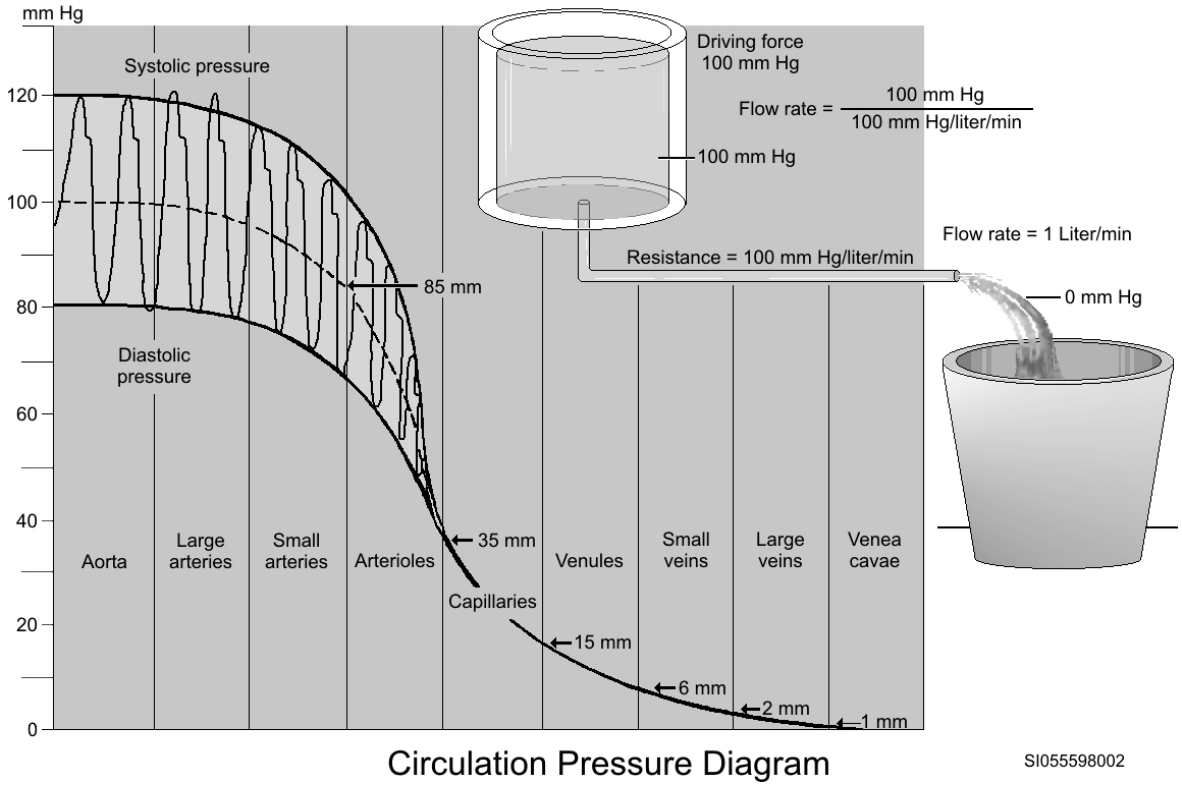

Figure 1–1. Circulation pressure diagram.

Resistance

Although normal blood flow is continually forward, elements exist which resist blood flow, even to the point of temporarily reversing it. The property of fluid that resists forward flow is called *viscosity*. Viscosity can be thought of as a property similar to the thickness of engine oil. Blood is a few times more viscous than water. Without this resistance, blood would flow forward with a continuous amount of high level energy. With resistance, the energy is reduced as the blood travels.

Because blood travels in concentric layers, or laminae, and because the central layer is thought to be faster than the other layers, blood cells produce a certain amount of friction with each other. The friction of red blood cells against each other and against arterial walls, where the velocity of blood is assumed to be zero, is one of the contributing factors that produce drag or viscosity.

Volume

Volume of blood is the amount of blood that circulates or flows throughout the body per minute. It is determined by the pressure *gradient*, the difference between high pressure areas and low pressure areas. It is also determined by the amount of resistance to flow.

Poiseuille's equation

Again, the three factors of pressure, resistance, and volume, influence blood flow simultaneously. The relationship between the three factors can be expressed in Poiseuille's equation:

$$V = \frac{\Pi r^4 (P_1 - P_2)}{8L\eta}$$

where V is the volume of flow; P_1 and P_2 are the proximal and distal fluid pressures, respectively, in a tube (pressure gradient); r is the radius of the tube or the line distance from the center of the tube to its surface; L is the length of the tube; η is the viscosity of the fluid.

Although this equation applies to steady flow states of low viscosity fluids, such as water, within uniform tubes, we can adapt it to blood flow within the arteries. Thus, we may derive certain relationships from the equation.

Because the length of blood vessels and the viscosity of blood remain constant, the parameters that mostly affect blood flow in the body are the radius of vessels and their pressure gradients. For example, a small change in a vessel's radius can result in a four times larger change in flow. Thus, as the radius decreases, the resistance increases because resistance in a blood vessel is largely a result of vessel dimension and the constant viscousness in blood. This fact provides another relationship between resistance and flow; that is, as resistance increases, the volume of flow decreases.

Peripheral resistance

Blood leaves the left ventricle and travels through the arteries in a pulsatile manner due to the pumping action of the heart. Keep in mind that the aorta and *large* branching vessels off the aorta offer little resistance to the high energy flow. In fact, the systolic pressure is increased due to the increasing stiffness of the vessel walls.

However, as blood exits the large arteries and enters the smaller diameter arterioles and capillaries, also known as vascular beds, resistance increases. By the time flow reaches the even tinier diameter capillaries, pressure drops significantly as blood enters the venules. The ability of the arterioles to constrict or dilate (*vasoconstriction* and *vasodilation*, respectively) controls the volume of blood that enters the capillary network. For example, in vessels leading to high-resistance vascular beds, such as the external carotid arteries, flow will temporarily reverse during diastole. Because the face does not require as much blood as the brain, facial blood vessels will constrict against the high flow. Flow traveling to low-resistance beds, such as that found within the internal carotid arteries, has continued forward flow in diastole. This is because of the brain's requirement for continuous blood supply.

202. Venous hemodynamics

Venous blood flow is blood that travels back to the heart. Capillary networks merge into small venules through which venous blood courses into increasingly larger veins, eventually terminating into the two main venous vessels attached to the heart (the superior and inferior venae cavae). Vein walls are more flexible than arterial walls, and the diameter of most major veins are larger than comparable arteries.

Venous blood moves through this system at progressively reduced pressures. For example, venous flow travels in the inferior vena cava (IVC) through the abdominal and thoracic cavities. By the time flow reaches the right atrium of the heart, it is at zero pressure. How does it continue to flow with the small amount of force pressure behind it?

The vein walls have valves that assist in preventing blood from reversing course. Also, respiration and skeletal muscle contractions help prevent venous blood from reversing. Respiration creates a difference in pressure (pressure gradient) between the portion of IVC passing through the thoracic cavity and the portion of the IVC passing through the abdominal cavity. The motion of the diaphragm

during inspiration increases the size of the thoracic cavity and reduces the intra*thoracic* pressure on the IVC. Simultaneously, the same diaphragm motion decreases the size of the abdominal cavity and increases the intra-*abdominal* pressure on the IVC. Thus, during inspiration, the pressure gradients between the two cavities propel blood back to the heart—from the high pressure intra-abdominal IVC to the low pressure intrathoracic IVC. There are six major respiration effects on venous flow and pressure as illustrated in the following table:

Respiratory Effects on Venous Pressure and Flow			
Respiration	***Pressure and Flow (volume)***	***Area Affected***	***Cause***
Inspiration	Volume ↑ Pressure ↓	Venous flow within thoracic cavity	Reduced intrathoracic pressure due to descent of diaphragm
Expiration	Volume ↓ Pressure ↑	Venous flow within thoracic cavity	Increased intrathoracic pressure due to ascent of diaphragm
Inspiration	Pressure gradient between peripheral vessels and the abdomen is decreased Reduced flow in lower peripherals	Venous flow within abdominal cavity and lower venous extremities	Increased intra-abdominal pressure due to descent of diaphragm
Expiration	Pressure gradient between peripheral vessels and the abdomen is increased Increased flow in lower peripherals	Venous flow within abdominal cavity and lower venous extremities	Decreased intra-abdominal pressure due to ascent of diaphragm
Inspiration	Pressure gradient between upper extremity vessels and the right atrium is increased Increased flow in upper extremities	Thoracic cavity and upper extremities	Reduced intrathoracic pressure due to descent of diaphragm
Expiration	Pressure gradient between upper extremity vessels and the right atrium is decreased Decreased flow in upper extremities	Thoracic cavity and upper extremities	Increased intrathoracic pressure due to ascent of diaphragm

As the table describes, the respiratory effect on venous blood flowing in the upper extremities is directly opposite that of the lower extremities. When you breathe in, venous flow from the legs into the abdomen stops. When you breathe out, flow resumes. The opposite occurs simultaneously in the arms.

Without continuous venous flow toward the heart due to respiratory changes in pressure and lower extremity muscle contractions, the weight or the effect of gravity on the entire column of blood from the heart to the distal extremities (hydrostatic pressure) would increase. Blood would pool in the gravity dependent lower extremities and dangerously reduce the volume of blood returning to the heart.

As mentioned before, the valves within the veins prevent blood reversal. When the leg muscles contract, venous blood moves up against hydrostatic pressure. Thus, the only thing to prevent hydrostatic pressure from forcing the blood back into the legs during muscle relaxation is the venous valve.

Self-Test Questions

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

201. Arterial hemodynamics

1. What factors affect blood flow simultaneously?

2. How does pressure drive fluid motion?

3. What parameters of Poiseuille's equation affect blood flow?

202. Venous hemodynamics

1. How is venous blood prevented from reversing?

2. What causes increased flow in the lower extremities during expiration?

1–2. Cerebrovascular Imaging

The primary purpose of the cerebrovascular system is to supply the brain with a continuous and abundant amount of oxygenated blood. To do this, blood must perfuse the brain with little resistance at high velocities. There are several main routes from the heart to the brain among the other vessels that supply head structures outside of the brain.

As sonographers, we are often called on to examine the primary routes that lead to the brain. This section will examine the principal part of the cerebrovascular system, the carotid arteries. We will quickly touch on the basic anatomy and functioning of the cerebrovascular system before moving on to the pathology and sonographic examination.

203. Cerebrovascular anatomy and physiology

The cerebrovascular system anatomy begins with the aorta and ends with the internal jugular veins. In sonography, we generally focus on the arterial system without neglecting the importance of certain venous structures. As we go along, we will also mention the most sonographically important veins that drain the head and neck.

Cerebrovascular anatomy (arterial)

The largest arterial vessel in the human body is the aorta, and its first part, the arch, has three major branches, which send blood to the head and neck at high velocity. The first vessel off the aortic arch is called the *brachiocephalic trunk*, which used to be called the innominate artery. This vessel travels slightly to the right and then bifurcates into right subclavian artery and right common carotid artery (CCA). Another way to describe it would be to say that the right CCA branches off the brachiocephalic trunk and that the trunk distal to this point continues on as the right subclavian artery. The right subclavian continues into the right arm while the right common carotid artery courses up the right side of the neck. The carotid is the primary artery of the neck.

The second vessel that commonly branches off the aortic arch is the *left CCA*, which courses up along the left side of the neck. The left CCA sometimes either branches directly off the brachiocephalic trunk or shares its origin with the aortic arch. The third branch of the arch is the *left subclavian artery* that runs into the left arm.

The two CCAs lie deep behind the sternocleidomastoid muscles and laterally to either side of the thyroid gland. The CCA has no branches but, occasionally, the superior thyroid artery may course off it. The CCA is straight throughout its length. Typically, the CCA will split (bifurcate) at the most proximal level of the thyroid cartilage into the internal carotid artery (ICA) and external carotid artery (ECA).

The ICA usually lies lateral and posterior to the ECA, but variations occur and you should not become complacent about the position of both vessels. The ICA usually courses straight but can have tortuous kinks and loops, which are considered normal variants. Frequently, the diameter of the ICA is slightly wider at its origin compared with the diameter of the rest of the vessel; this is an area that sonographers call the carotid bulb.

The ICA in the neck is branchless until it penetrates into the skull cavity, where it gives off the ophthalmic, middle cerebral, and anterior cerebral arteries. The cerebral branches of the ICA are the anterior and lateral parts of a communicating circle of vessels called the cerebral arterial circle, or the *circle of Willis*, located within the skull base. The ECA, on the other hand, has eight branches that start almost immediately after its origin in the neck, the first of which is usually the superior thyroid artery. With the first branch, the diameter of the ECA dramatically reduces.

The vertebral arteries originate off the left and right subclavian arteries. They travel through the foramen of the cervical vertebral bodies, usually from C6 to C1, into the cranial cavity. The diameters of both vertebrals are frequently asymmetric, with the left being commonly larger. One vertebral artery may be congenitally absent. After the vertebrals reach the inferior surface of the brain, they merge to form the basilar artery. The basilar artery's terminal bifurcation forms the posterior portion of the circle of Willis (fig. 1–2).

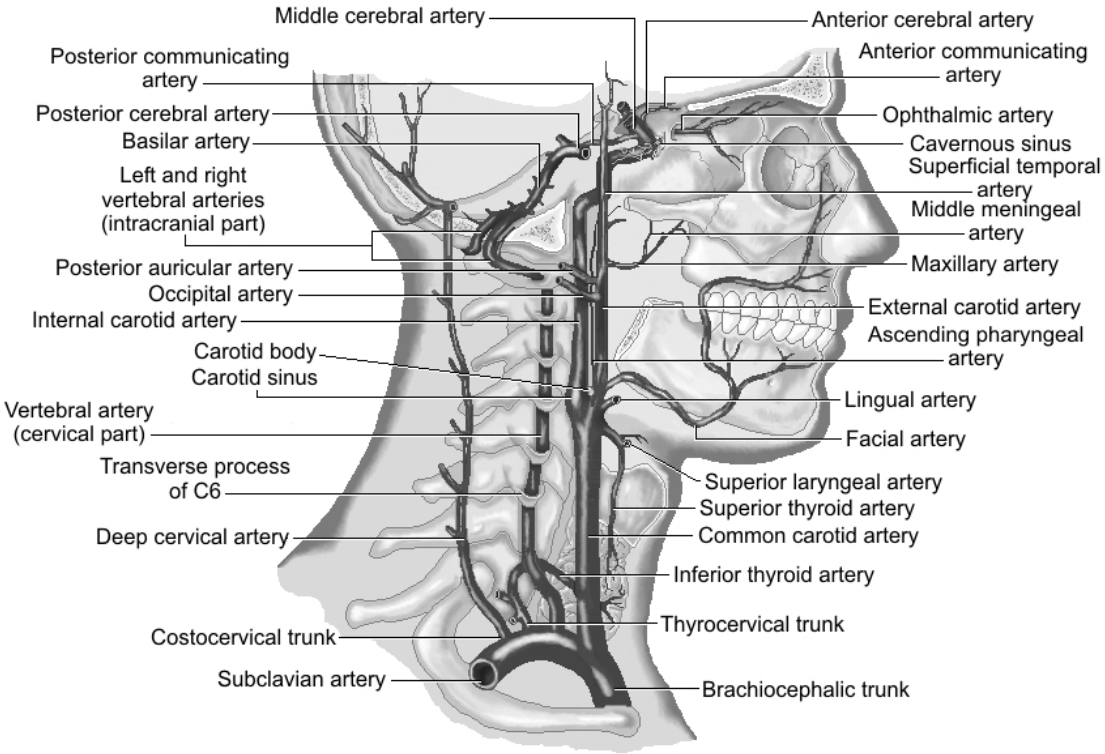

Figure 1–2. Arteries to brain.

Cerebrovascular function

The primary purpose of the cerebrovascular system is to supply the brain with oxygenated blood continuously. The principal route for blood pumped from the heart is through the CCAs and into the ICAs. The intracranial branches of the ICAs supply blood to the eyeballs, ears, most of the cerebrum and pituitary gland. The posterior brain is supplied with blood through the vertebral arteries. Because the brain requires uninterrupted blood supply, there is low resistance to the blood flow in the ICAs and vertebrals. Therefore, flow moves forward at a relatively high velocity compared with the rest of the body, even in diastole, the period of cardiac relaxation.

Although the ECA is the other half of the CCA bifurcation, it does not supply blood to the brain. Instead, its branches supply blood to the face, scalp, and neck. Because the face, scalp, and neck do not require blood at the same rate as the brain, it resists the incoming flow. In diastole, forward flow through the ECA is nearly stopped and may temporarily be reversed.

After the blood is used by the brain, it travels through veins that drain into a series of six dural venous sinuses. The dural venous sinuses are located within the dura mater, the fibrous outer covering of the brain. Most of the deoxygenated blood that drains from the dural venous sinuses passes through neck via the *internal* and *external jugular veins* (IJV and EJV, respectively).

The internal jugular veins run inferiorly down either side of the neck lateral to the carotid arteries. They join with the right and left subclavian veins behind the clavicles to form the right and left brachiocephalic veins. Much of the blood from the brain returns to the heart though the IJV.

The EJV drains blood from the face and scalp. The EJV is located superficially, or close to the skin, as it travels down the neck anterior to the sternocleidomastoid muscle. It merges with subclavian vein in the mid-clavicle region.

Finally, the vertebral veins drain nearly all of the structures in the neck. The vertebrals course parallel with the vertebral arteries through the cervical vertebral foramina and merge with the brachiocephalic veins.

204. Carotid pathology

The normal function of the carotid arteries can be interrupted by abnormalities. In this lesson, we will briefly look at some of the pathology that can occur with carotids and surrounding tissue. Familiarity with carotid pathology will help you to scan both normal and abnormal carotid arteries properly.

Indications

The basis for initiating the carotid sonogram, or the indication of an abnormality, includes stroke, transient ischemic attack (TIA), amaurosis fugax, cervical bruit, pre-operative evaluation, follow-up of known carotid disease, and post-operative follow-up. We will briefly discuss the most common symptoms: stroke, TIA, and amaurosis fugax.

Stroke

Stroke, or brain attack, is a sudden interruption of normal cerebral circulation lasting more than 24 hours. Usually, a stroke will cause irreversible brain damage (infarction). There are two types *ischemic* and *hemorrhagic*. The ischemic type involves the blocking or narrowing of an artery to such a point that either it stops blood from reaching a portion of the brain or it severely reduces the amount. Most strokes are of this type. The hemorrhagic stroke involves ruptured vessels that spill blood into or around the brain, putting damaging pressure on brain tissue. Whereas physicians prefer to use computed tomography (CT) to detect hemorrhagic strokes, sonography is useful primarily for cases of ischemic stroke.

One of the most common causes of stroke is *atherosclerosis*, the most common form of *arteriosclerosis*. Arteriosclerosis is the hardening of the arteries. Atherosclerosis is characterized by deposits of fat (lipids) within the inner layer (intima) of a blood vessel wall. The build-up of deposits

can cause narrowing of a vessel to the point where flow is severely reduced or blocked; thus causing the stroke. Also, pieces of fat deposition may break off within the larger extracranial vessels, forming *emboli* (clots) which travel to the smaller cerebral vessels and block (occlude) them.

Stroke can be caused by hypertension that damages vessels, contributing to atherosclerotic emboli formation. Another cause is valvular heart disease, which can form thrombus or clots around heart valves. Other causes for stroke are smoking, which also damages vessel walls, and a family history of stroke victims.

Some of the symptoms of a stroke are based upon the hemisphere of the brain affected. For instance, an infarction on the right side of the brain may cause neurological defects or symptoms on the left side of the body. The opposite would be true for the left side of the brain. A patient may have one-sided (unilateral) *paresis*, or weakness in the body and partial paralysis. Another symptom may be unilateral *paresthesia*, or a burning, tickling, or prickling sensation in the skin. *Aphasia* is a symptom where the patient has difficulty speaking or cannot speak. Abnormal or absent vision is a symptom called *amaurosis fugax*.

Transient ischemic attack

When the previously discussed neurological symptoms are fleeting, followed by complete recovery *within* 24 hours, the event is called a transient ischemic attack. Transient ischemic attacks can last anywhere from minutes to hours. They are more difficult to detect because many of the symptoms can be caused by diseases other than ischemia.

Amaurosis fugax

As stated before, amaurosis fugax is abnormal or absent vision. This blindness can be a partial or total blindness of one eye. Unlike the other symptoms, the origin of a possible embolism causing the blindness may be the internal carotid on the same side (ipsilateral). Carotid insufficiency due to a narrowing of the vessel buildup of plaque may also reduce flow to the orbit.

Abnormalities of the carotid

The above symptoms can appear with other disease processes that do not affect or are unconnected to the carotid arteries. However, physicians will usually refer these cases to the sonographer to rule out a direct insult on the carotid vessels. Some of the *most* common abnormalities that can cause significant obstruction of blood flow are the following:

- Plaque.
- Stenosis.
- Dissection.
- Subclavian steal.

Plaque

A longitudinal sonographic image of a normal carotid artery will display an inner wall layer, a millimeter-thin echogenic line that runs closely parallel to the outer wall. Any thickening of this inner layer, or intima, is considered atheromatous plaque.

Plaque is composed mostly of fat cells, or lipids, and fibrous cells (collagen, a connective tissue). As already hinted, the danger posed from plaque is its accumulation to the point of significantly narrowing the lumen of the carotid. Another danger is the formation of an ulcer, which may, for various reasons, shed pieces that could embolize to the brain. For example, the plaque ulcer may hemorrhage internally, causing the plaque to grow in size. Necrosis and hemorrhage may cause platelets to attach to the plaque and form clots. Either clots or plaque tissue may break off and travel to the brain as emboli, which is the most common cause of TIA.

Stenosis

Stenosis is the narrowing of the carotid lumen through either plaque buildup or an abnormality that closes or cuts off the open vessel pathway. Closing off the lumen can significantly affect the circulating blood (hemodynamically significant) and compromise the proper functioning of the brain. Stenosis of the ICA or even the CCA can become so bad that the lumen will be blocked off nearly or completely. Either plaque or thrombosis can cause the carotid to become obstructed.

Most physicians would prefer not to wait for the development of a stenosis to grow to the point of blocking a carotid artery. Therefore, many symptomatic patients are candidates for surgical removal of the plaque when stenosis has reached 70 percent of the internal diameter of the vessel. The surgical procedure used for carotid plaque removal is called an *endarterectomy*, which removes the intimal and most of the medial layers, leaving a smooth lining. Although the level when narrowing begins to affect the volume and rate of blood flow is above 50 percent, most patients do not benefit from surgery until 70 percent of the vessel diameter is blocked and flow is seriously challenged. Thus, plaque buildup has to be a hemodynamically significant stenosis for surgery to be performed.

Other carotid abnormalities

Aside from stenosis, other carotid abnormalities occur that affect blood flow such as dissection and subclavian steal. Along with your typical carotid scanning, you may find yourself confronted with one of these abnormalities. So that you are familiar with them, we will briefly discuss each one.

Dissection

A dissection of the carotid artery is a separation of the arterial wall. This separation is caused by a tear in the intima, which pulls the layer away from the media. Sometimes the medial layer is split or torn away from the outer or adventitia layer. When the defect occurs, blood enters the split in the wall, creating a false lumen. The false lumen may have only one opening, the original tear, for blood to enter, or it may have another opening that connects back to the true lumen. The intimal tear can collect thrombus, which may build up and possibly shed emboli.

Dissection of the carotid artery is caused by violent trauma to the neck, by degenerative diseases that weaken the arterial wall, by spontaneous unknown sources, or by dissection extending from the aorta.

Subclavian steal

Carotid sonography standard practice includes combined spectral, color, and grayscale images of the vertebral arteries. These arteries largely feed the posterior brain. Their evaluation, which can be extremely difficult, help make a diagnosis for associated neurologic symptoms. The primary usefulness for performing sonography on the vertebrals is to determine direction of blood flow.

Normally, vertebral flow is cephalic or forward toward the cranium. Vertebral spectral waveforms resemble ICA waveforms in that low-resistance flow is displayed. When one of the vertebrals displays a reversal of flow in a direction away from the cranium, this is a sign of blockage in the ipsilateral or same-side subclavian artery or brachiocephalic trunk. This phenomenon is known as *subclavian steal*, where the vertebral arteries are used as collateral pathways for blood flow that is blocked from its normal course.

Subclavian steal phenomenon is the term used to describe when plaque or thrombus block or cause severe stenosis of the proximal portion of the left sublcavian artery (the portion closest to the aortic arch) and the brachiocephalic trunk on the right. Occlusions of these vessels prevent arterial flow from flowing into either arm. Usually the blockage occurs unilaterally, mostly in the left subclavian artery.

The thing to remember is that blockage is in the *proximal* subclavian artery or brachiocephalic trunk, the portions of the vessels *before* the branching of the respective vertebral arteries. Thus, the left distal subclavian or the right subclavian will, via the vertebral artery, steal blood from the basilar artery located in the base of the brain. Blood in the vertebral artery connected to normal subclavian or

brachiocephalic trunk will flow forward in a normal fashion into the basilar artery, but a portion of the flow will reverse and enter the opposite vertebral (which would otherwise have no flow due to the blockage below). This reverse flow will then enter the distal subclavian on the left or the subclavian on the right.

205. Imaging normal and abnormal carotids

The carotid sonogram is extremely operator dependent. The skills that you need to perform carotid examinations require a lot of practice and knowledge of anatomy and pathology. At the heart of carotid sonography is the need to discover the abnormality hindering or blocking the normal flow of blood through the carotid arteries to the brain. Because this abnormality must be discovered indirectly from moving blood or directly from images of structures, or both, your approach should be as consistent and as accurate as possible.

Process before the carotid examination

Given the many causes of carotid disease and their various symptoms, it makes sense to interview the patient so that you may be able to evaluate the carotid system properly. Radiologists will be able to determine possible diagnosis better if they know backgrounds to examinations you perform. For this reason, questionnaires are just one of many ways to obtain this information. Age, family history, symptoms, medications, and patient condition should be obtained before the sonogram through informal interview, medical records, or from the written request.

Although there is no patient preparation for carotid examinations, you may find it helpful to keep certain things in mind concerning the patient. Be aware of limitations that may prevent you from obtaining optimum images and information. For example, short and thick neck muscles may make it difficult for ultrasound to penetrate to the carotids; recent surgeries may cause items such as surgical staples and dressings to be placed on the neck, which may limit your visibility of the carotids; or patients may not be able to lie flat because of medical conditions like arthritis of the neck and chronic obstructive pulmonary disease (COPD).

Another important step before performing the carotid examination is to review the findings of any previous diagnostic examinations of the carotids. This will give you an idea of the carotid anatomy of an individual and it will prepare you for any potential surprises. Try to select the same equipment and settings that were used previously.

Once the patient arrives for the examination, have him or her lie supine with his or her neck and upper shoulders comfortably supported with pillows or rolled-up towels. This action should help extend the neck and increase visibility of as much of the carotid arteries as possible. Placing a pillow beneath the head of the patient will likely cause the patient’s jaw to come down and interfere with your access to the neck. Many technologists will sit beside their patients’ heads; others will sit alongside the flanks of patients. There may be slight variations of these two methods of examining. The important thing is to choose one that allows you comfortable and easy access to the neck.

As with other sonography examinations, make sure that your equipment is set up and optimized for vascular studies. Keep in mind that your color and spectral Doppler filters and settings should already be at standard presets. As you proceed with the examination, then you should adjust these parameters to the patient. Typical transducers used in most sonography sections that perform carotid examinations are linear array probes with frequencies of at least 5 megahertz (MHz). The transducer should have the capability to drop the frequency down to 3 MHz for Doppler studies.

Grayscale imaging standard views

Each sonography section may have its own protocols for carotid examination, many of them based upon the preferences of the interpreting radiologists. We will point out the most typical standard protocol for grayscale (sometimes called real-time) imaging of the carotids.

The optimal grayscale appearance of the carotid artery is of a completely anechoic lumen bounded by echogenic walls. There should be a hypoechoic line within the wall that represents media layer of the artery. Thus, the echogenic line facing the lumen is the inner or intimal layer and the echogenic line on the wall side of the media layer represents the outer or adventitia layer. The carotid will be found medial to the internal jugular vein. The jugular vein can be identified by noting the periodic change in its diameter resulting from respiration. Also, with slight pressure from your transducer, the jugular vein may collapse. The jugular vein's wall is not readily contrasted with surrounding tissue because of its thinness, unlike the thick echogenicity found with carotid artery walls.

The purpose of scanning the carotid in grayscale is to document the structure of an individual's carotid and to locate abnormalities such as plaque. Depending upon the orientation of the carotid artery within the neck, a sonographer may place the transducer on the anterior, lateral, or posterolateral surface of the neck to access as much of the carotid artery as possible. Holding the transducer transversely and angling the surface of the transducer caudally, or toward the feet, against the clavicle should allow you to obtain an image of the origin of the CCA on the right side. The left CCA origin is deeper in the chest as it comes off the aorta and extremely difficult to see. At the opposite end of the neck, against the mandible angling up, or cephalad, is a good way to obtain a longitudinal image of the distal ICA.

Before documenting the carotid arteries, it is a good idea to sweep the transducer through the entire length of the vessel, both longitudinally and transversely. This will help you to locate any abnormality and to plan how you will document the abnormality with your standard views.

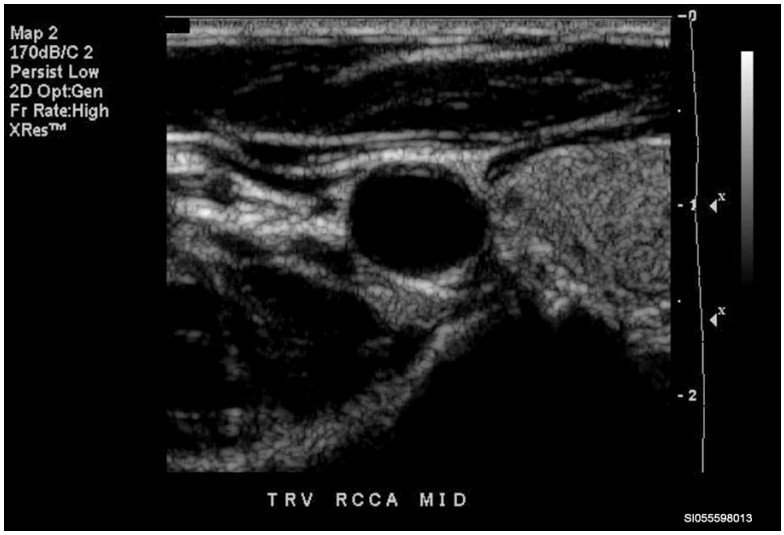

Figure 1–3. CCA, transverse.

Once the sweep is done, you can proceed to take more images that are accurate. Whether starting with longitudinal or transverse images, you should always make sure that the vessel of interest is placed in the center of the screen. Also, place one or two focal zones at the level of the vessel. Your overall gain should be adjusted so that the lumen of the vessel is completely black without obscuring low-level echoes typical of some abnormalities.

Start with transverse views of the CCA; usually three views are standard—proximal CCA, middle CCA (fig. 1–3), and distal CCA—with the distal being the furthest from the clavicle. Some sections will have a protocol that requires an additional view of the origin of the right CCA as it branches off the brachiocephalic trunk.

Moving cephalic, obtain a transverse image of the CCA as it bifurcates into the ICA and ECA. It's good practice to label both ICA and ECA in this view. Generally, this one transverse view of the ICA and ECA is all that's needed (fig. 1–4). However, if your original sweep or survey through the ICA and ECA displayed signs of plaque or other abnormality, you should include additional transverse views to demonstrate them.

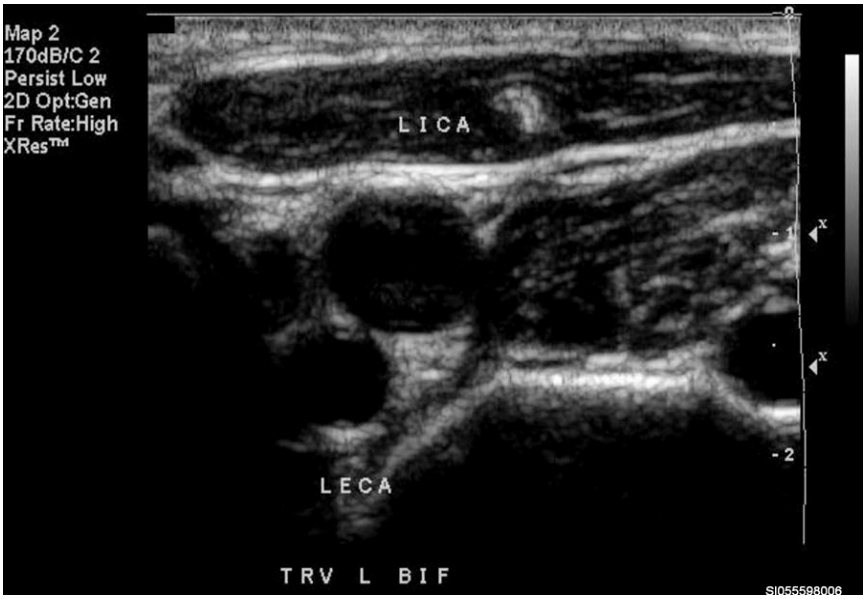

Figure 1–4. Carotid bifurcation, transverse.

Certain sonography sections require three longitudinal images of the CCA: proximal, middle, and distal. Other departments will want to see as much of the CCA that will fit on one image, so long as it is labeled. For instance, if you can get the proximal and middle sections of the CCA on one image, make sure that your label says precisely what is shown. The longitudinal image of the distal CCA should show something of the slight widening found at the bifurcation (fig. 1–5).

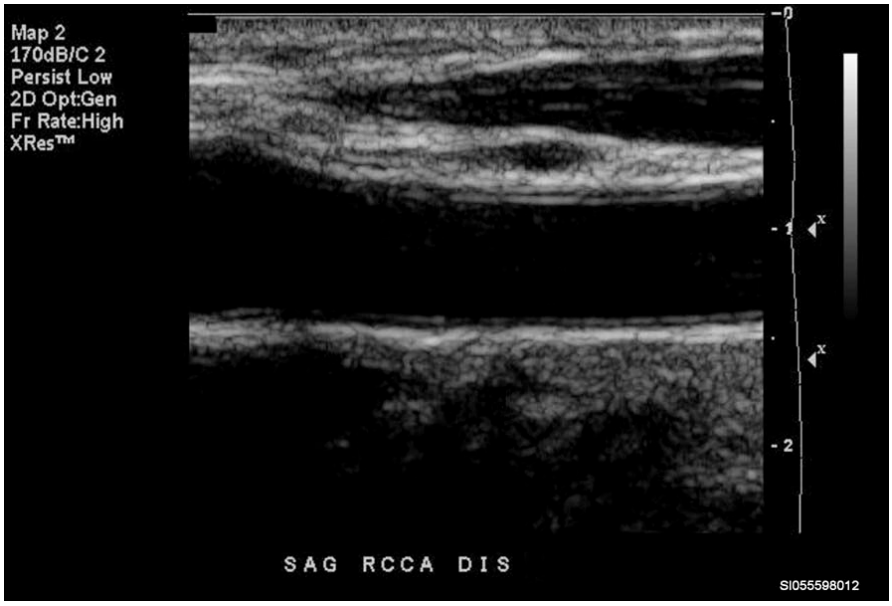

Figure 1–5. CCA distal, longitudinal.

The bifurcation image itself should display as much of the ICA and ECA as possible (fig. 1–6). There is certainly nothing wrong with obtaining grayscale images of as much of the lengths of ICA and ECA as you can. Some departments require it and some want it only when pathology is seen.

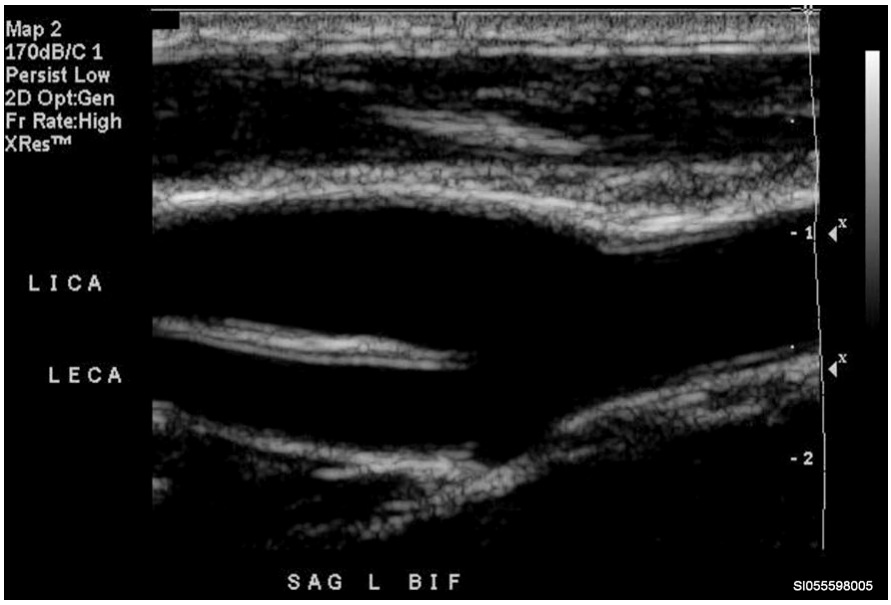

Figure 1–6. CCA/ICA/ECA, longitudinal bifurcation.

Color and power Doppler

In most institutions, using color flow Doppler to see the direction and character of blood flow within the carotids is an option. However, if your institution requires it, there is nothing wrong with it and it can be quite helpful. Color Doppler is obviously a quick way to determine direction of flow or even the presence of flow. The primary usefulness for color Doppler is to fill the lumen of the vessel with color as a landmark for placing your sampling box, which will be discussed more below. Also, it is helpful for outlining the shape of plaques against the vessel walls.

If you use color Doppler, try to fill the entire vessel from wall to wall with color (fig. 1–7). Set your color sensitivity so that it will not appear to cross over the vessel walls. Usually, turning down the filter will eliminate some artifacts such as flashes of color in surrounding tissue caused by slight motions.

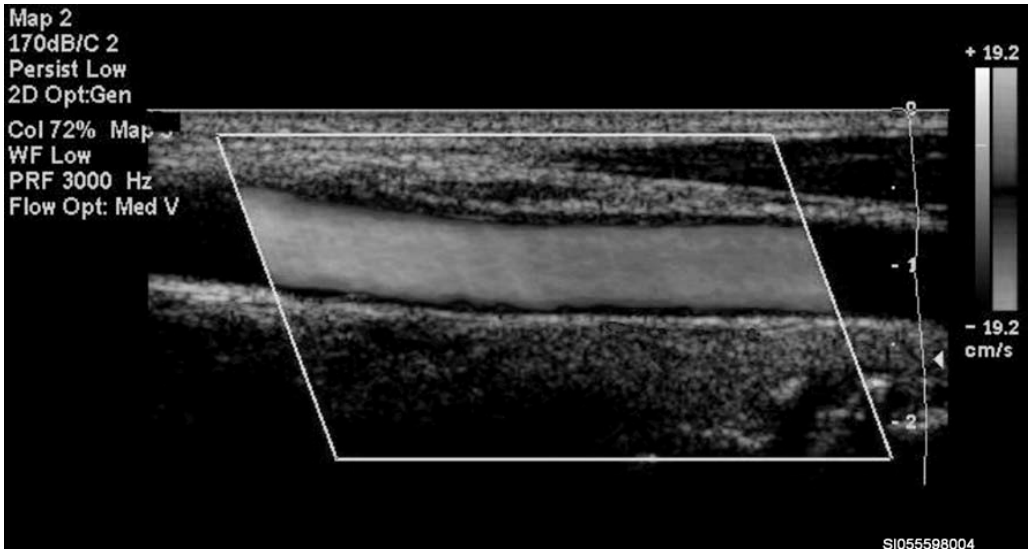

Figure 1–7. Doppler color image.

Finally, color Doppler is angle-dependent; that is, to adequately image a vessel in color, you should orient the transducer in such a way as to cause the vessel to be displayed at an angle to the ultrasound beam. Otherwise, the vessel will be perpendicular to the beam and moving blood will not be detected. Moving objects have to either approach or move away from the transducer to be detected by Doppler. Sometimes, the vessel's position in the neck prevents you from getting the angle necessary for color Doppler. Fortunately, many units have transducers with the capability to steer the Doppler ultrasound beam at least 30 degrees to the vessel, while simultaneously keeping the grayscale ultrasound beam at 90 degrees. This technology is usually seen as an electronic box on the display screen (fig. 1–8).

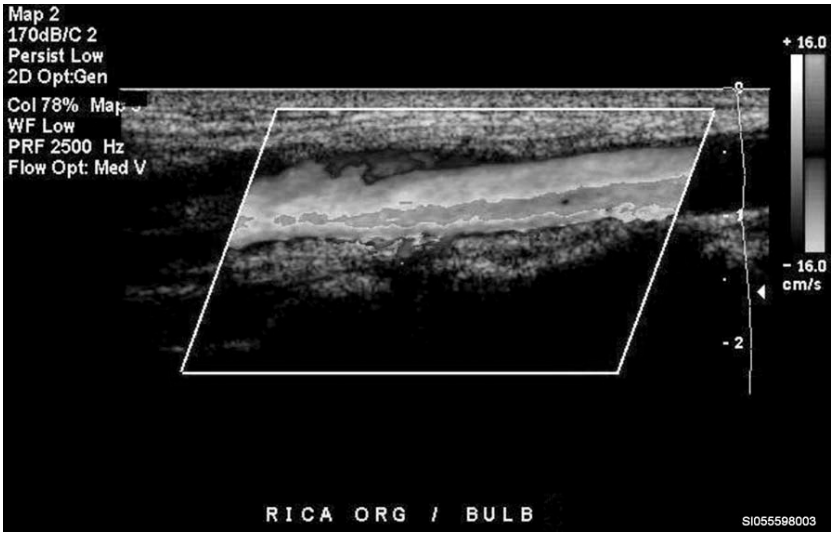

Figure 1–8. Doppler color box.

Sometimes, blood flow may be so slow (usually in the presence of an abnormality such as stenosis) that the filters in your instrumentation prevent detection. If lowering the filter, increasing the sensitivity (frequently called 'velocity range'), or angling the transducer does not help, you can attempt to use *power* Doppler, which is a color image obtained independent of the vessel angle for flow detection. Power Doppler, sometimes referred to as 'energy,' is usually more sensitive to slow flowing blood. Power is particularly useful in outlining the surfaces of plaque. However, power Doppler is extremely sensitive to motion. So try to ensure that the patient and your transducer are very still (fig. 1–9).

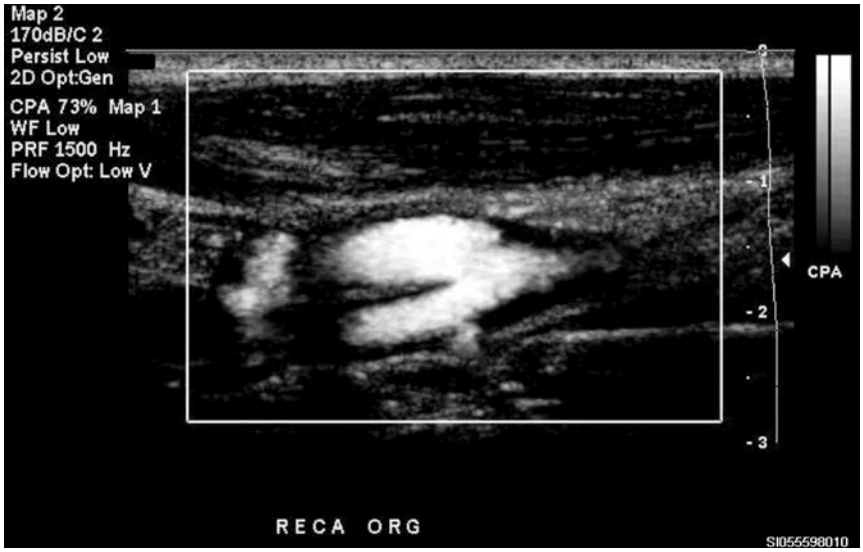

Figure 1–9. Image using power.

Doppler spectral analysis

Once the grayscale images of the carotids—with or without color Doppler—have been obtained, you should have mentally mapped out suitable locations to perform sampling of blood flow for spectral analysis. Spectral analysis, or quantitative analysis, analyzes the Doppler frequency shifts in return echoes (signals) from moving blood and digitally displays the range of velocities.

The information typically displayed on a spectral readout is velocity in centimeters per second (cm/s) on the vertical axis; time in seconds on the horizontal axis; and a legend providing characteristics of the sample such as angle, frequency of the transducer, pulse repetition frequency ((PRF), the number of pulses per second), and depth. Inside the vertical and horizontal axis will be the display of velocity ranges, usually called the waveform. Generally, this waveform is above a solid white line called a baseline, which conventionally represents the direction of flow in relation to the transducer. For example, waveforms above the baseline represent flow toward the transducer, and waveforms below the baseline represent flow away from the transducer.

Keep in mind that this relationship more precisely has to do with the orientation of blood flow to the ultrasound beam. If the beam is steered electronically, the direction of blood in relation to the beam (and not the transducer) is what determines if the waveform is displayed above or below the baseline. Thus, you may have control of the beam by simply steering the Doppler box without moving the transducer. Of course, you may also flip the waveform display by pressing a button—usually an invert button. However, it is conventional to represent forward or arterial blood flow above the baseline. Interestingly, some institutions prefer venous blood flow be displayed above the baseline, as well. Usually this is because some radiologists want to think of flow above the baseline as blood flowing in the *proper direction*—either away from the heart in the artery or toward the heart in the vein. Thus, any flow below the baseline represents a reversal of normal forward (venous or arterial) flow.

Most ultrasound displays will have the capability to show a spectral readout and a grayscale image simultaneously with a color Doppler box overlying it (a set-up called, ‘duplex’). This duplex image allows you to locate the sample box (sample volume gate) easier. The sample box allows you to adjust where your measurement is to be made within the color image. For proper spectral analysis, it also is useful to ensure that your Doppler angle is set between the standard 60 degrees and 30 degrees.

