

Section IV

IMAGING PROCEDURES *and* PROTOCOLS

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INTRODUCTION

Each imaging facility has its own, often unique, set of examination protocols. For any given CT examination, the protocol will vary according to the individual requirements of the imaging site. Factors that influence a specific site's protocol parameters include the type of equipment available (e.g., 64-slice versus 16-slice), the setting (e.g., inpatient versus outpatient), and the particular preferences of the radiologists in charge. Like all fields of medicine, CT is constantly evolving so it follows that examination protocols constantly undergo re-evaluation and refinement. A cookbook approach to CT scanning is impractical as it cannot possibly account for all possible variables. The protocols presented in this section are intended as examples of just a few of the many possible variations in current practice. There has been no attempt to survey institutions to determine the most prevalent protocols; protocols included here should not be considered either the most common or the best. They are simply one or two options among many. Many other protocol options can be found on the World Wide Web, posted by both equipment manufacturers and various university hospitals. The protocols in this text are in a tabular format to facilitate easy reference. When possible, the protocols include parameter variations for a 16- and a 64-detector system. Again, these are intended to be illustrative, providing the reader an idea of the parameters most likely to be adjusted when switching from one type of detector configuration

to another. Unless otherwise noted, protocols that include intravenous contrast enhancement use a 300 concentration, low-osmolality agent.

CT imaging increasingly relies on the integration of a variety of knowledge and skills. The first three sections of this text provide information on physics, patient care, and cross-sectional anatomy necessary to understand and carry out CT examination protocols. Hence, this section is intended to be a culmination of the sections that precede it. Within most categories of the examination protocols, one or two topics have been selected for expository writing. These essays are included to demonstrate the interdependency of the information provided in previous sections and to encourage the reader to assemble the knowledge gained thus far into a cohesive whole. Readers will notice that these essays provide information that is not strictly necessary to know to perform a high-quality examination. However, both new and seasoned CT technologists will appreciate the “extra” information, such as the signs and symptoms of various diseases and conditions, as these are so often observed in clinical practice.

ROUTINE SCANNING PROCEDURES

Although in actual practice there are exceptions to every rule, some standards of practice are assumed. We start with a discussion of these assumptions.

In all routine studies, one or two reference images (called “scouts” by some manufacturers) are acquired before scanning. The optimal reference image includes all areas to be scanned and therefore ensures that the anatomy to be imaged is within the range of the scanner (see Chapter 5).

Regardless of the type of scanner used, it is imperative that the operator input the correct directional instructions before scanning. This procedure requires indicating whether the patient is placed head or feet first into the gantry and whether he or she is lying supine, prone, or in the decubitus position. If the operator accurately enters this information into the system, the software correctly annotates the image as to left-right, anterior-posterior, and superior-inferior orientation.

After the scout image is obtained, the operator selects the location of cross-sectional slices. Scan parameters such as mAs, kVp, display field of view, and reconstruction algorithm(s) are selected. Most often, a preprogrammed group of scan parameters is selected so that the technologist must make only minor modifications to suit the particular patient or clinical indication.

It is important to correctly annotate the image as to whether contrast material is administered. All CT systems allow a notation concerning the use of contrast agents. Often, the notation +C is inputted after the introduction of contrast medium.

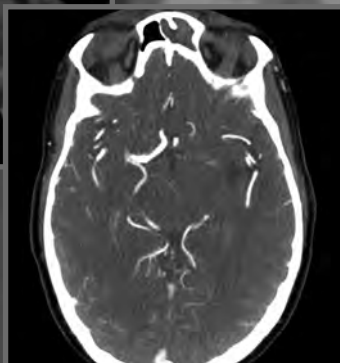
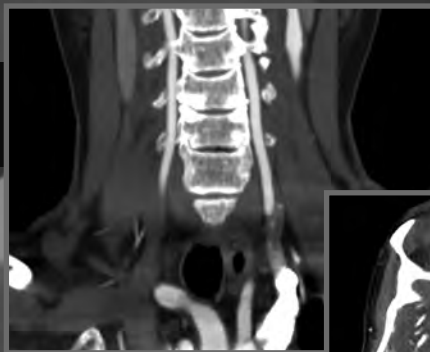
Routine procedure for some CT examinations includes scanning without contrast material, then repeating the study with intravenous contrast enhancement. Some protocols call for repeating the study at different phases of contrast enhancement. Whenever possible, the repeat scans should be taken at the same location as the unenhanced images.

A history should be taken for all patients. For examinations that require contrast enhancement, the history must include allergies as well as any renal complications (see Chapter 11).

Careful breathing instructions should be given to all patients undergoing body scanning. Some manufacturers suggest that technologists use a coaching procedure to increase the length of breath-hold for a helical study. This coaching requires the patient to take several deep breaths before taking one final breath and holding it for the maximum possible time, then exhaling slowly.

After completing a CT study, it is standard procedure to include a scout image that is cross-referenced by lines. These lines represent the location of each cross-sectional image. It is also typical to include patient data at the beginning or end of each examination. Data must then be stored in some fashion. The practice of transferring images to film is dwindling, but by no means has disappeared. When picture, archive, and communication systems (PACS) are used, images must be transmitted to the appropriate radiologist’s workstation (Chapter 9). This information may also be included in a facility’s examination protocols.

Chapter 19



NEUROLOGIC IMAGING PROCEDURES

Key Terms: supraorbital meatal line • glabellomeatal line • intracranial hemorrhage • Valsalva maneuver • split bolus • CT venography • thrombolytic therapy • completed or established stroke • cerebrovascular accident • stroke in evolution • progressive stroke • ischemic • hemorrhagic • thrombotic • embolic • subarachnoid hemorrhagic stroke • arteriovenous malformations • hypotensive stroke • transient ischemic attack (TIA) • atrial fibrillation • tissue plasminogen activator (t-PA) • penumbra • infarction • CT brain perfusion • rCBF • rCBV • MTT • central volume principle • basal ganglia • CVRC

GENERAL IMAGING METHODS FOR THE HEAD

The patient's head is positioned in the head holder for most protocols of the head. Depending on the design of the head holder, it can sometimes also be used for neck protocols. When the head holder is not used, a molded sponge is placed directly on the scan table and the patient's head is positioned within the sponge. In all cases, the patient should be made as comfortable as possible and immobilized as effectively as possible to prevent motion artifact on the images. This is often accomplished by placing small

wedge sponges on either side of the patient's head. In most cases it is not necessary to ask the patient to suspend breathing for CT studies of the head or neck. Anatomy displayed in cross-sectional slices will look slightly different depending on the angulation used. The slice angle is determined by the position of the patient's head (i.e., moving the chin up or down) and the angle of the gantry. It was once common to program the cross-sectional slices of the brain to be parallel to the orbitomeatal line; however, more recent practice favors using the supraorbital meatal line (also called the glabellomeatal line) to reduce radiation exposure to the lens of the eye. Figure 19-1 illustrates lines used for positioning. A disadvantage of many multi-detector CT systems is that they do not allow the gantry to be tilted when in helical mode. Therefore, axial (step-and-shoot) techniques are often used for routine brain imaging.

BOX 19-1 Key Concept

Recent practice favors programming slices of the brain parallel to the supraorbital meatal line (rather than the orbital meatal line) to reduce radiation exposure to the lens of the eye.

Changing the image plane from axial to coronal may provide additional information. There are two methods of achieving a coronal position for head scanning. One is to place the patient prone on the scanning table and ask the patient to extend the chin forward. An alternative approach is to place the patient supine and ask him to drop his head back as far as possible. This position usually requires a specialized head holder. In either position, the slice plane will be coronal. If the patient cannot extend the neck fully, the

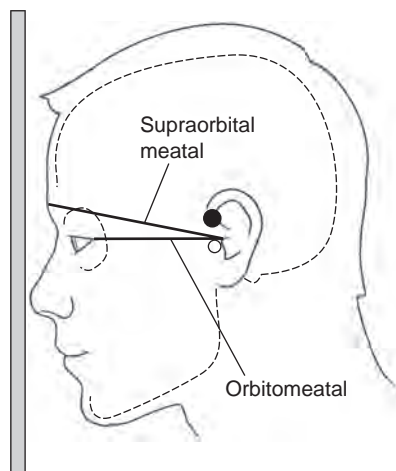


FIGURE 19-1 The basic localization points and planes used in CT positioning.

gantry may be angled to obtain a more coronal plane. The image obtained in either the prone or the supine coronal position is essentially the same. Obviously the images are flipped inferior-superior. The preferred position involves several factors, including patient comfort, radiologist preference, and the effect of gravity on anatomic structures (e.g., will fluid settle inferiorly or superiorly?).

Imaging the posterior fossa of the brain is a challenge in CT scanning. Because of the great difference in beam attenuation ability between the dense bone of the skull and the much less dense tissue of the brain, streak artifacts are common. This inherent limitation may be managed by decreasing slice thickness when scanning the posterior fossa and increasing the kVp setting.

BOX 19-2 Key Concept

Because of the dense bone of the skull, beam-hardening artifact is common in the posterior fossa.

Modern multislice CT scanners allow studies of the head to be routinely acquired with thinner slices than in the past—1.25 mm thickness is typical. These thin slices help to reduce beam-hardening artifacts. Images are often merged into thicker slices for viewing.

In examinations of the head, the helical CT mode is used mainly for the purpose of generating three-dimensional reformations or to minimize motion-related artifacts. In general, routine head studies are done using an axial mode, and CT angiography (CTA) studies of the head and neck are done using a helical mode.

Cross-sectional slices of the brain are viewed in multiple window settings. Standard window settings include soft-tissue (brain) 160/40 (approximate window width/window level) for slices in the posterior fossa, 100/30 for slices above; bone (particularly on trauma or postoperative patients) 2500/400; blood 200/60.

Narrow window widths are used to demonstrate the brain, as there is only a small difference in attenuation between the gray matter and the white matter (Chapter 4). Table 19-1 presents values of x-ray attenuation on unenhanced cranial CT. The slightly higher attenuation of the gray matter of the brain compared with white matter may be a result of both a lower gray matter water content and a higher blood volume.¹ Recall that on the Hounsfield scale zero refers to pure water; the value of cerebrospinal fluid (CSF) is slightly above that of water.