The basic waveform on the spectral display shows the amplitude (strength) of return echoes as they are received by the transducer in time. The amplitude is indicated by the brightness of the waveform. Flow begins close to the baseline and rapidly rises to a crest. The crest of the wave represents the systolic peak, which is the highest recorded velocity of forward flow. The flow that follows close to the baseline is the diastolic flow. The area inside the wave (window) is normally empty of echoes (fig. 1–10). If flow is turbulent or the sample volume is large enough to pick up slow-moving blood close to the vessel wall, the window will fill in with echoes (spectral broadening).

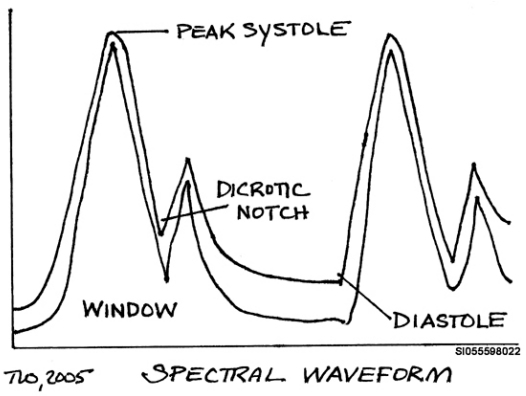

Figure 1–10. Spectral waveform.

Occasionally, false echoes are picked up on the spectral display below the baseline. They will usually be spaced at regular intervals and correspond to the peaks of the waveforms. This situation is called aliasing. It is caused by the PRF being too low or not sampling velocities fast enough to cope with high Doppler frequencies generated by high velocity blood flow. In order to prevent aliasing, the PRF must be increased to a rate faster than the Doppler frequency; in fact, it must be at least two times faster, a threshold called the Nyquist limit. Fortunately, the shallow depth of the carotids means a greater number of pulses per second (higher PRF) produced by the transducer, which will mean a

higher Nyquist limit, in turn making aliasing an infrequent occurrence. If aliasing does occur, many units will increase the PRF automatically as you increase the velocity scale (technically, a converted Doppler frequency shift scale). A unit may also allow you to increase the PRF with a button manually.

From the point where the wave starts to rise until the diastolic point, or the point just before the wave begins another rapid rise, represents one pulse. This pulse appears characteristically different in each part of the carotid artery. In the CCA, the entire waveform remains above the baseline, representing continuous forward flow. The appearance of the CCA waveform resembles a combination of ICA and ECA waveforms. The peaks are rounded and the diastolic portion is moderately close to the baseline (fig. 1–11).

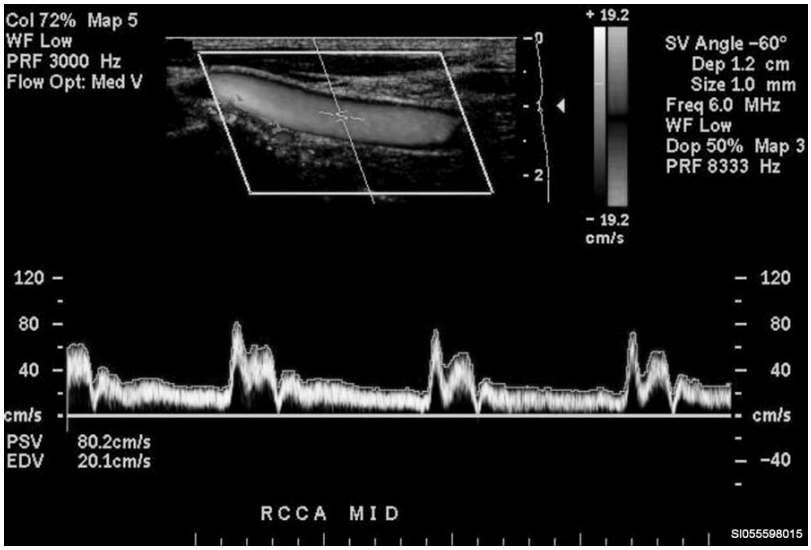

Figure 1–11. CCA waveform.

ICA waveforms have peaks that are more rounded and a higher level of diastolic flow than the CCA waveform. This represents the ICA delivering a greater amount of blood at a faster rate (fig. 1–12). Low resistance of blood flowing to the brain causes this appearance.

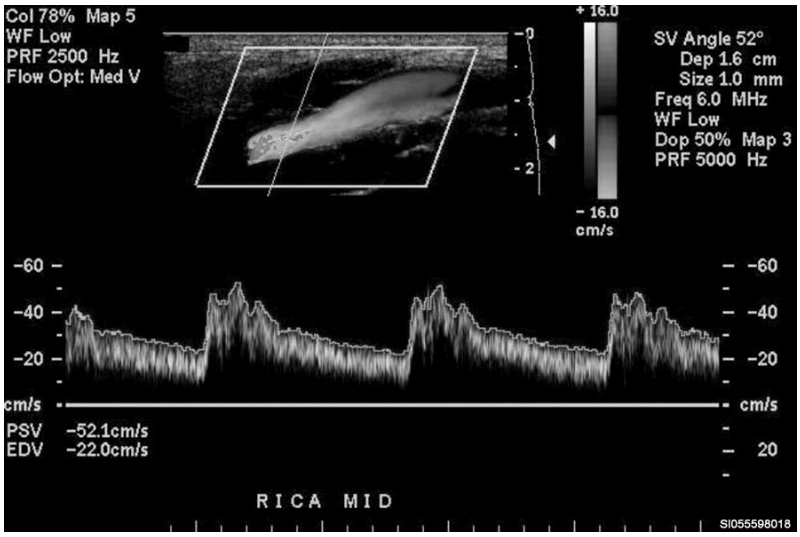

Figure 1–12. ICA waveform.

External carotid artery waveforms display high-resistance to flow. The peaks are sharp and tall, representing a higher velocity for a shorter period. Because the ECA supplies the face and scalp, the need for large amount of continuous blood flow is diminished relative to the brain's need. Thus, the diastolic portion of the waveform is quite near the baseline or either at the baseline, representing reduced or absent forward flow resulting from increased resistance.

Whereas grayscale may usefully show size, presence of branches, and orientation in the neck, spectral waveform appearance is the best way to distinguish between the normal ICA and ECA. This is because of the many possible variations of carotid appearance. Blood flow is usually consistent in the normal patient despite differences in carotid structure. However, you should keep in mind that the high resistance profile of the ECA and the low resistance profile of the ICA are characteristic of *normal* blood flow. If there is a blockage or reduced flow, these waveforms may appear quite different or even similar. In fact, the ECA is sometimes recruited to deliver low resistance flow as a collateral vessel when the ICA is blocked, a situation referred to as internalization.

Such cases can be quite confusing. Thus, for abnormal spectral waveforms, it is common to use another method to distinguish ECA from ICA—the temporal tap. By placing the sample gate at what you suspect is the ECA origin and gently tapping the side of the head just anterior to the ear opening, you should see a distortion in the waveform corresponding with your taps. This will indicate that you are sampling the ECA, which gives off the branch of the superficial temporal artery. However, you should use this method with caution. The ICA and even the CCA can sometimes show distortion in their waveforms from the reflections of temporal tapping. Perhaps a better way would be to use transverse color Doppler views and look for any small branches to indicate the ECA.

Most institutions require at least sampled measurements at the distal CCA (typically a few centimeters from the bifurcation) (fig. 1–13); at the proximal (fig. 1–14), middle, and distal ICA (although this last one may be extremely difficult to obtain because the vessel begins to turn at this point) (fig. 1–15); and the proximal ECA (which may have spectral broadening representing turbulence from the presence of branches downstream) (fig. 1–16).

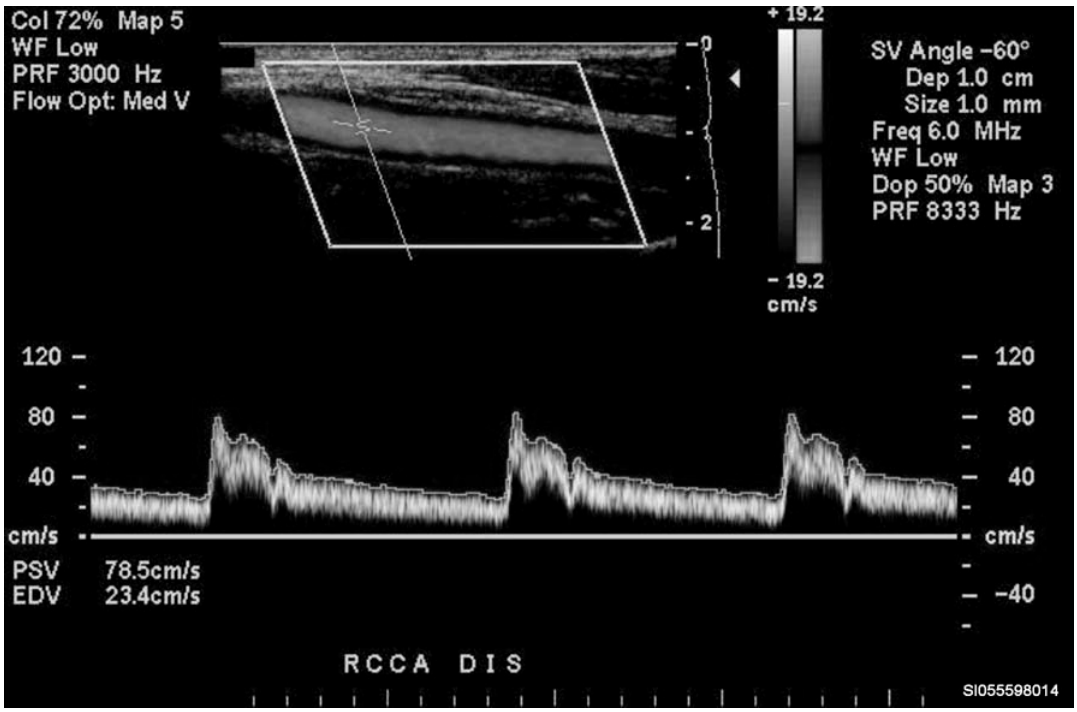

Figure 1–13. CCA sample.

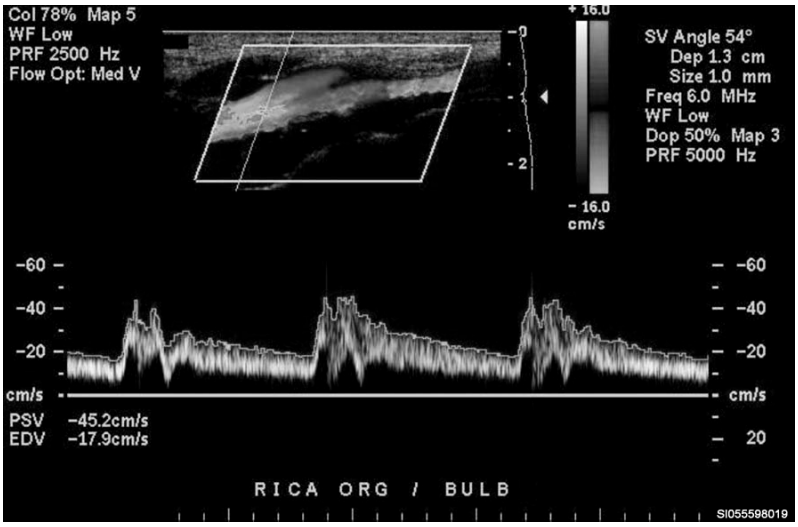

Figure 1–14. ICA sample, proximal.

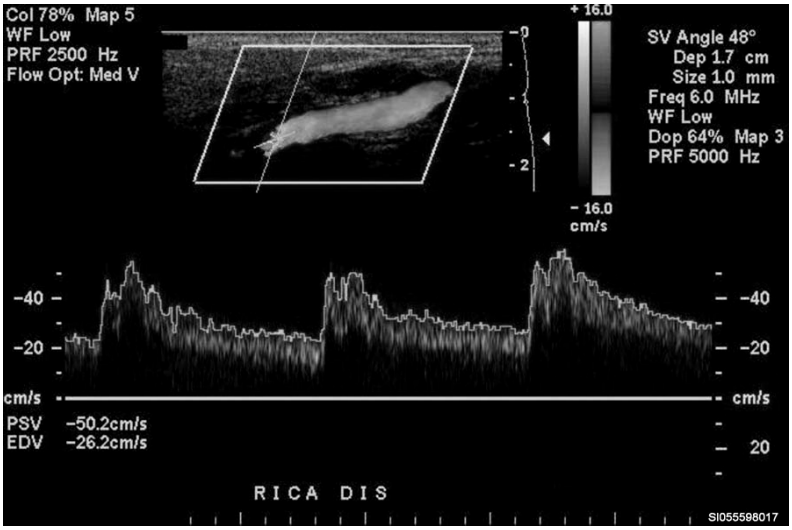

Figure 1–15. ICA sample, distal.

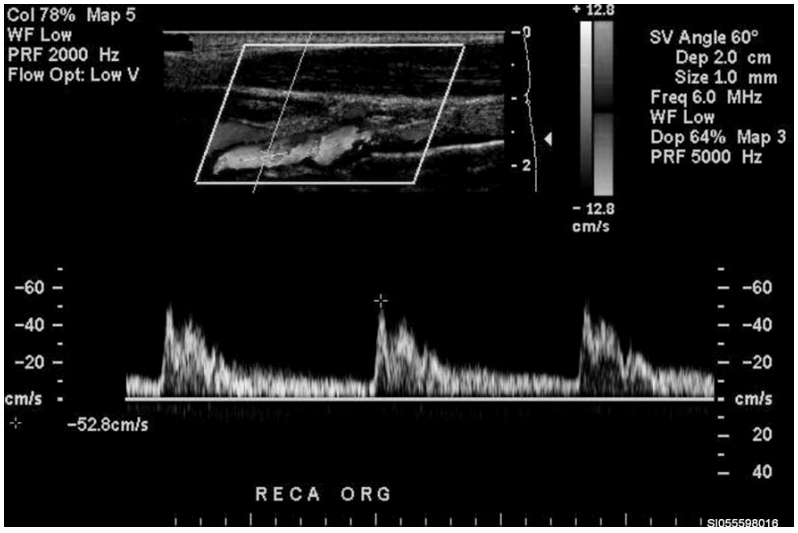

Figure 1–16. ECA sample, with turbulence.

At each of the spectral measurement locations, you should place a cursor at the peak systolic point and at the diastolic point of the spectral waveform. This action should display the peak systolic (maximum) velocity and the end diastolic (minimum) velocity of the waveform. Also, some ultrasound units have the capability to automatically display the average peak systolic and end diastolic velocities by tracing the outline of the entire waveform. Most sonography departments, however, will use only the maximum and minimum velocities for diagnostic studies. These measurements and their ratios become critical in determining the presence or extent of abnormalities such as stenosis.

Usually, the carotid examination is incomplete without a spectral measurement of the vertebral arteries. Some radiologists may only want color flow (which can convey information about the presence and direction of flow). Vertebral flow should always be forward toward the brain; with color Doppler, this is shown as the color above the black baseline on the color key. If you use duplex to image the vertebrals, the waveform will resemble that found in the ICA. This is appropriate because the vertebrals continuously supply the posterior brain with blood (fig. 1–17).

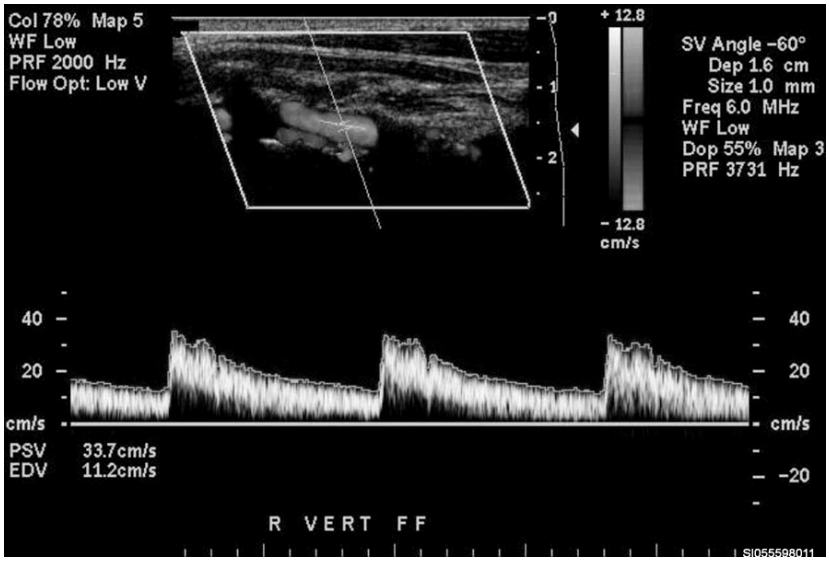

Figure 1–17. Vertebral sample.

Frequently locating the vertebral arteries can be difficult. You are more likely to find the vessel if you keep in mind that it is rarely distant from the ICA. With your transducer in the longitudinal position to see the ICA, slowly move or angle the transducer laterally. Your color box should be on, so that you should be able to see the pulsing forward flow of the vertebrals. A clue that you are seeing the vertebrals is the anechoic shadows that abruptly interrupt the color vessel at regular intervals. These are shadows produced by the transverse processes of the cervical vertebrae.

Techniques to consider for scanning various carotid abnormalities

Carotid sonography is a diagnostic procedure that primarily attempts to diagnose several problems found in the carotid artery anatomy.

1. Plaque buildup sufficient to cause narrowing of the vessel. (Stenosis)
2. Plaque ulcerating or causing clot formation, both which may shed pieces. (Embolization)
3. Inner layer of carotid wall separated from middle layer. (Dissection)

Carotid sonography also helps identify possible obstruction of an intracranial arterial vessel. To do this, blood flow rather than wall structure is analyzed. If stenosis is present within the carotid artery, the blood flowing before, through, and after a narrow channel is measured and analyzed.

Plaque

Identification of plaque and the attempt to highlight its appearance (whether ulcerated or not) is important. From the sonographer's perspective, the most important thing to accomplish is to determine the character of the plaque. If plaque is seen during the carotid examination, sonographers attempt to document its location, extent, echogenicity, echotexture, and surface appearance.

For you to locate plaque, the most common area to look is in the proximal ICA, within a few centimeters of the bifurcation. However, you shouldn't expect plaque only in that location; rather, expect it anywhere from the CCA origin all the way to the distal ICA. Do not be lulled into thinking that plaque will rarely be encountered in the ECA. Occasionally, there will be enough plaque in the proximal ECA to make the vessel difficult to detect.

The composition of plaque may also make locating it difficult. The sonographic echogenicity of the plaque may be reduced to the point where it is as hypoechoic or anechoic as the moving blood within the lumen. Usually this occurs in cases of soft plaque. The best way to detect these types of plaques is to fill the vessel, wall to wall, with color or power Doppler; this action will usually bring out most hypoechoic to anechoic plaques. Be sure to use this color Doppler approach both longitudinally and transversely.

The best way to evaluate and document the extent of plaque in the carotid is to use the longitudinal view. This should show the entire length of focal or diffuse plaque. It is also important to demonstrate plaque thickness as seen from the wall to the center of the lumen, best determined in the transverse view. It is important to recognize the various echogenic appearances of plaque. Your ability to recognize the appearances will enable you to tailor your scan to plaque severity properly. Generally, plaque appears in three levels of echogenicity:

1. Low.
2. Moderate.
3. High.

For low echogenic plaque, the plaque is made of both fibrous and fatty tissues (fibroadipose, or fibrofatty), but mostly fatty cells. Again, these fatty plaques are nearly hypoechoic and quite difficult to see. The best way to cope with low echogenic plaque is to use color Doppler first to locate it within the lumen. Then in grayscale, slightly increase your overall gain just to the point where you can see the plaque.

For moderately echogenic plaque, the fibrous tissue has increasing amounts of collagen, a protein fiber, which renders it more echogenic and easier to detect sonographically. You should have little difficulty seeing this type of plaque.

For highly echogenic plaque, the increased reflection is caused by calcification, which is in turn usually the result of hemorrhage or necrosis within the plaque. Obviously, the calcification will be easier to detect. Calcification tends to produce shadows that interfere with a clear view of carotid structure and even extent of plaque. Color Doppler is extremely helpful for outlining the channel of blood flow.

Another feature of carotid plaques that you must attempt to evaluate is the sonographic texture. Usually this falls into two categories: *homogeneous*, or smooth and uniform echoes; and *heterogeneous*, or complex patterns of combined high and low echogenicities. Heterogeneous plaque may suggest the presence of hemorrhage or ulcer. This is significant because more than half of patients with neurologic symptoms will have hemorrhagic or ulcerated plaque.

Finally, the surface characteristics, if readily detectable, should be documented. Usually, you will need color or energy Doppler to demonstrate the surface or lumen-side of the plaque accurately. Most moderate to highly echogenic plaques will readily display on a sonography screen in grayscale. However, be careful and remember that in some of these plaques, particularly heterogeneous plaques,

the surfaces may be coated with thrombus or clot. This thrombotic material may be as anechoic as the blood that flows around it, hiding its appearance on grayscale. Thus, Doppler should detect a void separating the flow from the apparent surface seen on grayscale. When plaque or intimal thickening is seen, it is a good technique to outline the surface with Doppler.

The surface will usually show three types of outlines: smooth, irregular, or an abrupt gap or pit representing ulceration. An irregular surface can sometimes mask an ulcer in the plaque. Clues to the presence of ulcer may be a lip that hangs over the anechoic space seen within the plaque, or color Doppler demonstrating flow swirling within the space. Otherwise, you may only detect a severely jagged, irregular surface. Ensure that you are documenting plaque in both longitudinal and transverse views. Recall your transverse sweep through the carotid; if you see plaque, you may notice areas where the surface seems pitted or has a lip of plaque hanging over a depression. These may indicate the presence of ulceration, particularly if the plaque is heterogeneous.

Stenosis

The principal purpose for performing carotid sonography is to diagnose stenosis, or narrowing of the lumen. The sonographic evaluation of stenosis observes the extent of plaque buildup that usually becomes more complex and heterogeneous with increasing size. As the severity of the stenosis increases, the velocities of the blood flowing through the narrow channel also increase to maintain the volume and rate that the brain requires. Thus, the principal means for sonographically evaluating the percentage of vessel stenosis is to examine the velocities. Color Doppler and grayscale are used to examine and document evidence of stenosis, along with spectral analysis of velocity waveforms.

If you understand that an increasingly narrow carotid lumen will cause high velocity flow in that channel, then you should understand that the single most effective measurement you can take will be the peak systolic velocity (PSV) at the narrowest point inside the stenotic zone. Recall that most plaque forms in the area of the bifurcation. Generally, plaque builds to a point where the proximal ICA is critically stenosed. Your job is to measure the PSV within the stenosis to determine if the velocity is hemodynamically significant; that is, velocity that significantly affects the normal circulation of blood. The velocity level is an indirect way to determine an estimation of how much of the ICA is stenosed. A certain velocity will indicate a certain percentage of stenosis, and prompt the physician to recommend the symptomatic patient for endarterectomy.

Numerous categories exist for determining which velocities represent levels of stenosis. Most departments settle on a set of standard parameters to use for analyzing stenosis, which may be different in another institution. However, there are some general parameters for stenosis evaluation.

Because most institutions agree that 70 percent stenosis is the threshold for endarterectomy in the symptomatic patient, most also agree that the most important criterion is the PSV. PSV at a certain level within the stenotic channel corresponds to a 70-percent reduction in the diameter of the vessel.

The PSV and the end diastolic velocities (EDV) should be taken at three levels:

1. Just before the stenosis (prestenotic zone).
2. Within the narrowest point in the channel (stenotic zone).
3. Just after the stenosis (poststenotic zone).

Recall that the spectral waveforms taken in carotids *without apparent plaque* are generally sampled in three areas of the ICA: proximal, middle, and distal. In those cases, the sample volume gate or box is placed within the central lumen and the cursor within the box is angled 60 degrees or less to the direction of blood flow. The cursor is also parallel with the vessel walls.

In the case of visible ICA stenosis, the sample volumes are placed in the above three areas and also within the narrowest point of the channel. However, you should remember that the *highest PSV* (usually, but not always, found within the narrow stenotic channel) is the velocity used to determine stenosis percentage. When placing the cursor in the stenotic zones, attempt to align the cursor along

the plane of the color flow jet rather than parallel with the vessel wall (as you would in the ICA *without* visible stenosis). This will enable you to obtain the most accurate velocity measurement possible *for a visible stenosis*. The flow jet is the area in the lumen that corresponds to the color furthest from the baseline on the color map.

Another common parameter is the ratio between the ICA stenosis PSV to the CCA PSV sampled a few centimeters before the bifurcation—usually just written ICA/CCA ratio. It is the ICA PSV divided by the CCA PSV. This ratio is used to grade stenosis as well as to correct for errors, and is particularly helpful when the PSV velocities are higher than normal without signs of plaque. Even in the presence of plaque, certain conditions can raise or lower velocities elsewhere in the vessel. For example, physiologic and instrumental changes, such as different equipment settings or patient hypertension, can cause an increase or decrease in velocities throughout the carotid artery, which can, in turn, cause you to overestimate or underestimate velocities in the ICA stenosis. Thus, the ratio eliminates relying on just the ICA stenotic PSV, which may be elevated due to conditions other than the stenosis.

The CCA PSV measurement should be in a non-stenotic area where the velocities are usually stable, which is normally a few centimeters before the bifurcation. In both the ICA and CCA, the EDV measurements are taken but are usually not focused on as a significant parameter until stenosis is extremely high. This usually is found in cases of near occlusion where the PSV suddenly drops down significantly (dampened) and the spectral waveform shows signs of broadening. You may also see spectral broadening throughout the ICA stenotic zone, particularly at or just distal to the narrowest point.

Stenosis in areas other than the ICA is examined in similar fashion. However, the hemodynamic significance will normally be determined by the radiologist.

General Parameters for Grading ICA Stenosis			
Reduction in Diameter	***Peak Systolic Velocity***	***End Diastolic Velocity***	***ICA/CCA Ratio***
Normal diameter (that is, no stenosis seen)	Less than 125 cm/sec	Should be less than 40 cm/sec	Less than 2
Less than 50%	Less than 125 cm/sec	Should be less than 40 cm/sec	Less than 2
50% to 69%	125 cm/sec to 230 cm/sec	40 cm/sec to 100 cm/sec	2 to 4
70% and above	Greater than 230 cm/sec	Greater than 100 cm/sec	Greater than 4

Occlusion

An occluded ICA will have no flow, which will be seen as an absence of a Doppler signal (spectral or color) when you place a sample box within the vessel. Grayscale sonography will also demonstrate a lumen completely filled with hypoechoic to echogenic plaque or thrombus.

To properly document carotid occlusion, you should attempt to do everything possible to detect flow. The reason for this is that sometimes an apparent occlusion is really an extremely stenosed carotid, which will have only a trickle of blood flow. Because a high grade stenosis can shed emboli (even if the flow is a trickle), it is a candidate for endarterectomy. Conversely, a completely blocked ICA is local and irredeemable. Thus, it is a good idea to use color and power Doppler as you search for a hint of blood flow. Remember that if there is flow in such a tight stenosis, it is flowing at an extremely slow rate. You should decrease the filter in order to pick up low flow Doppler shifts of slow moving blood. Power Doppler is particularly useful in this area. You should use both longitudinal and transverse views in order to detect Doppler flow. In the event that color and power Doppler yields no signal and you have documented these absences, some radiologist may have you sample the lumen with spectral Doppler which may yield a signal, however faint. When using spectral Doppler in this capacity, increase your gain to better display any faint echoes.

Occlusion of the carotids may also produce collateral flow to compensate for the blocked pathway of blood to the brain. Commonly, ICA occlusion will cause all of the CCA flow to divert into the ECA,

a situation called internalization of the ECA. To detect internalization of the ECA, you are likely to see a low resistance (high diastolic) waveform at the ECA origin rather than the typical high resistance waveform. Remember that branches off the ECA will give you a clue that you are examining the ECA and not the ICA. Also, CCA occlusion will not stop the body from developing a collateral pathway to reach a closed-off ICA. CCA occlusion is rare but very noticeable. When it occurs, ECA flow in the opposite carotid may flow across the head and neck and travel to the ECA branch of the occluded CCA. Once in the branch, the blood will travel in a reverse or retrograde fashion through the ECA, across the bifurcation, and into the ICA for forward flow. This can be demonstrated by taking Doppler readings of blood flow in the ECA, which should show reversed flow.

Dissection

You should learn to recognize a possible suggestion of carotid dissection. To do this, you should see either a flapping length of echogenic tissue stretching from the wall into the lumen, or you will see a fixed length of echogenic tissue in the lumen, which may taper to a point—-this may cause considerable confusion if blood is seen on color Doppler flowing turbulently within it. This false lumen may itself occlude with thrombus and become very difficult to detect. Sometimes the false lumen can expand to such a width that it causes the ICA or CCA to become stenosed or occluded.

If a dissection causes a stenosis, the velocities should be measured as if it were plaque that caused it. Along with the extent of the false lumen, it is a good idea to determine the flow characteristics in both the false lumen and true lumen. Thus, a combination of color and spectral Doppler is appropriate in dissection cases.

Subclavian steal

You can help diagnose subclavian steal with sonography by detecting reverse flow in the vertebral artery. Rarely will you see the blockage itself on sonography, particularly on the left, due to the depth behind the sternum. But you may be able to determine the severity of blockage from direction of flow in the vertebrals indirectly. Complete subclavian steal is represented by continuous flow reversal in the vertebrals. Partial subclavian steal occurs when flow travels forward in the vertebral and then reverses in a back and forth motion with the cardiac cycle. This action usually occurs in the presence of a high-grade stenosis of the subclavian or brachiocephalic trunk rather than a complete blockage. You should document the waveforms as they appear above and below the baseline, preferably in the same sample.

There are other abnormalities and variants associated with vertebral arteries that you may see, such as change in flow from vertebral stenosis. The vertebrals may also become occluded, blocking flow altogether. PSV and EDV should be taken for the vertebrals as a way to characterize the resistance levels of flow to the posterior brain.

206. Applying ergonomics

When examining the cerebrovascular system, you will find that the operation is delicate and requires a steady hand, particularly when sampling moving blood. In fact, the only thing that should be moving during spectral sampling is the blood and the slow movement of the sample volume gate; everything else from the patient to your transducer should be as still as possible. Otherwise, your readings will be inaccurate. Your motionlessness puts your body, particularly your arm and wrist, into the same position, or into a stressfully narrow set of positions for a long time. If repeated hour after hour, day after day, for months or years, you may develop a musculoskeletal disorder in your wrist or shoulder. To minimize such an occurrence, sonographers should be familiar with and use ergonomics.

Ergonomics is the concern for human factors when designing and operating equipment. This concern principally focuses on the reduction of human discomfort, difficulty, or injury. For the field of sonography, manufacturers developing ultrasound systems that are safer and more comfortable to

operate are using ergonomics in their design. Sometimes you may hear ergonomics being referred to as human engineering.

However, the responsibility for ergonomics does not stop with the manufacturers; sonography managers, supervisors, and workers have a need to use ergonomics in the sonography environment. Ergonomics is the best way to prevent musculoskeletal disorders that develop in sonographers who are expected to sit in the same position and repetitively place their arms and hands in certain positions.

As for the ergonomic operation of equipment, sonographers can control some of those factors. For instance, use variations in your routine when you perform sonograms. Perhaps the most important thing you should vary would be your posture as you sit or stand. Try changing your posture to a few different comfortable positions. Alternate every other patient. Also, how you hold your transducer can have a drastically negative impact on your wrist, shoulder, and even your back. Many times, simply relaxing or varying your grip when holding the transducer may prove beneficial.

Use the ergonomic designs found on equipment. The arm and head rests should be adjusted for your maximum comfort, depending on patient use. Raise or lower your chair for optimum comfort while you scan. Adjust the monitor on the ultrasound equipment so that you may easily see it without distorting or twisting your posture. Use electronic preset labels to minimize your use of the keyboard on ultrasound equipment. Many equipment designs have the capability of allowing nearly unlimited automatic labels that correspond to single button pushes.

Another method you can use is to relax your muscles periodically by stretching them and taking breaks. Also when transporting patients to and from or within the sonography section, use proper lifting, carrying, and pushing techniques. This will reduce the chance of injury on the job.

Finally, maintain good physical fitness, get plenty of sleep, eat nutritious meals and drink plenty of water. Exercise, rest, and healthy diet will give you the type of energy that will help you perform the demanding work tasks of sonography.

Self-Test Questions

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

203. Cerebrovascular anatomy and physiology

1. Both the brachiocephalic trunk and the left subclavian artery originate from what vessel?

2. At what level of the neck will the CCA typically bifurcate?

3. What does the ICA in the neck lack compared to the ECA?

4. The ECA supplies blood to what structures?

5. Deoxygenated blood travels to the heart through what two vessels in the neck?

204. Carotid pathology

1. List the indications for a carotid sonogram?

2. Sonography is useful primarily for which type of stroke?

3. Match the stroke symptom in column B with its description in column A. Each item in column B may be used only once.

Column A	*Column B*
____ (1) Weakness in the body.	a. Aphasia.
____ (2) Burning, tickling, or prickling sensation in the skin.	b. Amaurosis fugax.
____ (3) Difficulty or inability to speak.	c. Paresis.
____ (4) Abnormal or absent vision.	d. Parasthesia.

4. At what point of stenosis of the carotid artery will many symptomatic patients become candidates for surgical removal of the plaque?

205. Imaging normal and abnormal carotids

1. What limitations may prevent you from obtaining optimum images of the carotid arteries?

2. What is the purpose for reviewing any previous carotid examinations before performing your scan?

3. Why would you support a carotid sonography patient’s neck and shoulders with a pillow or a rolled up towel?

4. Describe the optimal grayscale image of the carotid artery.

5. What are the standard transverse views for a carotid examination?

6. What are some uses for color Doppler?

7. Between what two Doppler angles should you use for proper spectral analysis?

8. What does spectral broadening of the waveform represent?

9. Describe the ICA waveform.

10. What situation does internalization signify?

11. What is a clue that you are seeing the vertebral arteries when using color Doppler?

12. List the problems that carotid sonography attempts to diagnose.

13. What should a sonographer document when encountering plaque?

14. What is the most common location for plaque in the carotids?

15. What are the three levels of echogenicity in plaque?

16. Why should you outline the surface of plaque with Doppler?

17. For stenosis, velocity measurements should be taken at which locations?

18. What indicates subclavian steal phenomenon on a sonogram?

206. Applying ergonomics

1. What are some methods sonographers use to prevent muscoskeletal disorders?

2. As an ergonomic consideration, what is the most important thing you can vary in your routine while scanning patients?

Answers to Self-Test Questions

201

1. Pressure, resistance, and volume.
2. Fluid flows from high pressure areas to low pressure areas.
3. The radius of vessels (*r*) and their pressure gradients (P_1–P_2).

202

1. The vein walls have valves that assist in preventing blood from reversing course. Also, respiration and skeletal muscle contractions help prevent venous blood from reversing.
2. Decreased intra-abdominal pressure due to ascent of diaphragm.

203

1. Aorta.
2. Most proximal level of the thyroid cartilage.
3. Branches.
4. Face, scalp, and neck.
5. IJV and EJV.

204

1. Stroke, TIA, amaurosis fugax, cervical bruit, pre-operative evaluation, and follow-up of known carotid disease.
2. Ischemic.
3. (1) c.
   (2) d.
   (3) a.
   (4) b.
4. When stenosis has reached 70 percent of internal diameter of the vessel.

205

1. Short and thick neck muscles may make it difficult for ultrasound to penetrate to the carotids; recent surgeries may cause items such as surgical staples and dressings to be placed on the neck, which may limit your visibility of the carotids; or patients may not be able to lie flat because of medical conditions like arthritis of the neck and COPD.
2. To give you an idea of the carotid anatomy of an individual and it will also prepare you for any potential surprises.
3. To help extend the neck and increase visibility of as much of the carotid as possible.
4. A completely anechoic lumen bounded by echogenic walls. There should be hypoechoic line within the wall which represents media layer of the artery. Thus, the echogenic line facing the lumen is the inner or intimal layer and the echogenic line on the wall side of the media layer represents the outer or adventitia layer.
5. Proximal, middle, distal CCA; and the bifurcation labeled ICA and ECA.
6. Determining presence and direction of blood flow and outlining the shape of plaques.
7. 60 and 30 degrees.
8. Turbulence or a sample volume large enough to pick up slower moving blood close to vessel wall.
9. ICA waveforms have peaks that are more rounded and a higher level of diastolic flow than the CCA waveform. This high diastolic flow is due to low resistance of blood flowing to the brain.
10. The ECA is sometimes recruited to deliver low resistance flow as a collateral vessel when the ICA is blocked.
11. Anechoic shadows abruptly interrupt the color vessel at regular intervals.

12. (1) Plaque buildup sufficient enough to cause narrowing of the vessel. (Stenosis).
    (2) Plaque ulcerating or causing clot formation, both which may shed pieces. (Embolization).
    (3) Inner layer of carotid wall separated from middle layer. (Dissection).
13. Location, extent, echogenicity, echotexture, and surface appearance.
14. The proximal ICA, few centimeters from the bifurcation.
15. Low, medium, and high.
16. Some hidden thrombus may coat surface of plaque unseen due to its anechoic appearance on grayscale.
17. (1) Just before the stenosis (prestenotic zone).
    (2) Within the narrowest point in the channel (stenotic zone).
    (3) Just after the stenosis (poststenotic zone).
18. Reverse flow in the vertebral artery.

206

1. Variations in routine when performing sonograms; relaxing or varying your grip when holding the transducer; relaxing muscles periodically by stretching them and taking breaks; using proper lifting, carrying, and pushing techniques when transporting patients to and from or within the sonography section; maintaining good physical fitness; getting plenty of sleep; eating nutritious meals and drinking plenty of water.
2. Your posture as you sit or stand.

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. (201) A small change in a vessel's radius blood flow will produce a
   a. small change equally.
   b. change twice as large.
   c. change four times as large.
   d. change seven times as large.

2. (201) How does the arteriole control the volume of flow into the capillary network?
   a. Cardiac activity.
   b. Pressure gradient.
   c. Through the use of valves.
   d. Vasoconstriction and vasodilation.

3. (202) What type of effect does respiration have on flow in the upper extremities compared with flow in the lower extremities?
   a. Opposite effect.
   b. Identical effect.
   c. A causative effect.
   d. A proportional effect.

4. (203) How does the common carotid artery (CCA) relate to the thyroid gland?
   a. Anteriorly.
   b. Posteriorly.
   c. Medially.
   d. Laterally.

5. (203) How does the internal carotid artery (ICA) usually lie in the neck relative to the external carotid artery (ECA)?
   a. Lateral and posterior.
   b. Medial and posterior.
   c. Lateral and anterior.
   d. Medial and anterior.

6. (204) Based on neurologic symptoms, how does transient ischemic attack (TIA) differ from stroke?
   a. Strokes are fleeting.
   b. TIAs recover after 24 hours.
   c. TIAs recover within 24 hours.
   d. Strokes recover within 24 hours.

7. (205) For the carotid sonogram, when should you interview a patient?
   a. After the sonogram.
   b. Before the sonogram.
   c. During the sonogram.
   d. An interview is not necessary.

8. (205) You should add transverse views of the internal carotid artery (ICA) to your examination when you
   a. see lymph nodes.
   b. see the distal ICA.
   c. see signs of plaque.
   d. feel it will help distinguish the ICA from the external carotid artery (ECA).

9. (205) The *primary* usefulness for color Doppler is as a landmark for
   a. placing sampling box.
   b. identifying the carotids.
   c. locating external carotid artery (ECA) branches.
   d. outlining plaque ulcerations.

10. (205) The conventional relationship between the spectral waveform and the baseline is that the waveforms
    a. above baseline represent flow toward transducer.
    b. below the baseline represent flow in separate vessels.
    c. above baseline represent flow away from the transducer.
    d. below the baseline represent flow toward the transducer.

11. (205) What is the best way to distinguish between the normal internal carotid artery (ICA) and the external carotid artery (ECA)?
    a. Temporal tap.
    b. ICA/common carotid artery (CCA) ratio.
    c. Sudden sniffing.
    d. Spectral waveform appearance.

12. (205) What is a *common* method for distinguishing the external carotid artery (ECA) from the internal carotid artery (ICA) when spectral waveforms are abnormal?
    a. Sniff test.
    b. Temporal tap.
    c. Murphy's sign.
    d. Valsalva maneuver.

13. (205) Along with the common carotid artery (CCA) and internal carotid artery (ICA), what other vessel do most institutions require sample measurements?
    a. Vertebral artery.
    b. Subclavian artery.
    c. External carotid artery (ECA).
    d. Middle cerebral artery.

14. (205) What is the *best* way to detect soft plaque?
    a. Increase your filter.
    b. Use the spectral Doppler.
    c. Magnify the grayscale image.
    d. Fill vessel with color or power Doppler.

15. (205) What does heterogeneous plaque suggest?
    a. Ulcer.
    b. Dissection.
    c. Collaterals.
    d. Surgical stent.

16. (205) The purpose of calculating the internal carotid artery/common carotid artery (ICA/CCA) ratio is to
    a. determine patency of the external carotid artery (ECA).
    b. determine peak systolic velocity.
    c. grade stenosis and correct for errors.
    d. grade plaque ulceration in both vessels.

17. (206) From an ergonomic perspective, you would use electronic preset labels to
    a. increase the dexterity of your hands.
    b. minimize use of the ultrasound keyboard.
    c. minimize the potential for labeling errors.
    d. increase the diagnostic value of the image.

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

Student Notes

Unit 2. General Vascular Sonography

THIS UNIT provides a foundation of knowledge you will need to perform sonographic examinations of the abdominal aorta, the aortic branches, the venous vessels of the abdomen, and the blood vessels in the upper and lower extremities. Throughout the unit, we will briefly cover the normal anatomy and function of the various vessels. We will also provide you with standard approaches for obtaining sonographic images. We shall discuss recognizing and documenting abnormalities of these vascular structures.

2–1. Abdominal Imaging

In this section, we will discuss imaging of the abdominal aorta, from its structure and function to the most common abnormalities seen on sonography. Understanding the normal and abnormal appearance of the aorta involves recognizing and capturing those images that are considered standard in the field as well as evaluating abnormalities. This section will also cover some of the imaging problems that surround the branches of the aorta.

207. Anatomy and physiology of the abdominal vessels

For the sonographer, understanding the normal anatomy of the abdominal aorta begins with knowing its location in the body and ends with an examination of its structure. We will use this knowledge of the aorta's anatomy to help us understand its function.

Anatomy

The aorta is extremely important to the survival of the human body. Many complications can arise in organs of the body because of the interruption of normal blood flow through this vessel. Thus, it is necessary for you to increase your familiarity with this structure.

Aorta

The abdominal aorta lies entirely within the retroperitoneum, the area behind the peritoneum or abdominal cavity. The aorta is the largest artery in the body. Its abdominal portion begins at the level of the diaphragm and ends at the level of umbilicus, or T12 to L4 vertebral levels, respectively. The vessel is centrally located and runs just anterior to the spine and slightly to the left. Throughout its length, the abdominal aorta remains close to the spine. This feature distinguishes the aorta from the nearby inferior vena cava (IVC), which moves slightly away from the spine the further it runs caudal or toward the feet. Because of the curvature of the spine, the position of the abdominal aorta becomes progressively more anterior in the body as it runs caudally. At the caudal end, the abdominal aorta bifurcates into the right and left iliac arteries.

The normal diameter of the adult abdominal aorta varies from 2.0 to 3.0 centimeters (cm) at the diaphragm and gradually tapers down to 1.1 to 2.0 cm at the bifurcation. The aortic diameter is slightly larger in men than women and has a tendency to increase with advanced age.

Typical of most large medium-sized arterial vessels, the wall of the abdominal aorta is composed of three separate layers:

1. Tunica intima, the inner layer.
2. Tunica media, the middle muscular layer.
3. Tunica adventitia, the outer layer.

Together, the three layers form a wall of a tube, which encloses an empty space called a lumen, through which blood flows. Arterial walls are elastic, which means they can stretch to expand the diameter of the vessels.

Aortic branches

The branches of the abdominal aorta that supply visceral organs (stomach, liver, spleen, pancreas, kidneys, and intestines) are called *visceral or splanchnic* arteries. Several major branches off the anterior and lateral surfaces of the aorta will split to feed these structures. Three major splanchnic arteries are of principal concern in sonography:

1. Celiac trunk.
2. Superior mesenteric artery (SMA).
3. Renal arteries.

Typically, you may be called upon to perform Doppler on the celiac trunk or SMA, which are sometimes referred to as mesenteric arteries (fig. 2–1). Frequently, the renal arteries are also investigated.

Celiac trunk

The first branch off the abdominal aorta is the celiac trunk (axis). Supplying blood mostly to the liver and spleen, the celiac trunk arises off the anterior surface of the aorta to bifurcate a few centimeters later into common hepatic artery to the right and splenic artery to the left. The left gastric artery also branches off the celiac but is not normally seen during sonography.

Superior mesenteric artery

One centimeter distal to the celiac trunk, the SMA rises off the anterior surface of the abdominal aorta and immediately travels inferiorly in a course roughly parallel with the aorta. The SMA feeds blood to the small intestine and half of the large intestine.

Renal arteries

The renal arteries arise from the anterolateral surface of the abdominal aorta, just below the level of the SMA. The left renal artery is usually closer to the SMA level than the right. The right renal artery courses posterior to the inferior vena cava on the way to the right kidney. Occasionally, more than one renal artery will branch off the aorta to attach at the renal poles. Some renal arteries are duplicated along its course well before reaching the hilum.

Just before entering the kidney, the renal artery will normally form two branches, anterior (superior) and posterior (inferior), that subsequently divide upon entering the hilum. As soon as the anterior and posterior divisions pass through the hilum, they divide into segmental arteries. The anterior artery divides into four segmentals and the posterior artery supplies one segmental. The segmental arteries each further branch into interlobar arteries that stream through the kidney toward the cortex. The corticomedullary junction of the kidney is where the interlobar arteries terminate into arcuate arteries that send off tiny cortex arteries.