CT is the most frequently used initial examination for imaging of intracranial hemorrhage (ICH). The appearance of an ICH will change with the passage of time. This is because the red blood cells within the hemorrhage begin to deteriorate within several hours after leaving the vasculature. These changes are complex and depend on many factors, such as whether the patient is anemic or whether, and to what degree, blood has mixed with CSF. As a general rule, ICH will appear hyperdense to normal brain tissue for approximately 3 days, after which it will gradually decrease in density. This density loss begins at the periphery of the hematoma. As density diminishes, portions of the hematoma become isodense to brain tissue. This progressive density loss continues until the entire hematoma finally becomes hypodense to brain tissue. Although greatly simplified, ICH can be generally expected to appear hyperdense (i.e., white on the image) from onset to 3 days; from 4 to 10 days it is likely to contain a hyperdense center surrounded by concentric areas of hyperdense and hypodense tissue; from 11 days to 6 months it is likely to contain an isodense center surrounded by areas of hypodense tissue; by 6 months the ICH will be hypodense to brain² (Fig. 19-2). It is not the role of the technologist to interpret images. However, it is important that technologists recognize certain potentially critical pathologic changes so that when present, they can be brought to the attention of a radiologist. Although most patients with an ICH are seen through the emergency department where images are reviewed by radiologists and reported on quickly, some patients, particularly those with less acute presentations (e.g., headache) may arrive in the CT department as outpatients. In these situations, the technologist can play a vital role by bringing the

TABLE 19-1 X-ray Attenuation in Cranial CT (in Hounsfield Units)

Gray matter	35–45
White matter	20–30
Cerebrospinal fluid	4–8
Circulating blood	40–50
Clotted blood	60–110
Tissue calcification	80–150
Fat	–60 to –70
Air	–1000

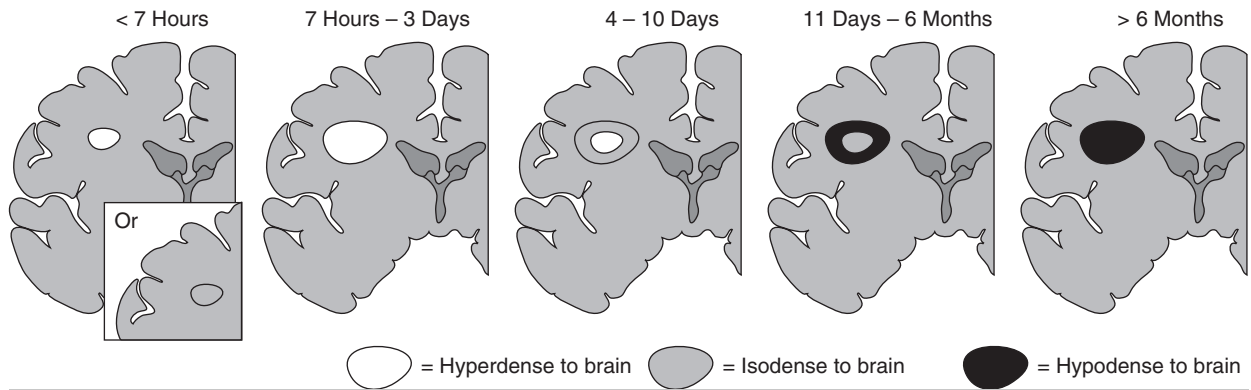


FIGURE 19-2 A simplified schematic of the evolution of intracranial hemorrhage as it appears on CT.

scan to the radiologist's attention so that these patients receive prompt medical attention.

CT is the primary imaging modality for emergent indications such as trauma and acute changes in neurologic status. For most applications concerning structural imaging of the brain and skull base, nonenhanced CT is usually adequate. IV contrast administration is indicated for infection and neoplasm, but in practice this is not frequently performed because those indications most often prompt an MRI, obviating the need for enhanced CT. However, in some situations MRI is contraindicated (e.g., patient with pacemakers) or unavailable, leaving enhanced CT the best diagnostic option.

BOX 19-3 Key Concept

It is not the role of the technologist to interpret images. However, it is important that technologists recognize certain potentially critical pathologic changes so that when present, they can be brought to the attention of a radiologist.

NECK PROTOCOLS

Routine scanning of the neck is typically performed with the patient supine and the neck slightly extended. It is most often performed in the helical mode. To reduce artifacts that degrade images in the lower neck, the patient should be instructed to lower the shoulders as much as possible. In some institutions images of the neck are acquired while the patient performs a modified Valsalva maneuver. This maneuver requires the patient to blow the cheeks out. This technique helps to distend the pyriform sinuses. Another technique that has been used to evaluate the aryepiglottic folds and pyriform sinuses is to ask the patient to pronounce a prolonged "e" during scanning.

Unless contraindicated, IV contrast media is used when scanning the neck. The goals in CT scanning of the neck are to allow sufficient time after contrast administration for mucosa, lymph nodes, and pathologic tissue to enhance, yet acquire images while the

vasculature remains opacified.³ Scanning too early after the contrast media injection could result in certain types of neoplastic and inflammatory processes going undetected. However, by delaying scan acquisition the injected contrast agent will no longer opacify the vasculature. One strategy for addressing these contradictory goals is a contrast injection technique referred to as a split bolus. The total contrast dose is split, often in half. The first dose is given and a delay of about 2 minutes is observed. This allows time for structures that are slower to enhance to be opacified. The delay is followed by a second bolus containing the remainder of the contrast; scanning is initiated soon after the second injection is complete, using this second injection to more fully opacify the vessels. The split bolus injection technique is also frequently used for maxillofacial studies in which contrast media is indicated.

BOX 19-4 Key Concept

Unless contraindicated, IV contrast media is used when scanning the neck. The goals in CT scanning of the neck are to allow sufficient time after contrast administration for mucosa, lymph nodes, and pathologic tissue to enhance, yet acquire images while the vasculature remains opacified.

CTA OF THE HEAD AND NECK

Advances in multidetector CT systems and image post-processing techniques have expanded the role that CT plays in the evaluation of cervicocranial vascular disease. Although cerebral catheter angiography or digital subtraction angiography (both performed in the interventional radiology department) are still generally regarded as the gold standard for the imaging of cerebrovascular disorders, those techniques are time-consuming and are associated with a small, but significant, rate of permanent neurologic complications.⁴ CT angiography has the advantages of being noninvasive and widely available. The time-saving nature of CTA over traditional angiography is particularly

important in the case of patients suspected of suffering an acute stroke in which treatment decisions must be made quickly (a more detailed discussion of stroke begins at the end of this page). In addition, cerebral CTA can be combined with brain perfusion imaging to assess the viability of brain parenchyma and its vascular supply. Cerebral and carotid CTA techniques provide important information about vessel walls; three-dimensional postprocessing of the image data depicts the spatial relationship of complex vascular lesions to the surrounding structures, providing valuable information for the surgeon. Rapid, high-resolution scans are taken while contrast is in the arterial enhancement phase. The goals of CTA for cervicocranial vascular evaluation can be summarized as follows: 1) to accurately measure stenosis of the carotid and vertebral arteries and their branches, 2) to evaluate the circle of Willis for completeness using three-dimensional reformations of cerebral vasculature in relation to other structures, and 3) to detect other vascular lesions, such as dissections or occlusions. A modification of CTA, called CT venography (CTV) is used for the depiction of venous anatomy. Scan parameters are quite similar to CTA, except images are acquired while contrast is in the venous enhancement phase.

SPINE PROTOCOLS

Compared with conventional radiography, CT examinations of the spine produce images with inherently high soft tissue contrast. This contrast permits the visualization of structures such as the intervertebral disks, ligaments, and muscle, as well as bone detail. Visualization of intradural structures is improved by the intrathecal administration of water-soluble contrast material (Chapter 12). CT examinations are performed after myelography to enhance or clarify myelographic findings of intradural and extradural abnormalities. MRI provides even higher soft tissue sensitivity than CT, and in certain circumstances, it is the modality of choice for imaging the spine (e.g., multiple sclerosis, hydromyelia, syringomyelia). For some conditions, such as spinal stenosis, MRI is equivalent to CT. In some situations, CT is considered superior to MRI, such as in the evaluation of bony abnormalities of the spine.

Scans of the spine are often obtained after intrathecal contrast material is given for a fluoroscopic myelography study. Intrathecal contrast medium may be helpful for the diagnosis of degenerative disk disease and other disk diseases, such as extradural neoplasm. Most reports suggest a delay of 1 to 3 hours between the intrathecal injection and scanning. This delay allows the contrast material to dilute. If the scans are performed while the contrast material is too dense, intradural structures may be masked. Rolling the patient once or twice before scanning is recommended to mix the contrast material that may have settled since the myelogram.

BOX 19-5 Key Concept

When CT is performed after intrathecal contrast administration for fluoroscopic myelography, a delay of 1 to 3 hours between the contrast injection and scanning is recommended. This delay allows the contrast material to become sufficiently dilute.

Proper localization is essential in scanning the spine. All studies should include scout images in both anteroposterior (AP) and lateral projections. The scouts will permit vertebral levels to be readily counted and classified to ensure that scans are taken at the appropriate levels. When scanning the lumbar spine, it is important to note whether the patient has a sixth lumbar vertebra (an anatomic variant) that requires additional scans.

SPECIFIC NEUROLOGIC PROTOCOLS

Concepts learned in previous chapters can be applied to scanning the head, neck, and spine. Neurologic CT protocols are often designed to meet the needs of a specific structure or organ (i.e., circle of Willis) or to adapt to a particular clinical indication (i.e., suspected spinal arteriovenous malformation). Many factors must be considered in the development of a scanning protocol. The following detailed discussion of stroke and CT brain perfusion is presented here to provide the reader with a broader understanding of the many issues that may be taken into consideration in the design of CT protocols. Examples of various protocols are provided at the end of the chapter.

A Profile of Stroke

Introduction

The American Heart Association reports that cerebrovascular disorders such as ischemic and hemorrhagic strokes constitute the third most frequent cause of death in North America. Equally alarming, stroke is the leading cause of long-term disability. Approximately 20 to 30% of stroke victims do not survive, and 55% of stroke survivors have a disability.⁵ Despite the significant advances in the treatment of stroke in recent years, cerebrovascular disorders continue to pose a considerable challenge to acute neurovascular management.

Therapeutic options, such as thrombolytic therapy, can limit the extent of brain injury and improve outcome after stroke when administered to patients who fall within narrow clinical guidelines. Many of these therapies are only effective if given early after the stroke has begun, hence the criticality of emergent imaging. However, these therapies are expensive and may result in potentially life-threatening complications, drawbacks that make it crucial that each case be assessed by its individual risk-benefit ratio. Most important, the location and extent of the ischemic lesion, combined with the severity of the blood flow reduction, are the main factors that predict outcome in the treatment of stroke.⁶ These

factors demand an assessment of cerebral blood circulation to determine whether a conservative or a more aggressive therapy is needed in the early stage of stroke.

To adequately explain cerebrovascular disorders it is necessary to begin with an analysis of the basic physiologic processes that result in the origin and development of stroke. This review will define common terms, list symptoms, and present risk factors. In the following section we discuss CT perfusion, a newer diagnostic option in the assessment of acute stroke.

Overview

Although the brain receives approximately 25% of the body's oxygen supply, the brain lacks the capacity for its storage. Brain cells require a constant supply of oxygen to maintain health and functionality. Blood provides this continuous supply through two main arterial systems: 1) the carotid arteries and 2) the basilar artery, formed by the vertebral arteries. These systems are connected in a unique way. They terminate in the circle of Willis, a vascular structure located on the floor of the cranial cavity. The circle of Willis loops around the brainstem, above the pons, giving off the major vessels supplying the brain. In general, it provides collateral blood supply to the brain for multiple arteries; however, it may not be completely interconnected in some patients, and of course, it does not help in supplying blood if the vascular deficit is distal to the circle of Willis.

A reduction of blood flow for even a short period of time can be disastrous and is the primary cause of a stroke. A completed, or established, stroke is the preferred medical term used to describe an acute episode of interrupted blood flow to the brain that lasts longer than 24 hours. Most completed strokes reach a maximal neurologic deficit within an hour of onset.⁷ Terms used to describe stroke in the medical field include cerebrovascular accident (CVA) and apoplexy, which literally means, "struck with violence" or "being thunderstruck." These older terms are no longer favored because they imply a random, unpredictable, or uncertain nature to the condition.

A stroke in evolution, or progressive stroke, describes a time-limited event in which the neurologic deficits occur in a progressive pattern. In cases when the carotid artery distribution is affected, there is little chance it will progress beyond 24 hours. However, disruption involving vertebrobasilar distribution may continue to progress for up to 72 hours.⁸

Types of Stroke

Stroke may be divided into two main categories: ischemic, caused by a blockage in an artery, and hemorrhagic, caused by a tear in the artery's wall that produces bleeding in the brain. A third less-prevalent type is hypotensive, which occurs as a result of blood pressure that is too low.

Ischemic Stroke Ischemic strokes are by far the most common, accounting for 80% of all strokes. Ischemia is defined as a deficiency of oxygen in vital tissues. Two main

types of ischemic stroke exist: 1) thrombotic, caused from a blood clot or a fatty deposit within one of the brain's arteries; and 2) embolic, resulting from a traveling particle that forms elsewhere and is too large to pass through small vessels and eventually lodges in a smaller artery. In addition to the two main types, a less acute form of ischemic stroke exists. This form is referred to as a lacunar stroke.

Thrombotic Stroke Thrombotic strokes occur when a clot that forms as the result of atherosclerosis blocks an artery that supplies blood to the brain. This condition is progressive and can be summarized as follows:

- Arterial walls slowly thicken, harden, and narrow (creating a stenosis) until blood flow is reduced.
- The abnormal arteries become vulnerable to injury, initiating an inflammatory response. It is hypothesized that this response plays a significant role in the evolution of stroke.
- The immune system reacts to the arterial injuries by releasing white blood cells at the site, specifically neutrophils and macrophages.
- Macrophages digest foreign debris, turning them into foamy cells that attach to the smooth muscle cells of the blood vessels, causing a buildup of these cells.
- Sensing further harm, the immune system releases factors called cytokines, which attract more white blood cells and perpetuate the entire cycle.
- These processes impede blood flow.
- To make matters worse, the injured inner walls of vessels fail to produce enough nitric oxide, a substance critical for maintaining blood vessel elasticity. Without adequate nitric oxide, arteries become calcified and lose elasticity.
- Hardened and rigid arteries are even more susceptible to injury. If they tear, a thrombus (clot) may form.
- If that happens, a thrombus could completely block the already narrowed artery, preventing oxygen from reaching a part of the brain, and stroke occurs.

Common focal sites of cerebrovascular atherosclerosis include the proximal common carotid artery, the origin of the internal carotid artery, the carotid siphon, and the proximal middle cerebral and vertebral arteries (Figs. 19-3, 19-4).

Embolic Stroke An embolic stroke is caused when an artery in the brain is suddenly blocked by embolic material, which is usually a thrombus that developed elsewhere in the body. The embolus travels within the cervicocranial arteries until it becomes wedged in an artery in the brain. If the embolic material lodges for very long, the resulting reduced blood flow, or hypoperfusion, results in an infarct. This infarct may become hemorrhagic when the embolus moves, or fragments, and reperfusion occurs.

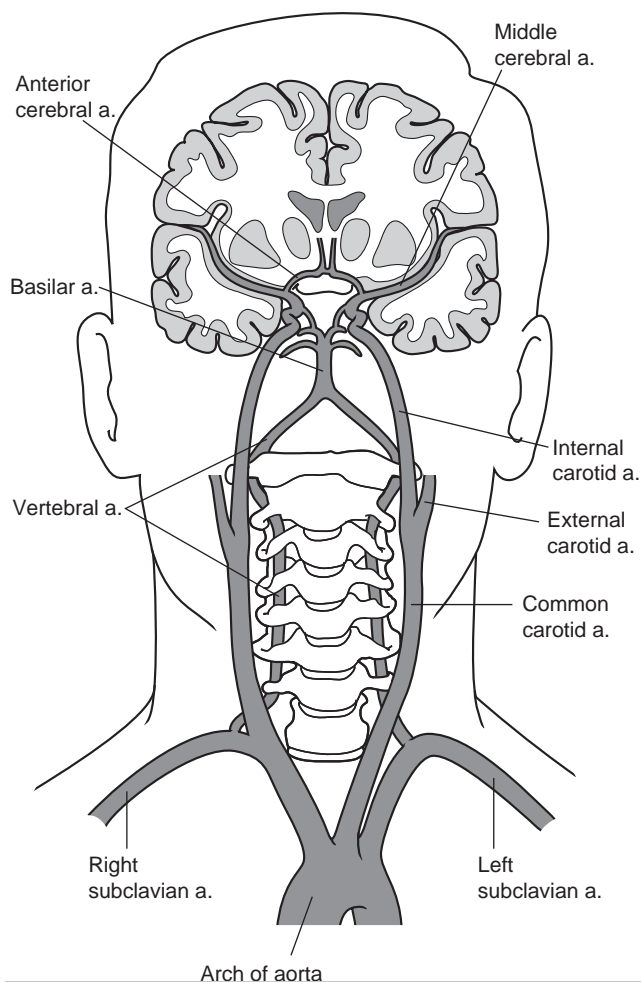


FIGURE 19-3 Branches of the aortic arch (anterior view).

The clinical findings depend on the location of the brain artery affected.

Emboli account for about 60% of strokes and may result from the following conditions:

- Atrial fibrillation, an abnormal rhythm (rapid quivering beat) in the atrium of the heart. The irregular beat may cause some blood to remain in the heart chamber where it forms clots, which then can break off and travel to the brain as emboli. Blood clots originally formed as a result of atrial fibrillation comprise approximately 15% of embolic strokes.
- At the site of artificial heart valves or as a result of heart valve disorders.
- After a heart attack or in association with heart failure, as a result of areas of the ventricles that do not move properly, allowing blood to stagnate and clot.
- From fat particles, tumor cells, or air bubbles that travel through the bloodstream. This type of emboli is rare.

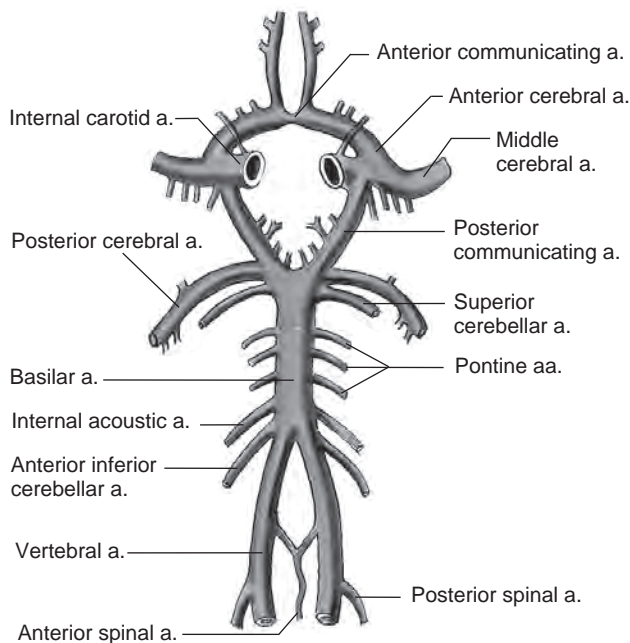


FIGURE 19-4 Circle of Willis.

Lacunar Stroke Small arteries, such as those that supply the deep cerebral white matter, are also susceptible to atherosclerotic changes. A lacunar stroke occurs when one of these small arteries is blocked. In these deep parts of the brain, no other blood vessels exist that can help supply blood to that region. Therefore, a blockage results in tissue death in that area. The word lacuna describes a small, hollow cavity or pit. After a lacunar stroke, the tiny infarcts that result have been described as hollowed out—thus the name lacunar.

Lacunar infarcts are a series of very tiny ischemic strokes. Symptoms present in the patient as clumsiness, weakness, and emotional variability. Lacunar stroke accounts for 25% of strokes and can sometimes serve as a warning sign that a major stroke may be imminent.

Hemorrhagic Stroke Rupture of a blood vessel in the brain causes leakage of blood into the brain parenchyma, CSF spaces around the brain, or both. Approximately 20% of strokes occur from hemorrhage. Hemorrhagic strokes are classified by how and where they occur.

BOX 19-6 Key Concept

Rupture of a blood vessel in the brain causes leakage of blood into the brain parenchyma, CSF spaces around the brain, or both.

Intracerebral Hemorrhage Intracerebral hemorrhagic strokes occur within the brain parenchyma itself, and a hematoma often results. These strokes account for

more than half of hemorrhagic strokes. Most often, they result from hypertension, which exerts excessive pressure on arterial walls already damaged by atherosclerosis. Heart attack patients who have been given drugs either to break up blood clots or to “thin blood” to reduce the likelihood of developing clots have a slightly elevated risk of this type of stroke.