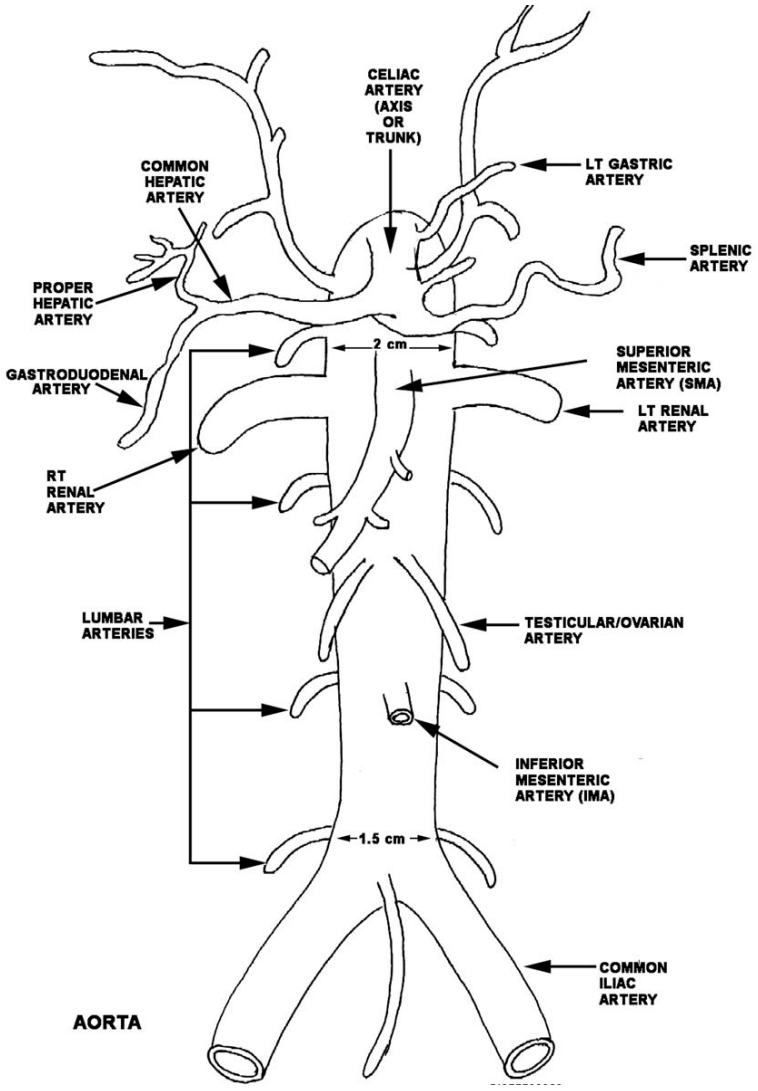

Figure 2–1. Diagram of the aorta and branches.

Venous vessels

The return of blood back to the heart rests with the veins of the abdominal trunk. The main vein is the IVC, which has as many major tributaries as the aorta has branches. We will briefly discuss the major tributary that is easier to detect sonographically.

Inferior vena cava

The IVC courses to the right side of the aorta and anterior to the spine. It begins where the two common iliac veins join just above the level where the sacrum joins the fifth lumbar vertebra and ends at the entrance to the right atrium of the heart.

Renal veins

Two major tributaries that merge into the IVC are the left and right renal veins. The left renal vein courses from the left kidney posterior to the SMA and anterior to the aorta before merging into the left side of the IVC. The right renal vein merges directly into the right side of the IVC. The renal veins course closely and parallel with the renal arteries.

Function of the abdominal vessels

The structure and location of the abdominal aorta determines its function. As the primary arterial vessel of the human body, the function of the abdominal aorta is to supply oxygenated blood to body tissue below the diaphragm. This function is carried out by the contractions of the aorta, which are largely regulated by the muscularity of the vessel wall. As we have seen, the muscle layer of the aorta is called the tunica media. This layer contracts in a delayed timing with the pulsations of the heart and squeezes large volumes of blood at a certain rate of pressure into smaller aortic branches.

For the abdominal aorta to be able to withstand the rigorous contraction and relaxation, the walls must be highly elastic. In fact, the wall's elastic property maintains the equilibrium between the high-pressure arterial system and the relatively low-pressure venous system. Because the wall expands and causes the vessel to hold a large volume of blood, the abdominal aorta also serves as a high-pressure reservoir. The expanded walls contain potential energy until the point where it recoils and contracts into kinetic energy. Thus, blood moves forward even when the ventricle of the heart is closed and relaxed.

Oxygenated blood moves out of the aorta into the abdominal organs and the lower extremities through the various branches. As you may have guessed, the function of the abdominal venous system is to move deoxygenated blood out of some of the abdominal organs and lower extremities into the IVC, the major conduit to the heart. Other abdominal organs, such as the gastrointestinal tract, spleen, and pancreas, drain into the portal venous circulation. Recall from volume one that the portal venous blood is filtered through the liver before entering the IVC (via hepatic veins) at the level just before the right atrium of the heart.

208. Pathology of the major abdominal vessels

The principal danger to the abdominal vascular system is aneurysms of the aorta, closely followed by obstruction of the IVC. This lesson briefly familiarizes you with the major abnormalities of these two important vessels.

Aneurysms

Normally silent and undetected, the aortic aneurysm is a potentially fatal abnormality. Depending on the size of the aneurysm, immediate surgery may be necessary. The sonographer's role with these abnormalities is to characterize the size and shape. To do this, it will be helpful to know a little more about this unique disease.

Typical aneurysms and pseudo aneurysms

Abdominal aortic aneurysms (AAA) are dilatations of the abdominal aorta. More specifically, they are aortic diameter dilatations greater than 3 cm. You may hear some radiologists refer to an AAA as being an increase in aortic diameter of 1.5 to 2 times the normal adjacent diameters.

Appearing mostly in men older than 65, AAA is strongly associated with atherosclerosis. Patients with AAA are frequently asymptomatic. However, if they do have symptoms, common among them is a swollen abdomen that pulsates and pain in the abdomen, back, or legs.

Because these aneurysms usually grow slowly at a rate of a few millimeters annually, diameters between 3 cm and 6 cm are generally followed with a sonographic examination at 6 to 12 months. Complications of AAA can occur with significantly increased diameter. For example, large aneurysms tend to have thrombus in them, which may shed emboli. These emboli can travel to the arteries in the legs and block them, causing leg tissue to be deprived of blood (ischemia).

Another complication is potentially fatal: a leak or a rupture. If the AAA ruptures, it can cause such intense back pain and internal bleeding that the patient may go into shock. Approximately half of AAA cases rupture, for which computed tomography (CT) is usually the diagnostic tool of

choice for physicians. Many rupture cases go straight to surgery, and most clinicians will schedule emergency surgery for AAA diameters of 7 cm or greater.

Classifications of abdominal aortic aneurysm

Sonographers use several classifications of AAA based either on physical description or on suspected causes. For physical description, the classifications usually describe the shape or extent of the AAA:

- Fusiform – A dilatation that tapers at both ends. This is the most common AAA.
- Saccular – An abrupt, rounded dilatation that can have the appearance of a mass alongside the aorta. These types can become quite large.
- Cylindrical – The entire length of the aorta is uniformly dilated, particularly noticeable distally, which is supposed to gradually taper from the renal artery level to the bifurcation.

Some institutions classify AAA by the underlying pathology, which is a way to distinguish real aneurysms from abnormalities that have the appearance of aneurysms. Thus, for some radiologists, the abnormality is either an aneurysm in the patient or it is not. That is, the aneurysm is a true aneurysm, where all three layers of the aortic wall remain intact.

A true aneurysm is opposed to a false or pseudoaneurysm. A hole or breach occurs in the aortic wall, where a jet of blood escapes into the surrounding tissue during systole and flows back into the aorta during diastole. The blood outside the aorta is walled off by a clot, forming a false wall that, on sonography, mimics the appearance of an aneurysm.

Arterial dissection

The rare arterial dissection has, in the past, been considered an aneurysmal classification, usually referred to as dissecting aneurysm. However, it is now recognized that what causes the dissection is also responsible for aneurysms; that is, weak vessel walls. In the past, the term, *dissecting aneurysm*, implied dissections could only happen from aneurysm. However, it is not clear that one causes the other. Dissections may or may not exist simultaneously with an aneurysm. Thus, a dissection is not a type of aneurysm.

When the intima tears away from the media or both layers tear away from the adventitia, the wall is usually weak enough where blood flowing at high pressure in between the layers can expand the diameter. Thus the dissection appears alongside an aneurysm. However, most people with aneurysms will *not* have a dissection, and most dissections will *not* appear with aneurysm. For this reason, most dissections are more accurately referred to as *arterial dissections* instead of the inaccurate dissecting aneurysm.

Stenosis

Another abnormality you may encounter in the abdominal aorta will be the narrowing resulting from plaque or thrombus. Plaques are seen more commonly in older patients because of hypertension, high cholesterol, diabetes mellitus, and smoking. Most stenotic material found in the abdominal aorta is usually plaque, which frequently has massive amounts of calcification. Plaque formation can be extremely irregular with jagged surfaces, which typically attracts thrombus formation. Abdominal aortic plaques form mostly on the posterior wall below the level of the renal arteries. They can grow to a point where they may form stenosis in the iliac arteries, compromising arterial flow to the legs. Stenosis in the renal artery can cause serious problems for kidney function.

Venous thrombosis

Occasionally, clot or thrombosis will form within the IVC, usually from improperly working valves that prevent the proper moving of blood back to the heart. As a result, blood stays in one place along the IVC column for too long, an abnormal state called *stasis*. Venous thrombosis can

form in the iliac veins or lower extremity veins and spread into the IVC. The danger is in reduced blood flow or the complete blocking of flow return to the heart. Thrombosis can also occur in the renal veins and develop subsequently into the IVC.

Grafts

Another abnormality you may have to evaluate is a surgical graft seen within the abdominal aorta. Generally, the concern is not with the graft itself but with problems that can develop (complications) from it.

Several types of grafts are used to repair damaged abdominal aortas or their iliac artery branches. Most are attached to the end of the proximal aorta and to the ends of either the distal aorta or the iliac arteries. Sometimes the distal portion is attached to the sides of the iliacs.

Common Abdominal Aortic Grafts	
Tube	End-to-end. Usually limited to abdominal aorta below the level of the renal arteries. Often wrapped with native aorta.
Aortoiliac	End-to-side. This graft will split into two branches to attach to the sides of the iliac arteries.
Aortobifemoral	End-to-end. Also splits into two branches to attach to the ends of the iliac arteries.
Prosthetic stent	Expandable stent placed within an aneurysm (endoluminal). Usually surrounded by serous fluid or blood collection post-surgery. The stents are introduced at another location and are threaded via catheter wire to the aneurysmal level.

Occasionally, fluid will collect at the anastomosis site. If seen just after surgery, the fluid collection is usually a collection of old blood (hematoma), which is normally reabsorbed by the body in a few weeks. Frequently, the fluid collection will be infected (abscess) and increase in size. Also, the anastomosis may dilate and form new aneurysms that may develop at the anastomosis site. Finally, stenosis and occlusion may result from thrombus formation at the anastomosis site.

209. Imaging the normal and abnormal aorta

The approach to the aorta is routinely done along with the typical abdominal sonogram in most departments. Thus, the preparation and techniques are roughly similar. When the focus is strictly on the aorta, however, the approach has slight modifications, which are discussed below.

Considering standard approaches

Sonographic imaging of the abdominal aorta is performed primarily to rule out the presence of an aneurysm, which is an abnormal dilatation of a blood vessel. To accomplish this task requires standard approaches to measurement and visibility.

Patient and equipment preparation

To prepare properly, patients for sonograms of the abdominal aorta should be instructed to fast four to six hours before the start of an examination. This fasting protocol is used for the same reason you would have a patient fast for an abdominal sonogram, which is to clear the body of bowel gas, which frequently obstructs and scatters sound. For those times when the patient has not properly fasted, such as in emergencies, you can apply gentle pressure with the transducer to move gas-filled bowel loops out of the field-of-view.

Examination of the abdominal aorta is generally performed with the patient lying in the supine position. Sometimes a large patient must be rolled onto his or her side or into a lateral decubitus position for sonographic access to the abdominal aorta.

Along with getting the patient prepared, you should consider the equipment. Use a four to five megahertz (MHz) transducer for the average patient. You may have to drop the frequency down to 3 MHz for larger patients. A sector scanner can be used but a curvilinear arrayed transducer

will normally get the most anatomy on the screen with fewer images. Ensure that your gain is low enough to eliminate stray echoes from the lumen of the abdominal aorta without sacrificing the ability to detect subtle, hypoechoic anomalies near the vessel wall.

Standard longitudinal images

Normally for longitudinal images, a radiologist will need only three that demonstrate the length of the abdominal aorta. This bare minimum should adequately demonstrate the presence or absence of disease. Do not be surprised if a radiologist requires you to obtain color Doppler images of the entire length of the aorta. Such an approach is useful for detecting subtle abnormalities, such as thrombus or dissections. Let's take a closer look at the three basic abdominal aorta longitudinal images:

1. Proximal.
2. Middle.
3. Distal.

Proximal abdominal aorta

Document the length of the abdominal aorta at the most superior end. Generally, you will have to place your transducer anteriorly just below the sternum with a slight angling of the transducer toward the head. The first structure that branches off the aorta's ventral, or anterior, surface is called the celiac trunk (axis) and, if seen, should be included in this image. The second structure branching off the ventral surface is the SMA, which should also be documented on the image. Keeping in mind the preferences of your radiologist, you should document the anteroposterior (AP) measurement of the proximal abdominal aorta from outer wall to outer wall (fig. 2–2).

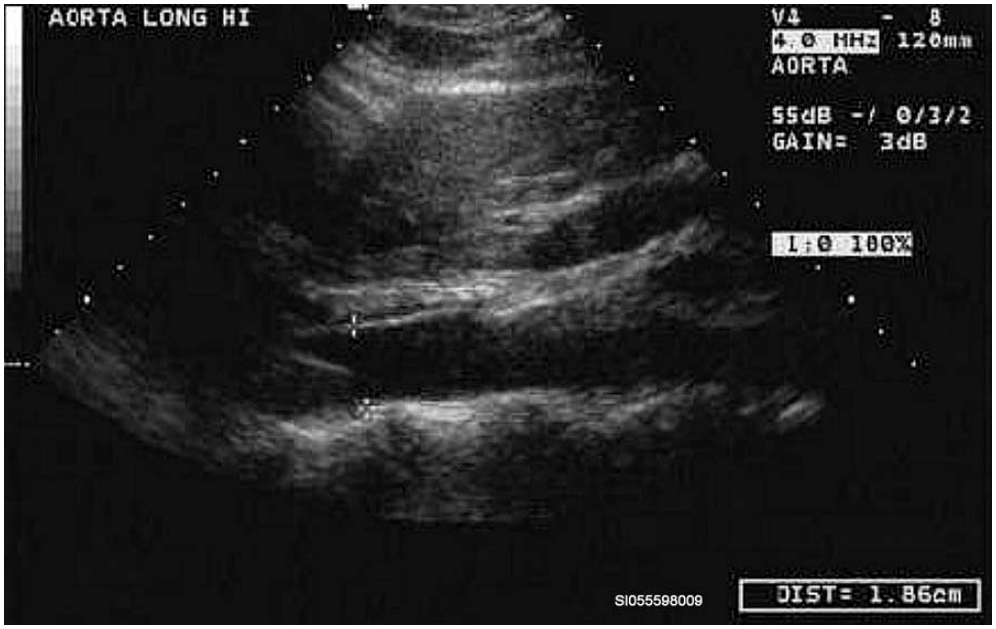

Figure 2–2. Longitudinal proximal abdominal aorta with AP measurement.

Middle abdominal aorta

Document as much of the next region caudally that will fit into the image. This will be the middle portion of the abdominal aorta. Measure the AP diameter at this level. Depending on the body habitus of the patient, you may have to apply a little more pressure with the probe. If bowel gas or patient size prevents you from seeing the aorta, turn the patient into a right lateral decubitus position and scan coronally from the left flank, usually against the side of the rectus muscle of the anterior abdomen.

Distal abdominal aorta

Document the inferior end of the abdominal aorta, using the same approach as you would for the middle abdominal aorta. Again, measure the AP diameter. Also document the proximal iliac arteries and, if required, measure the AP diameters. When scanning from the anterior abdomen, documenting the aortic bifurcation into iliac arteries in the longitudinal plane can be difficult. To solve the problem, briefly turn the transducer into the transverse plane and scan from the distal aorta through the bifurcation. Otherwise, the alternate coronal plane approach affords a ready view of the bifurcation and both iliac arteries in the same longitudinal image.

Standard transverse images

In sonography, no study is complete with just longitudinal images of a subject. We must also obtain transverse images for location and even for discovering abnormalities unseen in the longitudinal view. Let's look at three standard transverse images of the abdominal aorta.

Proximal abdominal aorta

Document the cross section of the abdominal aorta at its most proximal end. Remember that the aorta runs next to the spine and that the spine has a natural curve. These facts should help you orient your transducer so that the ultrasound beam will be as close to perpendicular to the plane of the aorta as possible. Once you are certain of the perpendicular orientation, measure the AP diameter. Some institutions will require you to include the celiac trunk or the SMA on the image. A good way to obtain a convincing image that contains celiac or SMA is to use color Doppler, which should show branches off the anterior surface of the abdominal aorta.

If necessary, the width measurement can be obtained transversely; however, you may have to position your transducer to the left flank of the body (as much as possible at this level). Because the sides of the vessel may be obscured by border shadowing, anterior beam approaches can cause you to guess the true location of the outer edge width of the abdominal aorta wall. The left flank approach will give you a more coronal view that may reliably display the sides of the aorta.

Middle abdominal aorta

Document the transverse view of the middle portion of the aorta. Typically, this is just a few centimeters distal to the SMA. Take the AP measurement at this level and, if required, the width measurements. Some institutions may have a protocol that calls for documentation of the right and left renal arteries at this level. This can be extremely difficult. However, as with the SMA and celiac trunk, you can use color Doppler to identify the color branching off the aorta. In this case the color branching will be to either side of the abdominal aorta.

Distal abdominal aorta

Document the transverse view of the distal abdominal aorta just before the level of bifurcation. It is appropriate to document the AP measurement at this level. Also, it is good practice to document the bifurcation itself because plaque tends to accumulate at this level. Some institutions require AP measurements of the iliac arteries as well.

Aneurysms

You should keep in mind that standard images are designed to catch most aortic abnormalities, from stenosis to dissections. Aneurysms of the abdominal aorta are often quite dramatic and you will not need standard images to detect them. However, you may still need them to detect other less obvious abnormalities that may simultaneously occur.

If you suspect a pseudoaneurysm, use color flow to attempt to identify the jet of blood between the aorta and the escaped (or extravasated) blood. It is also a good idea to measure length, width, and height of the entire pseudoaneurysm. The difficulty with pseudoaneurysm is it can be confused with structures around the aorta, such as infected lymph nodes (lymphadenopathy) or retroperitoneal masses. This is because both the pseudoaneurysm and the extra aortic mass have

hypoechoic sonographic appearances and can have similar round shapes. However, blood leaking from the abdominal aorta may surround the aorta or lay in a relatively thin line along the wall, which is helpful for distinguishing collection from a mass.

Sonography of abdominal aortic aneurysms

On sonography, you should generally see the AAA below the level of the renal arteries. Thrombus or plaque or both may appear within the aneurysm, particularly against the anterior or sidewalls. Thrombus usually appears as low-level echoes, while plaque may be slightly more echogenic, specifically if calcifications are present. For this reason, it is a good idea to ensure that your overall gain is not reduced to the point of obliterating all subtle low-level echoes.

The most important step for you to make is to measure the dimensions of the aneurysm. A commonly accepted approach is to measure the length and AP diameter of the AAA in the longitudinal view and the width in the transverse view. To get a clear view of the sides of the vessel, it is a good idea to measure the transverse width from the patient's left flank. The caliper placement from the flank of the sidewalls will appear to be another AP measurement on the screen.

You should attempt to describe or document the distance between the renal arteries and the beginning of the aneurysm. The measurement serves as important information for surgeons who may potentially operate on the aorta. In an aortic repair procedure, aortic flow into the renal arteries is left untouched as much as possible during the surgery. If the AAA is at the level of the renals or above (suprarenal), surgeons may have to close off and later reattach the renal arteries. The renal arteries are usually 1.5 cm inferior to the level of the SMA. Perhaps the best way to demonstrate the distance is from a coronal or flank approach.

Be aware that the AAA may be tortuous so a flank approach is perhaps a very good way to ensure that you have measured the diameter of the aneurysm at its widest point. Also, you should examine the kidneys for possible shrinking, due to stenosed renal arteries from thrombus, or for hydronephrosis due to the aneurysm putting pressure on and clamping off (impinging) the ureters.

Arterial dissection

On sonography, you should see an arterial dissection as an echogenic line, running roughly parallel with the aortic wall, in the center of the lumen on longitudinal views. The line represents the intima or both the intima and media layers torn away from the vessel wall. Usually, this torn away layer can be seen flapping in the lumen from the pulsations of flowing arterial blood. If just the intima is torn away, you should see a thin line. If both the media and intima are torn away, the line should be thicker. At times, the line will be fixed, particularly in the presence of thrombus to either side.

To recognize arterial dissection properly, you must understand some of the possible variations of sonographic appearance. The lumen in between the dissection and the wall is called the false lumen and it usually stretches from the intimal tear to the tapered end. Blood flows turbulently within this area and can only escape back out of the tear. Occasionally, another hole in the intima will open, allowing the blood to flow back into the true lumen. This picture can become quite confusing with the presence of thrombus filling the entire false lumen, which may hide the dissection. Use color Doppler to determine where flow jets or tiny channels of flow are located and to help determine where flow is directed. Another potentially confusing presentation is for the false lumen of a dissection with only one entrance and exit to expand and eventually narrow or close off the true lumen of the abdominal aorta. Use spectral Doppler to analyze velocity changes brought on by the dissection-caused stenosis. Just as you would measure plaque stenosis, sample at the narrowest point in the true lumen, if seen.

Most dissections extend from the aortic arch to about the middle of the abdominal aorta. Thus you may never see the intimal tear, but you may see the tapered blind end. Because dissections

can extend into the abdominal aorta from the proximal, they have the potential to clamp off the major branches of the aorta, such as the celiac trunk, SMA, and even, dangerously, the renal arteries.

It should be obvious that without the presence of dissection or pseudoaneurysm, a radiologist will assume an abdominal aortic dilatation to be a true aneurysm, and will rely mostly on a physical description for diagnosis.

Stenosis

As with stenosis in carotid arteries, arterial blood will increase its velocity through an aortic stenosis in direct proportion with the increasing narrowness of the channel. At some point the channel diameter will be so thin that velocity will diminish considerably.

As stated before, plaque formation can be extremely irregular with jagged surfaces, which typically attracts thrombus formation. This can make the sonographic appearance very difficult to capture. You should use color Doppler, which is, in these cases, valuable for outlining the surface of the plaque, particularly in the presence of shadowing from calcifications.

Some institutions require color Doppler and spectral waveform analysis of abdominal aortic stenosis. Remember that the depth of the vessel in the body will require some adjustments in your equipment, such as frame rate and color sensitivity. Keeping you color box sized only to the area of the abdominal aorta of interest will increase the sensitivity considerably. Longitudinal and transverse images of flow before, through, and after the stenosis should give the radiologist a good picture of patency and characteristics such as turbulence. Transverse color Doppler images are a good way to determine if any thrombus or plaques are blocking or narrowing the major aortic branches. In some cases where considerable tissue between the transducer and the aorta exists, no amount of instrument adjustment will give you optimal Doppler information. You may find many radiologists referring such patients to CT.

Radiologists will be concerned primarily with the shape of the waveform when conducting spectral analysis. Recognize that most abdominal aortic waveforms demonstrate high-resistance; that is, a sharp upstroke and high velocity for systole followed by a rapid down stroke to the baseline for diastole, with some reverse flow below the baseline. The systolic height of the waveforms diminishes as flow progresses from the proximal abdominal aorta to the iliac arteries. Radiologists will probably become more concerned with peak systolic and end diastolic velocities only in the presence of stenosis. For stenotic aortas, the waveform may demonstrate a normal empty window with typical high systolic peak and relatively low diastolic velocity just before the stenosis. Within the stenosis, the systolic peak will rise as will diastole; however, turbulence will fill in the spectral window in the diastolic portion. Just after the stenosis, systole and diastole return to pre-stenotic levels but with the window filled with spectral broadening echoes. For all waveforms that you capture, ensure that they are properly documented and be prepared to explain to radiologists the significance of the various shapes.

Grafts

Grafts are easily seen using sonography, particularly when outlined with fluid. Most grafts will be brightly echogenic and display a parallel series of lines. You should document this with grayscale image and insure that the parallel lines are clearly seen. Grafts are attached (anastomosed) to native aortic tissue, which shows up as an abrupt echogenic thickness of the walls at the connection point. This feature should also be documented. Finally, document any endoluminal stents, which will show an echogenic mesh pattern within an aneurysmal aorta. The stents themselves, once expanded, are usually surrounded by fluid on either side to the vessel wall, depending on the amount of expansion.

Because complications may arise, your job with grafts and stents will likely be limited to evaluating blood flow at the connecting proximal end and distal end of the graft using color and

spectral Doppler. As with other documentations, measure any collections and thrombotic stenosis sites.

210. Imaging abdominal vessels

Occasionally, you will be called upon to perform Doppler investigations of vessels inside the abdomen apart from the aorta. Most of these investigations will involve the major branches off the aorta and are usually performed in conjunction with other studies. In this lesson, we will briefly introduce major areas of Doppler evaluation and some important things to remember concerning each.

Most abdominal Doppler evaluations are limited to the renal arteries and the main portal vein. However, some institutions may routinely or in an emergency require you to perform Doppler sonography on other vessels.

Arterial

Occasionally, the major branches of the aorta are sonographically examined, sometimes in conjunction with aortic problems, such as aneurysm, dissection, or stenosis. Generally, the major branches that supply the digestive organs and kidneys are the primary focus in many departments. This is because patients are being clinically evaluated for problems such as disruption or obstruction of blood supply to the intestines (mesenteric ischemia). As with the abdominal aorta, the best transducer to use to scan the major abdominal arteries is a curvilinear array with at least 2 to 5 MHz frequency. Color and spectral Doppler should be used throughout.

Celiac trunk

The celiac artery with its branches is usually best seen in transverse and with color Doppler. If not visible, try a longitudinal view. Once visualized, the artery is sampled with an angle incident of 60 degrees or less. If the celiac is sampled close to its origin, it may generate a spectral waveform similar to the one found in the aorta; that is, a high resistance waveform. Sampling further along the length of the celiac will settle the waveform down into a pattern that represents a combination of the common hepatic and splenic arteries. Thus, you should see continuous flow in diastole, representing the low resistance of the liver and spleen to high perfusion of blood.

A peak systolic velocity of 200 cm per second or more may indicate stenosis. In such cases, some institutions may require samples of the hepatic artery and even the splenic artery.

Superior mesenteric artery

On sonography, the vessel is best seen in the longitudinal view. However, it can be well defined in the transverse view because of the echogenic fat that surrounds it. Certain sonographic landmarks make locating the SMA easier. For example, the most obvious is, in the transverse view, the left renal vein coursing between the SMA and the aorta. Also the pancreas is directly anterior.

Doppler waveforms reveal turbulent flow at the point of origin that becomes more uniform as the vessel turns inferiorly. Two normal states of waveform exist for the SMA and both depend on the digestive process. When a patient has fasted, the waveform displays a high resistance profile with a sharp peak systolic velocity (PSV) but an absent diastolic flow. Between 30 and 90 minutes after eating, the waveform displays a low resistance profile with a rounded PSV and a significant increase in continuous diastolic flow.

A signal that flow is abnormal in the SMA would be a low resistance waveform in a fasting patient. Stenosis of at least 70 percent can also be suggested by a PSV greater than 275 cm/sec.

Renal arteries

Of primary concern to a radiologist when considering renal arteries is the openness or patency of the vessels. Is blood flowing through the renal artery to the kidney and, because of its normal low

resistance, is it flowing at the proper rate? Most abnormalities that may prevent normal flow will be a stenosis or occlusion of the renal artery causing hypertension, typical reasons for examining the vessel.

Renal arteries are evaluated either directly or indirectly. Because the entire length of the renal artery is difficult to see via sonography, even with color Doppler, most institutions limit direct evaluation of renal artery stenosis to sonography of the proximal and distal portions of the artery. Doppler sampling at the proximal renal artery just off the lateral wall of the aorta and sampling at the distal end before branching into the kidney is the usual method for evaluating flow into the kidneys. If seen, the middle portion of the artery should be sampled.

Locating the renal artery can be problematic. Certain landmarks may ease efforts to see the arteries. For instance, the right renal artery is the only vessel that lies posterior to the longitudinal image of the IVC, appearing as a black circle just beneath. Once the right renal artery is located the left renal artery should be slightly superior and directly across the aortic lumen.

This direct sampling of the renal artery, however, is limited in documenting stenosis, as actual thrombus or plaques are typically impossible to see. The stenosis, therefore, is usually inferred from spectral waveforms and their velocities. A renal artery PSV of greater than 150 cm per second should be viewed with suspicion for at least 60 percent stenosis.

Another useful method for directly assessing renal artery stenosis is the renal-aortic velocity ratio (RAR). It is the renal artery PSV divided by the abdominal aortic PSV sampled from the same level as the renal artery origin. A ratio greater than 3.5 suggests renal artery stenosis.

Renal artery stenosis can also be estimated or inferred from sampling within the kidney itself. Because the kidneys are hypervascular structures, any hemodynamically significant stenosis or occlusion in the main renal artery will show in reduced flow, or perfusion, of blood in the major intrakidney arteries (segmental and interlobar). Because of easier access to the microvasculature of the kidneys, standard criteria have been developed for this indirect approach, and it is in use in many departments. Thus, sampling at the superior, middle, and inferior poles should indirectly suggest the percentage of stenosis within the main renal artery.

From these samples, certain calculations can be made from the waveforms. One particularly important calculation for this indirect method is called the *acceleration time*, which is simply the amount of time between the end diastolic velocity of one pulse and the peak systolic velocity of the next pulse. Acceleration time, sometimes called *upstroke*, is only a few microseconds and appears on spectral waveform to be a nearly vertical and sharp rise. Acceleration times greater than 0.07 seconds are usually considered abnormal and suggest obstruction or stenosis of at least 60 percent in the main renal artery. This stenosis causes the slowing down or blunting of systolic flow into the kidney and dampens the downstream intrarenal signals, an effect called *parvus-tardus* (or tardus-parvus; either combination refers to *both* pulsus tardus and pulsus parvus). *Tardus* is the delayed upstroke, and *parvus* is the rounded and lowered systolic peak.

Whichever approach used, direct or indirect, a radiologist may find it helpful to have a sonographer calculate the significance of certain measurements. One such measurement is the resistive index, which is the amount of resistance to incoming blood a kidney will display. A convenient method used by sonographers to calculate the resistance is through a brief formula:

$$RI = \frac{A - B}{A}$$

Calculate the resistive index (RI) by subtracting the end diastolic velocity (B) from the peak systolic velocity (A) and dividing the result by the peak systolic velocity. Normal kidney RI is usually less than 0.75.

Venous

Abnormalities may occur in the low-pressure half of the circulatory system, the venous system. For this reason, sonography can be a useful tool. Doppler methods work the same for venous flow as for arterial; that is, spectral and color yields useful information about direction, presence, and velocity of flow. The differences are slight and it must be remembered that forward flow for veins is the reverse direction of the artery.

Two principal abnormalities that require Doppler evaluation of the abdominal veins are thrombosis and tumor invasion. Both abnormalities will either interfere with normal venous flow or will cause complete blockage of the vessel. Sonographers performing abdominal examinations will encounter these two abnormalities mostly in the IVC and the renal veins (fig. 2–3).

Inferior vena cava

Doppler evaluation of the IVC is generally performed when the IVC is abnormally dilated—usually greater than 3.7 cm in AP diameter. In most patients with dilated IVC, the vessel will not collapse with expiration. Color flow and waveform sampling is done mostly in the upper abdomen where the IVC enters the level of the liver. Seeing the IVC at inferior levels can be difficult with the presence of bowel gas. Gentle pressure with the transducer or left lateral decubitus positioning combined with right flank transducer placement may help with moving bowel away from the IVC. Both longitudinal and transverse views are necessary for grayscale imaging, but longitudinal will be best for Doppler investigation.

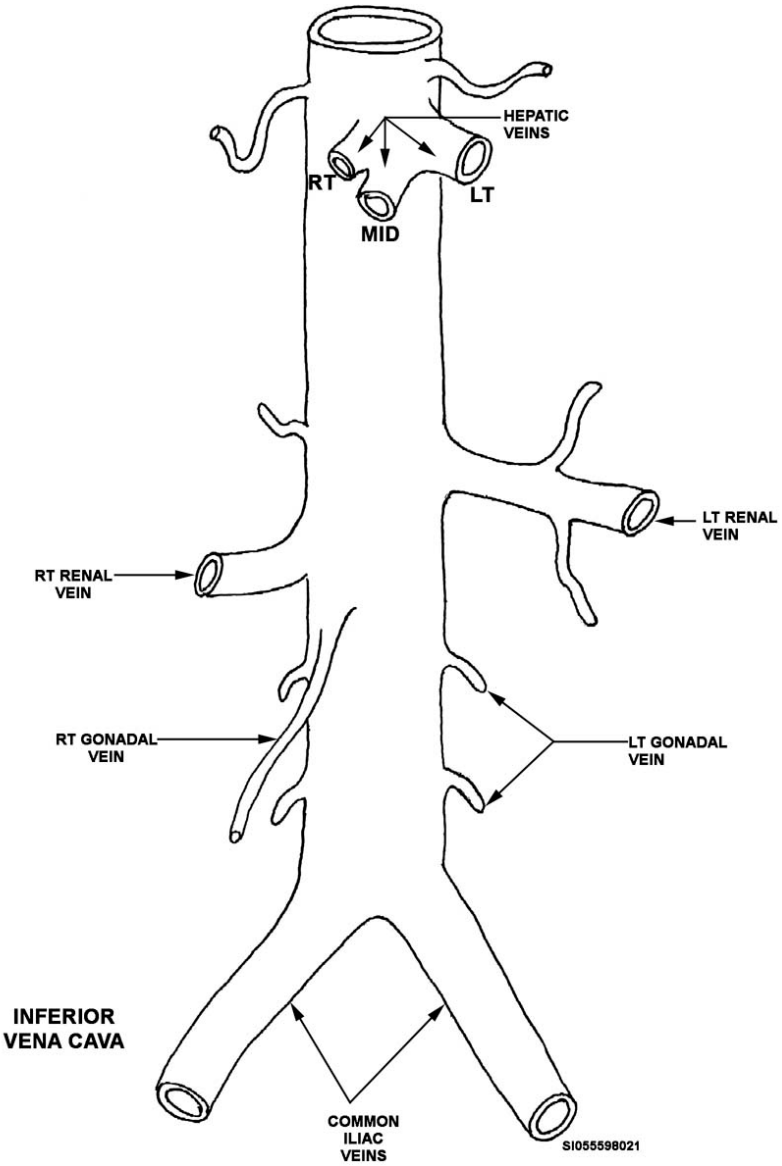

Figure 2–3. Diagram of the IVC.

Normal IVC flow is similar to venous flow throughout the body. Spectral waveforms show flow continuously moving toward the heart at low and steady velocities that vary with respirations (phasicity). However, the waveform may show pulsations the closer to the heart you sample. This is due to the rebounding effect of pressure waves coming off the opening and closing of the right atrium of the heart.

The most common IVC abnormality, thrombosis, is readily seen via Doppler. Color Doppler can flow around a thrombus and highlight the structure. In the presence of thrombus that nearly blocks the IVC, color Doppler may be able to detect small channels of venous flow. Two excellent ways to pick up the slow flow states in abnormal venous flow caused by thrombus is to lower your color and spectral Doppler filters and to use power Doppler. Recall that power Doppler does not require an angle orientation to the vessel to detect moving blood; instead, the power ultrasound beam can be directly perpendicular to the vessel.

Color and spectral Doppler is also a useful way to distinguish between thrombus formation and tumor. On grayscale, thrombus and tumor may both display varying appearances from homogeneous low-level echoes to heterogeneously complex echoes. The similar appearances can make distinguishing between the two extremely difficult. Thus, Doppler can demonstrate evidence for the presence of tiny arterial blood vessels within an IVC tumor, with the most common tumor being invasive renal cell carcinoma (RCC). Venous blood flowing in narrow channels through a thrombus formation should link up with venous flow in the lumen of an adjacent normal section.

Renal veins

When examining the IVC for the presence of thrombus, sonographers attempt to discover any extension of clot into the connected renal veins. Also, tumor invasion from the kidneys may travel along the renal veins to invade the IVC. Color Doppler is ideal for this type of work because color flows around and highlights the surface of thrombus within the vessel. Therefore, renal vein investigation using Doppler is important for diagnosing cases such as RCC and renal vein thrombosis (RVT).

Transverse views of the IVC should allow you good visualization of the renal veins. A useful landmark to orient you to the renal veins from a transverse IVC is the level where the left renal vein crosses in between the SMA and the abdominal aorta. Following the course of the left renal vein to the IVC should give you the same level as the shorter right renal vein entering the other side. Color Doppler will highlight these vessels for a more precise location.

Normal renal veins should demonstrate identical phasic waveforms found in the IVC. On the other hand, a different picture will appear with RVT. To diagnose thrombosis in the renal vein, the clot should be directly seen and flow should be absent. The diameter of the renal vein will increase if the thrombus nearly blocks or completely blocks the vessel. Color flow is helpful to determine if flow is present. If the vessel is dilated and the clot is seen, RVT is diagnosed.

Renal vein tumor extensions will show the same characteristics except the tumor may display arterial vessels. Otherwise, distinguishing between tumor and thrombus can be difficult. As with IVC investigations, color flow settings should be lowered to increase sensitivity to slow flowing blood.

Self-Test Questions

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

207. Anatomy and physiology of the abdominal vessels

1. What distinguishes the abdominal aorta from the IVC?

2. What happens to the abdominal aorta diameter between the diaphragm and the bifurcation?

3. List the three layers of the abdominal aorta wall.

4. List the three major splanchnic arteries.

5. What vessel does the right renal vein merge directly into?

6. What regulates the contractions of the abdominal aorta?

7. What are the two functions of the abdominal aorta?

208. Pathology of the major abdominal vessels

1. Besides a diameter greater than 3 cm, what other criterion may prompt a radiologist to classify an abdominal aortic dilation as an aneurysm?

2. List the three physical description classifications for the abdominal aortic aneurysm.

3. Where inside the abdominal aorta will plaques mostly form?

4. List the common abdominal aortic grafts.

209. Imaging the normal and abnormal aorta

1. What is the *primary* reason to perform abdominal aorta sonograms?

2. What are the standard longitudinal images in abdominal aorta sonography?

3. When is the abdominal aorta considered an AAA?

4. Why are AAAs of 3 to 6 cm followed at 6 to 12 months?

5. What is the most important step to make with an AAA?

6. What is a good way to ensure you have measured the aneurysm diameter at the widest point?

7. What is the appearance of dissection on sonography?

8. Where do AAA plaques mostly form?

9. When will radiologists probably become more concerned with peak systolic velocity and end diastolic velocity in the abdominal aorta?

10. Match the graft in column B with its description in column A. Each item in column B may be used only once.

Column A	***Column B***
____ (1) End-to-end. Usually limited to abdominal aorta below the level of the renal arteries. Often wrapped with native aorta.	a. Prosthetic stent.
____ (2) End-to-side. This graft will split into two branches to attach to the sides of the iliac arteries.	b. Tube.
____ (3) End-to-end. Also splits into two branches to attach to the ends of the iliac arteries.	c. Aortoiliac.
____ (4) Expandable stent placed within an aneurysm (endoluminal). Usually surrounded by serous fluid or blood collection post-surgery. The stents are introduced at another location and are threaded via catheter wire to the aneurysmal level.	d. Aortobifemoral.

210. Imaging abdominal vessels

1. What does continuous diastolic flow in the celiac trunk represent?

2. What renal artery PSV should be viewed for suspicion for at least 60 percent stenosis?

3. What method indirectly suggests percentage of stenosis in the main renal artery?

4. What formula is used to calculate resistance?

5. What is the normal kidney RI?

6. How can IVC thrombosis be readily seen?

7. What type of waveforms will be seen in normal renal veins?

2–2. Upper Extremity Abnormalities

Vascular abnormalities of the upper extremities, when compared with the lower extremities, are relatively uncommon. Most upper extremity arterial problems concern occlusion or stenosis from arteriosclerotic plaque. In this section, we will briefly touch on upper extremity sonographic imaging.

211. Upper extremity sonography

Most venous upper extremity abnormalities are the result of thrombosis developing from such things as catherization of the internal jugular vein. In this lesson, we will briefly touch on upper extremity anatomy and physiology and pathology.

Anatomy and physiology

Upper extremity arteries and veins normally follow closely parallel with each other. We will discuss the basic anatomy of the upper extremity arteries and their primary function. We will also cover the anatomy and physiology of the veins in the arm and the large vein draining the neck.

Arterial

The vessels that begin the vascular structure of the arms or upper extremities are the subclavian arteries. For the right arm, the right subclavian artery originates off the brachiocephalic trunk, the first branch of the aorta. On the left, the subclavian artery branches directly off the aorta. The subclavian artery runs behind the clavicle until it comes to the lateral portion of the first rib where it enters the axilla, or armpit. Thus, in this region, the same vessel is called the axillary artery. When the axillary artery passes into the upper arm region, it becomes the brachial artery. The brachial artery runs medial to the humerus until the level of the elbow where it bifurcates into the radial and ulnar arteries. The radial artery passes along the lateral aspect of the forearm and the ulnar artery courses along the medial aspect.

Veins accompany upper extremity arteries. Thus, the subclavian is closely paired with one vein; the axillary artery occasionally runs parallel to two veins; the brachial artery is always paired with two veins; and the radial and ulnar arteries each have a vein.

Also, a particular anatomic structure, which can be significant for certain disease processes, is located in the area where the subclavian artery leaves the chest region. At this point, the subclavian artery runs in between the distal ends of two neck muscles. This space is known as the *thoracic outlet*. Accompanying the subclavian through this extremely narrow opening are the brachial nerve and the subclavian vein.

The primary purpose of the upper extremity arteries is to supply oxygenated blood to the upper arm, forearm, wrist, hand, and digits. The arm is highly resistant to incoming flow and thus arterial blood comes in at high pressure. This high resistance is due to the relatively small need in the muscles and tissues of the arm for continuous blood compared to organs such as the brain. However, this situation is reversed when the arm is exercised, creating a need for more oxygenated blood in the muscles and, thus, a reduced resistance to flow.

Venous

There are two series of veins draining toward the heart that concern sonographers for upper extremity venous diagnosis: the veins of the neck and the veins of the arm. In the neck, specific interest is paid to the internal jugular vein (IJV), which runs closely beside the carotid artery. As it drains deoxygenated blood from the brain, the IJV joins the subclavian vein to form the brachiocephalic vein, which travels behind the sternum on the way to the superior vena cava.

The subclavian vein lies behind both the central portion of the clavicle and the subclavian artery. Blood in the subclavian vein comes from two tributaries that merge behind the lateral portion of the clavicle, the axillary and cephalic veins. The cephalic vein courses from the subclavian over the shoulder to run along the lateral part of the arm and is extremely superficial in location, just a few centimeters below the skin. The axillary vein runs paired with the axillary artery in the axilla region. The merger of the upper arm veins, the brachial and basilic veins, forms the axillary vein. The basilic vein is, like the cephalic vein, superficially located in the upper arm and forearm, but on the medial side. The basilic and cephalic veins are *not* paired with arteries.

The brachial vein is centrally located anterior to the humerus and behind the bicep muscle. This vein will usually split into two duplicate veins that run parallel and closely to either side of the brachial artery. The duplicate brachial veins normally rejoin at the level of the elbow before splitting again to enter the central forearm as radial and ulnar veins, each paired with an artery.

The axillary, brachial, radial and ulnar veins are considered the deep veins of the upper extremity. The cephalic and the basilic veins are considered superficial. Both deep and superficial are considered equally important when evaluating for abnormalities, unlike the lower extremity where the deep veins are more important. This is because both sets of veins in the upper extremity are both major pathways for venous drainage into the subclavian vein.

Pathology of the upper extremity

The principal reason for performing sonography on the upper extremity arteries is to rule out the presence of arterial occlusion or stenosis. Vascular disease in the upper extremity is relatively rare compared with the common abnormalities found in the vessels of the lower extremities. Atherosclerotic plaque is the most common arterial abnormality to occur in the upper extremity and is usually located at the first portion of subclavian artery. Because stenosis or occlusion of the proximal subclavian can cause the artery to steal blood from a collateral vessel—primarily the vertebral artery of the neck—the arm will usually not suffer outright blood loss. Thus, the abnormality can remain silently undetected.

Subclavian steal or a stenosis developed elsewhere in the subclavian artery, however, can cause detectable changes in blood flow. A stenosis in the subclavian artery is frequently discovered incidentally during a *lower* extremity segmental pressure examination, a specialized study that examines vascular flow in segments of the extremity. While performing a segmental pressure exam a sonographer may discover a difference in pressure between the two brachial arteries in either arm of greater than 30 millimeters of mercury (mm Hg). This is an indication of stenosis in the subclavian artery.

Sometimes the narrowing is severe enough to cause symptoms of claudication in the arm. Similar to what occurs in the lower extremities, claudication is pain in the arm muscles brought on by exercise, which is relieved by resting the arm.

Pseudoaneurysms also can occur, usually as a result of a surgical procedure involving catheterization of an artery. The pseudoaneurysm involves blood escaping through a hole to the outside of the blood vessel but contained within a wall of clotted blood. On diagnostic exams, the blood vessel will suddenly appear to have an increased diameter, giving the appearance of an aneurysm. The process is similar to that sometimes found in the aorta.