Subarachnoid Hemorrhage Subarachnoid hemorrhagic strokes occur when there is bleeding into the subarachnoid spaces and the CSF spaces. These strokes are usually caused by the rupture of an aneurysm.

Arteriovenous Malformation Arteriovenous malformations (AVMs) are composed of tangles of arteries and arterialized veins. There is brain tissue interposed between the vessels, but it is usually abnormal and often scarred from previous tiny hemorrhages. The patient may not have been aware of these tiny hemorrhages or may have experienced them as bad headaches.

Normally, blood enters brain tissue through major cerebral arteries, and then passes through smaller arterioles and subsequently into the capillary bed. It is at the capillary level that the exchange of oxygen and glucose in brain cells occurs. A drop in pressure usually occurs as the blood travels from the arteries to the veins. The blood, depleted of oxygen, then enters the venous system to continue its systemic circulation.

When an AVM exists, blood is shunted directly from the arterial system to the venous system. This shunting allows oxygenated blood to enter the veins. In AVMs, the flow is high and the pressure is elevated within the veins. The elevated pressure can cause the vessels to rupture, resulting in a hemorrhagic stroke.

Hypotensive Stroke Although rare, blood pressure that is too low can reduce oxygen supply to the brain enough to cause a stroke. Systemic hypoperfusion results when blood flow to the brain is lowered to a level too severe to be compensated by cerebral autoregulation mechanisms. These hypotensive episodes can be caused by cardiac pump failure, a major bleeding episode that causes a global decrease in cerebral blood flow, an overwhelming infection, or although rarely, from surgical anesthesia or overtreated high blood pressure. Hypotensive episodes cause infarction in the border zones between the major cerebral arteries, sometimes referred to as a watershed infarction, as well as widespread bilateral cerebral dysfunction. The primary areas of damage are within the circle of Willis between the anterior and middle cerebral arteries, and between the middle and posterior cerebral arteries in the parieto-occipital regions of the cerebral hemispheres⁶ (Fig. 19-4).

Symptoms of Stroke

Transient Ischemic Attacks A transient ischemic attack (TIA) is a reversible episode of focal neurologic dysfunction that typically lasts anywhere from a few minutes to a

few hours. Attacks are usually caused by tiny emboli that lodge in an artery and then quickly break up and dissolve, with no residual damage.

TIAs are a significant indicator of stroke risk. Approximately 5% of patients who experience TIAs go on to suffer a stroke within a month. Without treatment, one-third of these patients will experience strokes within 5 years. Because of the relationship between atherosclerosis, coronary artery disease, and stroke, TIAs are also warning signs for a heart attack.

The clinical presentation of TIAs varies slightly depending on whether the carotid or basilar artery is involved.

Symptoms of TIAs in the Carotid Arteries The carotid arteries are the more common sites of TIAs. The carotid arteries supply blood to the retinal artery. Emboli here cause symptoms originating in either the retina or the cerebral hemisphere. Reduction of oxygen to the eye results in a visual effect often described as a “shade being pulled down.” Poor night vision is another manifestation of carotid artery TIA. When the cerebral hemisphere is affected, the patient may experience problems with speech, partial and temporary paralysis, tingling, and numbness, typically on one side of the body.

Symptoms of TIAs in the Basilar Artery When TIAs result from occlusion of the basilar artery, both hemispheres of the brain may be affected. Therefore, symptoms often occur on both sides of the body and include the following:

- Temporarily dim, gray, blurry, or lost vision in both eyes
- Tingling or numbness in the mouth, cheeks, or gums
- Headache in the back of the head
- Dizziness
- Nausea and vomiting
- Difficulty swallowing
- Inability to speak clearly
- Weakness in the arm and legs, sometimes causing a sudden fall

Symptoms of a Major Ischemic Stroke The onset of symptoms in a major ischemic stroke may vary, depending on the source. If the stroke is caused by a large embolus that has traveled to and lodged in an artery in the brain, the onset is sudden. Headache and seizures can occur within seconds of the blockage. If the stroke results from a thrombosis that has formed in a narrowed artery, symptoms manifest more gradually, during minutes to hours. On rare occasions the onset can progress from days to weeks.

The symptoms of a major ischemic stroke can be extremely variable. Early symptoms can be identical to those of a TIA, because the clot may block a branch of the carotid or basilar arteries. However, unlike a TIA, the symptoms do not resolve. A thrombosis on one side of the brain usually affects the opposite side of the body,

with possible unilateral weakness, loss of feeling on one side of the face or in an arm or leg, or blindness in one eye. If the left hemisphere of the brain is affected, speech problems often occur. (In some patients, mostly those who are left-handed, a clot on the right side of the brain can affect speech.) Other symptoms may include an inability to express thoughts verbally or understand spoken words. The victim may experience major seizures and possibly coma.

Symptoms of Hemorrhagic Stroke The symptoms of hemorrhagic stroke depend, to some extent, on where and how the hemorrhage occurs. Symptoms from cerebral hemorrhage usually begin very suddenly and evolve over the course of several hours. They include headache, nausea and vomiting, and altered mental state. A subarachnoid hemorrhage may produce warning signs from the leaky blood vessel a few days to a month before the aneurysm fully develops and ruptures. Warning signs may include abrupt headaches, nausea and vomiting, and sensitivity to light. When the aneurysm ruptures, the stroke victim may experience a terrible headache; neck stiffness; vomiting; an altered state of consciousness; eyes that become fixed in one direction or a loss of vision; or stupor, rigidity, and even coma.

Symptoms of Hypertensive Stroke Symptoms resulting from hypertensive stroke are loss of vision, decreased alertness, and weakness that affects predominantly the shoulder, hand, and thigh.

Silent Brain Infarctions As many as 31% of elderly patients experience silent brain infarctions.⁷ These are small strokes that cause no apparent symptoms but are major contributors to changes in mental status in the elderly. Smokers and individuals with hypertension are at particular risk.

Stroke Risk Factors

Hypertension Hypertension contributes to 70% of all strokes. It is estimated that controlling blood pressure could prevent nearly half of all strokes.

Homocysteine and Vitamin B Deficiencies Abnormally high blood levels of the amino acid homocysteine, which occur with deficiencies of vitamin B₆, B₁₂, and folic acid, have been linked to an increased risk of coronary artery disease and stroke. Some experts believe that homocysteine is a major risk factor for stroke, second only to hypertension.^{9,10}

Cholesterol and Other Lipids It is well accepted that an unhealthy cholesterol balance plays a major role in atherosclerosis and consequent heart disease. However, the role cholesterol plays in stroke is less clear.

Atrial Fibrillation Atrial fibrillation is a disorder of heart rate and rhythm in which the atria are stimulated

to contract in a very rapid or disorganized manner rather than in an organized one. For individuals suffering from atrial fibrillation, their risk of stroke increases sixfold. By the age of 70, 10% of adults have this disorder.

Heart Disease A diseased heart increases the risk of stroke. In fact, people with heart problems have more than twice the risk of stroke compared with people whose hearts work normally.⁵ Heart disease and stroke are interconnected for many reasons. Chief among them is that the two disorders share common risk factors, including hypertension and diabetes.

Diabetes Mellitus Diabetes is a strong risk factor for ischemic stroke, perhaps because of often associated factors such as obesity, high cholesterol levels, and hypertension. Diabetes does not appear to increase the risk of hemorrhagic stroke.

Migraine Migraine sufferers, both men and women, have an increased risk for stroke, particularly before age 50. However, it is important to note that many people have migraines, and their risk is still low—2.7% for women and 4.6% for men, according to one study.¹¹ In both men and women with a history of migraines, the increased risk diminished with age.

Other Medical Conditions A number of other medical conditions may contribute to the risk of stroke. These include sleep apnea, the presence of antibodies called antiphospholipids, and sickle cell anemia. Pregnancy also carries a very small risk, mostly in women with pregnancy-related hypertension and women delivering via cesarean section.

Heredity An individual's genetic makeup may be responsible for many of the processes leading to stroke. Studies indicate that a family history of stroke, particularly on the paternal side, is a strong risk factor.

Smoking People who smoke a pack of cigarettes daily are at almost 2.5 times the risk of stroke than that of nonsmokers. The nicotine and carbon monoxide in cigarette smoke damage the cardiovascular system in many ways. The risk is proportional to the dose, that is, the heavier the smoking, the higher the risk. In addition, birth control pills and smoking have a synergistic risk effect. Thus, women who take birth control pills and smoke significantly increase their risk of stroke. Quitting smoking significantly reduces the risk of stroke.

Obesity Obesity is associated with stroke, most likely because being overweight reflects the presence of other risk factors, including insulin resistance and diabetes, hypertension, and unhealthy cholesterol levels. How a person carries extra weight also influences the risk of stroke. Weight that is centered around the abdomen

(i.e., “apple shape”) has a greater association with stroke than weight distributed around the hips (i.e., “pear shape”).

Alcohol and Drug Abuse Heavy alcohol use is associated with a higher risk of both ischemic and hemorrhagic stroke. However, studies have indicated that moderate alcohol use does not increase an individual’s risk of stroke and may even lower the risk of ischemic stroke.¹²

Intravenous drug abuse carries a high risk of stroke from cerebral embolism. Cocaine and methamphetamine are major factors in the incidence of stroke in young adults. Steroids used for bodybuilding also increase stroke risk.

Diagnosis and Treatment of Stroke

Although a detailed review of the many issues and options surrounding the treatment of stroke is beyond the scope of this text, it is worthwhile to touch on advances made in the past decade that have increased the importance of CT imaging in the diagnosis and treatment of stroke.

In June 1996, the U.S. Food and Drug Administration (FDA) approved a treatment for acute ischemic stroke. The treatment, known as tissue plasminogen activator (t-PA), was the first of its kind and revolutionized the way the medical community can respond to treating the 80% of stroke patients who experience ischemic strokes. t-PA is the only FDA-approved treatment for acute ischemic stroke. One dose of t-PA is required to treat an acute stroke patient at an average cost of approximately \$2,000 per dose. Despite the high cost, the treatment has been shown to reduce disability, leading to an overall cost savings in hospital, nursing home, and rehabilitation care expenses.

To be effective, t-PA must be administered within 3 hours of the first signs of stroke. This means that the stroke victim must be transported to the hospital, diagnosed, and administered the t-PA treatment before the 3-hour window has expired. Unfortunately, only a small percentage of patients who suffer a stroke reach the hospital in time to be considered for the t-PA treatment.¹³

BOX 19-7 Key Concept

To be effective t-PA must be administered within 3 hours of the first signs of stroke. This means that the stroke victim must be transported to the hospital, diagnosed, and administered the t-PA treatment before the 3-hour window has expired.

The target of t-PA therapy is the tissue known as the penumbra. After an acute ischemic stroke, areas of tissue death (infarction) occur because of a local lack of oxygen. The penumbra is ischemic tissue that (without successful intervention) is destined for infarction, but is not yet irreversibly injured. It is the penumbra that may be salvageable with the administration of t-PA. The fully infarcted tissue will not

benefit from reperfusion after t-PA and may be at increased risk of hemorrhage. However, if reperfusion of the penumbra occurs expeditiously, this tissue will recover and the patient improves. This is why the timing of t-PA therapy is so critical. Stroke experts commonly refer to the sense of urgency in stroke treatment with the expression “time is brain.”

Published guidelines recommend that the time between arriving at the emergency room to actual treatment should be 60 minutes or less.¹⁴ During this hour, various tests must be performed, including a neurologic examination, blood tests, and a CT scan of the head to determine whether hemorrhage has occurred and contraindicates t-PA.

BOX 19-8 Key Concept

Evidence of intracranial hemorrhage on pretreatment non-contrast head CT contraindicates t-PA therapy.

Even assuming that all the appropriate diagnostic testing can be accomplished in an hour, this leaves only 2 hours for recognition of the symptoms of stroke and transportation to the appropriate emergency room.

Fast diagnosis of both the presence and type of stroke is critical in saving lives and reducing the likelihood of severe disability. Patients must be carefully selected because the treatment itself can cause bleeding that can be lethal if the stroke is hemorrhagic in origin. Criteria that exclude patients from receiving t-PA are included in Table 19-2.

Although MRI of the head can also be used to evaluate stroke patients for possible thrombolytic therapy, CT is used more frequently. This is because the imaging tool must be used quickly, and CT is much more available than MRI. In addition, noncontrast CT is regarded as more practical because the imaging time is shorter, it is better tolerated by many patients, there are no contraindications as may occur for MRI, and all relevant information is provided with only one imaging sequence. A noncontrast CT of the brain is routinely performed to differentiate ischemic stroke from hemorrhagic stroke, to assess the state of cerebral circulation and tissue, and, secondarily, to assess the underlying disease.

BOX 19-9 Key Concept

A noncontrast CT of the brain is routinely performed to differentiate ischemic stroke from hemorrhagic stroke, to assess the state of cerebral circulation and tissue, and, secondarily, to assess the underlying disease.

An unenhanced CT is an effective tool in determining the presence of intracranial bleeding and therefore well meets the first goal of imaging. However, in the first 6 hours after onset of an ischemic stroke, an unenhanced

Table 19-2 Contraindications to t-PA¹³

<p>Evidence of intracranial hemorrhage on pretreatment noncontrast head CT</p> <p>Clinical presentation suggestive of subarachnoid hemorrhage, even with normal CT</p> <p>CT shows multilobar infarction (hypodensity greater than one-third cerebral hemisphere)</p> <p>History of intracranial hemorrhage</p> <p>Uncontrolled hypertension: At the time treatment should begin, systolic pressure remains > 185 mm Hg or diastolic pressure remains > 110 mm Hg despite repeated measurements</p> <p>Known arteriovenous malformation, neoplasm, or aneurysm</p> <p>Witnessed seizure at stroke onset</p> <p>Active internal bleeding or acute trauma (fracture)</p> <p>Acute bleeding diathesis, including but not limited to:</p> <ul style="list-style-type: none"> Platelet count < 100,000/mm³ Heparin received within 48 hours, resulting in an activated partial thromboplastin time (aPTT) that is greater than upper limit of normal for laboratory Current use of anticoagulant (e.g., warfarin sodium) that has produced an elevated international normalized ratio (INR) > 1.7 or prothrombin time (PT) > 15 seconds <p>Within 3 months of intracranial or intraspinal surgery, serious head trauma, or previous stroke</p> <p>Arterial puncture at a noncompressible site within past 7 days</p>
--

CT will remain normal in about one-third of patients because the ischemia does not reach the critical level of structural integrity. Studies such as CT perfusion and CT angiography are sometimes done in addition to the non-contrast CT scan to address the other goal of imaging, to assess the state of the cerebral circulation and tissue. CT perfusion techniques measure cerebral blood flow, whereas CT angiography of the carotid arteries and vessels of the circle of Willis can demonstrate stenosis or occlusion of extracranial and intracranial arteries.

CT Brain Perfusion Scans

Various attempts have been made to establish a CT method to allow qualitative and quantitative evaluation of cerebral perfusion. CT perfusion provides this information by calculating regional blood flow (rCBF) and regional blood volume (rCBV) and mean transit time (MTT). Perfusion studies are obtained by monitoring the passage of iodinated contrast through the cerebral vasculature. Attractive characteristics of this approach are the widespread availability of CT scanners, their high image quality, and relatively low costs. In addition, simply extending the routine CT examination eliminates time-consuming transport of patients between CT and MR scanners that serves to further delay treatment.

BOX 19-10 Key Concept

Perfusion studies are obtained by monitoring the passage of iodinated contrast through the cerebral vasculature.

CT perfusion software that interfaces with helical scanners can meet the clinical needs for evaluation of acute and subacute stroke. Such applications produce a quantitative measure of regional hemodynamics by demonstrating blood flow in each pixel of the cerebral

parenchyma that is imaged. The technique is based on the central volume principle, which states that cerebral blood volume can be calculated as the product of the total cerebral blood flow and the time needed for the cerebral blood passage: $CBF = CBV/MTT$. A workstation equipped with commercially available perfusion software can perform these complex calculations quickly. The goal in performing perfusion studies for patients with acute stroke is to distinguish infarcted tissue from the penumbra.

Technical Factors

The most common technique associated with CT perfusion scanning is based on the first pass of a contrast bolus through the brain tissue. With this technique a 50-mL IV bolus of a nonionic low osmolality contrast is injected at 4 to 5 mL/s. A helical scanner is used to produce a dynamic set of images at a single location. A 5-second scan delay is used; slices are typically 5 mm thick. Typical scan durations are in the range of 40 to 45 seconds.

The slices are produced by repeatedly scanning the same region at the same table position, a technique some manufacturers refer to as the cine mode. Multislice scanners allow several z position slices to be scanned simultaneously. Scans are typically acquired at 5-mm sections to lessen beam-hardening artifacts, and then reformatted into 10-mm-thick sections for viewing to improve the signal-to-noise ratio.

The brain perfusion protocol begins with an unenhanced scan of the whole brain. Although the level for scanning the enhanced portion of the study may be selected at the time of examination based on the unenhanced CT findings, a transverse slice through the level of the basal ganglia (Fig. 19-5) contains territories supplied by the anterior, middle, and posterior cerebral arteries, thus offering the opportunity to interrogate each of the major vascular regions.

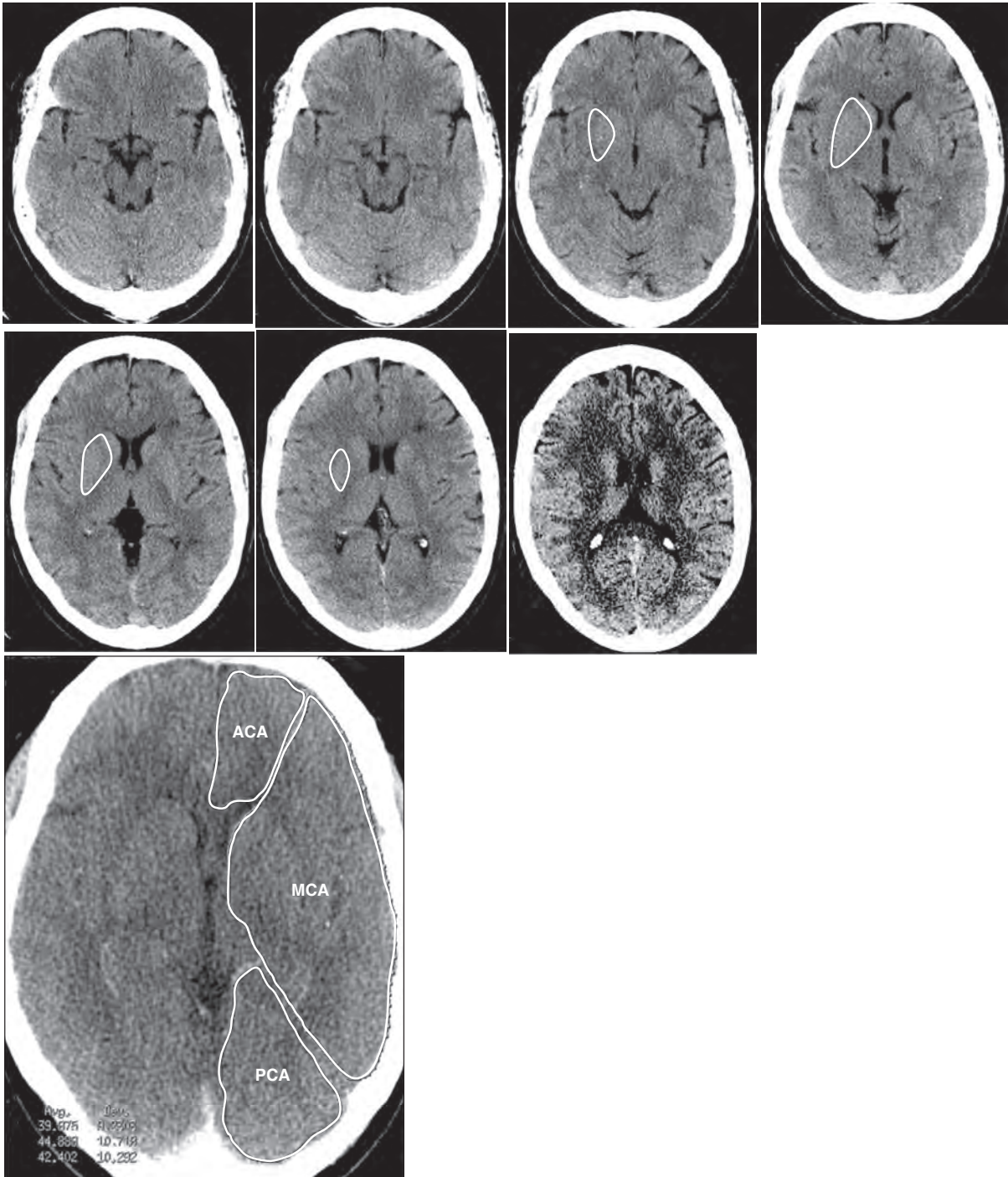


FIGURE 19-5 The level of the basal ganglia contains territories supplied by the anterior (ACA), middle (MCA), and posterior cerebral arteries (PCA). Therefore, this is the level most frequently scanned for brain perfusion studies. The outlined areas on the upper two rows of images are the basal ganglia. Images courtesy of the University of Michigan Health System.