Another abnormality is the thoracic outlet syndrome, which mimics atherosclerotic stenosis. Recall the narrow opening between the two neck muscles that the subclavian artery courses through on the way to the lateral portion of the first rib. The brachial nerve and subclavian vein also run with the artery through this space. Lying anteriorly over this opening is the clavicle. The close confinement of these structures can become pinched, or impinged, with certain arm movements that bring the clavicle down toward the outlet. This can cause flow to the arm to be reduced (ischemic) and produce symptoms of tingling, numbness and pain in the arm or hand.

The main reason for performing sonography of the upper extremity venous system is to rule out the presence of venous thrombosis in both the superficial and deep veins. Most patients with acute deep vein thrombosis (DVT) of the upper extremities will have dramatically swollen arms and palpable cords near the surface of the skin for superficial vein thrombosis. Chronic venous thrombosis will usually not produce such pronounced symptoms because of the formation of collaterals in the arm and neck area.

Thrombosis in the upper extremities usually results from placement of central venous catheters. The danger from clot forming in the arm and neck is the possibility of embolism dislodging from the clot and traveling to the heart, and on into the pulmonary artery where it can cause fatal results by interacting with the lungs.

212. Imaging the upper extremity

Although other methods are often used in conjunction with sonography to examine the upper extremity vessels, such as segmental arm pressure and plethysmorgraphy, we will concentrate on duplex sonography. To image the upper extremity, several general things must be kept in mind. First, the approach is rarely accomplished with only grayscale sonography. Color Doppler should be an integral part of the upper extremity evaluation because it helps with locating vessels and identifying suitable sites for spectral Doppler sampling. The combination of Doppler and grayscale is called duplex. Secondly, most patients should be supine for sonography examinations of the upper extremity vessels. Finally, you should always use a transducer with a frequency range of at least 5 MHz.

Arterial approach

Scanning the upper extremity arterial system should begin with the subclavian artery. This may be difficult for the simple reason that two portions of the artery are located beneath bone. The proximal portion or origin of the subclavian is usually located behind the sternum. You should place your transducer above the medial clavicle and angle toward the feet. This may or may not give you the origin of the subclavian and will produce better results on the right than left as the right subclavian branches off the brachiocephalic trunk, while the left subclavian takes off directly from the deeper located aorta. Try to obtain color and grayscale images of as much of the proximal subclavian artery as possible before it moves beneath the middle portion of the clavicle.

At the middle clavicle, you may have to place your transducer below the clavicle and angle slightly up to image the mid portion of the subclavian artery. Spectral Doppler sampling of the normal subclavian artery should give you a waveform that is triphasic. That is, the systolic peak is at a sharp and high velocity that rapidly falls to the baseline in diastole as a result of high resistance, and then continues below the baseline signifying temporary reversal of flow before returning above the baseline at about a third of the systolic velocity.

The lateral portion of the subclavian and the proximal portion of the axillary artery can be imaged from an axillary position of the transducer. That is, after raising or abducting the supine patient’s arm about 90 degrees from his or her body, you can place your transducer into the armpit. The arterial vessel visualized will be the axillary artery. The closer to the chest you follow the artery the more likely you will be looking at lateral subclavian artery. The normal waveform in the axillary should be identical to the subclavian.

The brachial artery can be seen in the medial upper arm, lodged in between the biceps and triceps. The vessel now can be followed as it courses around to the anterior portion of the humerus into the elbow region where it splits into the radial and ulnar arteries of the forearm. The normal waveform throughout is triphasic. However, this may change if the arm is warmed by exercise, creating more flow above the baseline, which indicates less resistance to flow.

As with the aorta and carotid arteries, stenosis should be examined with spectral Doppler sampling before, through, and in the velocity jet just after the narrow channel. Occlusions should be proven with color, power, and spectral Doppler by demonstrating a lack of flow in the artery. Suspected aneurysms should be documented as a grayscale image of the section of artery diameter being twice the diameter of adjacent proximal and distal sections. As color Doppler should also be used, you should examine the lumen of a suspected aneurysm for thrombus formation that can cause embolization to the hand.

Venous approach

The primary objective for performing sonography of the upper extremity veins is to demonstrate the patency and normal flow. Thrombus that is acute is usually hypoechoic but increasingly becomes more echogenic with age. If an upper extremity vein is nearly occluded or completely blocked with thrombus, you should see the entire diameter of the vessel with hypoechoic to heterogeneous material. The diameter of the vessel, also, will not change with pressure from your transducer, a method used to demonstrate the collapsibility of normal veins. Indeed, the vessel can become so filled with clot that the patient may complain of feeling a “cord” in the arm. Normally, a completely occluded upper extremity vein can be seen running parallel to an unobstructed artery visible through color flow. Do not automatically assume that blood is not getting through an apparently obstructed vein. Try using power Doppler to detect the tiny channels of extremely slow moving blood within the clot.

As you move the transducer along the venous system of the upper extremities, take spectral Doppler samples. You should see waveforms with a steady forward flow toward the heart but with a wavering characteristic due to respiration. Unlike the lower extremities, you should also see slight pulsations in the waveform from the reflections of the heart’s right-side contractions. Normally, the upper extremity flow should respond with increased flow velocity back to the heart on inspiration and decreased or no flow upon expiration. This is due to the change in thoracic pressure, which is just the opposite in the legs. Document these respiration changes, preferably with two images, one with inspiration and one with expiration, side-by-side. Also, aside from the subclavian and axillary veins, most of the veins in the arms are paired or duplicated, with all the deep veins running parallel with an artery. This arterial pairing excludes the superficial cephalic and basilic veins. Thus, you should use this feature of arterial pairing with veins as a landmark. If you see a vein (waveform, color, and vessel dimensions during breathing should help identify) without an accompanying artery, you are likely examining the superficial cephalic or basilic vein. Remember, the basilic is medial and the cephalic is lateral and superficial in the arm.

As with duplex examination of the upper extremity artery, your transducer positioning is largely a matter of maneuvering around the sternum and clavicle for visibility. The internal jugular vein in the neck can be examined along its entire length for thrombosis, but your efforts should be primarily placed on evaluating its merger with the brachiocephalic vein just behind the sternum. The subclavian vein origin should also be imaged if seen. Do not be surprised if the radiologist requires the entire internal jugular vein to be evaluated, particularly after a placement of a central venous catheter into the vein. Compression of the veins as you move the transducer along will show lack of wall collapse due to the presence of DVT.

Position your transducer inferior to the clavicle to obtain the best image of the length of the subclavian vein, as the vein is slightly deep to the artery. The superficial cephalic vein at the level of the lateral clavicle joins the lateral subclavian vein, which you can see from an area above the

clavicle. Follow the cephalic over the deltoid muscle of the shoulder down the lateral surface of the arm to the elbow. The cephalic vein can be followed longitudinally or transversely. However, to locate the cephalic, you should use light pressure with the transducer because the vessel lies only a few centimeters below the skin surface and is easily collapsible with pressure. This can hide the vessel from view. When examining this vessel in transverse it is a good idea to attempt to compress the vessel every few centimeters. If the vessel collapses, the likelihood of a thrombosis is greatly reduced.

Have the patient abduct the arm and place the transducer in the axilla or armpit to evaluate the axillary vein. Moving the transducer to the medial part of the upper arm should demonstrate the axillary vein branching into the brachial and basilic veins. Perform compression at this level along with Doppler imaging. Transverse and longitudinal views should be obtained throughout.

The brachial vein will itself split into duplicate pairs to either side of the brachial artery. The color Doppler view should show the direction of flow in the twin veins to either side of the artery flowing in the opposite direction. This is a useful landmark located centrally in the upper arm. Follow the brachials to the elbow where they frequently rejoin, using compression along the way.

Move your transducer back to the axillary where it splits into the brachial and basilic veins. Follow the basilic along the medial portion of the arm with light pressure, as this vessel, like the cephalic on the other side, is relatively superficial.

Most institutions are satisfied with evaluation of the veins in the upper extremity only to the elbow. The forearms are examined only when thrombus is seen to extend into the area. In those cases, color Doppler becomes even more critical in identifying the very small caliber of radial and ulnar veins. An easy way to locate them, however, is to look for the nearby color flash of arterial flow. This will be the forearm vein's paired artery.

Self-Test Questions

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

211. Upper extremity sonography

1. What is the axillary artery called once it passes into the upper arm region?

2. What accompanies the upper extremity arteries?

3. Which two upper extremity veins are not paired with arteries?

4. What is the most common abnormality of the upper extremity?

5. Define *claudication*.

6. Explain thoracic outlet syndrome.

212. Imaging the upper extremity

1. What artery is seen when placing the transducer to the armpit?

2. What is the *primary* objective for performing sonography of the upper extremity veins?

3. How do you obtain the best image of the length of the subclavian vein?

2–3. Lower Extremity Abnormalities

Most of the lower extremity vasculature concerns either loss of flow to the distal portions of the legs or flow unable to return from distal portions back to the heart. In this section, we will look at the major arteries and veins of the legs and discuss their function. We will also briefly discuss a few major approaches that sonographers use to evaluate these vessels.

213. Lower extremity sonography

Sonography has proven to be a relatively safe and non-invasive way to diagnose abnormalities of the lower extremity compared to such procedures as arteriograms and venograms. This lesson covers the lower extremity anatomy and physiology and pathology.

Anatomy and physiology

The major arteries of the lower extremity anatomy are mostly identical patient to patient. However, normal variations do exist but most of those involve the smaller vessels below the knee. Others, such as a duplicated superficial femoral artery, are rare. Conversely, the major veins of the lower extremities are frequently duplicated.

Arterial

The lower extremity arterial anatomy begins with the branching of the abdominal aorta into the common iliac arteries at the level of anterior abdominal umbilicus. The common iliac courses a short distance before bifurcating into the internal and external iliac arteries. The internal iliac artery feeds oxygenated blood to structures within the pelvis. The external iliac continues on into the leg at the inguinal canal level. Most lower arterial extremity sonography is concerned with the level inferior to the inguinal canal, or where the thigh bends relative to the abdominal trunk.

At the inguinal canal, the external iliac artery becomes the common femoral artery (CFA), which extends for several centimeters into the thigh before bifurcating into the superficial femoral artery (SFA) and the deep femoral artery (profundis femoris). The deep femoral, with its many branches, supplies blood to the upper thigh. The SFA, without major branches, continues along the medial thigh until it dives into the adductor canal. When it emerges from the canal just behind the knee, it is the popliteal artery (PA).

The popliteal travels the length of the posterior knee before bifurcating into the anterior tibial artery (AT) and the tibioperoneal trunk. The AT travels along the anterolateral side of the lower leg to the foot and feeds the anterior lower leg muscles. The tibioperoneal trunk extends for a few centimeters before bifurcating into peroneal and posterior tibial arteries. The peroneals, which feed the calf muscles, usually remain central within the lower leg and terminate just above the ankle, whereas the posterior tibial becomes increasingly more superficial as it runs to a point just behind the medial malleolus of the distal tibia. The branches of the posterior tibial artery supply the foot with blood.

Venous

In general, flow from the foot back against the hydrostatic pressure up to the heart is accomplished through the deep veins of the leg. The superficial veins of the legs also carry blood toward the heart but generally send the blood into the deep veins through connecting veins called perforators. Think of perforating veins as a series of veins that punch through or perforate the muscle fascia layer of superficial tissue into the deep tissues of the leg. Valves in the perforators ensure that normal flow is from superficial to deep veins.

From the proximal leg to the foot, the veins follow the same path as the paired arteries. At the inguinal ligament, the external iliac vein (EIV) continues for a few centimeters as the common femoral vein (CFV) before being joined by a superficial vein called the great (or long) saphenous vein (SV). Two veins merge to form the CFV a few centimeters below the saphenous junction: the deep femoral vein (profunda femoris) and the superficial femoral vein. The superficial femoral vein, like the deep femoral and the common femoral, is considered a deep vein. Thus, it is now called simply the femoral vein (FV) and distinguished from the deep femoral vein. The deep veins of the thigh lie posterior or deep to the paired arteries.

The FV continues distal in the thigh and dives like its paired artery into the adductor canal, where it is transformed into the popliteal vein. The FV is the main vessel that drains the leg. All veins, superficial and deep below the level of the deep vein, drain into it either directly or, below the knee, through the perforators. Most perforators are located just inferior to the knee level and are found in the calf.

The popliteal vein twists around the artery to lie anterior to it behind the knee. The popliteal vein is the common vessel that forms from the union of the posterior tibial (PT), AT, and peroneal veins (PV). The PV drains the calf muscles, while the PT and AT vessels drain the rest of the leg and foot.

The superficial venous system consists primarily of the SV, which extends along the medial thigh and lower leg on into the foot. The other major superficial vein branches directly off the popliteal vein and it is called the lesser or small saphenous vein.

Pathology of the lower extremity

The most common lower extremity arterial abnormality is stenosis or occlusion caused by the formation of arteriosclerotic plaque. However, symptomatic problems with the lower extremity arteries can also frequently be the result of invasive procedures such as percutaneous catherization and surgical placement of grafts. Some of the more frequent complications of these procedures that prompt physicians to use duplex sonography are pseudoaneurysms and arteriovenous fistulas (AVF).

AVF is the abnormal communication between an artery and a vein. Commonly, this occurs as a complication of vascular surgical procedures such as accidental catheter puncture through the common femoral artery into the common femoral vein.

DVT in the lower extremity is the formation of a clot within the lumen of the vein, either from stagnant venous flow or injury to vein walls. The clot can narrow or occlude the vein and prevent venous blood from departing the leg, which, because of continuing arterial inflow, will increase pressure in the leg below the level of narrowing or occlusion. DVT can also collect around venous valves and prevent their proper functioning, which is to prevent the low-pressure blood from returning back into a lower level. The eventual result of DVT is blood pooling into the lower extremities and painful swelling will occur. Also, the blood will seek other collateral channels or, if the valves of the perforator veins are damaged, will use tortuous collateral veins (varicosities) near the skin surface.

The development of DVT, because of its potential to dislodge pieces that can travel back to the heart and sometimes fatally into the pulmonary artery, is the principal reason for sonographic

evaluation of the deep veins in the leg. Examination of the superficial veins for thrombus is also important because flow is normally always from superficial to deep through the perforating veins.

Another use for sonography focuses on venous insufficiency. This abnormality is usually the result of damage to the valves of a vein from DVT. The valvular damage can extend to the perforating veins, which connect deep veins in the muscles with superficial veins close to the skin surface. The result of venous insufficiency is reflux of venous blood back into the distal leg, causing pain and swelling.

214. Imaging the lower extremity

Evaluation of the lower extremity can be quite complex with non-imaging approaches such as segmental arterial pressure examination. However, we will focus on the more common sonographic use of duplex for helping the radiologist diagnose vessel abnormalities.

Arterial

Sonography is a valuable method for locating stenosis and occlusions in the lower extremity before scheduled invasive repair procedures. Evaluating the lower extremity arteries with color Doppler allows you to locate obvious areas of stenosis or occlusion quickly. Spectral Doppler, as with cerebrovascular and upper extremity arteries, should always be used to determine the amount and rate of blood flow in the legs. Grayscale can be of use in identifying plaques quickly, specifically calcifications.

Normal arterial waveform patterns in the lower extremities, like the upper extremities, demonstrate high resistance in the resting state. Thus, you should look for a triphasic waveform in the normal lower extremity artery. Again, the triphasic waveform is a sharp and high peak systolic velocity followed by a rapid reversal of flow in early diastole, and then a continuation of forward diastolic flow at relatively low velocity. Normal spectral waveforms will have empty windows with little sign of spectral broadening. Velocities will normally decrease on spectral Doppler as you sample from the proximal leg to the foot.

The entire length of the leg's arterial system should be evaluated in both grayscale and color Doppler. This survey of the arteries will help you to locate the obvious abnormalities of stenosis or occlusion quickly. Following this survey of the system, you should use spectral Doppler at certain representative locations. Longitudinal views should be used when performing spectral sampling. The common femoral artery should be sampled. Also the proximal, middle, and distal femoral arteries should be sampled. Placing the patient in a prone position may make it easier to access the popliteal artery at the back of the knee. Branching off the popliteal arteries are the tibial arteries, which can be sonographically followed distally as far as possible with color. Sampling of the tibials in the proximal locations should be sufficient, but following the tibials from the ankle up toward the knee with color while sampling the ankle level can also be effective.

The normal high resistance waveforms can change decisively with the presence of stenosis or occlusion. Tissues that are ischemic tend to open vessels because of the need for more blood, reducing the resistance. Thus, severe stenosis or occlusion in the presence of collaterals can produce reduced systolic velocities but increased diastolic velocities.

A moderate stenosis downstream from your sampling site will usually increase the peak systolic velocity the closer you come to it. The narrower the stenosis, the more the window of the waveform will fill in with echoes (spectral broadening). The typical triphasic pattern will also be lost with severe stenosis or the closer you get to a moderate stenosis; that is, the reverse flow in early diastole will vanish to be replaced with forward flow. As with stenotic sites elsewhere in the body, sample before, through, and just after the narrowing. Occlusions will normally show either no flow or reduced velocity and continuous diastolic flow because of the presence of collaterals.

For AVF, you will either see a large amount of turbulence on color flow within the common femoral artery or you will see CFA blood flow directly into the adjacent common femoral vein. A way for you to determine if the communicating channel is large is to sample the CFV and perform the Valsalva maneuver; that is, after deep inspiration, have the patient tighten the abdominal muscles. This normally will stop venous flow. If flow still continues forward with little effect, even after release, the communicating channel is probably quite large.

Pseudoaneurysms also can be caused by surgical procedures. Color Doppler, with properly adjusted settings and filter, should be able to detect a swirling motion with the pseudoaneurysm. These abnormalities are difficult to distinguish from saccular aneurysms. However the presence of a small communicating neck, which you may or may not see, is helpful to confirm pseudoaneurysm. Sampling inside the neck should give you a spectral Doppler waveform with extremely high velocity and spectral broadening. There should also be a back and forth exchange of flow between the artery and the pseudoaneurysm.

Venous

Most institutions that perform sonography of the lower extremities have a simple protocol when asked to rule out DVT. Patients are supine with upper bodies slightly elevated with pillows or padding or the entire examination table is raised at the head (reverse Trendelenburg). Some institutions prefer patients to be in a sitting position. Either way, by making the lower extremity veins dependent, the hydrostatic pressure should increase the vein diameters and increase their visibility, particularly the smaller veins.

Grayscale longitudinal and transverse views should be used throughout along with color and spectral Doppler. Compression of the veins in the transverse view should be performed every few centimeters from the common femoral vein to the popliteal vein. The compression and color flow are particularly helpful for detecting new or fresh thrombus, which usually is very hypoechoic and can be hidden on grayscale. Thrombus prevents total collapsing of a vein from compression and is a useful indicator to its presence, as is color flowing around an otherwise unseen new clot. Otherwise, a thrombus is usually composed of medium-gray echoes.

Spectral waveforms can be taken throughout or after all grayscale and color images are obtained. The primary purpose of waveform analysis of lower extremity veins is to confirm either patency or obstruction of the vessels. There are generally three areas of concern with venous spectral analysis of the lower extremity veins:

1. Phasicity (Respiration and pulsation).
2. Valsalva maneuver response (Pressure).
3. Augmentation response (Compression).

Normal spectral waveforms for lower extremity veins will show continuous phasicity and will correctly respond to maneuvers such as Valsalva and augmentation. Phasicity is the change in velocity with due to both respiration and the distant cardiac pulsations. Phasic change indicates that the vein is open and clear between the point of sampling and the chest. Changes in thoracic pressure cause the velocities in the legs to either reduce or increase as it heads back to the heart. Pulsation waves from the right atrium contractions of the heart can be detected even as far distal as the distal femoral vein. Phasic change will vanish to be replaced by steady forward flow in cases of significant near obstruction of the veins above the CFV level.

Valsalva maneuvers also determine if blood flow is obstructed between the point of sampling and the chest. By having the patient breathe in deeply and bear down, the maneuver causes venous blood in the legs to cease flowing forward to the heart. Again, the diaphragm increases intra-abdominal pressure to the point where all low pressure flow in the legs is prevented from entering the level of the abdomen. When the breath is released, the flow resumes. In patients with obstructed CFV or EIV, the flow may be absent or reduced with no phasic changes from

respiration. You will notice that the Valsalva maneuver will not change this abnormal spectral pattern.

Finally, augmentation also determines patency only in the segment between manual compression of a vessel and the site of spectral sampling. For example, compressing the calf muscle with your hand, a cuff, or transducer will cause the velocities to increase sharply as a result of blood being squeezed forward. The spectral waveform showing this velocity increase, if taken at the level of the CFV, would mean no substantial obstruction in the veins between the two levels. Keep in mind that any of the three methods can show normal waveforms in cases of partial thrombosis. The vessels have to be occluded or nearly so to show a change in the venous flow.

Some institutions require you begin DVT examinations just above the inguinal canal at the level of EIV, while others are content with starting below the canal at the CFV. If thrombus is seen, you should immediately proceed to the proximal IVC in the abdomen and then follow it distally until it bifurcates. This is to ensure you document the true extent of DVT beyond the EIV.

Documentation should be taken at the level of CFV, preferably with landmarks in the longitudinal image such as the saphenous vein junction or the branching of the CFV into FV and DFV. Transverse compression should begin here.

Continue compressions every few centimeters as you continue distal along the entire visible FV. Document the proximal, middle, and distal FV levels. When the distal FV dives into the leg, have the patient lay on the side of the leg of interest while extending the other leg behind. The patient's leg should be bent at the knee as a way to relax the muscles and prevent collapsing of the now smaller caliber veins.

Examine the vein as it exits the adductor canal located on the medioposterior level of the distal thigh. Follow this vein, now called the popliteal vein, into the back of the knee. Some institutions will insist on you continuing to examine the veins as far distally into the calf as possible. Others demand the examination to cease at the knee. Whichever method is standard in your area, make sure to continue examination into the calf if you see thrombosis extending distal to the popliteal.

The superficial veins may also be a part of the protocol. The great saphenous vein may be examined by returning to the level of the CFV and scanning along medial thigh, from the origin of the vessel to its termination in the distal foot. The vessel is very superficial and is only a few centimeters below the skin surface. Representative images of the proximal and perhaps middle portions of the vessel can be taken once the survey of the entire vessel is complete. Your department may also require the small saphenous to be documented. The best location to begin the small SV is at the popliteal vein and lightly tracing the vessel down the center of the calf.

Many departments have various protocols for determining venous insufficiency. Most have a common approach of having the patient upright and placing the effected leg's foot on the floor with the opposite leg supporting the body's weight. A series of spectral waveforms are obtained from various levels of the deep veins, usually in conjunction with Valsalva maneuvers, as with DVT investigations. However, in this case augmentation is the primary method that may confirm malfunctioning valves in the veins (incompetent venous valves). For example, placing the transducer at the popliteal vein and squeezing the distal calf muscle and examining the waveform will show a brief period of forward flow (from the squeeze) followed by no sign of flow reversal. This means the valves are working (competent) and are preventing reverse flow. With incompetent valves, the flow will immediately reverse after augmentation and will continue to flow in reverse for a longer time in severe venous insufficiency cases.

The saphenous veins as well as the perforators may show incompetent veins by showing flow reversals with Doppler. Color Doppler can demonstrate the direction of blood within the perforators, between the superficial vessels and deep veins below the fascia. For instance, scanning along visible varicose veins may show a perforator diving down below the fascial layer

into the muscle of the calf. Color Doppler can confirm perforator valve incompetence by demonstrating blood flowing from the deep veins to the varicose veins at the surface; normal perforator valves prevent this type of reverse flow.

Self-Test Questions

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

213. Lower extremity sonography

1. The popliteal artery bifurcates into what two arteries?

2. What do the valves in the perforator veins ensure?

3. The superficial venous system consists primarily of what vein?

4. What problems can cause stenosis or occlusion in lower extremity arteries?

5. What is an AVF?

6. What is deep vein thrombosis?

214. Imaging the lower extremity

1. What waveform should you look for in a normal lower extremity artery?

2. What lower extremity arteries should be sampled with spectral Doppler?

3. In what positions should you place DVT patients?

4. List three areas of concern when using spectral analysis on lower extremity veins?

5. How often should you use compression along the femoral vein?

Answers to Self-Test Questions

207

1. The abdominal aorta remains close to the spine throughout its length; the IVC moves away caudally.
2. It gradually tapers.
3. (1) Tunica intima (inner layer).
   (2) Tunica media (middle muscular layer).
   (3) Tunica adventitia (outer layer).
4. (1) Celiac trunk.
   (2) Superior mesenteric artery.
   (3) Renal arteries.
5. Inferior vena cava.
6. Media layer (vessel wall muscle layer).
7. Carries oxygenated blood to the body below the diaphragm and serves as a high-pressure reservoir of blood.

208

1. An increase in aortic diameter of 1.5 to 2 times the normal adjacent diameters.
2. (1) Fusiform.
   (2) Saccular.
   (3) Cylindrical.
3. The posterior wall below the level of the renal arteries.
4. (1) Tube.
   (2) Aortoiliac.
   (3) Aortobifemoral.
   (4) Prosthetic stent.

209

1. To rule out the presence of aneurysm.
2. Proximal, middle, and distal abdominal aorta.
3. When the diameter is greater than 3 cm or when there is an increase in diameter of 1.5 to 2 times the normal adjacent diameters.
4. Because they grow slowly at a rate of a few millimeters annually.
5. Fusiform, saccular, and cylindrical.
6. Measure dimensions of the aneurysm.
7. Use flank approach.
8. An echogenic line, running roughly parallel with the aortic wall, in the center of the lumen on longitudinal views. The line represents the intima or both the intima and media layers torn away from the vessel wall. Usually, this torn away layer can be seen flapping in the lumen from the pulsations of flowing arterial blood. If just the intima is torn away, you should see a thin line. If both the media and intima are torn away, the line should be thicker. At times, the line will be fixed, particularly in the presence of thrombus to either side.
9. On the posterior wall below the renal arteries.
10. In the presence of stenosis.
11. (1) b.
    (2) c.
    (3) d.
    (4) a.

210

1. Low resistance of the liver and spleen.
2. 150 cm/sec.
3. Sampling the superior, middle, and inferior poles of the kidney.
4. $RI = \frac{A - B}{A}$ Where RI is resistive index, A is peak systolic velocity, and B is end diastolic velocity.
5. 0.75 or less.
6. Via Doppler.
7. Phasic waveforms identical to those in the IVC.

211

1. Brachial artery.
2. Veins.
3. Basilic and cephalic veins.
4. Arteriosclerotic plaque.
5. Pain in arm muscles caused by exercise and relieved by rest.
6. The subclavian artery courses through a narrow opening between the two neck muscles on the way to the lateral portion of the first rib. The brachial nerve and subclavian vein also run with the artery through this space. Lying anteriorly over the opening is the clavicle. The close confinement of these structures can become pinched, or impinged, with certain arm movements that bring the clavicle down toward the outlet. This can cause flow to the arm to be reduced (ischemic) and produce symptoms of tingling, numbness and pain in the arm or hand.

212

1. Axillary artery.
2. Demonstrate patency and normal flow.
3. Position transducer inferior to the clavicle.

213

1. AT and tibioperoneal trunk.
2. Blood flows from superficial veins to deep veins.
3. Saphenous vein.
4. Arteriosclerotic plaque, percutaneous catherization, and surgical grafts.
5. Abnormal communication between an artery and a vein.
6. Formation of a clot within the lumen of the vein, either from stagnant venous flow or injury to vein walls.

214

1. Triphasic.
2. The common femoral artery, the proximal, middle, and distal femoral arteries.
3. Patients are supine with upper bodies slightly elevated with pillows or padding or the entire examination table is raised at the head (reverse Trendelenburg). Some institutions prefer patients to be in a sitting position.
4. (1) Phasicity (Respiration and pulsation).
   (2) Valsalva maneuver response (Pressure).
   (3) Augmentation response (Compression).
5. Every few centimeters.

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. (207) The position of the abdominal aorta becomes progressively anterior in the body as it runs caudally because it
    a. follows the curvature of the spine.
    b. parallels the inferior vena cava (IVC).
    c. is displaced by the intestines.
    d. is displaced by the liver.

19. (207) The abdominal aorta supplies oxygenated blood to the
    a. extremities.
    b. head and neck.
    c. entire the body.
    d. body below the diaphragm.

20. (207) What is the first branch of the aorta?
    a. Celiac trunk.
    b. Common hepatic artery.
    c. Inferior mesenteric artery (IMA).
    d. Superior mesenteric artery (SMA).

21. (208) What is the basis for classifying an abdominal aortic aneurysm (AAA) as fusiform, saccular, or cylindrical?
    a. Velocity.
    b. Body location.
    c. Pathologic cause.
    d. Physical description.

22. (209) You would have a patient fast before an abdominal aorta sonogram to
    a. clear the aortic lumen.
    b. clear the body of bowel gas.
    c. distend the gallbladder wall.
    d. prepare for possible surgery.

23. (209) What approach may reliably display the sides of the abdominal aorta?
    a. Superior.
    b. Anterior.
    c. Left flank.
    d. Right flank.

24. (209) What should you use to help identify a pseudoaneurysm?
    a. Grayscale.
    b. Color flow.
    c. A 3 MHz transducer.
    d. A 5 MHz transducer.

25. (209) Why would you measure between an abdominal aortic aneurysm (AAA) and the renal arteries?
    a. Find ratio between aneurysms and renals.
    b. Include the renal arteries in any AAA image.
    c. Provide surgeons with the distance measurement.
    d. Determine the need for sampling the renal arteries.

26. (209) What Doppler should you use to help determine flow direction in an arterial dissection case?
    a. Color.
    b. Power.
    c. Energy.
    d. Spectral.

27. (210) The landmark that allows you to locate the right renal artery is the vessel that lays
    a. anterior to the inferior vena cava (IVC).
    b. posterior to the IVC.
    c. anterior to the abdominal aorta.
    d. posterior to the abdominal aorta.

28. (210) What is the time between the end diastolic velocity of one pulse and peak systolic velocity of the next pulse?
    a. Common carotid artery (CCA) ratio.
    b. Resistive index.
    c. Acceleration time.
    d. Pulse repetition period.

29. (210) The two principal abnormalities that require Doppler evaluation of the abdominal veins are thrombosis and
    a. stenosis.
    b. aneurysm.
    c. tumor invasion.
    d. abnormal kidney RI.

30. (210) Where is Doppler sampling of the inferior vena cava (IVC) performed most?
    a. Upper abdomen.
    b. Lower abdomen.
    c. At the bifurcation.
    d. Near the renal veins.

31. (211) When the arm is exercised, what happens to the blood flow resistance in the arm?
    a. Increased.
    b. Fluctuated.
    c. Reduced.
    d. Unchanged.

32. (211) Which of the following veins are more important when evaluating upper extremity abnormalities?
    a. Deep and superficial veins.
    b. Internal jugular veins.
    c. External jugular veins.
    d. Subclavian veins.

33. (211) Of the upper extremity arterial abnormalities, which of the following is the most common?
    a. Thoracic outlet syndrome.
    b. Pseudoaneurysm.
    c. Atherosclerotic plaque.
    d. Chronic venous thrombosis.

34. (211) Which of the following upper extremity abnormalities mimics thoracic outlet syndrome?
    a. Subclavian steal.
    b. Pseudoaneurysm.
    c. Atherosclerotic stenosis.
    d. Chronic venous thrombosis.

35. (212) Where should the upper extremity arterial sonogram begin?
    a. Brachial artery.
    b. Abdominal aorta.
    c. Subclavian artery.
    d. Radial and ulnar artery.

36. (212) While scanning the subclavian artery, what might you have to do when you reach the middle clavicle?
    a. Continue scanning over it.
    b. Use color and spectral Doppler.
    c. Keep transducer above the clavicle.
    d. Move the transducer below the clavicle.

37. (213) At what level does the external iliac vein become the common femoral vein?
    a. Popliteal.
    b. Deep vein.
    c. Inguinal canal.
    d. Adductor canal.

38. (213) What is the relationship between the posterior tibial, anterior tibial, and peroneal veins?
    a. Are perforator veins.
    b. Unite to form popliteal vein.
    c. Are all located above the knee.
    d. Unite to form tibiopopliteal vein.

39. (213) What is the principal reason for sonographic evaluation of the deep veins in the leg?
    a. Deep vein thrombosis (DVT).
    b. Venous insufficiency.
    c. Central venous catheter.
    d. Chronic venous thrombosis.

40. (213) How does venous insufficiency cause pain and swelling?
    a. Causes reflux of venous blood.
    b. Causes increase flow of venous blood.
    c. Causes arteriovenous fistulas.
    d. Causes pseudoaneurysms.

41. (214) What happens to the spectral velocity as you sample the femoral artery from proximal leg to foot?
    a. Decreases.
    b. Increases.
    c. Reverses.
    d. Vanishes.

42. (214) What position may make it easier to access the popliteal artery?
    a. Prone.
    b. Decub.
    c. Supine.
    d. Upright.

43. (214) What is the purpose of making the lower extremity veins dependent?
    a. Stop venous flow temporarily.
    b. Speed up venous velocities.
    c. Increase visibility of arteries.
    d. Increase visibility of veins.

44. (214) What maneuver can you use to help you determine the size of the communication channel in an arteriovenous fistula located in the lower extremity?
    a. Valsalva.
    b. Sniff test.
    c. Augmentation.
    d. Murphy's sign.

45. (214) How can compression of a vein indicate the presence of thrombus?
    a. Thrombus provides partial collapse of vein.
    b. Thrombus prevents partial collapse of vein.
    c. Thrombus provides total collapse of vein.
    d. Thrombus prevents total collapse of vein.

46. (214) In cases of partial thrombus, what type of waveforms might any of the three methods used for determining venous occlusion show?
    a. Absent.
    b. Normal.
    c. Abnormal.
    d. Tardus-parvus.

47. (214) What is the purpose for bending the knee of a patient resting on the side of the leg being examined for deep vein thrombosis (DVT)?
    a. Relax the muscles.
    b. Relax the nerves.
    c. Calm femoral vein velocities.
    d. Calm femoral artery velocities.

48. (214) On what level in the leg is the great saphenous vein located?
    a. Deeply.
    b. Superiorly.
    c. Centrally.
    d. Superficially.

49. (214) Which of the following flows is likely with common femoral vein obstruction?
    a. Phasic.
    b. Absent.
    c. High velocity.
    d. High resistance.

50. (214) With incompetent valves, augmentation will cause the venous flow to
    a. cease.
    b. reverse.
    c. increase.
    d. continue forward.

51. (214) Doppler can confirm perforator valve incompetence by showing flow from
    a. deep veins to varicose veins.
    b. deep veins to peroneal veins.
    c. superficial veins to deep veins.
    d. superficial veins to saphenous veins.

52. (214) What is a *common* patient position when examining for venous insufficiency?
    a. Trendelenburg.
    b. Upright.
    c. Supine.
    d. Prone.

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

Student Notes

Unit 3. Glandular Small Parts

IN THIS UNIT, we will discuss some major glandular small parts of the body: thyroid and testicle. Other endocrine (ductless and hormone secreting) glands exist; however, those are discussed in other units. The breast is discussed in unit 4 of this volume, the pancreas was discussed in volume 1, and the ovaries will be discussed in volume 3.

You will be frequently called on to examine the glandular small parts. Abnormalities of these structures occur with regularity and your familiarity with them will assist with accurate diagnosis. Familiarity with the various diseases is also part of the diagnostic process.

3–1. Thyroid Imaging

Sonographers approach the thyroid in a straightforward manner. The transducer is placed against the neck and the thyroid usually becomes immediately visible just below the muscle layer. Therefore, obtaining images of normal and abnormal thyroids is a simple procedure. However, some subtle abnormalities resist simple scanning and require an understanding of thyroid structure and function to detect accurately.

215. Thyroid anatomy and physiology

The anatomy of the thyroid helps inform our understanding of its normal function; that is, its physiology. Through understanding thyroid physiology, we can better grasp how diseases interrupt normal functioning.

Anatomy

The thyroid is an endocrine gland, which means it is one of the organs that secretes hormones directly into the blood stream instead of into ducts, as with exocrine glands. It is located in the lower part of the anterior neck to either side of the trachea. Its most superior portion is just below the level of the larynx, or voice box. Immediately anterior to the thyroid are the sternohyoid, omohyoid, and sternothyroid muscles. Together these thin muscles are called infrahyoid or strap muscles. Anterolateral to the strap muscles are larger muscles to either side called the sternocleidomastoid muscles. Lateral to the thyroid gland are the carotid arteries and the internal jugular veins. Posterior and toward the lateral margin of the thyroid are the triangular-shaped longus colli muscles. Occasionally, a portion of the esophagus will press up against the posterior and medial left side of the thyroid gland.

The gland itself is divided into two lobes, one to each side of the trachea. A thin strip of thyroid tissue travels across the anterior surface of the trachea and joins the two lobes together. This bit of thyroid is called the isthmus, found between the middle and lower portions of the thyroid lobes. In about a third of young patients, a narrow wormlike extension of thyroid tissue will arise from

the isthmus and stretch superiorly toward the larynx. This structure, a normal variant, is called the pyramidal lobe and progressively atrophies (or diminishes in size) with age.

Thyroid gland parenchyma, or functional tissue, is composed mostly of microscopic spherical sacs called follicles. The walls of these hollow sacs are made of follicular cells with a smaller number of parafollicular cells in between. Follicular cells are epithelial cells, which are simple cells that form the layers of most surfaces in the body.

Although thyroid glands in females have been characterized as slightly heavier and larger than in males, the most significant differences in size is found because of age. Adult thyroid lobes are more than twice as large as lobes in children. In general, tall and thin people tend to have thyroid lobes with longer lengths compared with average sized people. The opposite effect is present in shorter and larger individuals, whose thyroid lobes tend to be short and rounded. The isthmus can be anywhere from 2 millimeters (mm) to 6 mm in anteroposterior (AP) thickness.

Typical Thyroid Lobe Measurements in Centimeters (cm)			
Dimension	***Newborn***	***Child***	***Adult***
Length	1.8–2.0 cm	2.0–3.0 cm	4.0–6.0 cm
AP	0.2–1.2 cm	1.2–1.3 cm	1.3–2.0 cm
Width	1.0–1.5 cm		1.5–2.0 cm

Thyroid glands are supplied with oxygenated blood primarily from two arteries: the superior and inferior thyroid arteries. The superior thyroid artery is the first branch from the external carotid artery and the inferior thyroid artery is a branch off the thyrocervical artery (trunk). The external carotid and thyrocervical arteries originate from the common carotid and subclavian arteries, respectively. The superior thyroid artery enters the superior pole of the gland and feeds the upper thyroid. Conversely, the inferior thyroid enters the inferior pole and feeds the lower thyroid.

Venous blood is first drained from the depths of the gland to the surface, where a network or a mesh of venous blood vessels called plexus is located. Superior, middle, and inferior thyroid veins rise from the venous plexus and carry the deoxygenated blood to the major veins. Thus, the superior and middle thyroid veins drain into the internal jugular vein (IJV), and the inferior thyroid vein drains into the brachiocephalic vein.

In close proximity to the thyroid gland are the parathyroids, which are small and largely oval glands measuring approximately 5 mm in length, 3 mm in width, and 1 mm in AP diameter. Most individuals possess four parathyroids, a pair embedded into the posterior surface of the thyroid at the superior end and a pair embedded into the posterior surface at the inferior end. Variant locations are known to exist for parathyroids such as behind the trachea (retrotracheal), mediastinal (in the thymus gland), within the thyroid itself (intrathyroid), and around the carotid artery (carotid sheath or bifurcation). The parathyroids are important for their function. However, unless abnormal, they are rarely seen with sonography.

Physiology

The principal role of the thyroid gland is to enhance the metabolism of nearly all tissues in the body. Recall that metabolism is the total chemical and physical activity that occurs in cells, both the break down of large molecules to small (catabolism) and the buildup of small molecules to large (anabolism). The thyroid affects metabolism by producing hormones that target almost every cell in the body.

Thyroid hormones specifically affect metabolism in a variety of ways. First, they increase the rate that cells consume or use oxygen while the body is at rest (basal metabolic rate). Secondly,

because nearly every cell's metabolism is increased through thyroid hormones, heat builds up. Thus, the body's temperature is raised with an increase in thyroid hormones. Third, because metabolism enhancement is widespread, thyroid hormones stimulate the manufacturing or synthesis of proteins. Thyroid hormones have other functions such as acceleration of tissue growth in young people, increased blood flow and respiration because of increased oxygen use and heat elimination, stimulation of the central nervous system, and increased secretion rates of most other endocrine glands.

Three thyroid hormones are produced by the glands: tetraiodothyronine (also called *thyroxine*), triiodothyronine, and calcitonin. Tetraiodothyronine contains four atoms of iodine and is usually referred to as T_4. Similarly, triiodothyronine contains three iodine atoms and, thus, is referred to as T_3. Thyroid follicles produce all three hormones, with T_3 and T_4 made by the follicular cells and calcitonin formed by the parafollicular cells.

To manufacture thyroid hormones, the gland needs iodide (I^-), the negatively charged form of iodine that comes from dietary iodine absorbed by the gastrointestinal tract. The thyroid gland traps iodide from the passing blood stream and combines it with a specific glycoprotein (a protein molecule bound to a carbohydrate molecule) manufactured by the follicular cells. The combination produces the preliminary form of thyroid hormones, which are stored where they are made in the space or lumen of the follicle. The stored material in the lumen, proteins and iodine combined, is called colloid. The thyroid gland is the only gland in the body that stores its hormones in large quantities (usually a two to three month supply) before release. Once released the thyroid hormones are secreted into the bloodstream.

The process of iodide take-up and subsequent hormone production is controlled by both the hypothalamus and the gland (pituitary) suspended from its base. When levels of thyroid hormone decrease, the hypothalamus produces *thyrotropin-releasing hormone* (TRH). TRH directly affects the anterior pituitary gland to secrete *thyrotropin*, commonly called TSH (thyroid-stimulating hormone). The TSH causes the thyroid cells to increase production and secretion of thyroid hormones. TSH does this by stimulating the growth of thyroid follicles, which increase in size and number. Once thyroid hormones are released into the body, the hypothalamus stops secreting TRH, which in turn causes the pituitary to stop secreting TSH.

Along with thyroid hormones, the thyroid secretes calcitonin, produced by parafollicular cells. Calcitonin controls the body's calcium content by increasing the ability of bone to store calcium, which lowers the amount of calcium in the blood. The parathyroid glands release a hormone, parathyroid hormone (PTH), which has the exact opposite effect—it causes the release of calcium from bone into the blood circulation. Thus, PTH and calcitonin work in concert to keep calcium levels in balance.

Parathyroid hormone also increases the intestines ability to absorb calcium, magnesium, and phosphates by activating vitamin D in the kidney. Vitamin D is needed to give the intestines the ability to transport calcium through the intestinal cells into the blood stream. Calcium levels must be balanced (or in homeostasis) because certain body functions depend on it. For instance, calcium homeostasis is crucial for normal muscle contractions, nerve impulse transmissions, and blood clotting.

Thyroid and parathyroid glands usually produce specific amounts of hormones considered normal (euthyroid). Levels for these hormones are determined though a series of thyroid function tests, a combination of nuclear medicine, and laboratory analysis.

Thyroid Function Tests		
Tests	***Description***	***Typical values***
T_4	Determines amount of T_4 in the blood plasma (both free and bound to carrier proteins).	4.5–13 μg/dl (higher values in children and pregnant women)
T_3	Determines amount of T_3 in the blood plasma.	75–195 μg/dL
TSH-stimulating	Measures uptake of radioactive iodine tracer ^{131}I before and after the administration of TSH.	0.4–6 μU/mL
RAI Uptake	Uses a nuclear gamma detector to measure radioactivity. Radioactive iodine tracers, ^{123}I or ^{131}I, are given to patient orally. The thyroid gland is measured at 6 hours and 24 hours. Radioactivity from the gland is measured as a percentage of the radioactivity in the tracers originally given. Thus, the ability of gland to trap and absorb iodine is indirectly provided.	6 hours = 3–16% 24 hours = 10–30%
Calcium	Determines the amount of calcium in the blood plasma.	Male: ≤ 100 pg/mL Female: ≤ 30 pg/mL

216. Pathology of the thyroid and parathyroid

Although a small organ, the thyroid can cause serious problems for a person. Positioned close to the skin surface relative to other body organs allows the thyroid to be easily accessed with sonography. Frequently, you will be called on to examine and understand the various abnormalities of this unique gland.

Functional abnormality of the thyroid

Normally functioning thyroids are called euthyroid. Otherwise, any functional abnormality refers to the thyroid's ability to produce thyroid hormones; that is, the thyroid will be considered in a state of either hyperthyroidism or hypothyroidism.

Hyperthyroidism

An excessive amount of thyroid hormones circulating in the blood is called hyperthyroidism. Generally, this causes nearly every part of the body to be subjected to a hypermetabolic state. Typical symptoms are weight loss, nervousness, goiter, increased heart pulsations, excessive sweating, and protrusion of one or both eyeballs (exophthalmos). Severe cases (thyrotoxic crisis) can produce muscular weakness and high fevers.

Hypothyroidism

Hypothyroidism is the reduced amount of circulating thyroid hormones. This leads to clinical signs of thyroid insufficiency such as weight gain, slow metabolic rate, sleepiness, hoarseness, menstrual abnormalities, constipation, and sometimes myxedema (puffy eyelids and swollen hands and feet).

Structural abnormality of the thyroid

Various abnormalities can cause the thyroid to malfunction. However, some euthyroid patients may have visible abnormalities in or around the thyroid gland. Many different classifications of neck and thyroid disease exist, the two most typical being nodular and diffuse thyroid disease. First, we will look at the nodular disease.

Nodular thyroid disease

Frequently you will encounter the term ‘goiter’. This is what an enlarged thyroid gland is called, whatever the reason behind the enlargement. One or more nodules can cause goiter; or simply the thyroid gland itself is enlarged, which may or may not have a heterogeneous echotexture that suggest nodules on sonograms. An enlarged thyroid gland is its only significance, and you need not be overly concerned with the term. Many terms are used to describe abnormalities in the thyroid and it can become quite confusing. Remember that most benign masses or lesions in the thyroid are called ‘nodules’. Also, remember that ‘toxic’ and ‘nontoxic’ refer to the rate of hormone secretion; increased and decreased, respectively. Again, ‘goiter’ should be used only when the gland is enlarged because of these abnormalities.