A major limiting factor of older helical scanners was the 10- to 20-mm maximum anatomic coverage. Often a second bolus of contrast was necessary so that the patient could be scanned at a different location, typically in a more cephalad direction above the lateral ventricles. However, as scanners with expanded multidetector arrays have become more prevalent, this is no longer an issue. For

example, a 64-slice scanner will allow 40 mm of anatomic coverage for the perfusion study.

The first step in postprocessing the acquired images using the perfusion software is to select a reference artery and a reference vein. The reference artery should be 1) seen in cross-section, 2) one of the first to enhance, 3) produce a curve with a high enhancement peak, or 4) produce a

curve with a narrow width. The most common choice of reference artery is the anterior cerebral artery. The reference vein should be the largest venous structure available or one that produces an enhancement curve with the highest peak. The superior sagittal sinus is the vein most often used as the reference. An ROI is placed on the reference artery and the reference vein so that contrast-enhancement curves can be generated.

Analysis of the contrast enhancement curves guides the selection of preenhancement and postenhancement images (Fig. 19-6). The preenhancement image is that last image before contrast arrives. The postenhancement image is the point immediately after the first pass of the contrast bolus when the time-attenuation graph begins to flatten.

The perfusion software then generates color-coded maps demonstrating:

- Regional cerebral blood volume, or rCBV
- Blood mean transit time, or MTT, through cerebral capillaries
- Regional cerebral blood flow or rCBF

Quantitative data can be extracted from the maps by placing multiple ROIs in the brain parenchyma (Fig. 19-7).

Brain perfusion studies are frequently ordered with a CTA of the circle of Willis or the carotid arteries. In these situations the CTA should be performed first; the brain perfusion study can immediately follow.

Clinical Indications

CT perfusion is most frequently ordered in the evaluation of acute stroke. It is also ordered for vasospasm and tumor grading. In addition, the CT perfusion technique can be modified to determine cerebrovascular reserve and can be used in conjunction with temporary balloon occlusion protocols.

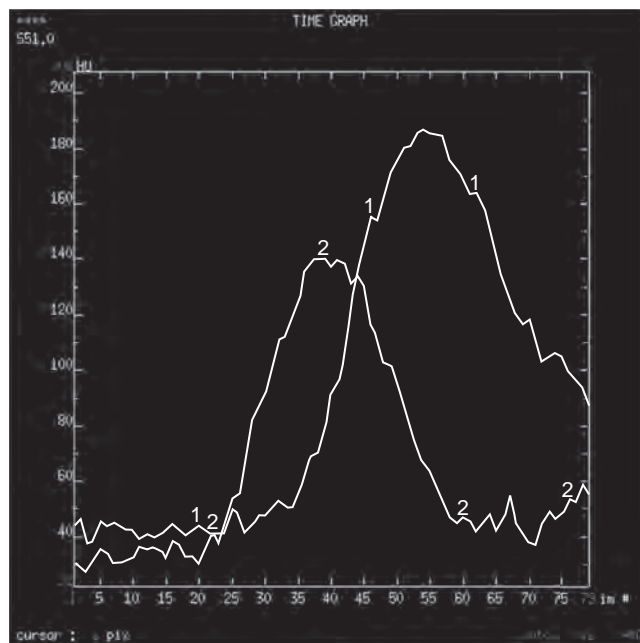


FIGURE 19-6 The contrast enhancement curves guides the selection of preenhancement and postenhancement images.

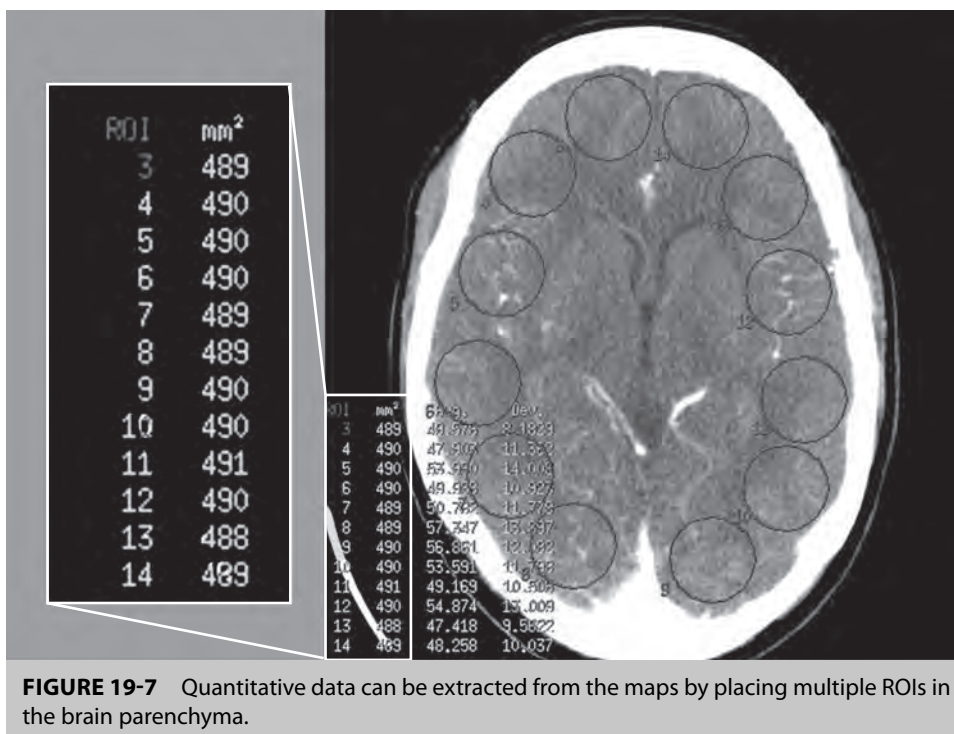


FIGURE 19-7 Quantitative data can be extracted from the maps by placing multiple ROIs in the brain parenchyma.

BOX 19-11 Key Concept

CT perfusion is most frequently ordered in the evaluation of acute stroke, but is also ordered for vasospasm or tumor grading. With some modification CT perfusion can also be used to determine cerebrovascular reserve during temporary balloon occlusion protocols.

CT Perfusion to Determine Cerebrovascular Reserve CT brain perfusion studies can be used to evaluate patients with chronic cerebral ischemia related to underlying vascular stenosis.¹⁵ In these situations, CT perfusion is used to determine cerebrovascular reserve capacity (CVRC). CVRC describes how far cerebral perfusion can increase from a baseline value after undergoing stimulation. It is essentially a “stress test” for the brain. Stimulation is provided through the intravenous administration of a drug such as acetazolamide (Diamox; Wyeth, Marietta, PA).

To evaluate CVRC a routine perfusion CT is performed. The patient is then given the acetazolamide, and the perfusion CT is repeated 20 minutes later with a second contrast injection. Pre and post acetazolamide scans should be acquired at the same anatomic level. ROIs on the resulting perfusion color maps should be placed in the same positions on pre and post acetazolamide scans. An allergy to sulfa drugs is a contraindication for acetazolamide. Acetazolamide is associated with certain side effects (e.g., weakness, numbness, bronchospasm); therefore, the patient should be monitored for at least 30 minutes after its administration.

CT Perfusion in Conjunction With Temporary Balloon Occlusion Balloon occlusion is a treatment option for some patients with aneurysm or intracranial or head and neck tumors. Before permanent occlusion is performed a temporary test occlusion is performed in the angiography suite to determine whether the patient can tolerate permanent occlusion. However, some patients who pass the temporary balloon occlusion test may still not tolerate permanent occlusion.¹⁶ Performing perfusion CT in conjunction with the temporary balloon occlusion test can help to identify these patients. The procedure is summarized as follows:

1. Patient undergoes angiography and balloon occlusion. Patients who pass the clinical portion of the examination are brought to the CT suite with the balloon in place.
2. A routine unenhanced head CT is performed.
3. The balloon is inflated by the neurointerventionalist and perfusion CT is performed.
4. The balloon is deflated and perfusion CT is repeated with a second contrast injection.

Summary

Dynamic perfusion CT imaging is a fast and effective means of determining cerebral perfusion. Combined with

unenhanced CT and CT angiography, it provides a means for prompt assessment of vascular anatomy and regional hemodynamics. CT perfusion is a valuable tool in the assessment of patients with acute stroke but can also be used for other cerebrovascular disease.

CHAPTER SUMMARY

CT is instrumental in the diagnosis of neurologic abnormality or the evaluation of injury to the head, sinuses, temporal bone, neck, and spine. The introduction of multislice CT realized the advantages of wider scanning range, shorter scanning time, and finer z axis resolution, all useful factors in neuroradiologic diagnosis. CT is the primary imaging modality for emergent indications such as trauma and acute changes in neurologic status, including ischemia and intracranial hemorrhage. CTA has become an attractive alternative to digital subtraction angiography for rapid evaluation of the cervical and cerebral vasculature. CT also provides an accurate display of the anatomy and pathology of the spine. The value of CT with intrathecal contrast medium injection is well accepted, and the use of CT as a supplemental study after routine fluoroscopic myelography is widely used in spine evaluation.

REVIEW QUESTIONS

1. When performing a CT study of the brain, what effect will moving the patient’s chin up or down have?
2. Describe how a patient can be positioned so that data can be acquired of the head in the coronal plane.
3. Describe the appearance of intracranial hemorrhage on the CT image.
4. Why might the split bolus method of contrast administration be used for CT studies of the neck? Describe the technique.
5. CT scans of the spine are often performed after a myelogram. What special considerations must be given for the intrathecal contrast material?
6. Explain why CT plays such a crucial role in the assessment and treatment of acute stroke.
7. What information do CT brain perfusion scans provide that is not available on unenhanced CT of the brain?