Benign or colloid adenomatous nodule (hyperplasia)

The most common type of thyroid abnormality is the benign adenomatous nodule. It can be in the form of singular or multiple lesions, but mostly multiple. Technically, the abnormality is not a true nodule in the sense of a tumor but rather a hyperplastic growth of thyroid follicles. Hyperplasia is an increased number of normal cells. However, because the growths have clear borders and a thin hypoechoic halo around them on sonograms, they resemble and are conveniently called nodules. The increase in the quantity of follicles means an increase in the production of colloid as well as fusion of colloid regions. For this reason you may hear references to ‘colloid nodules’; or, in the case of too much colloid expanding the gland size, you may hear the term ‘colloid goiter’.

Mostly appearing in middle-aged women, benign adenomatous nodules are either caused by a deficiency in iodine or are idiopathic, meaning the cause is unknown. Because iodine deficiency in the United States is rare due to iodized table salt, the usual reasons you may see it will be from an inherited disorder that prevents thyroid hormone production, or from medication that interferes with the thyroid’s ability to use iodine.

Dietary iodine is needed by the thyroid gland to form thyroid hormones that, when released, cause the pituitary gland to stop secreting TSH. Recall that TSH stimulates thyroid follicle growth and the production of colloid. In iodine deficiency, there is no incoming iodide (the form of ingested iodine— usually salt) that can be used to manufacture iodine inside the follicle walls. Once iodine is formed, it is used to make thyroid hormones inside the colloid of a follicle. If hormones are not produced, the hypothalamus will sense reduced amounts of iodide in the blood and will keep generating TRH, which in turn keeps the pituitary secreting TSH. Thus, the follicles will steadily be stimulated to grow in size and number (hyperplasia), which causes a buildup in colloid and eventually the size of the gland.

Therefore, adenomatous nodules are nontoxic, meaning they do not increase the rate of hormone secretion. When these lesions multiply and cause gland enlargement, they are often called diffuse nontoxic goiter. Otherwise, adenomatous nodules (that enlarge the gland) are called either multinodular goiter or adenomatous goiter. Be careful not to confuse diffuse nontoxic goiter with diffuse toxic goiter. Aside from the similar names, the two abnormalities may also share similar characteristics—largely an enlarged gland from iodine insufficiency. However, toxic goiter results from abnormal autoimmune activity and causes hyperthyroidism, and the nontoxic goiter does not affect the secretion rate of the gland.

Follicular adenoma

Follicular adenomas are neoplasms, or abnormal tissue different from surrounding tissue. Thus, unlike benign adenomatous nodules, the follicular adenoma is considered a true nodule. These

nodules are far less common than benign adenomatous nodules. When they occur, they are found mostly in females and are mostly solitary.

Slow growing, the follicular adenoma has extremely rare subtypes. For instance, the thyroid may contain a macrofollicular adenoma, which is composed of large colloid-filled cells in the shape of follicles; a microfollicular adenoma (also called fetal), which has small follicle-shaped cells packed tightly together; a embryonal adenoma, which has undeveloped cells packed so closely together that they form cords; and a Hürthle cell adenoma, another benign tumor which has the potential to become malignant. Many follicular adenomas are mixtures of all these sub-types.

Follicular adenomas can hemorrhage, causing the nodule to enlarge painfully, which tends to move against or compress normal thyroid tissue. Generally, they have no effect on the thyroid function and patients are euthyroid. Occasionally, a small percentage of follicular adenomas will produce thyroid hormones independent of the thyroid's normal control. This situation is known as hyperfunction or autonomy. Among the autonomous follicular adenomas, some will produce excessive quantities of thyroid hormones, a condition known as thyrotoxicosis.

Most physicians become alerted to the presence of the follicular adenoma during scintigraphy, a nuclear medicine examination. A radioactive agent or tracer is prepared that targets the thyroid gland; this is usually iodine–123 (^{123}I) or technetium–99m (^{99m}Tc). The tracer is intravenously introduced into a patient, and a gamma ray detector is placed over the thyroid. In the normal functioning thyroid, the tracer is absorbed or taken up and the detector forms images from the detected gamma rays emitted. Most of the thyroid tissue will be seen on the image. However, if a typical adenoma is present, there will be a region of the thyroid gland that will show less or no tracer uptake. This represents tissue that does not function in the same manner as normal thyroid tissue. The blank space is considered "cold." On the other hand, in hyperfunctioning follicular adenomas, the entire thyroid will show no uptake except for the area of the nodule. This isolated area of uptake represents a hyperfunctioning or "hot" nodule. Because a significant number of cold nodules using ^{123}I and hot nodules using ^{99m}Tc tend to be malignant, most physicians will order ultrasounds to characterize the mass.

Fine-needle aspirations (FNA) are not performed on these nodules because their cytologic, or cell, features are indistinguishable from follicular carcinoma. Because only histology (the study of microscopic structure of tissue) can show evidence of follicular carcinoma invading the vascular and thyroid tissue, the safest route is for physicians to surgically remove the adenomas or inject them with alcohol.

Papillary carcinoma

Thyroid cancer is rare. When it occurs, the most common type is papillary carcinoma, particularly in children. Occurring mostly in females younger than 40 years of age, papillary carcinoma is a slow growing malignancy that gives patients excellent chances for recovery (prognosis) after the surgical removal of the thyroid gland (thyroidectomy). A small percentage of papillary carcinomas spread through the lymph channels into nearby lymph nodes. Enlarged lymph nodes in the neck (cervical adenopathy) may offer a clue to the presence of malignancy when the papillary carcinoma is too small to be palpated.

Follicular carcinoma

The second most common malignant tumor of the thyroid behind papillary carcinoma is follicular carcinoma. Despite its rarity, this occasionally slow-growing malignancy is more aggressive and has a lower survival rate than papillary carcinoma. Nearly a third of people who have surgery to remove this cancer will die. Largely seen in middle-aged to elderly females, follicular carcinoma has two types: minimally invasive and widely invasive. This classification refers to the fibrous

connective tissue (capsule) surrounding it that is similar to the capsule seen around a follicular adenoma. Minimally invasive follicular carcinoma is thoroughly encapsulated on sonography and is nearly identical to a benign adenoma. Only through histology can carcinoma be distinguished from adenoma. Histologic evaluation of tissue obtained through FNA biopsy will demonstrate an intact capsule with carcinoma (arranged in tightly packed follicular shapes) invading the fibrous capsule as well as blood vessels. Conversely, widely invasive follicular carcinoma will also show invasion of blood vessels, but there is little or no capsule surrounding the tumor. For this type, the thyroid tissue itself will be invaded.

Once the tumor has invaded the blood vessels, it will spread to other parts of the body through the blood system (hematogenous), as opposed to lymphatic metastasis characteristic of papillary carcinoma. Thus, instead of nearby cervical lymph nodes being targeted, the carcinoma will travel to distant places in the body, mostly lung and bone and even brain and liver. Slightly less than half of all widely invasive follicular carcinomas will metastasize.

Medullary carcinoma

A small percentage of thyroid carcinoma is more lethal and rare than the follicular type. Medullary carcinoma is a particularly lethal malignancy that does not respond to chemotherapy or radiation therapy. It rises from parafollicular cells (C cells) as either a solitary tumor in one lobe or, more typically, as multiple tumors in both lobes.

Medullary carcinoma is a slow growing malignancy occurring in both middle-aged men and women. Most medullary carcinomas secrete calcitonin into the blood stream. This makes increased calcitonin levels in the serum a useful indicator for the possibility of medullary carcinoma, particularly when combined with the clinical symptom of a hard mass felt in the thyroid region.

This carcinoma can occur unpredictably (sporadic) or it can be inherited (often inaccurately referred to as ‘familial’, a term used interchangeably with ‘genetic’). The inherited form is the most common and is caused by an endocrine disorder called multiple endocrine neoplasia (MEN) type 2 (or 2A). MEN disorders cause functioning (hormone producing) tumors in more than one endocrine gland. Either sporadic or inherited forms will metastasize earlier than follicular carcinoma and will spread through both blood and lymph systems. Therefore, medullary cancer can be found in cervical nodes as well as bone, liver, and lungs.

Anaplastic carcinoma

The rarest and most malignant of thyroid carcinomas, and one of the most lethal malignancies in the body, is anaplastic carcinoma. It occurs mostly in the elderly, predominately in women. Its survival rate is less than 5 percent.

This mass rapidly enlarges and extends beyond the confines of the thyroid to invade the neck muscles and blood vessels. The situation is so rapid that it causes the patient pain, mimicking thyroiditis. Anaplastic carcinoma will usually compress or invade the trachea, causing death by asphyxiation.

Diffuse thyroid disease

The thyroid is subject to abnormalities that affect it from outside. Viral or bacterial infection, as well as autoimmune disorders, can cause disease throughout the gland. The two major diffuse thyroid diseases are thyroiditis and Graves’ disease. Thyroiditis is an inflammation of the thyroid gland. It is a group of diseases that can be caused by bacterial or viral infection and autoimmune disorders. Autoimmune refers to the body’s immune system targeting the body’s own tissues.

Chronic autoimmune lymphocytic (Hashimoto) thyroiditis

The most common form of thyroiditis is Hashimoto thyroiditis. This disease mostly occurs in middle-aged women and has a strong association with goiter and hypothyroidism. Recall that hypothyroidism refers to the reduction or absence of thyroid hormone being produced by the thyroid gland. Because the thyroid gland does not produce thyroid hormone, TSH is secreted at abnormal levels, which causes the increase of the size of the gland. In areas where iodine deficiency is not common, Hashimoto thyroiditis is the most common cause of hypothyroidism.

An autoimmune process causes Hashimoto thyroiditis by interrupting thyroid hormone production; that is, antithyroid antibodies and autoimmune cells (lymphocytes or white blood cells) attack thyroid cells. This is why the disease is sometimes called autoimmune lymphocytic thyroiditis. Despite the increased size of the gland due to inflammation, the swelling is painless. Patients are usually given thyroid hormones to relieve them of hypothyroidism and to reduce the size of the gland. Otherwise, no treatment is necessary.

Subacute granulomatous thyroiditis (de Quervain)

Subacute thyroiditis is caused mostly by viral infection and can come on suddenly or gradually, usually just after patient suffers upper respiratory tract infection. As with Hashimoto thyroiditis, subacute thyroiditis affects mostly middle-aged women. The major feature of this type of thyroid inflammation that differentiates it from Hashimoto thyroiditis is the presence of fever and pain.

Temporary hyperthyroidism can occur depending on the severity of the infection. If the inflammation is severe, thyroid tissue will initially breakdown to the point where much of the thyroid hormones are released into the bloodstream. TSH is not produced, yet the gland will remain swollen until it has run its course. Thyroid swelling can subside suddenly or sometimes weeks or months later.

Other forms of thyroiditis

Other types of thyroiditis can be acute or silent. The acute, or sudden, type of thyroiditis can proceed identically to an abrupt onset of subacute thyroiditis, with painful enlargement of the thyroid gland. The difference between the two can usually only be determined clinically; that is, acute thyroiditis is caused by bacterial infections such as *Streptococcus*, while subacute thyroiditis is a viral infectious process caused by such viruses as the Influenza virus. In some cases acute thyroiditis abscesses, which are collections of pus, can form. This is why it is sometimes called acute suppurative thyroiditis.

Another form of thyroiditis you may possibly encounter is a variant of subacute thyroiditis that is painless and affects women just after pregnancy, or in the postpartum period. Because it is painless, it is called silent. This disease is also sometimes referred to as lymphocytic thyroiditis because it has the same feature as Hashimoto thyroiditis, with lymphocyte invasion of the thyroid but without the autoimmune action behind it. However, unlike the hypothyroidism found in Hashimoto thyroiditis, silent thyroiditis produces hyperthyroidism from the destruction of thyroid tissue.

Chronic fibrous (Riedel) thyroiditis is extremely rare. The disease is essentially the replacement of thyroid tissue with dense fibrous tissue. The fibrous tissue occurs throughout the gland or can be seen in one lobe. As with most other forms of thyroiditis, Riedel thyroiditis affects mostly middle-aged women. The cause of the disease is unknown. Patients complain of painless neck enlargement, which is hard when palpated. A unique feature of this disease is that the fibrous tissue extends beyond the borders of the thyroid gland and can encase the tissues of the neck to include muscles, fat, and blood vessels. Riedel thyroiditis can extend to a dangerous point where

the trachea and esophagus are compressed, similar to anaplastic carcinoma. In fact, sonographically, both diseases are virtually identical and usually a biopsy is needed to distinguish between the two. The end-result of this disease is total destruction of thyroid tissue.

Diffuse toxic goiter (Graves' disease)

Graves' disease is an autoimmune disease that causes hyperplasia of the gland. Patients with Graves' disease have too much thyroid hormone (hyperfunction or thyrotoxicosis) in the blood, causing the patient to suffer symptoms of hyperthyroidism. In fact, it is the most common cause of hyperthyroidism. Patients will show signs of nervousness, weight loss, fatigue, profuse sweating, menstrual disturbance, and insomnia. As with other autoimmune disorders affecting the thyroid, this disease affects mostly women. Graves' disease patients show clinical signs of goiter, hyperthyroidism, and bulging eyes (Graves' ophthalmopathy).

Parathyroid disease

There are two types of parathyroid abnormalities: primary and secondary hyperparathyroidism. Both abnormalities concern the excessive production of PTH.

Primary hyperparathyroidism

Primary hyperparathyroidism is a common abnormality of the parathyroid gland. It is a condition caused by too much production of PTH. The hallmark symptom of the disease is excessive calcium levels in the blood and urine, which are normally only detected on routine blood tests. Otherwise, the patient is asymptomatic. Advanced cases may show severe symptoms such as nephrolithiasis (kidney stones) and bone diseases such as osteopenia (decreased density of bone). Occurring mostly in menopausal women, three abnormalities of the parathyroid cause the overproduction of PTH:

1. Single adenoma (glandular tumor of the parathyroid).
2. Multiple gland enlargement (multiple adenomas).
3. Carcinoma.

The most common cause is a solitary parathyroid adenoma. The adenoma enlarges the gland to a point that is detectable on sonography. This abnormality is found in any one of the four parathyroid glands.

A less frequent cause of primary hyperparathyroidism is multiple gland enlargement (multiple adenomas). A small percentage of the patients with multiple adenomas will have inherited a genetic disorder that causes multiple endocrine glands to contain functioning tumors (multiple endocrine neoplasia or MEN). However, you should be aware that most cases of multiple gland enlargements are due to *primary hyperplastic* parathyroid cells rather than multiple adenomas. With *primary parathyroid hyperplasia*, usually all four glands are affected simultaneously. However, the *enlargement* of the glands does not occur at equal rates. With multiple adenomas, the gland enlargement typically involves only two or three glands. Thus, it is extremely difficult to distinguish multiple adenomas from primary parathyroid hyperplasia without microscopic analysis of the tissue (histology).

Finally, an extremely rare third cause of hyperparathyroidism is carcinoma. This abnormality is also difficult to distinguish from an adenoma without histologic analysis.

Secondary hyperparathyroidism

Along with tumor changes in the parathyroid glands (adenoma and malignancy), primary hyperparathyroidism is detected through symptoms and laboratory data that indicate increased

levels of serum calcium. Conversely, *secondary* hyperparathyroidism is detected through symptoms and laboratory data that indicate low levels of calcium in the blood. As with the primary form of the disorder, secondary hyperparathyroidism also involves multiple enlarged glands.

Secondary hyperparathyroidism is normally caused by chronic renal failure. The kidney's filtration system malfunctions in this condition and tends to retain phosphates in the blood. Increase phosphates tend to suppress the normal levels of calcium in the blood. In cases of low serum calcium levels, the parathyroid glands kick into gear and secrete PTH. Because the condition is chronic, the secretion of PTH continues and the parathyroid glands enlarge. Vitamin D deficiency (rickets) also can cause compensatory hypersecretion of PTH and enlargement of the glands. Later stages of this disease may decrease bone density and increase serum alkaline phosphatase levels.

217. Imaging the normal and abnormal thyroid

Because you may not always see obvious abnormalities, you may be tempted to speed through a thyroid scan. However, slowing down to take a closer look may prevent the embarrassment of missing an abnormality in such a superficial structure. To help you know what to look for, we will briefly cover standard approaches to thyroid scanning before discussing obvious and subtle abnormalities.

Clinical considerations

Most clinicians who can palpate a mass in the neck will use FNA to obtain a biopsy sample of the mass. Usually, this method is the most effective for determining if the mass is a thyroid malignancy. Other diagnostic methods, such as sonography and radionuclide imaging, are used only for difficult cases. For example, sonography is useful for determining if a mass is located either within the thyroid gland or if a mass is an enlarged cervical lymph node.

Reasons that may prompt physicians to order sonograms of the thyroid gland are mostly centered on a number of common indications:

- Pre-surgical evaluation.
- Follow-up on patients after thyroid surgery.
- Follow-up on patients with proven malignancy.
- A neck mass is palpable, or can be felt (most common indication).
- Nuclear medicine studies reveal abnormal uptake of radioactive iodine.
- Patients at risk for occult (clinically unidentified or hidden) thyroid malignancy.

Most cases that you will see revolve around the need to determine characteristics of palpable neck masses. Your work in identifying and imaging the size and appearance of the thyroid gland or mass, or both, will usually determine the future need for further sonograms and other procedures.

The other indications are primarily to assess the state of the thyroid or thyroid region before or after a surgical procedure. In cancer cases, the concern for the surrounding tissue is due to the possibility that proven malignancy may spread (metastasize) to the nearby lymph nodes. Patients who have had malignant lesions removed may be scanned to determine leftover pieces of malignant tissue (residual), to determine the reappearance of new malignancy in the surgical area, or metastatic spread from elsewhere in the body.

Also, patients who may have had their necks irradiated as children are at increased risk for thyroid malignancy. Other patients have a family history of cancer-causing endocrine disorder, which we will briefly cover later. Some patients may have to undergo surgery for abnormal

parathyroid glands, which require sonograms to assess size and location. Finally, because of the vascular nature of the thyroid, it is important to know the precise sizes and locations of thyroid masses for interventional procedures such as FNA.

Patients that need a routine thyroid scan for any of the above reasons do not normally require preparation, unless the radiologist is accustomed to performing immediate FNA biopsy procedures on overtly suspicious masses or nodules. In such cases, have the patient avoid aspirin before the procedure to prevent interference with the body's normal ability to clot blood.

Standard thyroid sonography and normal appearance

Place the patient on your examination table so that you may easily access the patient's neck with one hand and the ultrasound controls with the other hand; almost identical to a carotid examination. A good idea is to place a pillow beneath the upper shoulders or a rolled-up towel behind the neck. This will help hyperextend the neck for better access to the lower portions of the thyroid gland. Some patients have thyroid glands with lengths that seem to stretch behind the sternum. In these cases, the extension of the neck will help, as will having the patient swallow and hold, which shifts the entire thyroid up toward the brain and into view on the sonography screen.

Using a 7 MHz or higher frequency transducer, placing your electronic focus at the level of the lobes, and adjusting your over-all gain to display medium gray echoes, should give you an image of the thyroid lobe with a smooth homogeneous echotexture. A hypoechoic to anechoic linear structure a few millimeters thick may be seen coursing through the middle of the lobe on longitudinal views; this usually represents a blood vessel. Turn on your color Doppler to confirm that blood is flowing within the linear structure. If you see no flow, try to adjust the angle and lower your filter. The normal thyroid should have the same homogeneous echotexture found in the testicles. The gland should be moderately echogenic relative to the hypoechoic strap and sternocleidomastoid muscles.

Typical documentation of the thyroid consists of longitudinal images of the lateral, middle, and medial portions of each lobe. Longitudinal images of the isthmus may be requested in some departments. You should image the long isthmus if it contains a mass or appears enlarged. For the middle longitudinal image, measure the AP and length diameters of each lobe (fig. 3–1). The AP diameter is mostly focused on, because if it is larger than 2 cm, the gland can be considered enlarged.

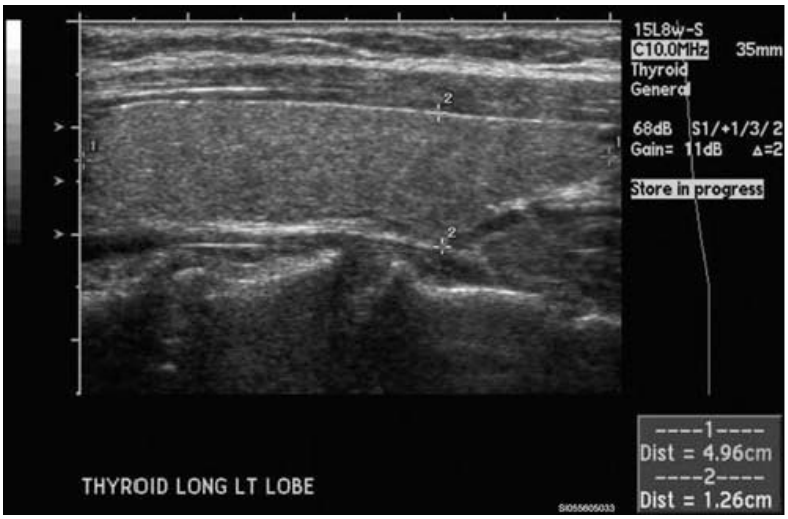

Figure 3–1. Longitudinal thyroid with measurements.

Transverse images of the thyroid consist of documenting superior, middle, and inferior poles of each lobe. Measure the width of each lobe at its widest point in the middle portion. Obtain a transverse image of both lobes simultaneously. This is best accomplished in the middle portion where the isthmus crosses over the trachea (fig. 3–2). Not only are both lobes widest at this point, but the entire gland is presented uninterrupted for comparison of the echotexture. Also, measure the AP diameter of the isthmus.

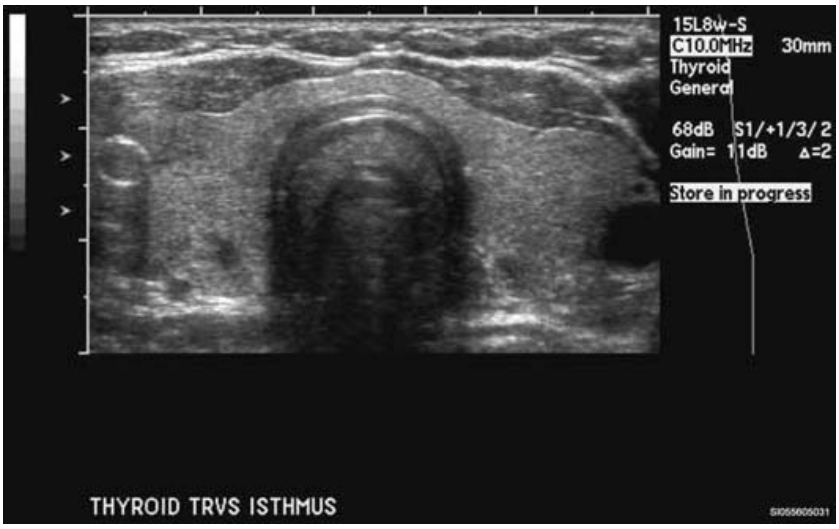

Figure 3–2. Transverse thyroid with isthmus.

Aside from these few standard images, some institutions may require you to display the length of each lobe with color Doppler or even to assess flow in the superior and inferior thyroid arteries with spectral Doppler. Briefly survey the surrounding region for enlarged lymph nodes (abnormally round as opposed to normally oval or thin) or other masses. If any abnormality is seen, document it in three dimensions and use color Doppler. Do not make the mistake of assuming that a hypoechoic mass in the medial position of the lower thyroid is a nodule. This will either be the esophagus, normally hidden in the shadow of the trachea, or the parathyroid gland.

Imaging nodular thyroid disease

When a patient comes to you to have their thyroid scanned, it is usually to determine the characteristics of a known mass. The question foremost in radiologists' and clinicians' minds is if any masses are either benign or malignant. Sonography cannot distinguish with certainty if a nodule is benign or malignant, because both types of thyroid masses have similar sonographic appearances. Thus, any thyroid mass seen on sonogram has the potential to be malignant. However, most masses in the thyroid are benign nodules, which are quite common, and examining every single nodule seen would be an inefficient way to determine malignancy.

Nevertheless, sonography is used because some sonographic clues tend to suggest malignancy in thyroid nodule cases. For example, if a nodule has a thick and irregular hypoechoic halo around the periphery, it is probably malignant. Also, malignant nodules tend to have fine microcalcifications clustered together as opposed to coarse calcifications spread throughout the nodule from benign degenerative changes, or calcifications around the periphery of the nodule. Despite the presence of these clues, sonography alone cannot definitively confirm if a nodule is malignant or benign. The diagnostic procedure for that would be FNA of the nodule cells for laboratory analysis.

Benign or colloid adenomatous nodule (hyperplasia)

Sonographically, adenomatous nodules are isoechoic relative to the surrounding thyroid tissue, with a thin, hypoechoic halo (fig. 3–3). This halo may represent blood vessels, which you may confirm by placing a color Doppler box over the nodule or gland if multinodular. As the size of the adenomatous nodules increase, so does their echogenicity. Normally, this is a result of hemorrhaging within the nodule. Occasionally, you will see cystic areas or spaces, which represent degeneration and necrosis. The cystic areas are filled with blood, colloid, and serous fluid. Hyperechoic foci with comet-tail artifacts may appear within the cystic areas, representing dense colloid (fig. 3–4). You may sometimes see calcification around the nodule in an eggshell appearance or scattered throughout the nodule (fig. 3–5). If you see what looks like an adenomatous nodule, document the size and its relationship to thyroid blood vessels. In cases of multinodular goiter, it is good practice to measure and Doppler the largest nodule. Do not be overly stressed if a radiologist requires you to measure every nodule you see. One of them may be an unrelated follicular adenoma or carcinoma hiding among the hyperplastic follicles!

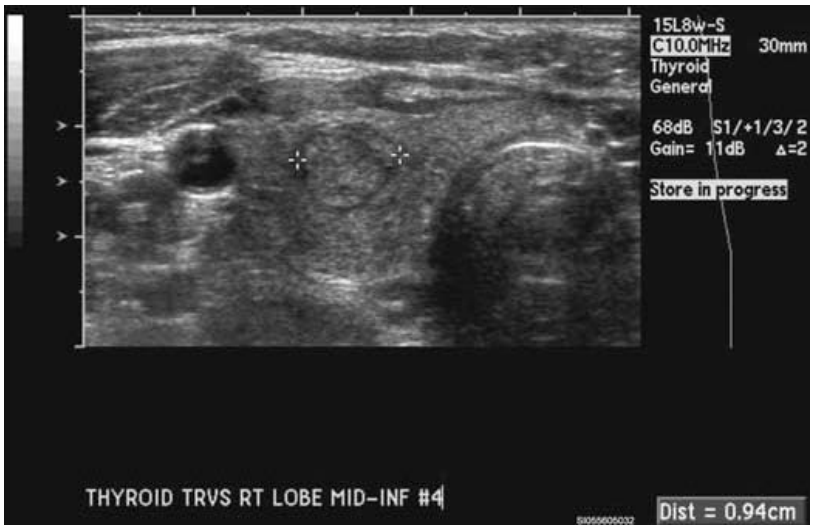

Figure 3–3. Thyroid nodule.

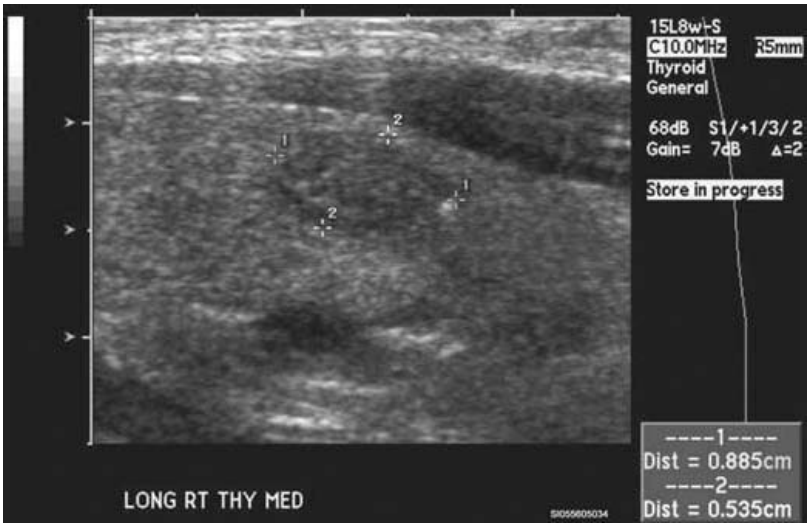

Figure 3–4. Thyroid nodule with hyperechoic foci.

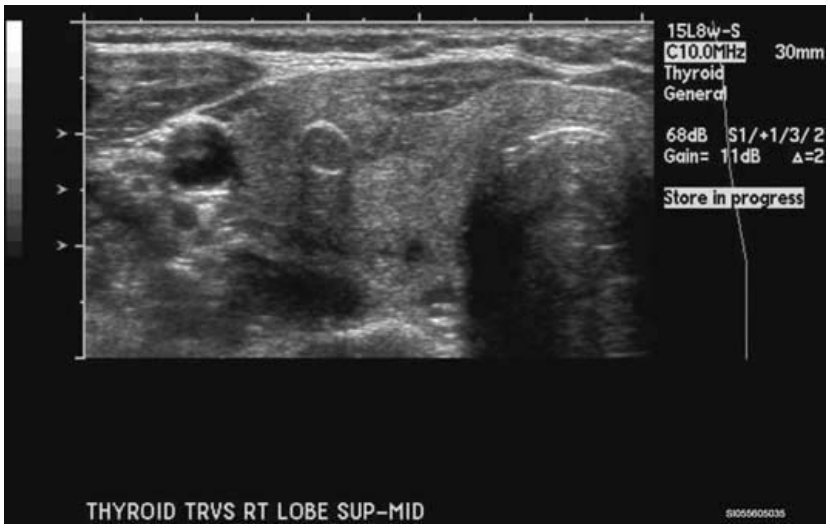

Figure 3–5. Calcification around thyroid nodule.

Follicular adenoma

On sonography, you will normally see a solitary solid nodule. An isolated nodule prompts suspicion in many radiologists, because multiple nodules tend to be benign while a solitary nodule is viewed as a possible malignancy in the thyroid. The echogenicity of the follicular adenoma appears on sonography to be anywhere from extremely hypoechoic to extremely hyperechoic. About half will have a hypoechoic rim band around them similar to benign adenomatous nodules, only thicker. The hypoechoic halo is made up of compressed thyroid tissue and blood vessels. Otherwise, the internal echotexture of the adenoma is homogeneous. Occasionally, you may see cystic spaces within the adenoma, confusing its identity with the benign adenomatous nodule. The cystic spaces represent degeneration; however, in this case, it will usually only be seen in the hyperfunctioning adenoma. As with any mass, measure the adenoma in three dimensions.

One way to distinguish the follicular adenoma from the benign adenoma is to use color Doppler. Follicular adenomas are extremely vascular. Blood vessels in the nodule are arranged in a fashion reminiscent of a wheel spoke, with the vessels at the surface of the nodule radiating inward toward the center. Color Doppler should readily demonstrate this spoke pattern, whereas the benign adenomatous nodule will demonstrate most blood flow along the periphery overlaying the thin halo.

Papillary carcinoma

On sonography, you will usually see a solid nodule. Typical appearances will be a solitary, hypoechoic mass of varying sizes and an irregular border. Approximately a fourth of papillary carcinomas also show fine microcalcifications called psammoma bodies. As with any abnormality, measure it in three dimensions.

Occasionally, you may see enlarged lymph nodes as round hypoechoic masses outside the thyroid on the same side of the papillary mass. This is why it is a good idea to look around the nearby neck region if you should see any of the typical sonographic features of papillary carcinoma. Usually long and slender, the roundness of an enlarged lymph node and the sight of a single nodule in the thyroid are clues to the presence of malignancy. Some metastatic nodes will also

have calcifications identical to nearby papillary carcinoma. If you easily see round lymph nodes, this suggests that papillary carcinoma is present within the thyroid. Some nodes with metastatic papillary carcinoma will start to degenerate, giving rise to cystic areas within it.

Another limited clue may be the increased blood flow that appears in some papillary carcinomas. Color Doppler will show elevated flow within the mass. However, be aware that some papillary carcinomas will show no evidence of flow, particularly within the smaller tumors.

Follicular carcinoma

On sonography, you may see an isoechoic, solitary mass; but more likely the mass will be hidden among other nodules in an unrelated case of multinodular goiter. This makes follicular carcinoma extremely difficult to detect. Usually sonographic clues will allow you to document the tumor for further evaluation. For instance, whereas the benign adenoma has a thin hypoechoic halo, the carcinoma will have an irregular and thickened halo or margin. Of all the nodules in a multinodular goiter, measure the nodule that is the largest or has an irregular halo.

Another clue is that follicular carcinomas tend to be more vascular internally than adenomas. Use your color Doppler to see if there are chaotic and winding blood vessels within the mass as opposed to outside the mass.

Medullary carcinoma

Typical sonographic appearances for medullary carcinoma are of a hypoechoic nodule with irregular margins, coarse calcifications located in the center of the mass that tend to shadow, and hypervascularity. Usually, it is the appearance of the calcification within the mass, along with clinical symptoms, that tends to suggest medullary carcinoma.

Anaplastic carcinoma

On sonography, you will see a large heterogeneous mass with inferior borders sometimes obscured due to its massive size and extension into the thoracic cage. Usually there is no way to measure such a large mass without computed tomography or magnetic resonance imaging. Smaller anaplastic carcinomas can be seen; however, these cancers enlarge so rapidly that by the time symptoms of pain occur, the disease has progressed beyond sonographic ability to adequately measure. Thus the sonographer should measure the dimensions as much as possible.

Imaging diffuse thyroid disease

Frequently an abnormal thyroid will not display a discrete mass on sonography. Instead, the entire gland may be affected and only the echotexture will be unusual in appearance. This type of subtle abnormality is called diffuse thyroid disease. We will look at a few of the more common ones.

Chronic autoimmune lymphocytic (Hashimoto) thyroiditis

On sonography, the thyroid is heterogeneous with multiple areas of hypoechoic echoes interlaced with echogenic linear bands. The hypoechoic areas resemble nodules, which is why this disease can be confused with multinodular goiter on sonography. These areas represent infiltration of the thyroid cells by lymphocytes and other autoimmune cells. Also, the destruction of thyroid follicles leaves behind necrotic tissue. The echogenic bands are fibrous tissue.

Because most of the nodules that form in Hashimoto thyroiditis are only a few millimeters in size, no measurements are usually needed. However, the sizes of the thyroid lobes themselves can be obtained. Color Doppler will reveal either typical or reduced thyroid blood flow. In a few cases, you may see increased vascularity, which is similar to the vascularity seen with Graves' disease.

Subacute granulomatous thyroiditis (de Quervain)

Typical sonographic appearance of de Quervain thyroiditis is of a partially or completely enlarged thyroid with either the entire gland being hypoechoic or areas of each lobe, which may be confused with nodules. The outer border of the gland will be irregular or lobulated. Vascularity on color Doppler will either be normal or decreased. Occasionally, you may see tiny microcalcifications; these are the granulomatous reaction to thyroid follicular destruction.

Diffuse toxic goiter (Graves' disease)

On sonography, you will see an enlarged gland that is typically homogeneous but will occasionally become heterogeneous. Nearly always hypoechoic in comparison with normal thyroid tissue, you might notice a lobulated contour on the surface of the thyroid. A good way to distinguish this appearance from some similar appearances of Hashimoto thyroiditis is to use color Doppler. The color flow will increase beyond normal thyroid levels and stay at a high level even in diastole. Although Hashimoto can show hypervascular flow, it is not present in most cases. The presence of hyperthyroidism and the high-level color flow (sometimes called "thyroid inferno") tends to confirm Graves' disease as opposed to the hypothyroidism of Hashimoto thyroiditis.

Imaging parathyroid abnormalities

On routine thyroid sonography, parathyroid glands are rarely seen. Thus, if seen, they are usually enlarged. However, most cases of parathyroid identification will involve laboratory analysis before sonography and you will probably be expecting them.

Primary hyperparathyroidism

The typical sonographic appearance of the parathyroid gland enlarged by a single adenoma is of a hypoechoic oval or teardrop-shaped mass just a few centimeters in length. Some can grow up to 5 cm in length. Although any one of the four parathyroid glands can be affected, you should look for the solitary adenoma in the posterior and inferior portion of the thyroid. The other three glands are usually smaller (atrophic) relative to the adenoma. Use color Doppler to prevent you from confusing an extremely hypoechoic adenoma for the cross-section of an enlarged blood vessel.

As stated before, with primary parathyroid hyperplasia, usually all four glands are affected simultaneously. However, the enlargement of the glands does not happen at equal rates. Because of this, it may appear on sonography that two or three are enlarged but the other one or two glands are of normal size. This situation can be confused with multiple adenomas, which also shows gland enlargement in only a few of the glands. Also, if you have a patient who has MEN, type 1, and you see multiple enlarged glands, recognize that this is likely to be multiple adenomas rather than primary hyperplasia, and suggest this to the radiologist. Apart from MEN, type 1, *most* multiple gland enlargements will be hyperplasia of all four glands, despite the sonographic appearance of asymmetric sizes. Microscopic tissue analysis is the only accurate way to distinguish between the two.

Usually only one clue will help diagnose a malignancy in the parathyroid on sonography. Look for a parathyroid gland larger than those with adenomas or hyperplasia. Another clue you can use is to note and document the heterogeneous echotexture of the malignant mass, compared to the homogeneous appearances of adenomas and hyperplastic glands.

Secondary hyperparathyroidism

Sonographic appearance of secondary hyperparathyroidism is nearly indistinguishable from multiple adenomas and primary hyperplasia.

Self-Test Questions

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

215. Thyroid anatomy and physiology

1. What is the difference between endocrine and exocrine glands?

2. What structures are situated laterally to the thyroid gland?

3. What normal thyroid variant appears in about a third of young patients?

4. What two types of cells form the walls of thyroid follicles?

5. What two arteries supply the thyroid with oxygenated blood?

6. Where are parathyroid glands typically located?

7. What is the principal role of the thyroid gland?

8. List the ways thyroid hormones affect metabolism.

9. In terms of hormones, what makes the thyroid gland unique from other glands in the body?

10. What effect does parathyroid hormone have on bone?

216. Pathology of the thyroid and parathyroid

1. What condition can result from a small percentage of follicular adenomas, and why?

2. People with MEN type 2 are at increased risk for which type of thyroid carcinoma?

3. What is significant about a neck mass that produces pain similar to thyroiditis?

4. What is the most common form of thyroiditis?

5. What causes Hashimoto thyroiditis?

6. Who is mostly affected by Hashimoto and de Quervain thyroiditis?

7. What is the hallmark symptom of primary hyperparathyroidism?

8. List three abnormalities of the parathyroid gland that cause overproduction of PTH.

9. What abnormalities cause secondary hyperparathyroidism?

217. Imaging the normal and abnormal thyroid

1. List the common indications for thyroid sonography.

2. What is the purpose for having a patient swallow and hold during a thyroid exam?

3. When should you image the long isthmus?

4. What prevents sonography from distinguishing between benign or malignant thyroids?

5. What are some sonographic clues that suggest malignancy in a thyroid nodule?

6. Describe sonographic appearance of the benign adenomatous nodule.

7. What are some typical appearances of papillary carcinoma?

8. What feature makes follicular carcinoma nearly identical sonographically with follicular adenoma?

9. What will color Doppler reveal with Hashimoto thyroiditis?

3–2. Male Reproductive Imaging

Another glandular small part commonly scanned by sonographers is the external portion of the male reproductive system; that is, the contents of the scrotum. While the reproduction capability of this system is important, the hormone production of the scrotal glands is just as significant. Because of its small size, nearly every cell in the testicles exists for these two purposes, and the slightest abnormality can have an enormous impact on function. Fortunately, scrotal abnormalities are usually seen with sonography, sometimes early enough in a disease's course to help determine appropriate or even life-saving treatment.

218. Testicular anatomy and physiology

Testicular anatomy reflects its functions—to produce sperm and testosterone. Nearly every structure of the two testicles and their surrounding environment contributes to these actions.

Anatomy

The male scrotum is a sac or pouch that contains the principal male reproductive organs—two testes. Rarely, patients have one testis (anorchia), or one or more additional testes (polyorchidism). The wall of the scrotum is made of skin, connective tissue, and several layers of fascia, or fibrous tissue that encases muscles or groups of muscles. Scrotal layers, each with varying degrees of muscle fibers, from outer to inner, are the raphe (the *external ridge of skin* located on the *surface* of the scrotal sac along the mid-plane), the scrotal septum (connective tissue that divides the scrotum into two chambers—often incorrectly called the 'median raphe'), the dartos muscle or fascia (which also makes up part of the scrotal septum), the external spermatic fascia, the cremasteric fascia, and the internal spermatic fascia.

A space is formed by the tunica vaginalis, which lies beneath the fascial layers. In most individuals, the space contains a small amount of serous or clear fluid. The tunica vaginalis is made of two layers of serous tissue—an outer or parietal layer that adheres to the scrotal wall and an inner or visceral layer that adheres to the covering of the testis and epididymis. Beneath the visceral layer of the tunica vaginalis is the tunica albuginea of testis, a single thick and fibrous membrane. A space, called the bare area, is located at the posterior portion of each testis not covered by this membrane.

The typical size of a testis is 3 to 5 cm in length, 3 cm in AP diameter, and 3 cm in width. Thus, most testicles will have an oval shape. The sizes diminish with increasing age.

The tunica albuginea dives into the posterior portion of the testicle, forming the thick fibrous tissue called the mediastinum testis. The tissue of the mediastinum testis radiates out into the body of the testicle as fibrous walls of more than 250 separate lobules or divisions. Each lobule contains a few tightly coiled tubes called seminiferous tubules (fig. 3–6). The seminiferous tubules are made of two types of cells: spermatogenic (germ or sex) cells and Sertoli (sustentacular, or support) cells. In between the seminiferous tubules are cells called Leydig (interstitial) cells.

The seminiferous tubules converge into a series of short and relatively straight tubules (sometimes called tubuli seminiferi recti) before entering the fibrous mediastinum testis. Once inside the mediastinum, the tubules form into a mesh of channels called the rete testis. The rete testis converges into at least 10 efferent ductules (tiny ducts), which exit the testicle and immediately begin to merge and coil into a single ductus epididymidis, or duct of epididymis.

Thus, the epididymal head is the combination of efferent ducts and the tightly coiled ductus epididymidis. The epididymal body and tail are composed only of the coiled ductus epididymidis. At the tail of the epididymis, the duct turns a sharp angle and becomes the ductus (vas) deferens, which ascends cephalic and parallel with the epididymal body. The ductus deferens joins blood vessels, nerves, and lymphatics to form the spermatic cord, which courses up into the pelvis through the inguinal canal. Once inside the pelvic cavity the ductus deferens loops over the ureter and down behind the posterior surface of the bladder. At the base of the prostate (its superior portion), the ductus deferens joins the seminal vesicle to form the ejaculatory duct, which passes through the prostate.

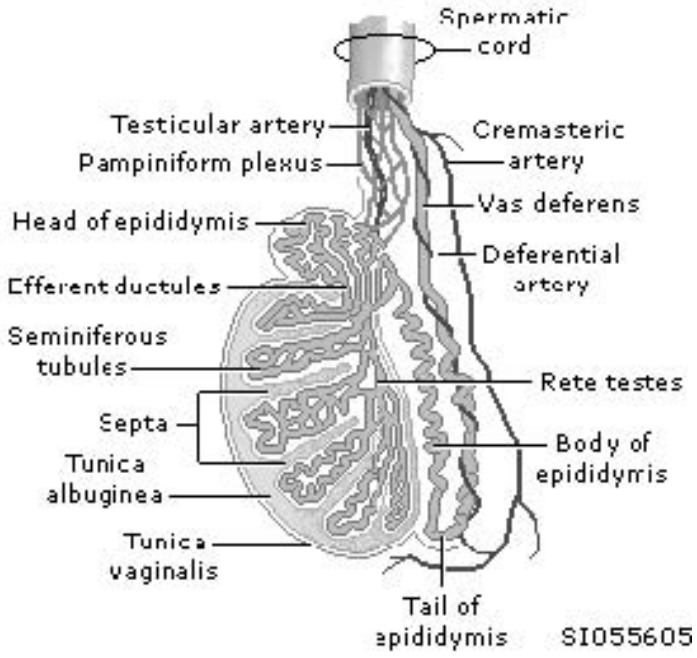

Figure 3–6. Diagram of the testis and scrotal sac.

The testes and scrotal environment are supplied with blood via three pairs of main arteries: the testicular artery, the cremasteric artery, and the artery to ductus deferens. On each side, these three arteries course together with the right and left spermatic cords through the inguinal canals

into the scrotal sac. The testicular artery is sometimes known as the internal spermatic artery. It is the artery that supplies the testis with most of its blood. Originating from the aorta slightly inferior to the renal arteries, it feeds mostly the testis as well as parts of the epididymis and ureter.

The cremasteric artery is occasionally called the external spermatic artery. Originating from the inferior epigastric artery, which is a branch of the external iliac artery, the cremasteric artery feeds the cremaster muscle fibers that interweave the tissues covering the spermatic cord. The cremaster muscle is a continuation of the internal oblique muscle of the abdominal wall. The cremasteric artery also feeds the tissues covering the spermatic cord.

The artery to ductus deferens is commonly called the deferential artery. Originating off the internal iliac artery, this vessel primarily feeds the ductus deferens and supplies the testis and ureter. Occasionally, the artery to ductus deferens will arise off a branch of the internal iliac artery such as the umbilical artery or its branch, the superior vesicle artery.

Veins exiting the testis at the mediastinum gather together at the posterior portion of the testis, forming a network of veins called the pampiniform plexus. The epididymis and various tunica layers also send venous tributaries to join with the pampiniform plexus. A significant part of the spermatic cord consists of the pampiniform plexus, which courses anterior to the ductus (vas) deferens. After coursing through the inguinal canal into the pelvis, the many veins of the plexus merge together to form the internal spermatic vein, one on the left and one on the right. The right internal spermatic vein courses into inferior vena cava at an acute angle, and the left internal spermatic vein follows a tortuous course before merging abruptly into the left renal vein at a 90-degree angle.