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NEUROLOGIC PROTOCOLS

Head		
Examples of clinical indications: Without contrast: intracranial hemorrhage, early infarction, dementia, hydrocephalus, cerebral trauma		
Without and with contrast: Mass, lesion, arteriovenous malformation, metastasis, aneurysm, for symptoms of headache, seizure		
Scouts: AP and lateral		
Scan type: Axial		
Scan plane: Transverse		
Start location: Just below skull base		
End location: Just above vertex		
IV contrast: (if contrast is ordered) 100 mL at 1.0 mL/s. Scan delay = 5 minutes		
Oral contrast: None		
Reference angle: Angle gantry parallel to supraorbital meatal line (avoid lens of eyes)		
DFOV: ~23 cm		
SFOV: Head		
Algorithm: Standard		
Window settings: 140 ww/40 wl posterior fossa; 90 ww/35 wl vertex		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	2.0 s	1.0 s
Acquisition (detector width × number of detector rows = coverage)	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	5.0 mm/5 mm (2 images per rotation)	5.0 mm/5 mm (4 images per rotation)
kVp/mA (posterior fossa)	140/150	140/330
kVp/mA (vertex)	120/150	120/330
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: ~23		
Slice thickness/interval	2.5 mm/2.5 mm	2.5 mm/2.5 mm

Skull Base (Posterior Fossa)		
Examples of clinical indications: Posterior fossa and brainstem tumors, hemorrhages, AVM, dural sinus, thrombosis		
Scouts: AP and lateral		
Scan type: Axial		
Scan plane: Transverse (coronal images may also be of benefit)		
Start location: Foramen magnum		
End location: Through petrous ridges		
IV contrast: (if contrast is ordered) 100 mL at 1.0 mL/s. Scan delay = when all contrast is administered		
Oral contrast: None		
Reference angle: Angle gantry parallel to infraorbital meatal line		
DFOV: ~23 cm		
SFOV: Head		

(Continued)

Skull Base (Posterior Fossa) (Continued)		
Algorithm: Standard		
Window settings: 140 ww/40 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	2.0 s	1.0 s
Acquisition (detector width × number of detector rows = coverage)	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	1.25 mm/1.25 mm (8 images per rotation)	1.25 mm/1.25 mm (16 images per rotation)
kVp/mA	140/170	140/340
Reconstruction 2:		
Algorithm: Bone		
DFOV: 18–20		
Window settings: 4000 ww/400 wl		
Slice thickness/interval	1.25 mm/1.25 mm	1.25 mm/1.25 mm

Temporal Bones		
Examples of clinical indications: Without contrast: cholesteatoma, inflammatory disease, fractures, evaluate implants With contrast: IAC tumor, hearing loss, acoustic neuroma, Schwannoma		
GROUP 1.		
Scouts: AP and lateral		
Scan type: Axial		
Scan plane: Transverse		
Start location: Just below the mastoid process		
End location: Just above petrous ridge (include entire mastoid, internal auditory canal, and external auditory canal)		
IV contrast: (if contrast is ordered) 100 mL at 1.0 mL/s. Scan delay = when all contrast is administered		
Oral contrast: None		
Reference angle: Angle gantry parallel to infraorbital meatal line (be sure patient's head is straight and not rotated in the head holder)		
DFOV: ~9.6 cm (center RAS coordinates for right side ~R35)		
SFOV: Head		
Algorithm: Bone		
Window settings: 4000 ww/ 400 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	1.0 s	1.0 s
Acquisition (detector width × number of detector rows = coverage)	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	0.625 mm/0.625 mm (16 images per rotation)	0.625 mm/0.625 mm (32 images per rotation)
kVp/mA	140/170	140/170
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
GROUP 2.		
Scout: Lateral		

Scan type: Axial		
Scan plane: Coronal		
Start location: Just anterior to the TMJ		
End location: Just posterior to the mastoid (include entire mastoid, internal auditory canal, and external auditory canal)		
Reference angle: Angle gantry perpendicular to the infraorbital meatal line		
DFOV: ~9.6 cm (center RAS coordinates for right side ~R35)		
SFOV: Head		
Algorithm: Bone		
Window settings: 4000 ww/ 400 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	1.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	1.25 mm/1.25 mm (8 images per rotation)	1.25 mm/1.25 mm (16 images per rotation)
kVp/mA	140/200	140/200
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 9.6 cm (center RAS coordinates for left side ~L35)		
Slice thickness/interval	1.25 mm/1.25 mm	1.25 mm/1.25 mm

Sinus Screen		
<i>Sinus screening is intended as an inexpensive, accurate, and low radiation dose method for confirming the presence of inflammatory sinonasal disease. If confirmed and the patient will then have endoscopic sinus surgery, the coronal images provide a “road map” for the surgeon.</i>		
Examples of clinical indications: Recurrent or chronic sinusitis		
Scout: Lateral		
Scan type: Axial		
Scan plane: Coronal		
Start location: Mid sella		
End location: Through frontal sinus		
IV contrast: None		
Oral contrast: None		
Reference angle: Angle gantry perpendicular to the orbital meatal line		
DFOV: 16 cm		
SFOV: Head		
Algorithm: Standard		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Scan Type	Helical	Axial
Gantry rotation time	1.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm

(Continued)

Sinus Screen (Continued)		
Reconstruction (slice thickness/interval)	2.5 mm/2.5 mm (4 images per rotation)	2.5 mm/2.5 mm (8 images per rotation)
kVp/mA	120/150	120/150
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: ~23		
Slice thickness/interval	2.5 mm/2.5 mm	2.5 mm/2.5 mm

Trauma Facial Bones		
Examples of clinical indications: Characterization of facial fractures and soft tissue injury		
Scouts: AP and lateral		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just below mandible		
End location: Just above frontal sinus		
IV contrast: None		
Oral contrast: None		
Reference angle: Angle gantry parallel to infraorbital meatal line		
DFOV: 18 cm		
SFOV: Head		
Algorithm: Standard		
Window settings: 350 ww/ 50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.8 s	0.8 s
Acquisition (detector width × number of detector rows = coverage)	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	1.25 mm/0.625	1.25 mm/0.625
Pitch	0.562	0.531
kVp/mA	120/250	140/250
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 18		
Slice thickness/interval	1.25 mm/0.625 mm	1.25 mm/0.625 mm
Reformations:		
Coronal reformations using both standard and bone algorithm image data		
Window setting: 140 ww/40 wl (standard algorithm); 4000 ww/400 wl (bone algorithm)		
DFOV: ~18		
Thickness/spacing: 1.2 mm/0.6 mm		

Orbits		
Examples of clinical indications: Without contrast: trauma, foreign body With contrast: intraorbital masses, thyroid ophthalmopathy, inflammation, infection		
GROUP 1.		
Scouts: AP and lateral		
Scan type: Axial		
Scan plane: Transverse		
Start location: Just below orbital floor End location: Just above orbital roof		
IV contrast (if contrast is ordered): 100 mL at 1.0 mL/s. Split bolus—two 50-mL injections. Two-minute delay between injections; scans initiated once second injection is complete. Oral contrast: None		
Reference angle: Angle gantry parallel to infraorbital meatal line		
DFOV: 16 cm		
SFOV: Head		
Algorithm: Soft		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	1.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	2.5 mm/2.5 mm (4 images per rotation)	2.5 mm/2.5 mm (8 images per rotation)
kVp/mA	120/200	120/200
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 16 cm		
Slice thickness/interval	2.5 mm/2.5 mm	2.5 mm/2.5 mm
GROUP 2.		
Scout: Lateral		
Scan type: Axial		
Scan plane: Coronal		
Start location: Mid sphenoid sinus End location: Anterior frontal sinus		
Reference angle: Angle gantry perpendicular to the infraorbital meatal line		
DFOV: 16		
SFOV: Head		
Algorithm: Soft		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	1.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	2.5 mm/2.5 mm (4 images per rotation)	2.5 mm/2.5 mm (8 images per rotation)
kVp/mA	140/200	140/200

(Continued)

Orbits (Continued)		
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 16		
Slice thickness/interval	2.5 mm/2.5 mm	2.5 mm/2.5mm

Sella		
Examples of clinical indications: Pituitary mass, microadenoma, and parasellar masses		
GROUP 1.		
Scouts: AP and lateral		
Scan type: Axial		
Scan plane: Transverse		
Start location: Just below sellar floor		
End location: Through dorsum sellae		
IV contrast: 150 mL at 1.0 mL/s. Scan delay = 75 seconds		
Oral contrast: None		
Reference angle: Angle gantry parallel to infraorbital meatal line		
DFOV: 14 cm		
SFOV: Head		
Algorithm: Soft		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	2.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	1.25 mm/1.25 (8 images per rotation)	1.25 mm/1.25 mm (16 images per rotation)
kVp/mA	140/140	140/280
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 12 cm		
Slice thickness/interval	1.25 mm/1.25 mm	1.25 mm/1.25 mm
GROUP 2.		
Scout: Lateral		
Scan type: Axial		
Scan plane: Coronal		
Start location: Anterior clinoid		
End location: Through dorsum sellae		
Reference angle: Angle gantry perpendicular to the infraorbital meatal line		
DFOV: 12		
SFOV: Head		
Algorithm: soft		
Window settings: 350 ww/50 wl		

	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	2.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	1.25 mm/1.25 mm (8 images per rotation)	1.25 mm/1.25 mm (16 images per rotation)
kVp/mA	140/140	140/280
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 12		
Slice thickness/interval	1.25 mm/1.25 mm	1.25 mm/1.25 mm

Brain Perfusion		
Examples of clinical indications: Acute stroke, vasospasm, to determine cerebrovascular reserve, in conjunction with temporary balloon occlusion		
GROUP 1. Noncontrast Head Refer to Table 14-2 (If a brain perfusion is ordered with a circle of Willis (CoW), or a CoW/carotid study, scan the CoW or CoW/carotid first, then perform the brain perfusion examination.)		
GROUP 2. Contrast Scan		
Scan type: Cine (table increment = 0)		
Scan plane: Transverse		
Slice location: Center at the level of the basal ganglia		
IV contrast: 50 mL (370 concentration) at 4.0 mL/s; 20 mL saline flush at 4.0 mL/s. Scan delay = 5 seconds		
Reference angle: Same gantry tilt and RAS coordinates as the noncontrast portion		
DFOV: 25		
SFOV: Head		
Algorithm: Standard		
Window settings: 90 ww/35 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	1.0 s	1.0 s
Acquisition (detector width × number of detector rows) = coverage	1.25 mm × 16 = 20 mm	0.625 mm × 64 = 40 mm
Scan time (continuous scanning at the same table position)	50 s	50 s
Reconstruction (slice thickness/interval/rotation) *image reconstructed for each 0.5 s of tube travel	5.0 mm/5.0 mm/0.5 s (8 images per rotation)	5.0 mm/5.0 mm/0.5 s (16 images per rotation)
kVp/mA	80/200	80/200
GROUP 3. 2nd Contrast Scan With Diamox (when ordered)		
Diamox is administered by IV injection. 20 minutes after Diamox injection, scan is repeated. All parameters remain the same as group 2, including injection of another 50 mL of 370 concentration iodinated contrast media.		
After group 3 scans are complete, patient is escorted to the recovery area, where he or she will be monitored for 30 minutes. Page the neuroradiologist if patient exhibits any unexpected signs or symptoms, especially those indicative of a stroke.		