Physiology

The function of the male reproductive organs is to produce sperm and testosterone. The production of sperm begins within the walls of the seminiferous tubules. The sperm-making cells (spermatogenic) are located from the outer layer to the inner layer of the tubule wall. Supporting the spermatogenic cells are the Sertoli cells. After puberty, the spermatogenic cells actively begin to develop, with increasing levels of maturity from outer layer to inner layer of the tubule wall. Outside of the tubules, the Leydig cells assist with development by producing the androgen, testosterone. An androgen is a hormone that promotes male characteristics. Leydig cells secrete testosterone directly into the blood circulation (leaving the testis principally through the pampiniform plexus, a network of veins). Testosterone also influences the development of the early spermatogenic cells.

As spermatogenic cells develop, the Sertoli cells nourish, dispose of excess cells, and control movement and release of matured sperm cells into the inside of the tubule (the lumen). Sertoli cells also produce fluid for sperm transport and for providing the sperm cells with proteins and hormones. Spermatogenesis, or sperm development, takes approximately 64 days.

Once sperm cells are produced, they are transported with the seminal fluid from the seminiferous tubules toward the tubuli recti or straight tubules. After traveling from the recti through the rete testis network, the seminal fluid is sent to the epididymis via the efferent ductules.

Inside the epididymis, sperm acquires motility (the ability to move on its own) and is also stored for weeks. The smooth muscle walls of the ductus epididymidis contract and relax (peristalsis), which propels the seminal fluid toward the ductus (vas) deferens. Inside the deferens, sperm is again stored for up to several months. Peristalsis moves the fluid up into the spermatic cord on its way to the pelvic cavity and the prostate.

219. Testicular pathology

Abnormalities of the male reproductive system can be categorized into those originating within the testes (intratesticular) and all others originating outside the testes (extratesticular). Most of the focal lesions or masses are seen intratesticularly; and all others, such as fluid and evidence of inflammation, are located extratesticularly.

Intratesticular abnormality

When it comes to abnormalities of the scrotum, most physicians are concerned about the occurrence of intratesticular masses. Given the tendency of these masses to be malignant, this view is understandable. Thus, you should be familiar with the various masses, benign and malignant, that may occur.

Benign cystic abnormalities

Approximatelyone-tenth of men will have testicular cysts, usually middle-aged or older. Two types of benign cysts occur within the testis: tunica albuginea cysts and intratesticular cysts. Apart from size, both are structurally identical. The primary difference between the two is that tunica cysts form within the thick fibrous membrane (tunica albuginea) that covers the testis while intratesticular cysts are surrounded by testicular tissue (parenchyma).

Tunica albuginea cyst

Tunica albuginea cysts are usually discovered when a middle-aged patient complains of either a painless lump or of painful focal swelling. These cysts usually appear along the lateral surface of the testis near the mediastinum and epididymis. The cause is unknown; however, several theories have been proposed, such as the cysts are remnants of embryonic development. The cysts contain serous (clear, resembling serum) fluid and are generally a few millimeters in diameter.

Intratesticular cyst

Other benign cysts are intratesticular cysts. These cysts are identical to tunica cysts but are located within the testis. Causes of intratesticular cysts can be from past inflammation of the rete testis, which can back up the ductules (hence the appearance near the mediastinum), or they can be from past trauma. Unlike tunica cysts, intratesticular cysts appear more in older men and are not normally palpable.

Ectasia of the rete testis

Dilation of the rete testis is called ectasia (dilation of a tubular structure). Do not be concerned if you hear some refer to this condition as *tubular* ectasia; there really is no other type of ectasia. The cause of ectasia of the rete testis is thought to be epididymal obstruction from either inflammation or trauma.

Epidermoid cyst

These are extremely rare benign masses. They grow at an extremely slow rate. Most clinicians will be able to feel a hard and smooth mass that does not cause pain for the patient. An epidermoid is thought to be a benign variant of teratoma. Generally, only microscopic analysis of the cells (histology) will distinguish an epidermoid cyst from a malignant teratoma.

Malignant testes abnormalities

Most tumors of the testis are germ cell tumors. Germ cells are the reproductive cells, either mature or immature. In the male, the mature germ cells are the sperm cells (spermatozoa), and the

immature germ cells are the spermatogenic cells. Both are found in the seminiferous tubules; the spermatogenic cells are located within the walls, and the sperm cells are attached to the inner wall or within the lumen of each tubule. It is from these cells that germ cell tumors arise. Germ cell tumors are composed of either pure cell types or a mixture of one or more cell lines. Most single cell type tumors are seminomas. Mixed-cell tumors constitute one or more of other types, such as embryonal carcinoma, teratoma, and choriocarcinoma. Although testicular germ cell tumors can be composed purely of one of these types, such occurrences are rare.

Seminoma

The most common germ cell tumor in the adult testes is the seminoma. It occurs in men ranging in age from 25 years to 55 years, but peak incidences are in the fourth decade of life (30–40 years old). The cause is unknown; however, there is a known risk of occurrence in young patients who have suffered undescended testes (cryptorchidism). The usual clinical presentation is of a patient with painless and palpable mass, mostly on one side. A small percentage will have pain if the tumor is necrotic or hemorrhages. There are no significant laboratory values other than a slight increase in human chorionic gonadotropin (hCG) in one-tenth of cases.

The seminoma appears in three different types, but only two are of significance to physicians: the typical or classic type and the spermatocytic seminoma. Most seminomas are classic and remain within the confines of the testis. However, one-fourth will extend beyond the tunica albuginea, usually through the lymphatics. They can be as small as several centimeters in size or can largely replace the testis. They occur singularly or can be multiple within the same testis. Conversely, the spermatocytic type never metastasizes and occurs in men who are 50 years old or more. This type is very large and can expand the testicle to an abnormal size. With seminomas, the prospect for recovery (prognosis) after five years is excellent. This is mostly due to the extreme responsiveness of the tumor to radiation therapy or chemotherapy.

Embryonal carcinoma

The second most common germ cell tumor is the embryonal carcinoma (occasionally referred to as embryonal *cell* carcinoma). The tumor occurs in men between 20 and 35 years of age. However, a variation of it occurs in infants called an endodermal sinus or yolk sac tumor, which is the most frequent of germinal tumors in infants. Both tumors are highly aggressive and do not respond well to either radiation therapy or chemotherapy. Thus, only a quarter of patients survive after five years. Embryonal carcinomas typically invade the tunica albuginea and spread to the epididymis and spermatic cord.

Clinically, most patients will have a palpable scrotal mass. Just over half of adult patients will show elevations of hCG. Infants and adults with tumors mixed with components of yolk sac tumor will usually show elevations of alpha-fetoprotein (AFP).

Teratoma

Five percent of germ cell tumors are teratomas, a tumor that is usually composed of all three germ cell layers: endoderm, mesoderm, and ectoderm. Further, most teratomas are mixed with other germ cell types, with embryonal carcinoma and teratoma being the most common. Teratomas range from being benign to malignant and occur mostly in men between 20 and 35 years of age. In infants and young children, the benign form occurs and is called a *mature* teratoma. The immature, malignant form occurs in adults and is the most aggressive of germ cell tumors.

Choriocarcinoma

Less than 5 percent of germ cell tumors are choriocarcinomas, which makes it the rarest. When it occurs, it is extremely malignant and will metastasize quickly. Unlike other testicular masses, choriocarcinomas are small and thus, clinically, the patient will have no pain or enlargement in the scrotum. However, hCG levels will be significantly elevated, which may lead to gynecomastia, an over-development of male mammary glands. The patient may show signs of metastatis through the blood. For example, the patient will be either spitting blood from bronchial bleeding or the patient will be vomiting blood from gastrointestinal bleeding.

Other testes abnormalities

Not every focal abnormality within the testis is a germ cell tumor. A small percentage arises from stromal tissues that support reproductive cells, such as Leydig and Sertoli cells.

Gonadal stromal tumors

Recall that Leydig cells (interstitial cells) are located between seminiferous tubules. Leydig cells produce androgens, the hormones that provide male characteristics, of which the principal secretion is testosterone. Also, Sertoli cells make up most of the cells in the walls of the seminiferous tubules. Sertoli cells nourish and protect immature germ cells as they develop to maturity.

Gonadal stromal tumors are either pure examples of these cells or a mixture of both. Either one or both may also be mixed with other types such as thecal, granulosa, or lutein cells. Stromal tumors may also combine with germ cell tumors, which are called gonadoblastomas.

Less than six percent of testicular neoplasms are gonadal stromal tumors, of which most are Leydig cell tumors. The majority of these tumors are benign in both children and adults. Just over one-tenth of the tumors are malignant. Significantly, these tumors produce androgens and other hormones (estrogens) that result in precocious (unexpectedly early) puberty in children and gynecomastia in adults. Patients have painless and palpable masses clinically. These tumors are usually small but can become quite large, filling an entire testis.

Microlithiasis

Microlithiasis is a rare condition in which tiny calcification bodies are scattered throughout the testis. Although this abnormality has been historically categorized as benign, a number of recent studies have been performed which have increasingly demonstrated an association with testicular malignancy. Not every radiologist will accept this trend and it remains controversial. A cause of microlithiasis is also debated, with most generally suggesting malfunctioning Sertoli cells within the seminiferous tubules.

Typically, no clinical symptoms exist for microlithiasis. Therefore, they are commonly incidental findings on sonography. However, microlithiasis tends to be associated with infertility or reduced fertility (subfertility), Klinefelter syndrome, Down syndrome, cryptorchidism, and pulmonary alveolar microlithiasis (calcifications in the alveoli of the lungs).

Metastases

Apart from germ cell and gonadal stromal cell neoplasms, you may encounter testes with secondary malignant neoplasms originating from sites elsewhere in the body. The spread of malignancy from a site in the body (the primary site) is called metastasis. Most metastatic testicular tumors are malignant lymphomas, of which the majority is of the non-Hodgkin type. Leukemia is the second most common testicular secondary neoplasm. The prostate and lungs are the sites where most testicular metastases originate. Other sites, such as colon, kidneys, stomach,

and pancreas, will also generate secondary tumors in the testes. Metastatic carcinoma to the testis is extremely rare, but can occur.

Testicular lymphoma and leukemia occur in males older than 50, with some types of leukemia limited to testicular involvement in children. They usually occur bilaterally with multiple lesions but can also spread throughout the testis.

Extratesticular (scrotal) abnormality

Nearly every abnormality that can occur outside the testis within the scrotum can negatively affect the testis. Thus, it is important to diagnose extratesticular diseases. Most of the abnormalities that you encounter will usually be caused by an extratesticular abnormality. Although a host of diseases can occur in the scrotum, such as epididymal tumors, we will look at some of the more common abnormalities.

Hydrocele

Normally, the tunica vaginalis has a miniscule amount of serous fluid between its two layers, the visceral and parietal. When enough fluid is present to surround most of the testis and epididymis, the abnormality is called a hydrocele. Usually the fluid is serous fluid; however, some cases may be blood (hematocele) or pus (pyocele).

Hydroceles have either congenital or acquired causes. A common congenital cause is improper closing of the processus vaginalis, which connects the peritoneal cavity to the scrotal sac of the fetus. The processus vaginalis is a pouch from the peritoneal cavity that slips through the inguinal canal in males and forms the early tunica vaginalis within the scrotum. It normally closes around birth. Thus, fluid leaks from abdomen into the scrotum in newborns or infants. This abnormality typically resolves itself within 18 months.

Most acquired causes of hydrocele are unknown, or idiopathic. Up to nearly a half of causes are due to some type of trauma to the scrotal region. Some other causes are inflammation, torsion, or tumor. Unless attributed to these causes or of significant size, hydroceles are typically painless. Most cases of inflammation may have either blood or pus within the fluid. A good clinical indication of pyocele is the presence of pain.

Spermatocele and epididymal cyst

Cysts within the epididymis are usually not true cysts. Rather they are focal dilatations of the efferent tubules in the head and along the ductus epididymis of the body and tail. Most dilatations are in the epididymal head and are filled with sperm cells (spermatozoa), lymphocytes, and fat. These are called spermatoceles. Rarely, a dilatation will occur that has nothing in it but serous fluid or will appear in the body or tail of the epididymis. Sonographers call these structures epididymal cysts. However, many physicians are unconcerned with the differences between the two types, and the spermatocele is considered to be the more common of the two.

Mostly asymptomatic, spermatoceles are essentially the result of an obstruction between the ductus (vas) deferens at the tail of the epididymis along to where the efferent tubules connect to the rete testis. Common causes are prior infection of the epididymis (epididymitis), vasectomy, and inguinal hernia repair.

Varicocele

Remember that the pampiniform plexus of veins run within the spermatic cord as the cord exits the scrotal sac via the inguinal canal. When these veins become abnormally dilated, the condition is known as a varicocele. Two types of varicocele exist, depending on the nature of the suspected cause: primary and secondary. Primary varicoceles are the most common and occur mostly in

adolescents or young men. The cause is attributed to absent or improperly functioning (incompetent) valves within the internal spermatic vein. Without working valves in the internal spermatic vein, venous blood flow reverses course and runs back through the pampiniform plexus, especially when the patient is in an upright position. Thus, the veins of the spermatic cord become abnormally dilated. The causes for incompetent valves in the spermatic vein are unknown; therefore, you may hear primary varicoceles referred to as idiopathic varicoceles. Although varicoceles can appear bilaterally, most cases of primary varicoceles occur on the left side where the internal spermatic vein drains into the left renal vein at a 90-degree angle.

The secondary varicocele is caused by external pressure on the internal spermatic vein. In fact, varicoceles that occur suddenly on the right side or bilaterally, particularly ones that are unaffected in diameter with a change in patient positioning, should raise the suspicion of compression of the internal spermatic vein. Enlarged liver, renal or other abdominal masses, lymphadenopathy, or even a tortuous superior mesenteric vein compressing the left renal vein, can cause retrograde flow within the internal spermatic veins and resulting in varicoceles.

Most patients complain of a painless mass that feels like a "bag of worms." Complaints of pressure or a dull throbbing, especially with changes in patient position, are common. Some are completely asymptomatic and are discovered incidentally on sonography for an unrelated problem. Because the properly venous blood flow from the testis carries heat away from the scrotal environment, a varicocele tends to raise the temperature of the testis. This affects the production of spermatozoa. Thus, some physicians may send infertile or sub-fertile patients to the sonography department to determine if an asymptomatic varicocele is the source.

Epididymitis

Epididymitis is inflammation of the epididymis. When the inflammation spreads to the testis, it is called epididymo-orchitis. Numerous organisms infect the prostate or the lower urinary tract, causing inflammation that spreads along the ductus (vas) deferens into the epididymis. One of the most common organisms responsible is *Escherichia coli,* which typically occurs in middle-aged or older men but also in children. In younger men and adolescents, sexually transmitted organisms are usually responsible for infection, such as *Neisseria gonorrhoeae* (gonorrhea) or *Chlamydia* (chlamydiosis). Other causes of infection can be the result of surgical operations such as vasectomy or viral infections such as mumps.

There are two types of epididymitis, acute or chronic. The acute variety is characterized by sudden intense pain in the scrotum, usually accompanied by fever and extremely painful urination. The scrotal wall will be thickened and extremely painful when touched. A fever (with its associated elevation in white blood cell count) is a useful marker that distinguishes inflammation of the epididymis from torsion of the spermatic cord, a condition that produces similar pain and results on sonography. As the urinary tract and prostate stage the infection, the usual pathway to the scrotum is via the lymphatics and ductus (vas) deferens, both of which are encased within the spermatic cord. Consequently, the first part of the epididymis affected is usually the tail, but often the body and head are inflamed as well. Just under a quarter of epididymitis cases will have extension of inflammation beyond the epididymis into the testis. Epididymitis is normally treated with antibiotics. Nearly identical features occur in chronic bouts of epididymitis, including reoccurrences of acute inflammation. However, pus (pyocele), abscesses, and hemorrhage collections (hematocele) may appear in the scrotal sac. Epididymo-orchitis is likely during extended bouts of inflammation.

Torsion

Torsion is a twisting of the spermatic cord, which causes ischemia and infarction of the attached testis. Although you may hear it referred to as torsion of the testis, of course, the testis itself is not twisted. The spermatic cord contains blood vessels that supply the testis. When the cord twists, blood flow is clamped off and the testis suffers from ischemia, the interruption of blood supply. The severity of torsion is based on the number of twists, with partial torsion represented by one turn of 360 degrees and complete torsion of several more turns. If the condition persists, the cells that make up the testis will begin to die (necrosis) for lack of blood, a complication called infarction. Physicians are specifically concerned about gonadal necrosis, and all efforts are focused on salvaging the affected testis.

The most common cause of spermatic cord torsion is the congenitally abnormal surrounding of the testis and epididymis by the tunica vaginalis. Normally, the tunica vaginalis covers the anterior and lateral portions of the testis and leaves the posterior portion bare. It is through this posterior portion that the testis is attached to the wall of the scrotum. When the tunica vaginalis surrounds the entire testis, attaching to part of the spermatic cord, it allows the testis to hang suspended within the scrotum by the vessels and vas deferens. The condition is called a bell clapper deformity. Thus, the testis becomes mobile enough to allow the cord to twist upon itself. The bell clapper deformity is nearly always a bilateral occurrence.

Most incidences of torsion occur in young males, peaking in preadolescents to adolescents. Infants can experience torsion, but this type is usually due to a twisting high up on the cord within the inguinal canal. Undescended testis and the surgical repair of it (orchiopexy) can prompt this inguinal twisting of the spermatic cord.

Most patients complain of sudden (acute) onset of severe pain, vomiting, and fever. In some patients, pain gradually intensifies. Approximately a third will resolve spontaneously. Because torsion is a surgical emergency, classifications of severity are based upon the time of pain onset. All torsions that occur within 24 hours are considered *acute*; those that occur within one to 10 days are *subacute*; and those that are in existence beyond 10 days are classified as *chronic*.

Acute torsion or the first 24 hours concern surgeons. Most try to correct torsion surgically within five to six hours, a period where most testes can be salvaged. The salvage rate for acute torsion:

- 5–6 hours: 80 percent–100 percent.
- 6–12 hours: 70 percent.
- > 12 hours: 20 percent.
- > 24 hours: unsalvageable.

Scrotal hernia

Scrotal hernia is simply the presence of abdominal contents within the scrotal sac. Not all scrotal hernias contain bowel (small or large intestines); some are composed of omentum, the membrane that encases the bowel. Usually, scrotal hernias are diagnosed easily in the clinic. However, the atypical hernias such as hardened (indurated) hernias are sent to sonography for evaluation.

Occurring at any age, scrotal hernias are caused by bowel slipping through the deep inguinal ring and into the scrotum through the inguinal canal. The abnormality is common and can be caused by exertion or through a congenital defect, such as a processus vaginalis that has not properly closed after birth.

Undescended testis

Commonly referred to as *cryptorchidism*, undescended testis is, obviously, the failure of the testis to descend into the scrotum. Therefore, the testis remains anywhere along the descent path, from within the abdomen to just above the scrotum. Most undescended testes are located within the inguinal canal.

Despite being one of the most common of genitourinary abnormalities in infants, only 3 to 4 percent of full-term newborns have undescended testes. Most cases are unilateral, although a fourth will be bilateral. Causes proposed range from interruption of hormonal stimulation from the pituitary gland to abnormal development of the inguinal canal.

The primary concern in infants and children with undescended testis is the risk of infertility and malignancy. Although asymptomatic, most physicians will choose to surgically remove the undescended testis from the inguinal canal and plant it within the scrotum (orchiopexy). If the testis is untreated, the risk for malignant germ cell tumor increases, particularly seminoma.

220. Imaging normal and abnormal scrotum/testes

Before we discuss scanning the normal scrotum and various abnormalities, we should cover some common reasons or indications for performing sonography of the scrotum and testes. We will then cover standard images you should obtain to maximize the likelihood of picking up abnormalities of the scrotum.

Normal imaging of the scrotum/testes

Most sonographic examinations of the scrotum are sent to evaluate the causes of scrotal swelling, such as fluid or mass. Scrotal abnormalities are evaluated to determine if they are intratesticular or extratesticular. This is critical because most intratesticular masses are malignant. Patients with known metastatic disease or prior testicular cancers are followed-up. Suspicious intratesticular abnormalities such as microlithiasis (explained below) are also followed-up. Infrequent indications are infertility cases as well as undescended testes.

Sonographic evaluation for acute scrotal pain is accomplished primarily to determine if the root cause is inflammation or torsion. Sometimes known inflammation cases are sent because patient condition deteriorates to a point where complications such as abscesses arise. Other cases, which may or may not cause pain, are sent to sonography such as trauma or varicoceles.

Whatever the cause, the approach to sonographic examining these patients remains simple. There is no preparation required. Most patients are able to lay flat on their backs and have a folded towel wrapped around both upper thighs high enough to lay the scrotal sac on it. Another towel should cover the penis in such a manner where only the scrotal sac is visible for examination. The transducer should be a linear or curvilinear array with at least 5 MHz, but 7 MHz or greater produces excellent resolution. Doppler should be available with your ultrasound unit and set to detect slow and subtle blood flow. Some departments may require standoff pads for evaluating superficial structures or the scrotal wall. Although only a few millimeters separate the transducer from the scrotal anatomy, you should still scan carefully because some of these abnormalities can prove to be quite subtle.

The usual appearance on sonography of the normal testis is of a smooth-textured (homogeneous) ovoid organ with medium gray (medium level) echoes. This appearance is somewhat similar to the sonographic appearance of the thyroid gland. On older equipment, you may not see the tunica albuginea covering the testis, but some of the latest equipment with superior resolution can clearly display a thin and highly echogenic line marking the outer testis. Normally, you will see an echogenic band within the testis extend the length along one side. This is the mediastinum

testis, and it is a landmark that identifies the posterolateral portion of the testis. The medium-echoed structure outside the testis and attached very near to the mediastinum is the head of the epididymis, which marks the superior pole. The epididymis is coarser in texture than the smooth appearance of the normal testis. It also is either isoechoic or slightly hypoechoic to the echogenicity of the testis. Neither the tunica vaginalis nor the fascial layers of the wall will be seen with sonography. However, a thin layer of fluid may be seen around the testis, which is actually the space between the two layers of tunica vaginalis. Finally, the scrotal wall is seen as a medium-echoed structure close to the transducer usually 2 to 8 mm thick.

The standard views for scrotal sonography should contain testes, epididymides, and color and spectral Doppler images demonstrating evidence of blood flow within the testis. Focal zones should be situated at the level of the testis; multiple focal zones are even better, but tend to slow the frame rate in slightly older machines. Longitudinal images of the medial (fig. 3–7), middle (fig. 3–8), and lateral (fig. 3–9) portions of the testis should be obtained. The echogenic mediastinum is useful as a landmark for the lateral portion. Measurements of the length and AP diameter of the middle portion of the testis should always be included (fig. 3–10).

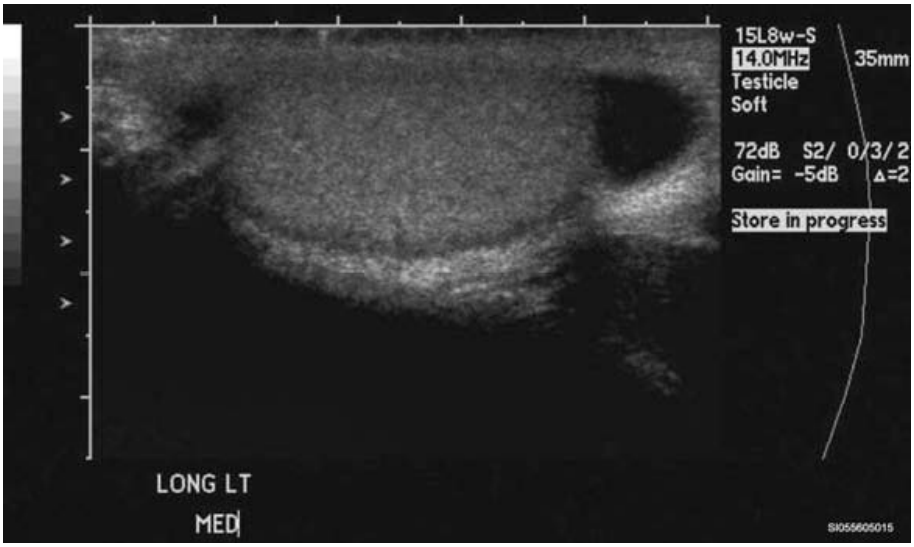

Figure 3–7. Longitudinal testis medial.

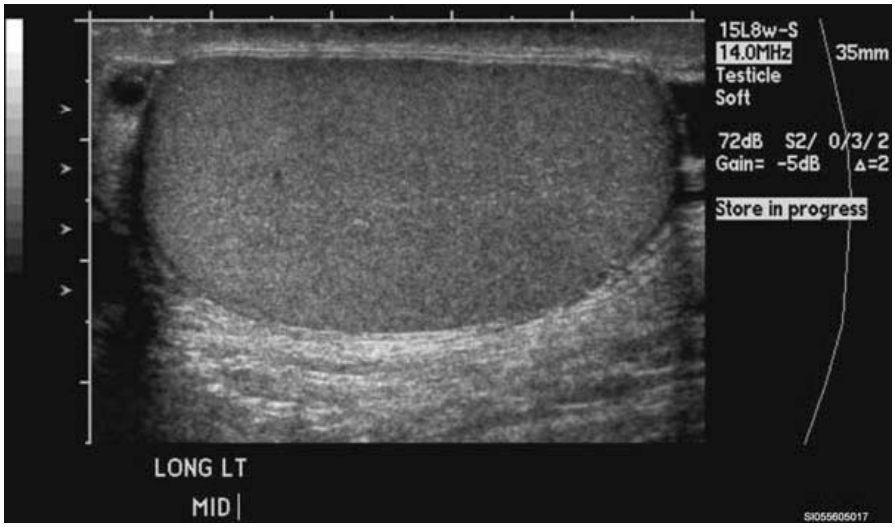

Figure 3–8. Longitudinal testis middle.

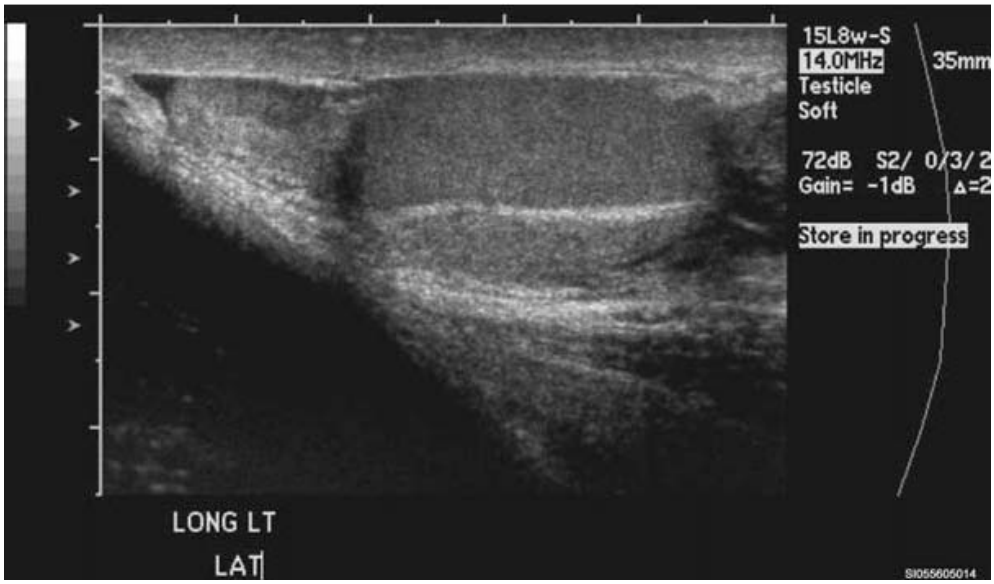

Figure 3–9. Longitudinal testis lateral.

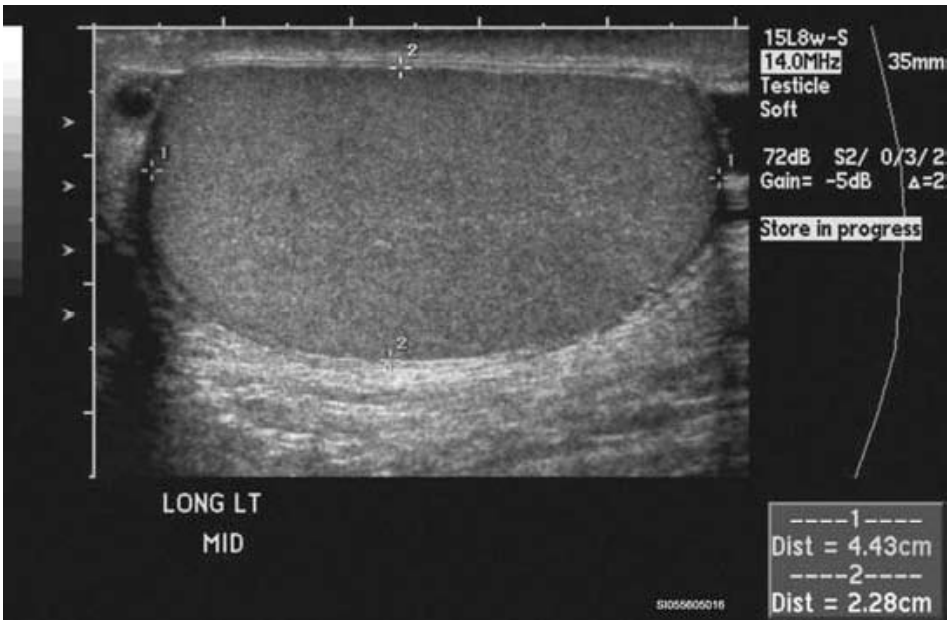

Figure 3–10. Longitudinal testis measurements.

Transverse images of the superior, middle, and inferior poles of the testis should be obtained. At the widest part of the middle portion, the width measurement should be taken (fig. 3–11). Color Doppler may be required in some departments for the transverse view of the testis. This is to demonstrate the presence of normal blood flow. Longitudinal color Doppler images of the testis may also be required. An image of both testes (bilateral) in the transverse view is necessary for comparison to detect differences in echotexture, echogenicity, and size (fig. 3–12).

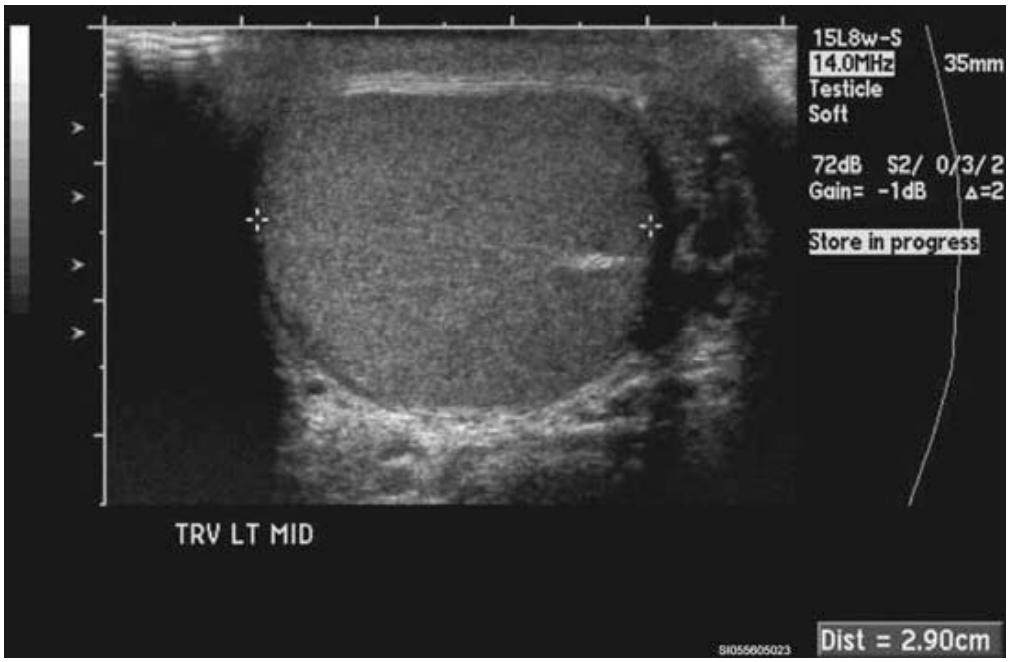

Figure 3–11. Transverse testis measurement.

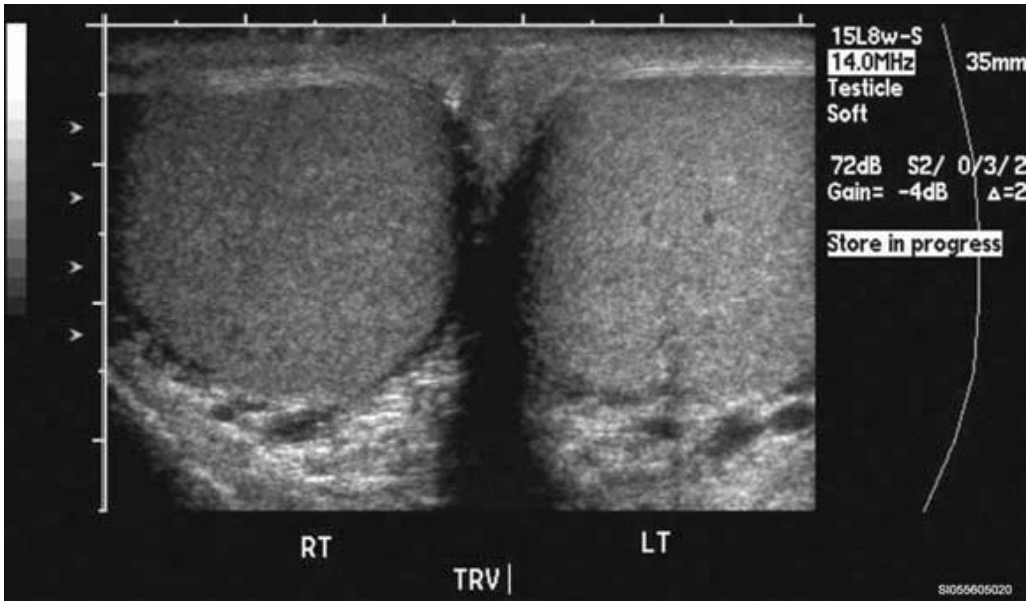

Figure 3–12. Transverse testes bilateral.

Images of the extratesticular anatomy are normally limited to the head of the epididymis, with AP diameter measurement and color Doppler images added (fig. 3–13). However, in the presence of abnormalities, such as varicoceles and hydroceles, the scrotal sac is imaged in the area of abnormality in both long and transverse views along with Doppler imaging.

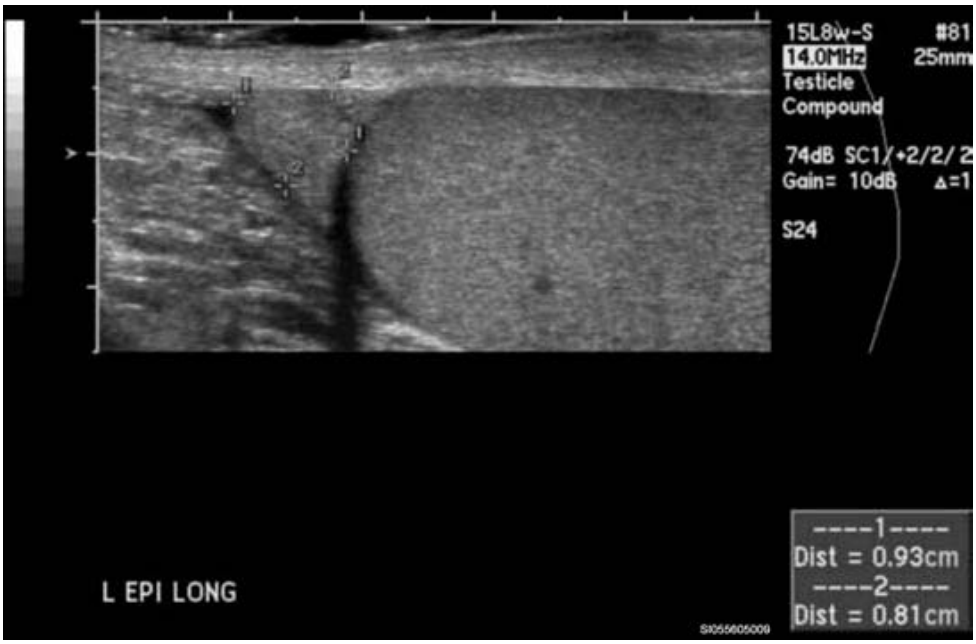

Figure 3–13. Longitudinal epididymis head.

Imaging benign cystic abnormalities

Like elsewhere in the body, they must have the classic sonographic criteria of simple cysts to be diagnosed as benign cysts. The criteria for simple cysts are well-defined margins, thin walls, anechoic centers, and enhanced posterior acoustic transmission. Otherwise, particularly with the presence of solid elements, they become suspicious for being cystic tumors; and most tumors within the testes are malignant.

Tunica albuginea cyst

On sonography, tunica cysts can be singular, multiple, or mutlilobed. They may press (invaginate) into the testis parenchyma, simulating an intratesticular cyst. Otherwise, they fit the classic criteria of simple cysts. Measure any cyst that you see in at least the AP dimension. It is helpful for such a small cyst to magnify the image to a point where half the testis fills the display. This action will help with accurate placement of your measurement cursors. On follow-up sonography, tunica cysts should appear unchanged. To ensure you are not examining a cross-section of a surface blood vessel, turn your transducer 90 degrees to see if the lumen lengthens out. Also, place a color Doppler box over the area, to see if it fills with color, representing blood flow. Do not forget to lower filter settings and increase sensitivity settings for color Doppler.

Intratesticular cyst

As previously stated, intratesticular cysts are identical to tunica cysts. To locate them within the testicle, sweep the beam across the upper pole near the mediastinum (fig.1–14). Be sure to prove whether the cyst is solitary within a testis by displaying the testicle in a longitudinal image. For multiple cysts, prove the case by obtaining longitudinal images of a testis or obtaining transverse bilateral images of the testes (fig. 3–15). As with tunica cysts, measure these cysts in an identical fashion. Compared with tunica cysts, these cysts have a wider range of sizes; anywhere from 2 mm up to several centimeters.

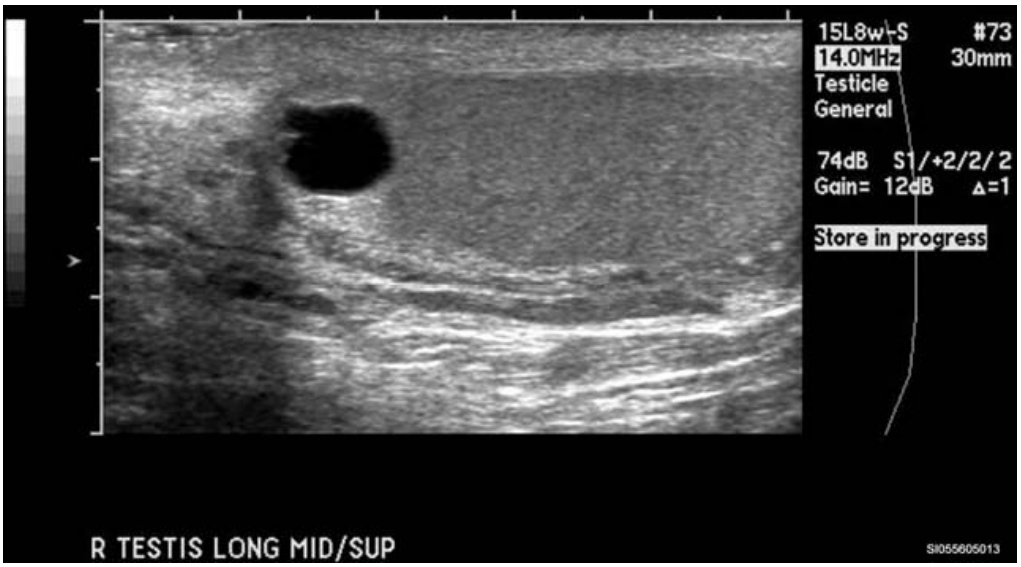

Figure 3–14. Longitudinal intratesticular cyst.

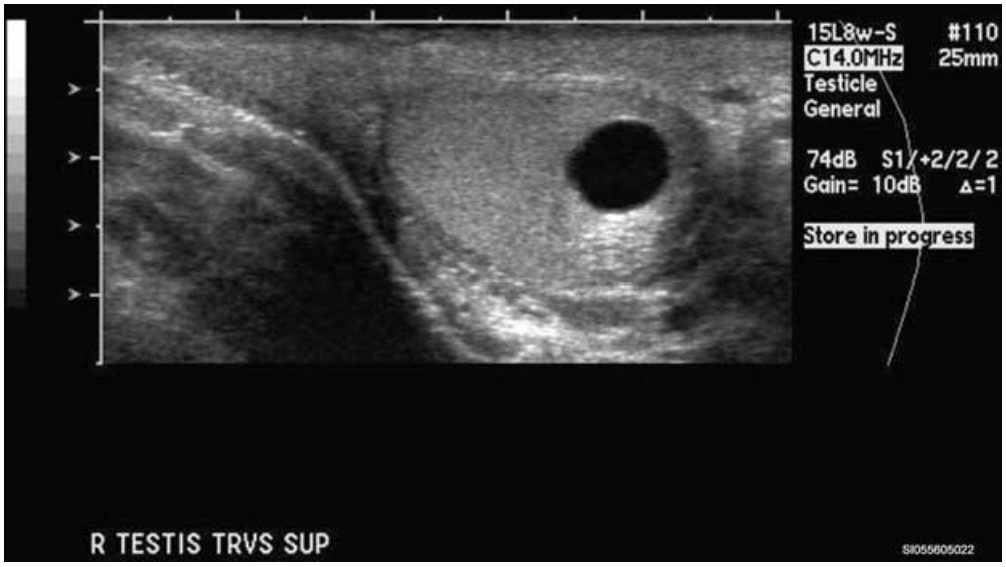

Figure 3–15. Transverse intratesticular cyst.

Ectasia of the rete testis

Occasionally, ectasia (dilation of a tubular structure) of the rete testis will appear on sonography, resembling a collection of cysts in the region of the mediastinum. In this condition, tubules of the rete testis network (normally unseen during sonography) are dilated. On sonography, the ectasia may be seen bilaterally as clusters of anechoic areas of varying sizes, but most are very small. You may also see a spermatocele (another type of anechoic cyst discussed below) in the epididymis on the same side (ipsilateral). Make sure that you use color Doppler over the area to ensure that it is not a vascular problem. Measuring each individual anechoic area is unnecessary; however, some radiologist may want to see the entire ectatic rete testis region measured in length, AP, and width dimensions.

Epidermoid cyst

Another type of cystic structure that may appear in the testes is an epidermoid cyst. They can have a predominately cystic appearance; however, the walls will be thick and echogenic. Most are 1 to 3 cm in diameter and appear singularly in one testis.

On sonography, an epidermoid cyst resembles a solid tumor. For example, the epidermoid will be hypoechoic with a hyperechoic and thickened rim, giving it a solid appearance. At times, a calcification will be seen within the center, but most calcifications will appear within the thick and echogenic rim. For this reason, sonographers generally do not consider this to be a cyst but rather a solid tumor with some cystic characteristics. Because of its atypical appearance (for a cyst) that cannot be distinguished from some teratomas on sonography, most radiologists will be suspicious that an epidermoid is a malignant mass. Your job as a sonographer is to measure and document longitudinal and transverse images of the epidermoid.

Imaging malignant testes abnormalities

Sonography provides a highly effective way to detect tumors of the testes. However, sonography cannot determine if a lesion is malignant or benign. Because most tumors of the testes are germ cell tumors, and most germ cells tumors are malignant, sonographic detection is nearly synonymous with diagnosis. For this reason, most institutions will surgically remove either the tumor (orchiotomy) or the testis (orchiectomy) when any mass is detected.

Thus, for the sonographer, merely detecting masses in the testes is the primary goal. However, sonography can also provide a physician with clues as to the nature of a testicular mass, as types of malignant tumors have differences in aggressiveness. The level of tumor aggressiveness can affect determining the potential for tumor spread (metastasis), which may become important for scheduling timelines of radiation or chemical therapeutic treatment and anticipating outcomes of treatment (prognosis). Although tissue samples serve to confirm malignant type, sonography can cause physicians to plan treatment and to look elsewhere in the body—through other diagnostic tests—in search of metastatic sites.

Seminoma

The sonographic appearance of the classic seminoma is a hypoechoic and homogeneous mass in the testis. The tumor can have either well-defined borders or poorly defined margins. As with most types of tumors in the testes, if the tumor is greater than 1.5 centimeters, you will likely find increased vascularity. Measure the length, width, and AP diameter of the mass and document the color Doppler image.

Embryonal carcinoma

On sonography, you should see a large heterogeneous mass with irregular borders that are poorly defined. You may see the contour of the testis as distorted if the tumor expands into the tunica albuginea. Because of necrotic material and hemorrhaging you may see cystic areas. Do not be surprised to see calcifications within the tumor. Embryonal carcinomas, especially because most are mixed, share the complex sonographic appearance of choriocarcinomas and some teratomas. Because embryonal tumors aggressively metastasize via the lymph channels, scanning for enlarged lymph nodes aids in diagnosis. Aside from measuring and documenting a suspected embryonal carcinoma and any color Doppler characteristics, you may find it useful to examine the area around the abdominal aorta and near the kidneys for the presence of lymph nodes. This becomes especially necessary for patients complaining of back pain. Transverse views of the abdominal aorta with a wide enough view should pick out abnormal nodes. Late-stage tumors can spread to the lung and liver.

Teratoma

Sonographically, the teratoma is identical to other non-seminoma germ cell tumors such as the embryonal carcinoma. However, the calcifications or echogenic foci are particularly dense with shadowing, largely due to presence of bone and cartilage. Some physicians consider the epidermoid cyst to be a variant of a teratoma and can also have dense echogenic areas.

Choriocarcinoma

As with other non-seminomatous germ cell tumors, the appearance of the choriocarcinoma on sonography is a heterogeneous mass with cystic areas representing hemorrhaging and necrosis. Calcifications are frequently seen. Choriocarcinomas possess irregular and ill-defined borders. Again, this tumor is usually smaller than other testicular tumors. Measure and document as you would any mass within the testis.

Imaging other testes abnormalities

Other abnormalities are immediately noticeable on sonography such as tumorless calcifications. Still other focal abnormalities may be malignant masses spread from elsewhere in the body.

Gonadal stromal tumors

When seen on sonography, these tumors are homogenous and hypoechoic, with well-defined margins. The larger stromal tumors may show cystic areas from hemorrhage and necrosis and may be heterogeneous. Because these tumors cannot be sonographically distinguished from malignant germ cell tumors, they are usually surgically removed. Removal usually cures abnormal hormonal effects as well.

Microlithiasis

Microlithiasis should not be confused with the appearances of isolated groups of calcifications that occur with past infections, inflammation, or trauma.

Microlithiasis has been sonographically classified as one of two types: *diffuse* and *limited*. Depending upon the radiologists' views about malignancy association, microlithiasis cases will fall into either of these two groups.

Diffuse cases are the appearance of five or more tiny calcifications in one sonographic image of the testis. Limited cases are less than five per image. Radiologists who follow these divisions tend to be less suspicious of limited cases. If concerned, radiologists may recommend annual sonographic follow-up. Conversely, less concerned radiologists may simply recommend annual physical or self-examinations whenever any microliths appear, whatever the number.

On sonography, the typical appearance of microlithiasis is of many hyperechoic foci dispersed throughout the testis. It can be bilateral or unilateral. Each focus is usually no greater than 1 mm in diameter and presents no shadows on the sonographic image. However, diameters of up to 3 mm have been reported. Imaging specific testicular locations of microliths is important in limited cases. Make sure that for every longitudinal image of limited microliths, you try to include a corresponding transverse view. Measuring these calcifications is usually unnecessary unless required by your radiologist. For diffuse cases, a few representative longitudinal and transverse images are all that is needed.

Metastases

Generally, metastatic tumors are indistinguishable from primary germ cell tumors. On sonography, most malignant testicular lymphomas are hypoechoic but can range all the way to

hyperechoic or a mixture of both. The tumor is usually diffuse with ill-defined borders. The appearance of leukemic involvement of the testes is nearly identical to lymphoma. However, chronic lymphocytic leukemia may appear nodular with well-defined margins.

Imaging scrotal abnormalities

The contents of the scrotum apart from the testes and the epididymides are usually limited to a small amount of fluid. Anything else should be considered abnormal. Frequently, the appearance of an abnormal scrotum can confuse a sonographer, making distinguishing between intratesticular and extratesticular pathology difficult. Care is required during scanning.

Hydrocele

On sonography, anechoic fluid surrounds the epididymis and testis anteriorly and laterally (fig. 3–16). The testis will be attached to the scrotal wall posteriorly. The hydrocele may be unilateral of bilateral. Occasionally you will see low-level echoes, which represent cholesterol crystals. For extremely large hydroceles, some radiologists will have you measure the length, AP, and width of the fluid.

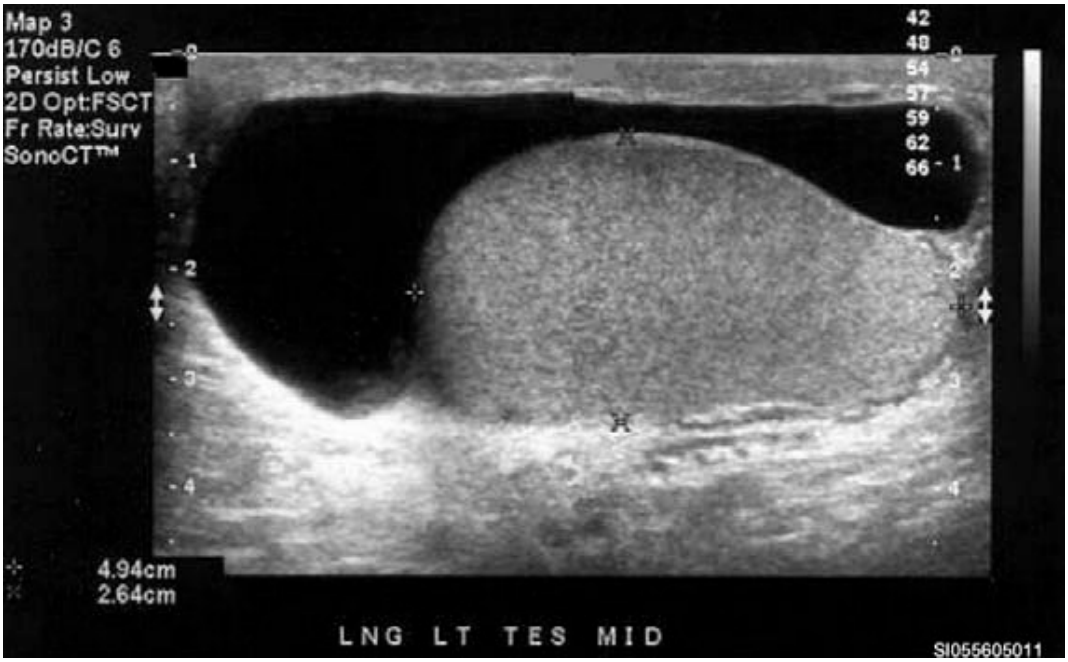

Figure 3–16. Scrotal hydrocele.

Spermatocele and epididymal cyst

Normally, sonographers distinguish spermatoceles from epididymal cysts by noting the appearance of low-level echoes within spermatoceles. This rarely occurs within the rare epididymal cyst but is a typical characteristic of the spermatocele. The anechoic nature and its appearance mostly within the body and tail help the sonographer identify a potential epididymal cyst. Scan for spermatoceles mostly in the epididymal head. Most measure a few millimeters in diameter but can grow up to several centimeters.

Varicocele

On sonography, the obvious sign is of dilated tortuous veins beside the upper pole of the testis near the head of the epididymis or posteriorly (fig. 3–17). The veins will be generally dilated 2 mm or more. A good way for you to confirm this is to measure the AP diameter of a few veins with the patient supine. Then have the patient hold his or her breath and tighten his or her abdominal muscles (this action is called the Valsalva maneuver). If the diameter dilates, the condition is a varicocele. A better way to demonstrate this is to display the increased reverse flow that causes the veins to dilate. Use color Doppler over the veins to achieve this. A quick method that is usually convincing to a radiologist is to document dual color images—one without Valsalva (fig. 3–18) and one with Valsalva (fig. 3–19). However, lack of Valsalva dilation does not, by itself, rule out the presence of a secondary varicocele.

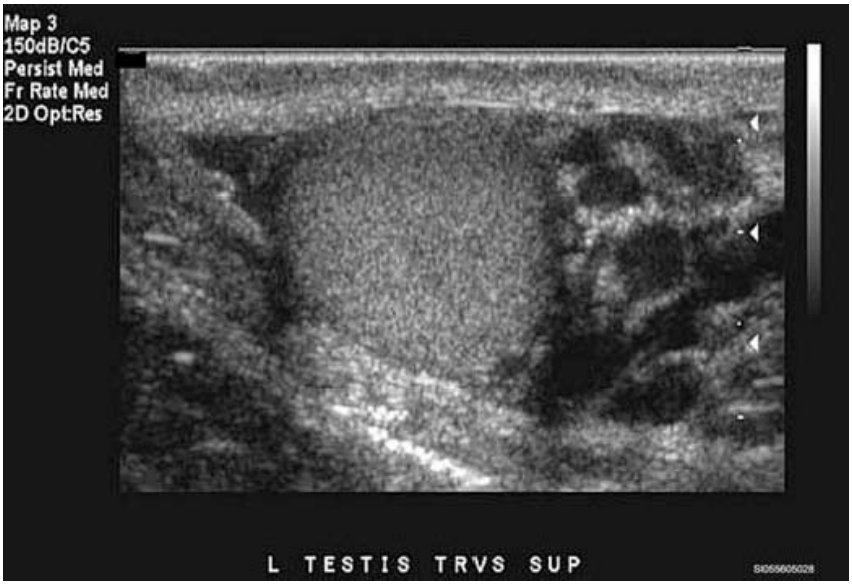

Figure 3–17. Varicocele.

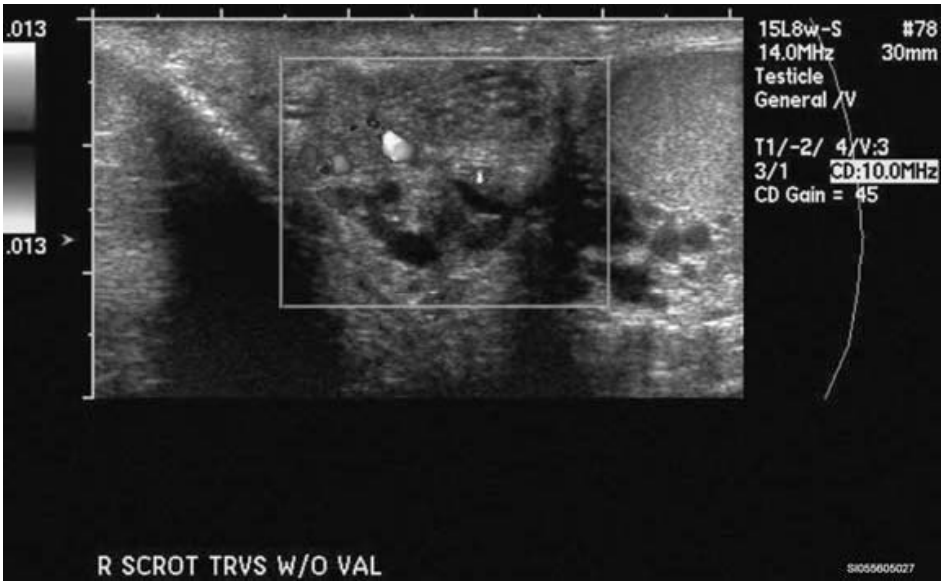

Figure 3–18. Varicocele evaluation without Valsalva

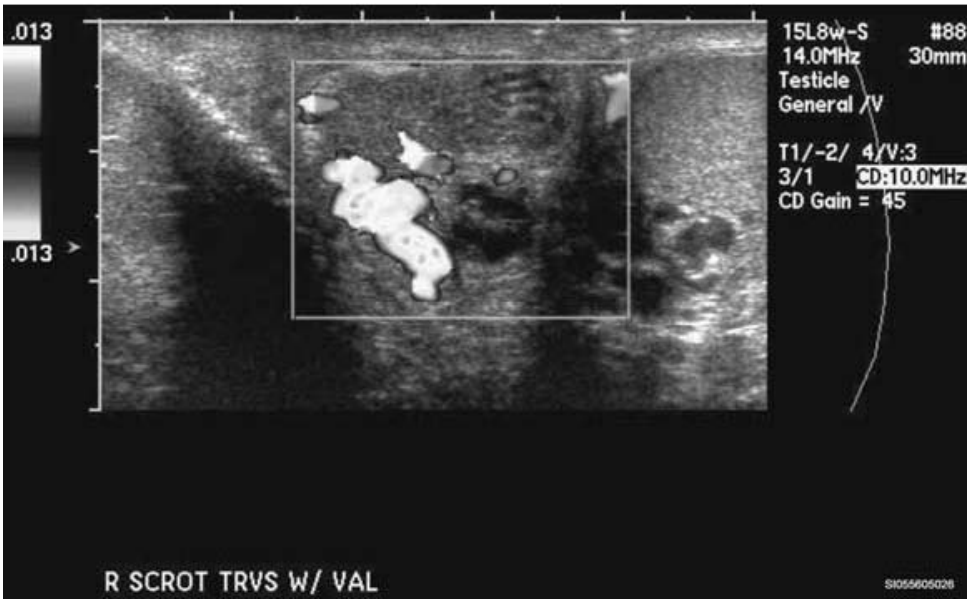

Figure 3–19. Varicocele evaluation with Valsalva.

Epididymitis

On sonography, the classic appearance of epididymitis is of a hypoechoic, coarse, and thickened epididymis. Epididymal thickness is typically easy to see in infection cases because of the significant increase in size of the body and tail from normal diameters of less than 4 mm. However, to confirm the presence of epididymitis further, it is advantageous to employ the use of color Doppler. Color Doppler should demonstrate increased blood flow in and around the epididymis, which normally shows very little high flow states.

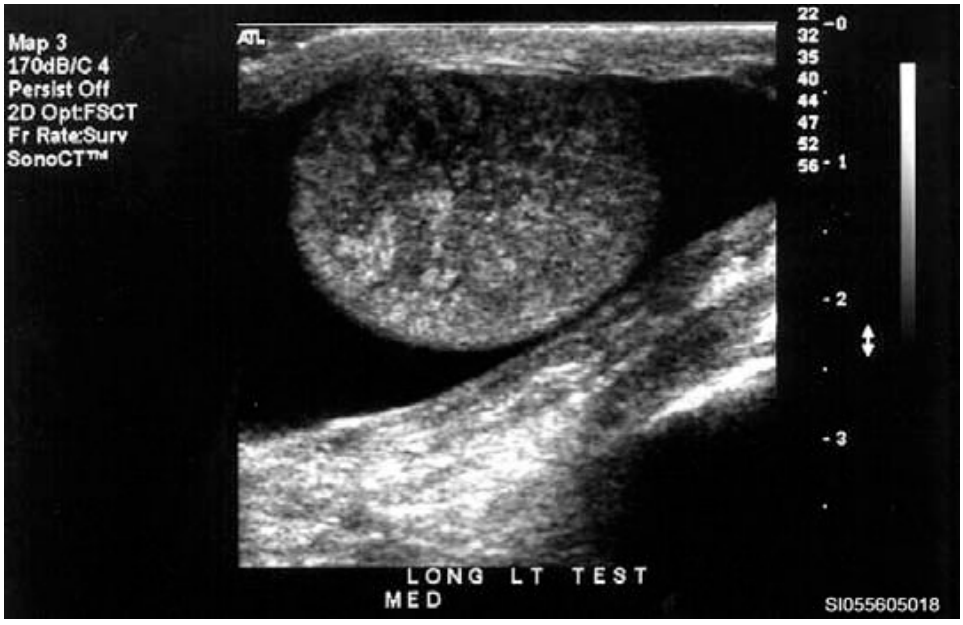

Figure 3–20. Epididymo-ochitis.

Other abnormalities seen may be a thickening of the scrotal wall or skin beyond 7 mm, an increase in fluid between the layers of the tunica vaginalis (reactive hydrocele), and diffuse or

focal spread of infection within the testis (epididymo-orchitis). Epididymo-orchitis can be seen as a hypoechoic testis with heterogeneous echotexture (fig. 3–20). The testis may be enlarged. Focal hypoechoic areas may occur instead of the diffuse variety so a careful scan through the entire testis is in order. It is also a good idea to image both testes in a transverse view with color Doppler. Most institutions will require color Doppler of both testes and epididymides to distinguish between the increased vascularity of epididymo-orchitis and the absent vascularity of torsion of the spermatic cord. This method will demonstrate the increased vascularity of epididymo-orchitis either unilaterally or bilaterally.

Torsion

The time constraint between onset of pain and surgery makes your sonographic scanning critical. In a lot of institutions, set protocols for torsion are modified in these emergencies. What concerns the radiologist and the referring physician most is determining if acute pain of a patient is caused by torsion or epididymitis. The fastest way to determine torsion of the testis is to use color Doppler immediately. Using color Doppler to demonstrate the absence of blood flow within the testis is considered diagnostic of torsion and significantly distinguishes this abnormality from epididymitis. Grayscale imaging is helpful but is not specifically tailored to distinguish torsion from any other abnormality. For example, the grayscale image may show a normal appearance, or a heterogeneous and hypoechoic parenchyma similar to other abnormalities such as diffuse seminoma, orchitis, or lymphoma. Usually, the only grayscale characteristic of the testis pointing to torsion is enlargement.

Blood flow demonstrated on Doppler does not rule out a partial torsion or a spontaneous resolution of torsion. Because venous flow within the testis will be compromised first, you may still encounter arterial signals, depending on the degree of turns. Also, if the torsion suddenly resolves before you scan, you may see increased blood flow beyond the amount seen in the normal testis. This might cause you to classify the abnormality as orchitis. Checking the white blood cell count from the patient’s laboratory values should clear up this error, as infections will cause an elevation.

Scanning torsion or an inflamed scrotum can be difficult due to the extreme tenderness of the skin. To minimize the amount of time it takes you to acquire the necessary images, ensure that your color and spectral Doppler settings are optimized to detect the low flow states typical of the testes and epididymis. Using power Doppler is an excellent but additional method to determine slow flow, preventing you from incorrectly declaring torsion of a testis. A color image of both testes in the transverse view may clarify the sensitivity of your equipment. Although the bell clapper deformity can predispose torsion bilaterally, the event usually occurs unilaterally. Thus, an image of the both testes using color Doppler should show normal flow in one and the absence of flow in the other. A few longitudinal and transverse images with color and spectral Doppler samples of blood flow, or lack of, are essential. Attempt to obtain spectral imaging of both venous (fig. 3–21) and arterial flow (fig. 3–22). If there is time, measure the dimensions of both testes for size comparison. Just as with epididymitis, you may see a reactive hydrocele surrounding the testis. A hydrocele can be helpful in imaging a spiral or coiled spermatic cord at the superior pole of the testis. Use color Doppler to check for flow within the cord. Also, a hydrocele can help you demonstrate the testis unattached to scrotal wall, confirming the bell clapper deformity.

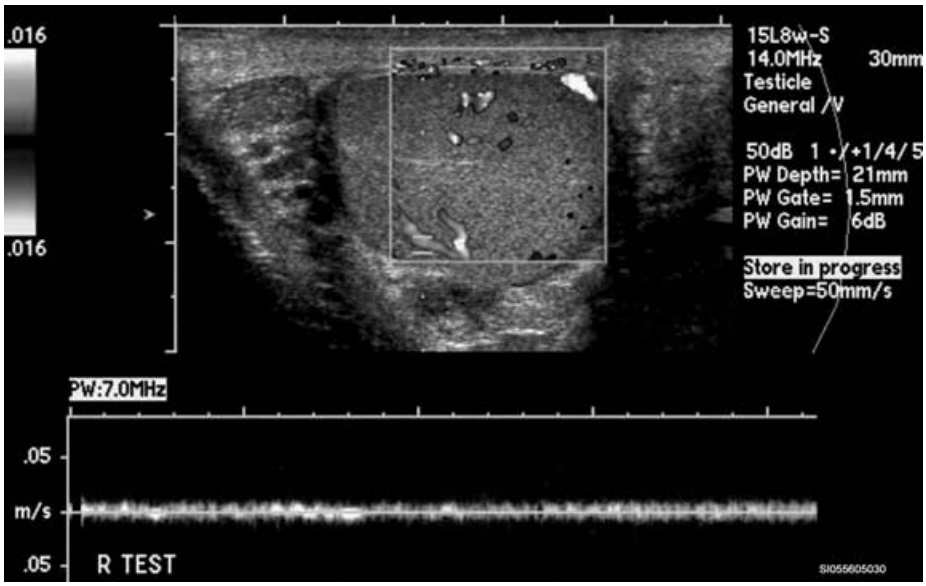

Figure 3–21. Venous flow in the testis on spectral Doppler.

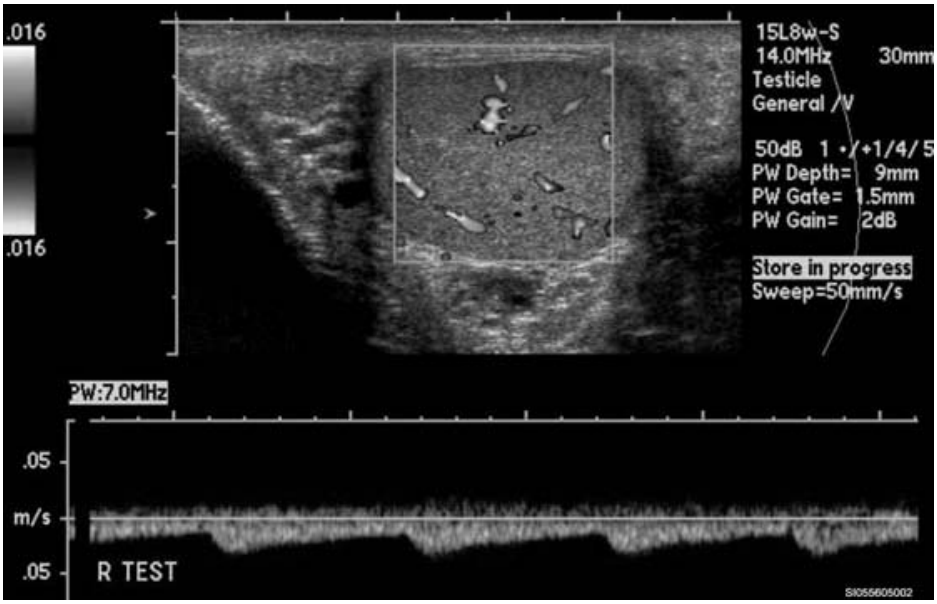

Figure 3–22. Arterial flow in the testis on spectral Doppler.

Trauma

Another cause of acute scrotal pain is trauma. Blunt trauma is a forceful blow against the scrotum such that the testis is injured. The main concern for physicians is whether the testis has ruptured, which requires immediate surgical repair if it is to be saved.

The first step for the sonographer to determine if the testis has ruptured is to focus on any interruption of the tunica albuginea. Sonographically, the ruptured testis will have a heterogeneous echotexture with an irregular margin, represented by discontinuation of the tunica albuginea. Testis parenchyma will be protruding out into the scrotum.

Other abnormalities that occur with trauma may be the appearance of collections of blood either outside the testis (hematocele) or within the testis (hematoma). The age of the collection should determine its sonographic appearance. For instance, early hematoceles are usually anechoic and can simulate reactive hydroceles. As they age, the collections become more echogenic with septations. Also, trauma may induce the cremasteric muscles to contract enough to cause torsion spontaneously. Use color flow to see if trauma to the testis has interrupted blood flow.

Scrotal hernia

Sonographically, a classic scrotal hernia is diagnosed when echogenic bowel loops in motion (peristalsis) are seen within the scrotal sac. Occasionally, hyperechoic foci will be seen with dirty shadowing, which represents bowel gas. Sometimes no bowel is seen; only an echogenic mass which represents omental fat. The absence of peristalsis makes the omental herniation appear similar to pyoceles and hematoceles.

You should use color to verify blood supply in the bowel wall, as sometimes the inguinal canal may clamp off flow. You should also document sequential images of most of the scrotal mass. This demonstrates the changing appearance of the bowel with peristalsis. A very good way to confirm herniation, particularly in the presence of just omental fat, is to use the Valsalva maneuver (again, instruct the patients to tighten their stomach muscles and to hold their breath), which should expand the size of abdominal contents within scrotum. The scrotal hernia is easier to see with the presence of reactive hydroceles, which may sometimes occur.

Undescended testis

On sonography, infants will demonstrate a normal appearing testis approximately 1 cm in diameter lodged within the inguinal canal. The rare case in adults will probably show an atrophic testis. Because a fourth of undescended testis cases are bilateral, you should also scan the opposite inguinal canal even if a testis is seen within the sac (in which case, the patient would actually have three testes, or polyorchidism).

Self-Test Questions

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

218. Testicular anatomy and physiology

1. List the scrotal fasciae.

2. What is the difference between tunica vaginalis and tunica albuginea?

3. What is the relationship between the tunica albuginea and the separate lobules of the testis?

4. Where in the testis does the production of sperm begin?

5. Aside from promoting male characteristics, what is another function of testosterone?

219. Testicular pathology

1. What is ectasia of the rete testis?

2. What is the most common germ-cell tumor in the adult?

3. What is the difference between immature and mature teratoma?

4. What is the rarest germ-cell tumor?

5. Apart from potential malignancy, list some of the associations for microlithiasis.

6. What are the two most common secondary malignant neoplasms of the testes?

7. What is indicated by the presence of pain accompanied with fluid surrounding the testis?

8. Describe the difference between primary varicoceles and secondary varicoceles.

9. What are the symptoms of acute epididymitis?

10. What are the classifications of torsion severity?

11. List the testis salvage rates for acute torsion.

220. Imaging normal and abnormal scrotum/testes

1. Why is evaluation of acute scrotal pain accomplished?

2. What sonographic landmark identifies the posterolateral portion of the testis?

3. What standard images should sonography of the testes contain?

4. What are the classic sonographic criteria for simple cysts?

5. What should you do to ensure you are not examining a cross-section of a surface blood vessel during a sonographic examination of the testis?

6. Describe the sonographic appearance of an epidermoid cyst.

7. Why will most institutions surgically remove either tumor or testis when any mass sonographically detected in the testis?

8. Describe the classic sonographic appearance of testicular seminoma.

9. What is the purpose in scanning for enlarged lymph nodes during a case of embryonal carcinoma of the testis?

10. Describe the sonographic description of gonadal stromal tumors.

11. Where should you scan for spermatoceles?

12. Describe the sonographic appearance of epididymo-orchitis.

13. Describe the sonographic appearance of a scrotal hernia.

Answers to Self-Test Questions

215

1. Endocrine glands secrete into bloodstream; exocrine glands secrete into ducts.
2. Carotid arteries and internal jugular veins.
3. Pyramidal lobe.
4. Follicular and parafollicular cells.
5. Superior and inferior thyroid arteries.
6. A pair is embedded into the posterior surface of the thyroid at the superior end, and a pair is embedded into the posterior surface at the inferior end.
7. To enhance the metabolism of nearly all tissues in the body.
8. (1) Increase the rate that cells consume or use oxygen while the body is at rest (basal metabolic rate).
9. Raise body temperature.
10. Stimulate the manufacturing or synthesis of proteins.
11. Accelerate tissue growth in young people.
12. Increase blood flow and respiration.
13. Stimulate the central nervous system.
14. Increase secretion rates of most other endocrine glands.
15. Only gland in the body that stores its hormones in large quantities (usually a two to three month supply) before release.
16. Causes the release of calcium from bone into the blood circulation.

216

1. Thyrotoxicosis. Occasionally, a small percentage of follicular adenomas will produce thyroid hormones independent of the thyroid's normal control. This situation is known as hyperfunction or autonomy. Among the autonomous follicular adenomas, some will produce excessive quantities of thyroid hormones, a condition known as thyrotoxicosis.
2. Medullary carcinoma.
3. The neck mass is likely an anaplastic carcinoma which can compress or invade the trachea, causing death by asphyxiation.
4. Hashimoto thyroiditis (chronic autoimmune lymphocytic thyroiditis).
5. Autoimmune process that interrupts the production of thyroid hormones.
6. Middle-aged women.
7. Excessive calcium in the blood and urine.
8. (1) Single adenoma (glandular tumor of the parathyroid).
9. Multiple-gland enlargement (multiple adenomas).
10. Carcinoma.
11. Chronic renal failure and vitamin D deficiency (rickets).

217

1. (1) A neck mass is palpable.
   (2) Nuclear medicine studies reveal abnormal uptake of radioactive iodine.
   (3) Patients at risk for occult (clinically unidentified or hidden) thyroid malignancy.
   (4) Pre-surgical evaluation.
   (5) Follow-up on patients with proven malignancy.
   (6) Follow-up on patients after thyroid surgery.

2. In cases where patients have thyroid glands with lengths that stretch behind the sternum, having them swallow and hold shifts the entire thyroid up toward the brain and into view on the sonography screen.
3. If it contains a mass or appears enlarged.
4. Both types of thyroid masses have similar sonographic appearances.
5. A thick and irregular hypoechoic halo around the periphery; fine microcalcifications clustered together.
6. Isoechoic relative to the surrounding thyroid tissue, with a thin hypoechoic halo; occasional cystic areas or spaces, which represent degeneration and necrosis; hyperechoic foci with comet-tail artifacts may appear within the cystic areas, representing dense colloid; calcification around the nodule in an eggshell appearance or scattered throughout the nodule.
7. A solitary, hypoechoic mass of varying sizes and an irregular border. Approximately a fourth of papillary carcinomas also show fine microcalcifications called psammoma bodies.
8. Encapsulated on sonography.
9. Typical or reduced blood flow.

218

1. (1) Dartos.
   (2) Cremasteric.
   (3) External spermatic.
   (4) Internal spermatic.
2. The tunica vaginalis is made of two layers of serous tissue and the tunica albuginea is made of a single thick and fibrous membrane.
3. The tunica albuginea tissue forms the fibrous walls of each lobule.
4. The seminiferous tubules.
5. Influences the development of the early spermatogenic cells.

219

1. Dilated tubules of the rete testis network.
2. Seminoma.
3. Immature is benign and mature is malignant.
4. Choriocarcinoma.
5. Infertility or reduced fertility (subfertility), Klinefelter syndrome, Down syndrome, cryptorchidism, and pulmonary alveolar microlithiasis (calcifications in the alveoli of the lungs).
6. Lymphoma and leukemia.
7. Pyocele.
8. Primary varicoceles are the most common and occur mostly in adolescents or young men. The cause is attributed to absent or improperly functioning (incompetent) valves within the internal spermatic vein. Most cases of primary varicoceles occur on the left side where the internal spermatic vein drains into the left renal vein at a 90-degree angle. The secondary varicocele is caused by external pressure on the internal spermatic vein. Varicoceles that occur suddenly on the right side or bilaterally, particularly ones that are unaffected in diameter with a change in patient positioning, should raise the suspicion of compression of the internal spermatic vein. Enlarged liver, renal or other abdominal masses, lymphadenopathy, or even a tortuous superior mesenteric vein compressing the left renal vein, can cause retrograde flow within the internal spermatic veins and resulting in varicoceles.
9. Sudden intense pain in the scrotum, usually accompanied by fever and extremely painful urination. The scrotal wall will be thickened and extremely painful when touched.
10. (1) Acute: occurring within 24 hours.
    (2) Subacute: within 1–10 days.
    (3) Chronic: in existence beyond 10 days.

11. (1) 5–6 hours: 80 percent–100 percent.
    (2) 6–12 hours: 70 percent.
    (3) > 12 hours: 20 percent.
    (4) > 24 hours: unsalvageable.

220

1. To determine if the root cause is inflammation or torsion.
2. Mediastinum testis.
3. Longitudinal images of the medial, middle, and lateral portions of the testis; measurements of the length and AP diameter of the middle portion of the testis; transverse images of the superior, middle, and inferior poles of the testis; at the widest part of the middle portion, the width measurement should be taken.
4. Well-defined margins, thin walls, anechoic centers, and enhanced posterior acoustic transmission.
5. Turn your transducer 90 degrees to see if the lumen lengthens out.
6. Hypoechoic with a hyperechoic and thickened rim, giving it a solid appearance. At times, a calcification will be seen within the center, but most calcifications will appear within the thick and echogenic rim.
7. Because most tumors of the testes are germ cell tumors, and most germ cells tumors are malignant, sonographic detection is nearly synonymous with diagnosis.
8. A hypoechoic and homogeneous mass in the testis. The tumor can have either well-defined borders or poorly defined margins.
9. Embryonal tumors aggressively metastasize via the lymph channels.
10. Usually small but can become quite large, filling an entire testis; homogenous and hypoechoic, with well-defined margins. The larger stromal tumors may show cystic areas from hemorrhage and necrosis and may be heterogeneous.
11. Epididymal head.
12. A hypoechoic testis with heterogeneous echotexture. The testis may be enlarged. Focal hypoechoic areas may occur instead of the diffuse variety; Doppler demonstrates increased vascularity of epididymo-orchitis either unilaterally or bilaterally.
13. Echogenic bowel loops in motion (peristalsis) are seen within the scrotal sac. Occasionally, hyperechoic foci will be seen with dirty shadowing, which represents bowel gas. Sometimes no bowel is seen; only an echogenic mass which represents omental fat.

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.

53. (215) The lobes of the thyroid gland are located to each side of which structure?
    a. Esophagus.
    b. Trachea.
    c. Carotid.
    d. Jugular.

54. (215) What is the difference in thyroid lobes between tall/thin people and short/large people?
    a. Tall/thin people have shorter lobe lengths.
    b. Tall/thin people have longer lobe lengths.
    c. Short/large people have smaller lobes.
    d. Short/large people have larger lobes.

55. (215) The thyrotropin-releasing hormone causes the
    a. thyroid gland to release thyroid-stimulating hormone (TSH).
    b. thyroid gland to release thyrotropin-releasing hormone (TRH).
    c. anterior pituitary to release TSH.
    d. anterior pituitary to release TRH.

56. (216) In areas where iodine deficiency is uncommon, what is the relationship of hypothyroidism and Hashimoto thyroiditis?
    a. They are identical.
    b. No relationship.
    c. Hashimoto thyroiditis is the least common cause of hypothyroidism.
    d. Hashimoto thyroiditis is the most common cause of hypothyroidism.

57. (216) What *major* feature differentiates de Quervain thyroiditis from Hashimoto thyroiditis?
    a. Larger gland.
    b. Fever and pain.
    c. Larger nodules.
    d. Elevated calcium levels.

58. (216) What dangerous feature does Riedel thyroiditis share with anaplastic carcinoma?
    a. Extends to lymph nodes.
    b. Compresses the trachea.
    c. Increases pain in throat.
    d. Metastasizes to brain.

59. (217) Of the following actions, which one reflects the relationship between using color Doppler and revealing blood flow within an anechoic linear structure coursing through a thyroid lobe?
    a. Increase gain.
    b. Have patient exhale.
    c. Adjust patient’s neck.
    d. Adjust angle and lower filter.

60. (217) Why would you obtain a transverse view of both thyroid lobes where the isthmus crosses over the trachea?
    a. For comparison of echotexture.
    b. To obtain better isthmus measurement.
    c. To evaluate the length of the thyroid gland.
    d. For a better view of the trachea or esophagus.

61. (217) You would use a sonography in thyroid nodule cases because sonographic clues tend to suggest
    a. malignancy.
    b. metastasis.
    c. normality.
    d. benignity.

62. (217) If you see any of the typical sonographic features of papillary carcinoma of the thyroid, you should look around the nearby neck region for
    a. enlarged lymph nodes.
    b. carotid abnormalities.
    c. follicular carcinoma.
    d. multinodular goiter.

63. (217) Why is Hashimoto thyroiditis sometimes confused with multinodular goiter on sonography?
    a. Multiple hypoechoic areas resemble nodules.
    b. Both diseases secrete thyroxine autonomously.
    c. Both diseases secrete calcitonin autonomously.
    d. Identical vascular appearance on color Doppler.

64. (217) What is a good way to distinguish Graves' disease from Hashimoto thyroiditis on sonography?
    a. Use color Doppler.
    b. Decrease overall gain.
    c. Increase the transducer frequency.
    d. Adjust the pulse repetition frequency.

65. (217) If one parathyroid gland is affected with adenoma, the other three become
    a. atrophic.
    b. enlarged.
    c. inflamed.
    d. malignant.

66. (217) What condition can be confused with *primary* parathyroid hyperplasia?
    a. Multiple endocrine neoplasia (MEN), type 2.
    b. Multiple adenomas.
    c. Secondary hyperplasia.
    d. Primary hyperparathyroidism.

67. (218) What happens to the size of the testis with increasing age?
    a. Enlarge.
    b. Diminishes.
    c. Fluctuates.
    d. Remain the same.

68. (218) How does seminal fluid travel from the ductus epididymidis to the ductus deferens?
    a. Osmosis.
    b. Peristalsis.
    c. Absorption.
    d. Spermatogenesis.

69. (219) What distinguishes intratesticular cysts from tunica albuginea cysts?
    a. Tunica vaginalis surrounds tunica cysts.
    b. Epididymis parenchyma surrounds tunica cysts.
    c. Tunica albuginea surrounds intratesticular cysts.
    d. Testis parenchyma surrounds intratesticular cysts.

70. (219) Which germ-cell tumor of the testis is the most aggressive?
    a. Choriocarcinoma.
    b. Immature teratoma.
    c. Embryonal carcinoma.
    d. Spermatocytic seminoma.

71. (219) What is the difference between a hematocele and a pyocele?
    a. Hematocele contains serous fluid and a pyocele contains blood.
    b. Pyocele contains serous fluid and a hematocele contains blood.
    c. Hematocele contains serous fluid and a pyocele contains pus.
    d. Pyocele contains pus and hematocele contains blood.

72. (219) What makes a pyocele clinically different from a hydrocele?
    a. Pain.
    b. Fainting.
    c. Weight loss.
    d. Hypertension.

73. (220) The *primary* reason for sonographic evaluation of acute scrotal pain is to determine if the cause is inflammation or
    a. torsion.
    b. orchitis.
    c. seminoma.
    d. epididymitis.

74. (220) On sonography, what testis landmark can be used to identify the head of the epididymis?
    a. Mediastinum.
    b. Vas deferens.
    c. Tunica albuginea.
    d. Seminiferous tubules.

75. (220) What is the relationship between epidermoid cysts and solid masses within the testis?
    a. Solid masses are less numerous than epidermoid cysts.
    b. Epidermoid cysts are degenerate into solid masses.
    c. Epidermoid cysts do not resemble solid masses.
    d. Epidermoid cysts resemble solid masses.

76. (220) What is the sonographic feature that embryonal carcinomas of the testes share with choriocarcinomas and some teratomas?
    a. Solid.
    b. Complex.
    c. Hyperechoic.
    d. Homogeneous.

77. (220) The sonographic feature that distinguishes choriocarcinoma from other testis tumors is that it is
    a. larger.
    b. smaller.
    c. isoechoic.
    d. hyperechoic.

78. (220) What would color Doppler demonstrate about the blood flow in and around the epididymis in cases of epididymitis?
    a. Increased.
    b. Reversed.
    c. Reduced.
    d. Absent.

79. (220) What must you use to distinguish torsion from epididymitis?
    a. Increased gain.
    b. Color Doppler.
    c. Murphy's sign.
    d. Valsalva maneuver.

80. (220) Why would you use the Valsalva maneuver in a case of suspected scrotal hernia?
    a. Expands the size of abdominal contents in the scrotum.
    b. Reduces the size of abdominal contents in the scrotum.
    c. Expands the size of the epididymis in the scrotum.
    d. Reduces the size of the rete testis in the scrotum.

81. (220) When you scan the opposite inguinal canal in cases of undescended testis, what percentage of these cases are bilateral?
    a. 10 percent.
    b. 15 percent.
    c. 20 percent.
    d. 25 percent.

Please read the menu for Unit 4 and begin. ➔

Unit 4. Other Sonography Imaging

MOST OF THE facilities you will be working in will have a mammography staff as part of the diagnostic imaging team. You will be called upon to assist the mammographic section in their diagnosis of a host of breast problems. Where you fit in will be to clarify a problem for the radiologist to make a proper diagnosis. In addition, sonography is increasingly being used for purposes beyond traditional body imaging of structures such as the breast and abdomen. Because of improvements in resolution, minute detail is more readily visible than was possible even five years ago. In this unit, we will cover breast imaging and briefly describe some of the uses of sonography in specialized roles—most of which have been with us for a long time, but are being used more frequently.

4–1. Breast Imaging

To be an effective member of this diagnostic team requires familiarity with breast anatomy and physiology. An understanding of the sonographic appearance of breast structures will help you to correlate your findings with those of your mammography partners. Finally, your knowledge of the various breast abnormalities and their sonographic characteristics will enable you to recognize a range of masses efficiently; both benign and malignant. In this section, we will focus on the sonographic approach to breast imaging, and by doing so you will better assist the radiologist as he or she combines mammographic reporting in his or her diagnosis.

221. Breast anatomy and physiology

Most of the female breast is functional or glandular tissue (parenchymal) with stromal or supporting tissue providing a frame. The anatomy of the breast is laid out in such a way as to point to its primary focus for providing milk to a nursing infant. Thus, glands and ducts throughout the breast all point toward the nipple.

Breast anatomy

The female has two collections of breast tissue situated anterior to and covering the chest muscles on the left and right. Size and shapes of breasts vary from woman to woman and frequently within the same woman, depending on age, genetic factors, and individual hormone influence.

From the outer skin down deep to the chest wall, all ducts of the breast are arranged in a concentric circle, like spokes in a wheel, pointing to the central axis of the nipple. It is helpful to

think of the breast as being divided into three layers or zones, each going deeper toward the chest wall:

1. Premammary zone (subcutaneous layer).
2. Mammary zone (mammary layer).
3. Retromammary zone (retromammary layer).

The subcutaneous layer, or premammary zone, is composed of skin, connective tissue, and fat, all being subcutaneous tissue similar to that elsewhere in the body. In menstruating women, this region is thin relative to the mammary layer beneath, and in older women, it is relatively thick. The outer skin displays the nipple in the anatomical center of the breast surrounded by a circular region of varying sizes and pigments called the areola of breast. The areola surface has numerous tiny projections that serve as outlets for the areolar glands (sometimes called Montgomery glands) beneath. Below the skin are fat lobules separated by suspensory ligaments of breast, fibrous connective tissue commonly referred to as Cooper ligaments (you'll often hear the possessive, *Cooper's*). The Cooper ligaments hold up the breast tissue, giving it much of its shape and stretch from the skin down through the subcutaneous layer with its fat lobules to the anterior surface of the mammary zone. Cooper ligaments also frequently form a network all the way through the layers to the chest wall.

The anterior surface below the premammary zone is actually thick connective fascia and separates the premammary zone from the mammary zone. The second layer, the mammary zone, is the thickest layer and contains the functional or glandular tissue of the breast. This layer is divided into at least 15 lobes, each with ducts and hundreds of lobules, and separated by dense fibrous tissue. Inside each lobule is less dense fibrous tissue that surrounds the ducts and, along with varying amounts of fat, is interspersed throughout. Also inside each of the lobules are the functional units of the breast called terminal ductolobular units (TDLU), composed of tiny sac-like glands called acini or alveoli, as well as intralobular stroma or connective tissue. Branching from the TDLU are tiny ducts called secondary tubules, pointing toward the center of breast and the nipple. Each secondary tubule departs their respective lobule and eventually merges with other secondary tubules to form one of at least 15 lactiferous ducts (also called mammary ducts) that drain the main lobes. Lactiferous ducts widen slightly into lactiferous sinuses or ampulla just before ascending up into the nipple.

Below the functional mammary layer is the posterior mammary fascia or deep fascia. Beneath the fascia is a thin layer of fat lobules called the retromammary zone. Deep into this layer lies the pectoralis major muscle of the chest.

A triangular shape sliver of breast tissue sometimes extends into the armpit in some women. This area, the axilla, has many lymph nodes, which sometimes factor into mammographic problems requiring sonographic clarification.

Breast physiology

The function of the breast (specifically, the mammary glands) is to produce and secrete milk (lactation) for the nourishment of infants after childbirth. To accomplish this, the glandular tissue is strongly influenced by hormonal forces in producing milk as well as moving milk through and out of the breast. Continuous development of the tissue varies with age and monthly menstrual cycles, all of which correspond with levels of hormone production. The important hormones are estrogen and progesterone, which increase significantly during pregnancy due to their major source, the placenta. Estrogen causes an increase in the development of the acinar and ducts in preparation for delivery. The increase in glandular tissue and activity causes the breast to swell in size. Progesterone completes the processes begun by estrogen.

At the time of birth, the placental production of estrogen and progesterone ceases and the presence in the blood of those hormones drops dramatically. The hypothalamus gland in the brain senses this reduction in estrogen and progesterone, and ceases to produce a factor that prevents the anterior pituitary gland from producing prolactin. Prolactin is a hormone that stimulates the acini to produce milk.

The milk is secreted from the TDLU into the lactiferous ducts and stored within lactiferous sinuses. When a child sucks on the nipple and draws the stored milk from the sinus, the posterior pituitary gland is stimulated to produce a hormone called oxytocin. This hormone causes the lactiferous ducts to contract and squeeze secreted milk forward and continuous lactation begins.

To prevent damage and infection to the nipple area while breastfeeding, the areolar glands (which enlarge during pregnancy) secrete a lubricant that seems to reduce chapping also.

222. Breast pathology

Clinical symptoms of patients with palpable but benign breast masses are likely limited to pain or bilateral nipple discharge. Palpable benign causes are frequently due to glandular changes of normal breast tissue, which may be tied to the menstrual cycle and its fluctuating hormone levels. These changes, such as fibrocystic changes and duct ectasia, can produce hardened lumps (sonographically echogenic glandular ridges along the anterior of the mammary layer) and are sometimes called aberrations of normal development (ANDI).

Conversely, patients with malignant breast masses tend to have symptoms of painless skin dimpling, ulcerations, nipple retraction, palpable masses that are extremely hard, and unilateral spontaneous or bloody nipple discharge.

Benign disease

Non-malignant diseases of the breast can be focal or diffuse. We will look at some of the more common occurrences.

Benign cystic disease

Simple cysts of the breast are common in middle-aged women. However, they occur less frequently in menopausal women who do not receive hormone (estrogen) therapy. They can be singular or multiple, unilaterally or bilaterally. They are asymptomatic unless very large, causing pain. In nearly all cases, simple cysts of the breast are benign.

Various conditions can cause benign complex cysts. One of the more common benign conditions is fibrocystic change or disease. Occurring mostly in young women after 20, fibrocystic change is a condition that involves the increase of fibrous stroma or connective tissue and varying degrees of cystic dilation of the terminal ducts of the breast. The terminal ducts are connected to the mammary glands or acini, located within the TDLUs. Most fibrocystic change patients complain of pain in the breast and lumpiness.

Fibroadenoma

The most common benign solid tumor of the breast in women under 35 is the fibroadenoma. Arising from the TDLU, fibroadenomas are composed of a mixture of fibrous connective tissue, epithelial or glandular tissue (acini), and ducts. The tumors are generally 2 to 4 cm and are typically singular but may be multiple. Most are asymptomatic. In adolescents, a fibroadenoma may occur that is 5 cm or more in size called a giant “juvenile” fibroadenoma, which tend to rapidly grow and cause pain. They are otherwise indistinguishable from typical fibroadenoma.

Papilloma

A papilloma is a benign epithelial tumor that projects from surrounding tissue. In breasts, the intraductal papilloma arises from the lactiferous duct, mostly in the subareolar region. Common in middle-aged women, they cause nipple discharge of either serous or bloody fluid. The carcinoma form of papilloma is rare and normally seen only in postmenopausal women.