CTA—Circle of Willis		
Examples of clinical indications: Locate cerebral aneurysm or AVM in patients with SAH/ICH		
GROUP 1. Noncontrast Head Refer to Table 14-2		
GROUP 2. Arterial Phase Scan		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just above the frontal sinuses		
End location: Just below the skull base		
IV contrast: 60 mL (370 concentration) at 4.0 mL/s. 20 mL saline at 4.0 mL/s. Scan delay = from timing bolus (use carotid artery at approximately level of C4 for ROI)		
Oral contrast: None		
Reference angle: No gantry tilt		
DFOV: 25 cm		
SFOV: Head		
Algorithm: Standard		
Window settings: 140 ww/40 wl posterior fossa; 90 ww/35 wl vertex		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.5 s	0.5 s
Acquisition (detector width × number of detector rows) = coverage	1.25 mm × 16 = 20 mm	0.625 mm × 64 = 40 mm
Reconstruction (slice thickness/interval)	1.25 mm/0.625	1.25 mm/0.625
Pitch	0.938	0.984
kVp/mA	120/500	120/500
Reformations: Coronal and Sagittal		
Window setting: 140 ww/40 wl		
DFOV: ~18		
Slice thickness/spacing: 2.0 mm/2.0 mm		
Render mode: MIP		

CTA—Circle of Willis/Carotid		
Examples of clinical indications: Acute stroke, carotid atherostenosis, carotid dissections		
GROUP 1. Noncontrast Head Refer to Table 14-2		
Group 2. Arterial Phase Scan		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just below the aortic arch		
End location: Just above the frontal sinus		
IV contrast: 80 mL (370 concentration) at 4.0 mL/s; 40 mL saline at 4.0 mL/s, Scan delay = from timing bolus (use carotid artery at approximately level of C4 for ROI)		
Oral contrast: None		
Reference angle: No gantry tilt		

DFOV: 25 cm		
SFOV: Large body		
Algorithm: Standard		
Window settings: 250 ww/30 wl—through foramen magnum; 140 ww/40 wl—through base of skull; 90 ww/35 wl—through vertex		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.5 s	0.5 s
Acquisition (detector width × number of detector rows) = coverage	1.25 mm × 16 = 20 mm	0.625 mm × 64 = 40 mm
Reconstruction (slice thickness/interval)	1.25 mm/0.625 mm	1.25 mm/0.625
Pitch	0.938	0.984
kVp/mA	120/500	120/500
Reconstruction 2: For C-spine evaluation (only needed if history includes trauma)		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: ~13		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm
Reformations: Coronal and Sagittal for both CoW and Carotids		
Window setting: 800 ww/200 wl		
DFOV: ~20		
Slice thickness/spacing: 2.0 mm/2.0 mm		
Render mode: MIP		

CTV—Cranial Venography		
Examples of clinical indications: Evaluation of cerebral venous disorders, dural sinus thrombosis		
Scouts: AP and lateral		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just below the skull base		
End location: Just above the vertex		
IV contrast: 100 mL at 4.0 mL/s. Scan delay = 30 seconds (16-detector)/45 seconds (64-detector)		
Oral contrast: None		
Reference angle: No gantry tilt		
DFOV: 25 cm		
SFOV: Head		
Algorithm: Standard		
Window settings: 350 ww/40 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.5 s	0.5 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 64 = 40 mm
Reconstruction (slice thickness/interval)	1.25 mm/0.625 mm	1.25 mm/0.625 mm
Pitch	0.562	0.531
kVp/mA	120/300	120/300

(Continued)

CTV—Cranial Venography (Continued)		
Reconstruction 2:		
Algorithm: Standard		
Window setting: 350 ww/40 wl		
DFOV: 25		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm
Reformations: Coronal and Sagittal		
Window setting: 800 ww/200 wl		
DFOV: 25		
Slice thickness/spacing: 2.0 mm/2.0 mm		
Render mode: MIP		

Neck (Soft Tissue)		
Examples of clinical indications: Neck mass, vascular abnormality		
(If patient has metal dental work, split scan into two groups and angle to reduce artifact)		
Scouts: AP and lateral		
Scan type: Helical		
Scan plane: Transverse		
Start location: Mid orbit		
End location: Clavicular heads		
IV contrast: 125 mL at 1.5 mL/s. Split bolus—1st injection 50 mL, 2-minute delay; 2nd injection 75 mL, scans initiated 25 seconds after the start of the second injection.		
Oral contrast: None		
Reference angle: Angle gantry parallel to hard palate		
DFOV: 18 cm		
SFOV: Large body		
Algorithm: Standard		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.8 s	0.8 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	2.5 mm /1.25 mm	2.5 mm/1.25 mm
Pitch	0.562	0.531
kVp/auto mA	120/150–800	120/150–800
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 18 cm		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm
Reconstruction 3: From just below mandible to clavicular heads		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: 30		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm

Cervical Spine		
Examples of clinical indications: Fracture, dislocation		
Scouts: AP and lateral		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just above skull base		
End location: Mid T1 (include all cervical spine vertebrae, unless a level is specified)		
IV contrast: (only when requested by radiologist) 100 mL at 1.5 mL/s. Scan delay = when injection is complete		
Oral contrast: None		
Reference angle: No gantry tilt		
DFOV: ~13		
SFOV: Large body		
Algorithm: Standard		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.8 s	0.8 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Reconstruction (slice thickness/interval)	2.50 mm/1.25 mm	2.50 mm/1.25 mm
Pitch	0.562	0.531
kVp/auto mA	140/125–325	140/125–325
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: ~13 cm		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm
Reformations: Coronal and Sagittal		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: Full		
Slice thickness/spacing	2.0 mm/2.0 mm	2.0 mm/2.0 mm
Render mode	Average	Average

Thoracic Spine		
Examples of clinical indications: Fracture, dislocation		
Scouts: AP and lateral		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just above T1		
End location: Just below T12 (include all thoracic spine vertebrae, unless a level is specified)		
Oral contrast: None		
Reference angle: No gantry tilt		
DFOV: ~16		
SFOV: Large body		
Algorithm: Standard		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.8 s	0.8 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Slice thickness	2.50 mm/1.25 mm	2.50 mm/1.25 mm
Pitch	0.562	0.531
kVp/auto mA	140/100–350	140/100–350
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: ~13 cm		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm
Reformations: Coronal and Sagittal		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: Full		
Slice thickness/spacing	2.0 mm/2.0 mm	2.0 mm/2.0 mm
Render mode	Average	Average

Lumbar Spine		
Examples of clinical indications: Fracture, dislocation		
Scouts: AP and lateral		
Scan type: Helical		
Scan plane: Transverse		
Start location: Just above L1		
End location: Just below S1 (unless a level is specified by radiologist)		
IV contrast: (only when requested by radiologist) 100 mL at 1.5 mL/s. Scan delay = when injection is complete		
Oral contrast: None		

Reference angle: No gantry tilt		
DFOV: 14–16		
SFOV: Large body		
Algorithm: Standard		
Window settings: 350 ww/50 wl		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.8 s	0.8 s
Acquisition (detector width × number of detector rows) = coverage	0.625 mm × 16 = 10 mm	0.625 mm × 32 = 20 mm
Slice thickness	2.50 mm/1.25 mm	2.50 mm/1.25 mm
Pitch	0.562	0.531
kVp/auto mA	140/150–380	140/150–380
Reconstruction 2:		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: ~13 cm		
Slice thickness/interval	2.5 mm/1.25 mm	2.5 mm/1.25 mm
Reformations: Coronal and Sagittal		
Algorithm: Bone		
Window setting: 4000 ww/400 wl		
DFOV: Full		
Slice Thickness/spacing	2.0 mm/2.0 mm	2.0 mm/2.0 mm
Render mode	Average	Average

CTA Spine
Examples of clinical indications: Localization of the shunt of spinal dural arteriovenous fistulas, spinal arteriovenous malformation, blunt trauma (suspected vascular injury)
Scouts: AP and lateral
Scan type: Helical
Scan plane: Transverse
GROUP 1. Arterial Phase
Start location: Skull base
End location: Sacrum (or levels specified by radiologist)
IV contrast: 120 mL (370 concentration) at 6 mL/s. Scan delay = bolus tracking; place ROI in the aorta just below diaphragm; manually trigger when enhancement value approaches 125 HU
Oral contrast: None
Reference angle: No gantry tilt
DFOV: 20
SFOV: Large body
Algorithm: Standard
Window settings: 350 ww/50 wl

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CTA Spine (Continued)		
	16-Detector Protocol	64-Detector Protocol
Gantry rotation time	0.5 s	0.4 s
Detector coverage	1.25 mm × 16 = 20 mm	0.625 × 64 mm = 40
Slice thickness	1.25 mm/0.625	1.25 mm/0.625
Pitch	0.938	0.984
kVp/auto mA	140/100–750	140/100–750
GROUP 2. Delayed Scans		
Repeat parameters from group 1. Begin group 2 immediately after 1st group is complete.		
Reformations: Coronal and Sagittal		
Algorithm: Standard		
Window setting: 350 ww/50 wl		
DFOV: Full		
Slice thickness/spacing	2.0 mm/2.0 mm	2.0 mm/2.0 mm
Render mode	Average	Average