Another benign form of papilloma is the intracystic papilloma, which arise from the TDLUs. The cysts can cause pain if they become inflamed.

Mastitis

Although other benign focal abnormalities occur within the breast, such as milk of calcium cysts, lipomas in the subcutaneous layer, and cystic collections of necrotic fat (fat necrosis), these are extremely rare. Another more common breast abnormality is mastitis, inflammation of diffuse breast tissue. The typical breast mastitis case is caused by infection. Rarely, it may be the result of prior irradiation. The infectious organism typically responsible is *Staphylococcus*. Patients commonly complain of extremely painful and tender breast either bilaterally or in a single area. Nipple discharge is also seen with mastitis cases.

Mastitis is divided up into two types: breast inflammation occurring after childbirth (puerperal) and inflammation at all other times (non-puerperal). During pregnancy, the lactiferous duct will sometimes become blocked or occluded, causing the duct to expand into a retention cyst called a galactocele. This structure, in combination with the introduction of infectious inflammation, sometimes erupts into a massive abscess (a collection of pus) within a few months of delivery. Puerperal mastitis is located close to the periphery of the breast. Occasionally certain types of mastitis will cause periductal inflammation in the subareolar region, prompting the lactiferous ducts to expand with debris. This condition is called *ductal ectasia*.

Conversely, centrally located abscesses can be both puerperal and non-puerperal. These large collections tend to possess an elongated shape, coursing roughly parallel to the duct of origin. Because mastitis can cause an inflamed duct to rupture, fluid is spilled into the breast tissue.

Malignant disease

Of the various breast cancers that can occur, by far the most common is carcinoma. Carcinomas arise from epithelial cells, which are structural cells that cover nearly every surface and organ in the body. Women who are at risk for breast carcinoma are generally provided with diagnostic mammograms coupled with sonography annually. One percent of breast cancers occur in men.

Risk Factors for Breast Cancer	
Age	Rare in women younger than 35. Mostly occur in women over 50.
Family history/ Genetics	History of breast cancer in mother, sister, daughter. Autosomal dominant.
Hormone levels and status	Early or late menopause. Women who have never given birth (nulliparous) or who become pregnant after reaching 35 years of age.
Alcohol/ chemicals/ radiation	Excessive exposure.

Two types of carcinoma occur in the breast: carcinoma *in situ* (non-invasive or non-infiltrating) and invasive (infiltrating) carcinoma.

Carcinoma in situ

Of the *non-invasive* carcinomas, the most common is intraductal carcinoma or ductal carcinoma in situ (DCIS). Arising from the TDLUs, DCIS fills and expands the mammary ducts but does not break through. Because this malignancy can develop into invasive carcinoma, biopsy is used when expanded ducts filled with hypoechoic material are seen on sonography. DCIS frequently appears as a mass extending throughout a duct.

Invasive carcinoma

Invasive ductal or infiltrating ductal carcinoma is the most common breast cancer in women. Usually, the invasive carcinoma presents clinically as a hard fixed mass in the breast. Invasive carcinoma does precisely what its name implies and spreads into the breast tissue beyond the duct of origin. Other types of invasive carcinoma are lobular carcinoma, medullary carcinoma (which resembles a cyst because of its sharp round border and homogeneously hypoechoic center), and colloid (mucinous) carcinoma. All of these types are determined after laboratory analysis of a biopsy tissue sample.

Invasive carcinoma spreads primarily through the lymphatics of the breast. The lymph channels all rise toward the nipple and converge before coursing toward the axillary lymph nodes.

223. Imaging normal and abnormal breasts

Breast sonography seems to be a simple thing to do because of the superficial nature of the tissue. However, because glandular tissue changes with age and hormonal levels, the sonographic appearance of the breast will also change. The three major layers of the breast vary in size and echogenicity to such a degree that a sonographer must have knowledge of other factors such as patient history, clinical information, and prior mammogram information. Considering these factors, along with practical experience examining normal breast tissue, will help you to locate and document masked and subtle abnormalities.

Indications

The major reason for performing breast sonography is to determine the characteristics of either palpable abnormalities or focal abnormalities seen on mammography. Frequently, sonography can prove to be useful for other reasons.

Indications for Breast Sonography	
Locate and Characterize Masses	Palpable masses felt through either clinical or self-examination and in conjunction with mammography. Nonpalpable masses seen on mammography. Initial step for women under 30 and pregnant or lactating women.
Localization and Guidance	Useful for needle biopsy and cyst aspiration of abnormal tissue, decreasing the need for surgery.
Evaluation	Breast implant problems such as leakage and rupture.
Planning	Used to help map out plans for radiation therapy of prior breast cancer patients

The main concern is breast cancer, and some women at risk will often have breast masses evaluated with both mammography and sonography. Certain symptoms are common for breast cancer and may prompt sonographic evaluation, such as advanced age, family history of breast cancer, and personal history of breast cancer. On mammography, suspicions of cancer are raised when irregular borders are seen in masses (often referred to as “spiculated”) or focal clusters of microcalcifications are discovered. Patients may also have inflammation (mastitis) or trauma.

Sonographic approach

To properly evaluate breast tissue, you will need at least a 7 MHz linear-array transducer or higher with multiple focal zones in the near-field. Most transducers with frequencies above 7 MHz have near field focusing capable of superior imaging as close as 1.5 to 2.0 cm beneath the skin surface. Breast masses are typically seen no deeper than 3 or 4 cm, and higher frequency transducers are able to render these structures with outstanding resolution. For patients with large breasts having deep breast abnormalities, either reposition patient (to flatten breast out as much as possible) or switch to a lower frequency for adequate beam penetration.

It is critical that you work closely with the radiologist to determine the best way to evaluate mammographic findings or the referring physician's concerns. The patient's medical history and prior sonographic or mammographic studies must be reviewed (preferably before you scan). Unless you are scanning a patient younger than 30 or pregnant/lactating, you should have mammograms available for review. If not, proceed with the radiologist's instructions. Requests should be specific on what breast abnormality is to be evaluated and the general location.

Patients should be dressed in such a manner where only the breast to be examined is exposed. For abnormalities located in the medial portion of the breast, a supine position for the patient is appropriate. If the abnormality is laterally situated to the nipple, roll the patient up slightly on the contralateral side (the side opposite the breast of interest) to flatten the breast out against the body. This will bring any potential masses closer to your transducer. For large breasts, roll them up into a near decubitus position. For all positions, have the patient raise the arm of the side examined above their head and resting on a support.

Sonographic appearance and standard views

In general, young women will have more dense breasts in the glandular tissue, while older women (particularly post-menopausal) will have mostly fat within the breast tissue. The three zones of the breast have distinct appearances on sonography.

Sonographic Appearance of Normal Breasts	
Premammary (subcutaneous)	Skin layer is echogenic and few millimeters thick. Fat lobules beneath are hypoechoic and this area is very thin in young women and thick in older women (fig. 4–1). Interspersed throughout the fat lobules are the highly echogenic linear bands (Cooper ligaments) stretching (on sonography) from the mammary layer to the skin surface.
Mammary	Echogenic layer. Young women: thick and contains some areas of hypoechoic fat; occasionally, hypoechoic linear structures (ducts) are seen; may be echogenic and dense from fibrous tissue. Pregnant or lactating women: Extremely thickened and echogenic with a homogeneous echotexture. Older and post-menopausal women: Thin or almost entirely replaced with hypoechoic fat; Cooper ligaments remain highly echogenic and give a polyhedral shapes to the fat lobules.
Retromammary	Hypoechoic fat lobules similar to premammary in echogenicity. Very thin layer (a few centimeters) made of fat lobules. Sits atop the linear bands of hypoechoic muscle representing the pectoralis major muscle of the chest.

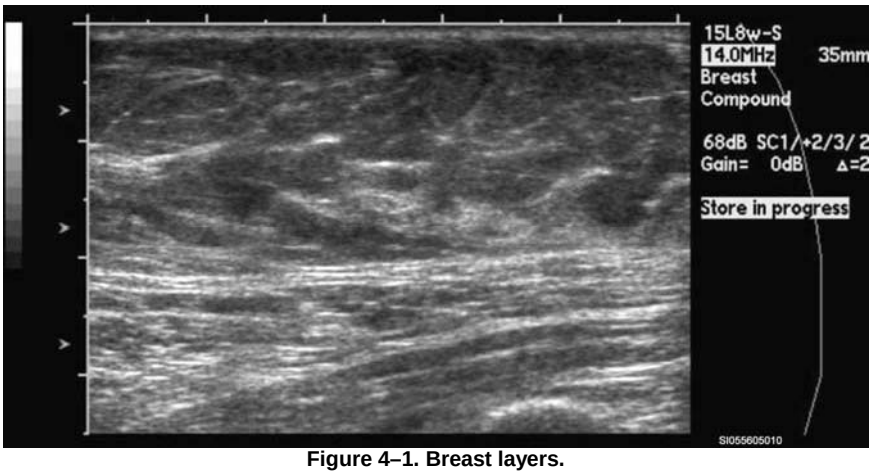

Figure 4–1. Breast layers.

The retromammary layer appears thicker on mammography because breast tissue is pulled away from the chest wall during the procedure. Conversely on sonography, the retromammary layer is compressed during the supine examination beneath the sonographer's transducer. Thus, masses that seem further out from the retromammary layer on mammography may actually appear closer to the layer with sonography. Therefore, careful scanning and correlation with mammographic location is critical.

Most sonography departments will require you to use either a quadrant method or a clock method to label location for your images. For the quadrant-method, the right and left breasts are split into four sections through the nipple.

1. Upper Outer Quadrant – RUOQ and LUOQ.
2. Lower Outer Quadrant – RLOQ and LLOQ.
3. Upper Inner Quadrant – RUIQ and LUIQ.
4. Lower Inner Quadrant – RLIQ and LLIQ.

For the clock method, you should imagine the breast as similar to the face of a clock with the center being the nipple. Directly cephalic to the nipple is the 12:00 position and it increases clockwise from 1:00 to 11:00 in even intervals around.

Frequently, you will be asked to label the orientation of a mass in relation to its location. For example, you've located a right breast mass at the 3:00 position. You would label its orientation either as longitudinal (sagittal) or transverse to the body. Some radiologists may have you label the long axis of a mass according to its orientation to the nipple. That is, the nipple has imaginary lines radiating out like wheel spokes (radial). Our 3:00 mass will be imaged and labeled as radial and, with a 90-degree rotation, antiradial. Further methods of labeling will even have you label the depth of a mass in relation to the three mammary layers (A, B, and C).

Mammographic imaging of masses is generally performed through a cranial-caudal (CC) approach through the breast (under compression) or a medial lateral oblique (MLO) with the breast pulled away from the chest wall (also under compression). Although sonography can approach breast masses from nearly all directions, it may prove helpful to try to use the same mammographic view. A transverse view anteriorly corresponds to the CC mammographic projection. Although difficult with a transducer, you may be able to come close to MLO view (remember the breast is pulled away during the mammogram) by doing a longitudinal view along the lateral edge of the breast, coronal with the body. Again, use the quadrant or clock method to annotate your findings.

Imaging breast abnormalities

A host of breast masses may be present but your main job is to determine if the masses are cystic or solid and their locations. Once a solid mass is identified, you must attempt to characterize its features. This is important because the radiologist is responsible for positively ruling out or confirming the presence of a malignancy.

Benign versus malignant masses

Benign masses of the breast tend to be cystic. Solid benign masses have borders that are mostly smooth and sharply defined on sonography. Sonographic findings characteristic of malignancy can be seen in the borders, shapes, and internal appearances of breast masses.

General Sonographic Characteristics of Malignant Breast Masses		
Border (Surface)	***Shape***	***Internal Appearance***
Irregular Spicular (spiked) /hypo- or hyperechoic Thick echogenic rim Angular (infiltration) Microlobulation	Taller than wide (1 cm or less) Extension or branches into a visible duct	Acoustic posterior shadowing beginning from within the mass Clusters of microcalcifications within mass Hypoechogenicity

Benign disease

We will look at typical sonographic approaches.

Benign cystic disease

Simple cysts of the breast can appear singular or multiple unilaterally or bilaterally. To be considered simple cyst, many radiologists require you to demonstrate specific criteria:

- Smooth, solid, and thin border or margin.
- Anechoic lumen.
- Posterior acoustic transmission or enhancement.
- Round or oval shape.
- Thin posterior shadowing off lateral edges of the cyst (fig. 4–2).

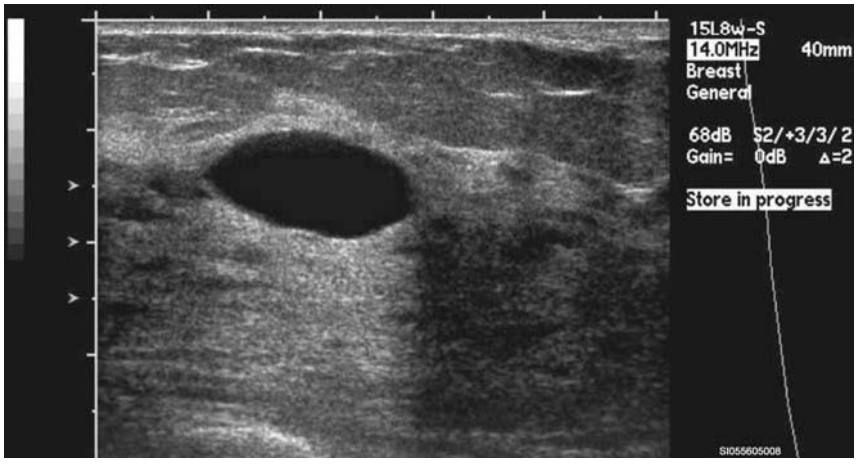

Figure 4–2. Classic breast cyst.

Without these criteria being met, the cysts should be considered complex. The range of appearances for complex cysts runs from internal low-level echoes within the cyst to mural nodules and thick septations.

On sonography, fibrocystic change typically will display multiple cysts within the glandular layer of the breast. Some cysts may have thin septations. Mild cases of fibrocystic change will have cysts appearing throughout the breast of varying sizes but normally only a few centimeters in size, corresponding to the mottled appearance of breast tissue on mammography.

Fibroadenoma

On sonography, the tumor is mostly hypoechoic compared to surrounding glandular layer or isoechoic if surrounded by hypoechoic fat. The borders are clearly defined and smooth. However, some fibroadenomas may have rounded lobulations. The tumor is mostly oval with the long axis parallel to the skin surface (wider-than-tall) (fig. 4–3). Because a fibroadenoma is not really a part of the parenchyma and displaces surrounding tissue, you may see what appears to be a thin echogenic capsule around it—particularly with the superior resolutions of modern equipment. This false capsule (pseudocapsule) represents compressed glandular tissue. The fibroadenoma is slightly mobile and moves independently of surrounding tissue. Upon palpation, they are felt to be firm but rubbery. You will occasionally see posterior shadowing; however, most will show no effect below the tumor. Measure these tumors in all three dimensions (sagittal/transverse or radial/antiradial), and note the location using the clock or quadrant methods.

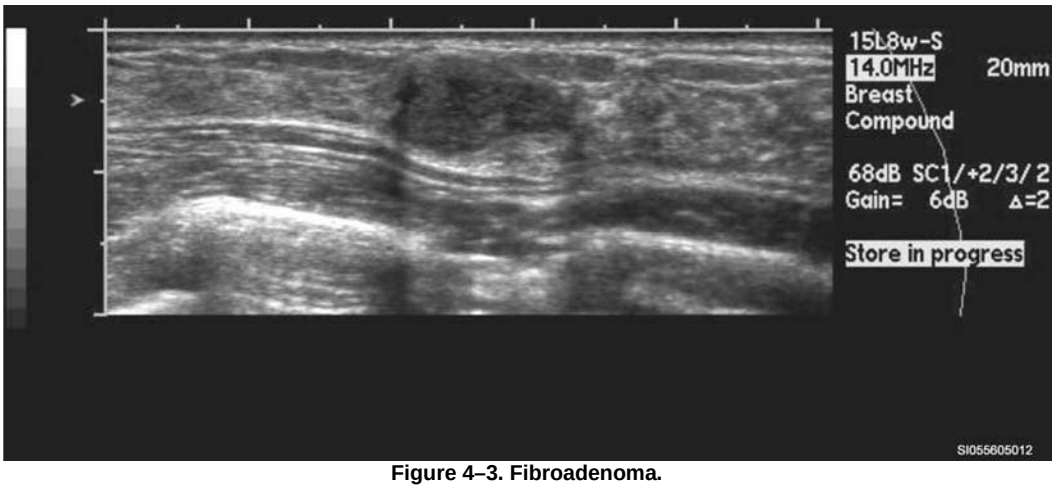

Figure 4–3. Fibroadenoma.

Papilloma

They appear on sonography as isoechoic to duct walls and will usually cause the affected duct to distend with fluid. Intraductal papillomas are solitary and measure only a few millimeters, so you likely will be able to locate it only within a distended duct.

The intracystic papilloma appears on sonography as a projection from cyst walls into the lumen. These mural nodules are the key sonographic feature of intracystic papillomas. The cysts may have thick walls and septations.

Mastitis

On sonography, peripheral or central abscess are complex but largely cystic, sometimes with enhanced acoustic transmission. The tenderness of the skin directly over the area and the large, disorganized appearance of the collection on sonography is nearly confirmation for abscess. Generally, abscesses are analyzed by tissue sample (aspiration) and then clinically drained.

Malignant disease

Of the various carcinomas within invasive and noninvasive breast cancers, each generally shares malignant features and their appearances overlap on sonography. However, certain distinguishing sonographic features lend themselves to suggesting a type of carcinoma present.

Carcinoma in situ

Although contained in the duct, DCIS occasionally displays the typical sonographic signs of malignancy; that is, a mass taller-than-wide, hypoechoic, with extreme shadowing, lobulations, angled margins, and calcifications. However, visualization of DCIS can be difficult because these lesions can be quite small. If they contain a small single calcification, you may be able to detect it within the center of the duct in cross-section, almost as a target sign. If occurring in the subareolar region, DCIS can be confused on sonography with ductal ectasia, which can fill ducts with debris in this area.

Invasive carcinoma

This carcinoma shares the typical sonographic features of breast malignancy, especially with branches of ducts extending from a mass. Therefore, the mass requires biopsy for confirmation (figs. 4–4 and fig. 4–5).

By the time the mammogram and sonogram occurs in about half of breast cancer patients, the cancer will have spread to nodes in the axilla. A normal lymph node is hypoechoic and oval shaped. Inside it is an echogenic line representing a hilum, through which nerves and blood vessels pass. If infected or metastasized, it will typically enlarge into rounded shaped and will become hypervascular. If you notice a mass displaying the hallmarks of malignancy, your radiologist may require you to search the axillary with the transducer for enlarged and rounded lymph nodes.

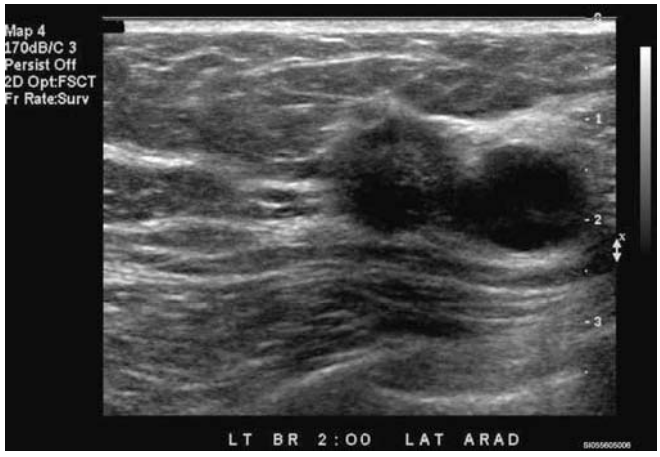

Figure 4–4. Shadowing breast mass exhibiting a malignant character beside a cystic mass.

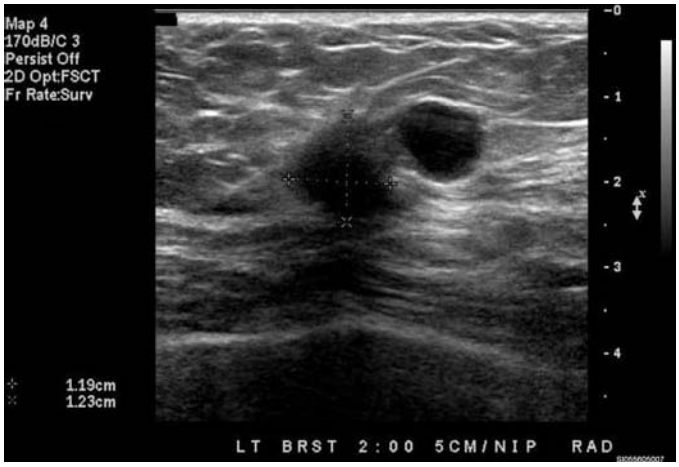

Figure 4–5. Measuring shadowing breast mass.

Self-Test Questions

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

221. Breast anatomy and physiology

1. What influences the size and shape of breasts?

2. List the three layers of the breast.

3. What separates the fat lobules below the breast skin?

4. What constitutes the mammary layer?

5. What is the relationship between TDLUs and breast milk?

222. Breast pathology

1. Clinical symptoms of pain are likely found in which type of breast mass?

2. What is the difference between a typical fibroadenoma and a giant fibroadenoma?

3. Describe a centrally located abscess of the breast.

4. Match the risk factor for breast cancer in column B with its more specific description in column A. Each item in column B may be used only once.

Column A	*Column B*
____ (1) Excessive exposure.	a. Hormone levels and status.
____ (2) Women who become pregnant after reaching 35 years of age.	b. Age.
	c. Alcohol/chemicals/radiation.
____ (3) Rare in women younger than 35.	d. Family history/genetics.
____ (4) History of breast cancer in mother, sister, daughter.	

5. Of the two types of breast carcinoma, which is the most common?

223. Imaging normal and abnormal breasts

1. What type of transducer will you need to evaluate breast tissue properly?

2. Describe the sonographic appearance of the mammary layer of the breast.

3. List the four sections of the four-quadrant method and their sonographic labels.

4. Describe general sonographic characteristics of malignant breast masses.

5. What are the key sonographic features of intracystic papillomas?

4–2. Neonatal Brain Imaging

Although sonography of the neonatal brain is not new, it is being performed in more institutions because of the excellent images obtained compared with the past. Relatively simple to perform, the notorious complexity behind neonatal imaging is largely due to a need for understanding neonatal brain anatomy. For our purposes, only a basic description is needed. As you advance in sonography, you will be able to build on this foundation with more in-depth studies of brain anatomy.

224. Neonatal brain fundamentals

Recall from your diagnostic imaging training that the sutures of the skull are formed by the incomplete fusions of bone plates. At many of the angles and intersections of these sutures are spaces called fontanelles. Of particularly interest to sonographers is the anterior or frontal fontanelle primarily, and the posterior or occipital fontanelle secondarily. The fontanelles may remain open anywhere from six months up to over a year after birth. The anterior fontanelle is a space formed by the junction of the frontal bone at the front of the skull above the orbits with the two parietal bones on either side of the head. The posterior fontanelle is a space formed by a similar arrangement of the posterior parietal bones with the occipital bone at the back of the skull.

Typical reasons for physicians to request a sonogram of the neonatal head are development abnormalities, brain lesions, hemorrhage, and inflammation.

Anatomy and physiology

The brain housed within the skull is divided into two hemispheres superiorly sitting atop a midbrain section and brain stem, from which the spinal cord courses through the foramen magnum and becomes the spinal cord. Three layers of membranes, called meninges, cover the brain.

1. Dura mater (tough outer covering).
2. Arachnoid (middle membrane).
3. Pia mater (inner layer).

The surface of each brain hemisphere is convoluted with twists and turns of raised tissue called gyri. Fissures or spaces called sulci separate every couple of gyri. Each sulcus may be thought of as a

groove or depression. Prominent sulci or fissures separate the brain into four separate regions or lobes, the frontal (anterior and superior), parietal (on either side posteriorly), temporal (on either side inferior to both frontal and parietal lobes), and occipital lobe (posteriorly).

A space exists between two hemispheres of the brain (right and left) called the interhemispheric fissure or falx cerebri. These two hemispheres represent the largest lobe (frontal lobe), called the cerebrum, the area attributed to thought, consciousness, memory, emotion, and language. Within the either half of the cerebrum are two spaces called lateral ventricles. Each ventricle produces and is filled with cerebral spinal fluid (CSF), which coats the subarachnoid (beneath the arachnoid membrane) cavities of the brain and spinal cord. CSF fluid helps maintain and balance the internal pressure of the brain and spinal cord within their bone casings (skull and vertebrae), as well as bathes these structures with proteins and glucose.

The two ventricles form channels that slip through tiny openings in the brain called interventricular foramina (foramen of Monro) and join together into a deep midline third ventricle. A narrow tube called the cerebral aqueduct (aqueduct of Sylvius) drains CSF inferiorly into a fourth ventricle, located between the cerebellum in the posterior cavity (fossa) of the skull and a portion of the brain stem.

Neonatal head pathology

One developmental abnormality is of the cerebellum and brain stem pulled down into and through the foramen magnum (Arnold-Chiari malformation, type II, clamping off CSF flow at the fourth ventricle and causing hydrocephalus or expansion of the ventricular system).

Other neonatal developmental abnormalities of the head referred for sonography are large cysts in the area where cerebellum should be, Dandy-Walker malformations, and hydrocephalus caused by congenital (from birth) obstruction of the aqueduct of Sylvius (aqueductal stenosis).

Prominent reasons for sonography of the neonatal head are to rule out hemorrhage within the brain, above the dura mater (epidural), or below the dura mater (subdural). Inflammation of the meninges and ventricles are also reasons for prompt scanning of the neonatal brain. Finally, brain masses are obvious reasons for using sonography as a way to characterize a lesion in the brain as solid or cystic.

225. Imaging the neonatal head

Using at least a 7.5 MHz sector array transducer with a small footprint allows sonographers to obtain the complex images of the neonatal brain. Many radiologists establish neonatal head protocol based on certain structures that they want to see on the image. The structures and the order in which they are imaged vary with the radiologists and institutions. The following images are basic protocol typical of most departments:

1. Coronal images of the interhemispheric fissure.
2. Sagittal and coronal (or transverse) views of the corpus callosum (the tissue that connects the hemispheres).
3. Lateral ventricles.
4. The third ventricle.
5. Contents of the posterior fossa.

To obtain these images, most sonographers use the open anterior fontanelle as a window through the bone of the skull. Nearly all of the images can be obtained by manipulating transducer at this fontanelle. Common coronal scan planes are generally based on acquiring certain landmarks.

Common Coronal Scan Planes and Landmarks	
Anterior	Anterior frontal horns of the lateral ventricles
Middle	Lateral ventricles, third ventricle, corpus callosum
Posterior (A)	Cisterna magna
Posterior (B)	Choroid within the atria of the lateral ventricles
Posterior (C)	Posterior brain matter

The sagittal scan planes found in many departments are based on three major scan planes: a central midline scan and an approximately 10-degree angulation of the transducer to either side for lateral plane views. To obtain images of the cerebellum and posterior fossa that are not well seen with coronal views, some radiologists will ask for axial views obtained from a fontanelle located on the posterior and side of the head behind the ear (mastoid fontanelle).

Self-Test Questions

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

224. Neonatal brain fundamentals

1. Which fontanelles of the neonatal skull are of particular interest to sonographers?

2. List the three layers of the brain meninges.

3. What is the narrow tube that drains CSF into the fourth ventricle?

4. What are the typical reasons for physicians to request a sonogram of the neonatal head?

5. List some neonatal developmental abnormalities of the head.

225. Imaging the neonatal head

1. Describe basic neonatal head protocol typical of most departments.

2. Match the scan plane in column B with the landmark in column A. Each item in column B may be used only once.

Column A	*Column B*
____ (1) Anterior frontal horns of the lateral ventricles.	a. Middle.
____ (2) Lateral ventricles, third ventricle, and corpus callosum.	b. Anterior.
	c. Posterior.
____ (3) Choroid within the atria of the lateral ventricles.	d. Posterior.
____ (4) Posterior brain matter.	e. Posterior.
____ (5) Cisterna magna.	

4–3. Invasive Procedures

The usefulness of sonography for assisting surgical procedures cannot be understated. The ability to see where a needle or catheter is going through successive layers of tissue, real-time, and cheaply compared to the costs of surgical instruments, is invaluable. In this section we will briefly look at some of the interesting ways sonography can enhance interventional procedures.

226. Performing invasive ultrasound

Without sonography, physicians who perform invasive procedures (those that enter the body through piercing the skin) are technically doing so blind. Such a method relies on the physician's knowledge of typical anatomical and pathological structures, with varying degrees of extensiveness. However, with sonography, the accuracy of performance increases to a near standard, and physicians are able to see where they are scanning and what they are doing in real-time.

Ultrasound guidance

Whatever invasive procedure used, one of three guidance methods will typically be employed:

1. Indirect needle guidance.
2. Free hand technique.
3. Needle guidance.

The indirect guidance is nothing more than marking the skin over the site where sonographic images were taken to demonstrate the target. The free-hand technique involves the operator seeing the needle being placed into the target from virtually any angle with a free hand while watching the image real-time via sonography. In the needle guidance method, the needle is attached to the transducer and is visualized inserting into the target along a pre-determined path.

Aspiration/localization

Aspiration is technically a form of biopsy in that fluid rather than solid tissue is extracted from the body using a needle under ultrasound guidance. Fluid is then examined by laboratory analysis. Most aspiration procedures call for thin diameter needles, such as the 22 or 20 gauge needles to aspirate or drain various fluids from cysts to abscesses. This is why it is sometimes referred to as fine-needle aspiration (FNA). Various types of needles having removable parts that allow catheters to be inserted are available, as well as needles that accommodate equipment such as guide-wires and three-way stopcocks for drainage procedures. Renal cysts, breast cysts, or abscesses throughout the body are some of the areas of interest for FNA. Breast masses typically call for the use of the free-hand guidance method, whereas a more direct approach is used for other procedures. FNA using larger bore 18 gauge needles are sometimes used to extract cells from solid nodules in the thyroid. Thoracentesis (the passage into the chest with a hollow needle, trocar, and cannula for draining fluid from the pleural space), or simply marking on the skin a location before thoracentesis, is significantly enhanced when guided by sonography. Intervention procedures (i.e., thoracentesis) for thorax fluid collections are accomplished through curvilinear transducers that can view the chest tissue through the rib cage. FNA is also used for thoracentesis.

Sonographic localization procedures are largely used to augment mammography. Ultrasound guidance for the placement of a guide wire before a surgical procedure is the extent of the sonographer's involvement. Normally localization using sonography is for masses already known. Sonography is used in the event a patient will be unable or unwilling to undergo mammographic localization.

Ultrasound-guided needle biopsy

Ultrasound-guided needle biopsy is strictly used for solid tissue extraction of cells. Large needles are typically required beginning with 16-gauge or larger. Most needle-biopsies (also called *core* biopsies)

use spring-loaded biopsy guns, in which the needle is loaded into a handheld mechanism and inserted into the body percutaneously (through the skin) and guided under sonography to the edge of a target. A button is pressed and the needle is thrust forward into a target lesion and then manually withdrawn with a sample amount of target tissue (the core) within the needle. This is quite useful for hard, tough tumors or masses. The entire procedure, before, during, and immediately after the firing of the biopsy gun, is recorded. Most biopsy guns are held separately from the ultrasound transducer. You may work in a department where the radiologist fires the biopsy gun while you hold the transducer directly over the site of interest.

Because of the possibility for uncontrolled internal hemorrhaging, sonography helps to see the post-biopsy effects on any vascular structures in the path of the needle. Patients with blood-clotting problems should be identified before attempting ultrasound-guided needle biopsy.

Interoperative portable sonography

Frequently referred to as *intra*operative sonography, the procedure is simply the use of sonography during operating room surgery. Thus, the procedure is an invasive one. Normally, this involves moving sonographic units (thus, making them portable) from the ultrasound section to the operating room.

Interoperative portable sonography is used to provide real-time benefit of direct visualization of disease during the surgical procedure. Surgeons find interoperative sonography particularly useful for locating and characterizing masses and fluid collections during operations of such structures as the liver, the kidneys, pancreas, and the biliary system. Frequently, new characterization of disease is discovered that may have been masked during routine pre-operation sonography. Compared to other surgical imaging devices, such as bulky C-arm equipment, sonography in the operating room is quicker, uses no ionizing radiation, and provides easier direct organ imaging.

Sonographers always should use sterile techniques and personal protective equipment when in the surgical environment. Each facility has their own protective procedures, but certain precautions are universal. For example, surgical gloves, mask, and gown are basic items needed. Also, most sonographers will have sterile probe covers, usually made of latex with coupling gel inside for sound transmission through the material. Various designs of surgical probe covers are available for the interoperative procedure. Also manufactured are sterile plastic covers or barriers for the ultrasound unit itself. Many surgeons will have everyone in the surgical suite scrub (surgical handwashing before donning surgical clothing), with no exceptions. If you do have to scrub, always remember to consider every surface non-sterile, and do not touch anything unless clearly told you can touch it. The reverse is also true; if unscrubbed, assume everything in the room to have an invisible sterile field and ask where you are permitted to work. This latter distinction still involves the complete sterilization of your equipment and at least semi-sterilization of the hand that holds the probe.

227. Thorax sonography

Because previous units have covered most of the anatomy concerning parts requiring invasive sonography, such as the liver or kidneys, and because sonographers are sometimes called upon to perform ultrasound guidance for an invasive procedure of the thorax (thoracentesis), we will briefly cover the anatomy and physiology of the chest or thorax.

Anatomy and physiology

The ribcage surrounds the thoracic cavity posteriorly and laterally, with a cartilaginous attachment to the ends of the ribs anteriorly meeting up with the sternum or breastbone centrally. In between individual ribs are intercostal muscles, which allow the rib cage to expand and contract. Sonographers use the intercostal space as windows for thoracic imaging. Inferiorly, the diaphragm separates the thoracic cavity from the abdominal cavity.

Beneath the ribs is the parietal pleura, a membrane that covers the entire inner wall of the thoracic cavity to include the superior surface of the diaphragm. On the inferior surface of the diaphragm, the portion that faces the abdomen, there is a peritoneum covering. The layer beneath the parietal pleura is the visceral pleura, which is separated from the parietal by a space called the pleural space. A small amount of pleural fluid is present in the space to lubricate the movement of the lungs against the thoracic wall during respiration. The visceral pleura separate and completely encase the lungs, partitioning those structures from the mediastinum, which contains esophagus, trachea, and heart. Thus, right and left pleural cavities are distinctly closed off from each other.

Of course, two air-filled lungs take up most of the thoracic cavity. The sole purpose of the lungs is to intake oxygen and to expel carbon dioxide. This gaseous exchange (respiration) takes place within thousands of individual lung sacs called alveoli. The ability to inhale and exhale air is largely a result of the rib cage, intercostal muscles, and the pleural membranes sliding past each other without friction. The lack of friction is, again, due to the presence of fluid in the pleural space. The capability of the lungs to expand and contract is largely due to the motion of the diaphragm.

Thorax pathology

Abnormalities of the chest can be characterized into three major areas:

1. Pleural.
2. Lung.
3. Mediastinal.

An increase in the normal amount of fluid within the pleural space is called a *pleural effusion.* Typically, sonographic involvement is concerned with locating a pleural effusion collection. Two types of pleural effusion are transudative (caused by heart failure or uremia, which is excessive urea and nitrogen wastes in the blood) and exudative (caused by infectious inflammation or malignancy). Infectious inflammation can cause pleural fluid to become prurulent, a condition called *empyema*, or pus in the pleural space. The pleura itself may thicken with both effusion and empyema, restricting the expansion of the lung and interfering with respiration.

Other pleural abnormalities can occur. For instance, *pleural plaques*, dense collagenous or calcified tissue caused by pneumonia, trauma, and exposure to certain chemicals and toxins. Tumors may occur from the lung tissue and can be found in the pleural space or as a part of the pleura itself.

The lung itself can be infiltrated with fluid and inflammatory cells within the air spaces caused by disease processes such as pneumonia. The affected lung tends to harden into a dense mass. This condition is called *consolidation*. Other lung abnormalities are atelectasis (absence of air in the lung with collapsed alveoli) and solid lung masses.

Cysts and masses typically collect within the mediastinum. Common types are *bronchogenic cysts* and *teratomas*. Usually sonographers are called on only to determine if a mass is solid or cystic.

Self-Test Questions

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

226. Performing invasive ultrasound

1. List three ultrasound guidance methods.

2. Most aspiration procedures call for which type of needles?

3. How are sonographic localization procedures related to mammography?

4. What is the strict use of ultrasound-guided needle biopsy?

5. What is the purpose for interoperative portable sonography?

227. Thorax sonography

1. What is the significance of the intercostal space to sonographers?

2. What is the sole purpose of the lungs?

3. List the three major abnormalities of the chest area.

4. What causes pleural plaques?

Answers to Self-Test Questions

221

1. Age, genetic factors, and individual hormone influence.
2. (1) Premammary zone (subcutaneous layer).
   (2) Mammary zone (mammary layer).
   (3) Retromammary zone (retromammary layer).
3. Suspensory ligaments of breast, fibrous connective tissue commonly referred to as Cooper ligaments.
4. Functional or glandular tissue of the breast. This layer is divided into at least 15 lobes, each with ducts and hundreds of lobules, and separated by dense fibrous tissue. Inside each lobule is less dense fibrous tissue that surrounds the ducts and, along with varying amounts of fat, is interspersed throughout. Also, inside each of the lobules are the functional units of the breast called TDLU, composed of tiny sac-like glands called acini or alveoli as well as intralobular stroma or connective tissue.
5. Milk is secreted from the TDLU into the lactiferous ducts.

222

1. Benign.
2. Unlike typical fibroadenomas, giant fibroadenomas occur in adolescents, tending to rapidly grow and cause pain.

3. Can be both puerperal and non-puerperal; large collections with an elongated shape, coursing roughly parallel to the duct of origin. Because mastitis can cause an inflamed duct to rupture, fluid is spilled into the breast tissue.
4. (1) c.
   (2) a.
   (3) b.
   (4) d.
5. Invasive ductal or infiltrating ductal carcinoma.

223

1. At least a 7 MHz linear-array transducer or higher with multiple focal zones in the near-field.
2. Echogenic layer; Young women: thick and contains some areas of hypoechoic fat; occasionally, hypoechoic linear structures (ducts) are seen; May be echogenic and dense from fibrous tissue; Pregnant or lactating women: Extremely thickened and echogenic with a homogeneous echotexture; Older and post-menopausal women: Thin or almost entirely replaced with hypoechoic fat; Cooper ligaments remain highly echogenic and give a polyhedral shapes to the fat lobules.
3. (1) Upper Outer Quadrant: RUOQ and LUOQ.
   (2) Lower Outer Quadrant: RLOQ and LLOQ.
   (3) Upper Inner Quadrant: RUIQ and LUIQ.
   (4) Lower Inner Quadrant: RLIQ and LLIQ.
4. Border (surface) is irregular (spiculated, thick, or hypo/hyper echoic), angular, with microlubualtions; the shape is taller-than-wide, with extension or branches into a visible duct; and an internal appearance of hypoechogenicity, clusters of microcalcifications within mass, and acoustic posterior shadowing beginning from within the mass..
5. Mural nodules, projections from cyst walls into the lumen.

224

1. The anterior or frontal fontanelle primarily, and the posterior or occipital fontanelle secondarily.
2. (1) Dura mater (tough outer covering).
   (2) Arachnoid (middle membrane).
   (3) Pia mater (inner layer).
3. Cerebral aqueduct (aqueduct of Sylvius).
4. Development abnormalities, brain lesions, hemorrhage, and inflammation.
5. Large cysts in the area where cerebellum should be, Dandy-Walker malformations, and hydrocephalus caused by congenital (from birth) obstruction of the aqueduct of Sylvius (aqueductal stenosis).

225

1. Coronal images of the interhemispheric fissure, sagittal and coronal (or transverse) views of the corpus callosum (the tissue that connects the hemispheres), the lateral ventricles, the third ventricle, and the contents of the posterior fossa.
2. (1) b.
   (2) a.
   (3) d.
   (4) e.
   (5) c.

226

1. (1) Indirect needle guidance.
   (2) Free hand technique.
   (3) Needle guidance.
2. Thin diameter needles, such as the 22 or 20 gauge needles.

3. Augment.
4. Solid tissue extraction of cells.
5. To provide real-time benefit of direct visualization of disease during surgical procedures. Surgeons find interoperative sonography particularly useful for locating and characterizing masses and fluid collections during operations of such structures as the liver, the kidneys, pancreas, and the biliary system. Frequently, new characterization of disease is discovered that may have been masked during routine pre-operation sonography.

227

1. Sonographers use the intercostal space as windows for thoracic imaging.
2. To intake oxygen and to expel carbon dioxide.
3. (1) Pleural.
   (2) Lung.
   (3) Mediastinal.
4. Pneumonia, trauma, and exposure to certain chemicals and toxins.

Complete the UREs.

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.

82. (221) What is the relationship of the subcutaneous layer and the premammary zone in the breast?
    a. Premammary zone is the subcutaneous layer.
    b. Premammary zone is inside the subcutaneous layer.
    c. Subcutaneous layer is below the premammary zone.
    d. Subcutaneous layer is above the premammary zone.

83. (221) Where inside the mammary layer of the breast are terminal ductolobular units (TDLU) located?
    a. Lobules.
    b. Nipples.
    c. Lobes.
    d. Ducts.

84. (222) What is the *common* factor of both puerperal and non-puerperal mastitis?
    a. Inflammation of the breast.
    b. Formation of a galactocele.
    c. Occurs after delivering child.
    d. Peripheral abscess formation.

85. (222) What characteristic makes ductal carcinoma in situ (DCIS) different from invasive carcinoma?
    a. Ductal.
    b. Metastatic.
    c. Noninvasive.
    d. Hyperechoic.

86. (223) What is the *main* reason for performing breast sonography?
    a. Analyze breast implants.
    b. Locate the lactiferous ducts.
    c. Ascertain the size of breasts.
    d. Characterize palpable masses.

87. (223) Of the following, which sonographic characteristic will a radiologist require you to demonstrate as a criterion for a simple cyst?
    a. Mural nodule.
    b. Thin septations.
    c. Irregular border.
    d. Anechoic lumen.

88. (223) What is the sonographic appearance of a fibroadenoma compared to fat?
    a. Hyperechoic.
    b. Hypoechoic.
    c. Isoechoic.
    d. Anechoic.

89. (224) What helps maintain internal pressure of the spinal cord?
    a. Pia mater.
    b. Falx cerebri.
    c. Cerebral spinal fluid.
    d. Fine-needle aspiration.

90. (224) Which of the following developmental abnormalities involves the cerebellum being pulled down into the foramen magnum?
    a. Brain hemorrhage.
    b. Aqueductal stenosis.
    c. Dandy Walker malformation.
    d. Arnold-Chiari malformation, type II.

91. (224) What is a reason for requesting sonography of the neonatal head?
    a. Stenosis.
    b. Dysphagia.
    c. Inflammation.
    d. Hypertension.

92. (225) Through which skull structure will some radiologist ask for axial views of the cerebellum?
    a. Mastoid fontanelle.
    b. Anterior fontanelle.
    c. Posterior fontanelle.
    d. Occipital fontanelle.

93. (226) What ultrasound guidance method involves attaching a needle to the transducer and visualizing the needle along a pre-determined path?
    a. Needle guidance.
    b. Indirect needle guidance.
    c. Free-hand needle technique.
    d. Three-way stopcock guidance.

94. (226) Of the following techniques, which extracts fluid from the body rather than solid tissue?
    a. Free hand.
    b. Aspiration.
    c. Core biopsy.
    d. Localization.

95. (226) Breast masses typically call for which guidance method?
    a. Needle guidance.
    b. Free-hand technique.
    c. Indirect needle guidance.
    d. Three-way stopcock guidance.

96. (226) What is interoperative sonography?
    a. Sonography replacing surgery.
    b. Sonography during surgery.
    c. Sonography before surgery.
    d. Sonography after surgery.

97. (226) What surrounds the thoracic cavity posteriorly and laterally?
    a. Ribs.
    b. Pleura.
    c. Sternum.
    d. Diaphragm.

98. (226) What structure covers the entire inner wall of the thoracic cavity?
    a. Parietal pleura.
    b. Visceral pleura.
    c. Tunica albuginea.
    d. Tunica vaginalis.

99. (227) An increase in the normal amount of fluid within the pleural space is called pleural
    a. plaque.
    b. empyma.
    c. effusion.
    d. consolidation.

100. (227) What is absence of air in the lungs?
a. Atelectasis.
b. Pleural effusion.
c. Bronchogenic cyst.
d. Pleural consolidation.

When you complete this course, please complete the student survey on the Internet at this URL: http://www.maxwell.af.mil/au/afiadl/. Click on Student Info and choose 9502 Survey.

Student Notes

Glossary

Abbreviations and Acronyms

AAA	abdominal aortic aneurysms
AFP	alpha-fetoprotein
AP	anteroposterior
AT	anterior tibial artery
AVF	arteriovenous fistulas
CCA	common carotid artery
CFA	common femoral artery
CFV	femoral vein
cm	Centimeters
CSF	cerebral spinal fluid
CT	computed tomography
DCIS	ductal carcinoma in situ
DVT	deep vein thrombosis
ECA	external carotid artery
EDV	end diastolic velocities
EIV	external iliac vein
FNA	fine-needle aspiration
FV	femoral vein
hCG	human chorionic gonadotropin
Hg	Mercury
ICA	internal carotid artery
IJV	internal jugular vein
IVC	inferior vena cava
MEN	multiple endocrine neoplasia
MHz	megahertz
mm	millimeters
PA	popliteal artery
PRF	repetition frequency
PSV	peak systolic velocity
PT	posterior tibial
PTH	parathyroid hormone
PV	peroneal veins

RCC	renal cell carcinoma
RVT	renal vein thrombosis
SFA	superficial femoral artery
SMA	superior mesenteric artery
SV	saphenous vein
TDLU	terminal ductolobular units
TIA	transient ischemic attack
TRH	thyrotropin-releasing hormone

Student Notes

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