Cranial CT has assumed a critical role in the practice of emergency medicine for the evaluation of intracranial emergencies, both traumatic and atraumatic. A number of published studies have revealed a deficiency in the ability of EPs to interpret head CTs.1-6 Significantly, a number of these same studies do show that with even a brief educational effort, EPs can gain considerable proficiency in cranial CT scan interpretation.2,3 This is important because there are many situations where the EP must interpret and act upon head CT results in real time without assistance from other specialists such as neurologists, radiologists, or neuroradiologists.7,8 The advantages of CT scanning for CNS pathology in the ED are well known, and include widespread availability at most institutions, speed of imaging, patient accessibility, and sensitivity for a detection of many pathologic processes (particularly acute hemorrhage).

**Basic Principles of CT**

The fundamental principle behind radiography is the following statement: *X-rays are absorbed to different degrees by different tissues.* Dense tissues such as bone absorb the most x-rays, and hence allow the fewest passing through the body part being studied to reach the film or detector opposite. Conversely, tissues with low density (e.g., air and fat) absorb almost none of the x-rays, allowing most to pass through to the film or detector opposite. Conventional radiographs are two-dimensional images of three-dimensional structures; they rely on a summation of tissue densities penetrated by x-rays as they pass through the body. It should be noted that in plain radiographs, denser objects, because they tend to absorb more x-rays, can obscure or attenuate less dense objects.
As opposed to conventional radiographs, with CT scanning an x-ray source and detector, situated 180 degrees across from each other, move 360 degrees around the patient, continuously detecting and sending information about the attenuation of x-rays as they pass through the body. Very thin x-ray beams are utilized, which minimizes the degree of scatter or blurring that limits conventional radiographs. In CT, a computer manipulates and integrates the acquired data and assigns numerical values based on the subtle differences in x-ray attenuation. Based on these values, a gray-scale axial image is generated that can distinguish between objects with even small differences in density.

**ATTENUATION COEFFICIENT**

The tissue contained within each image unit (called a pixel) absorbs a certain proportion of the x-rays that pass through it (e.g., bone absorbs a lot, air almost none). This ability to block x-rays as they pass through a substance is known as attenuation. For a given body tissue, the amount of attenuation is relatively constant and is known as that tissue’s attenuation coefficient. In CT, these attenuation coefficients are mapped to an arbitrary scale between −1000 Hounsfield units (HU) (air) and +1000 HU (bone) (Box 69-1). This scale is the Hounsfield scale (in honor of Sir Jeffrey Hounsfield, who received a Nobel prize for his pioneering work with this technology).

**WINDOWING**

Windowing allows the CT scan reader to focus on certain tissues within a CT scan that fall within set parameters. Tissues of interest can be assigned the full range of blacks and whites, rather than a narrow portion of the gray scale. With this technique, subtle differences in tissue densities can be maximized. The image displayed will depend on both the centering of the viewing window and the width of the window. Most CT imaging includes windows that are optimized for brain, blood, and bone (Fig. 69-1).

**ARTIFACT**

CT of the brain is subject to a few predictable artifactual effects that can potentially inhibit the ability to accurately interpret the images. Besides motion and metal artifact (self-explanatory), the two most common effects are called beam hardening and volume averaging. It is important to understand these effects and to be able to identify them, because they can mimic pathology as well as obscure actual significant findings.

Beam hardening is a phenomenon that causes an abnormal signal when a relatively small amount of hypodense brain tissue is immediately adjacent to dense bone. The posterior fossa, where there is extremely dense bone surrounding the brain, is particularly subject to this phenomenon. It appears as either linear hyper- or hypodensities that can partially obscure the brainstem and cerebellum. Although beam hardening can be reduced with appropriate filtering, it cannot be eliminated.

Volume averaging (also called partial volume artifact) arises when the imaged area contains different types of tissues (e.g., bone and brain). For that particular image unit, the CT pixel produced will represent an average density for all the contained structures. In the above instance of brain and bone, an intermediate density will be represented that may have the appearance of blood. As with beam hardening, certain techniques can minimize this type of artifact (e.g., thinner slice thickness, computer algorithms), but it cannot be eliminated, particularly in the posterior fossa.

### Appearance and Density of Tissues on Cranial CT

**Appearance**
- Black → White
- −1000 HU → +1000 HU
- Air, fat, CSF, white matter, gray matter, acute hemorrhage, bone

**Important Densities**
- Air = −1000 HU
- Water = 0 HU
- Bone = +1000 HU

CSF, cerebrospinal fluid; HU, Hounsfield units.

![Figure 69-1](image_url) CT scan windowing: A, brain. B, blood. C, bone.
Normal Neuroanatomy As Seen on Head CT Scans

As with radiologic interpretation of any body part, a working knowledge of normal anatomic structures and location is fundamental to the clinician’s ability to detect pathologic variants. Cranial CT interpretation is no exception. Paramount in head CT interpretation is familiarity with the various structures, ranging from parenchymal areas such as basal ganglia to vasculature, cisterns, and ventricles. Finally, knowing neurologic functional regions of the brain helps when correlating CT results with physical examination findings.

Although a detailed knowledge of cranial neuroanatomy and its CT appearance is clearly in the realm of the neuroradiologist, familiarity with a relatively few structures, regions, and expected findings allows sufficient interpretation of most head CT scans by the EP. Figures 69-2 through 69-5 demonstrate key structures of a normal head CT scan.

As long as one is systematic in the search for pathology, any number of techniques can be utilized in the review of head CT images. Some recommend a “center-out” technique, in which the examiner starts from the middle of the brain and works outward. Others advocate a problem-oriented approach, in which the clinical history directs the examiner to a particular portion of the scan. In the author’s experience, both of these are of limited utility to the clinician who does not frequently review scans. A preferred method, one that has been demonstrated to work in the ED, is to use the mnemonic “blood can be very bad” (Box 69-2). In this mnemonic, the first letter of each word prompts the clinician to search a certain portion of the cranial CT scan for pathology. The clinician is urged to use the entire mnemonic when examining a cranial CT scan because the presence of one pathologic state does not rule out the presence...
of another one. Following is a detailed description of the components of the mnemonic.

- **Blood**

The appearance of blood on a head CT scan depends primarily on its location and size. Acute hemorrhage will appear hyperdense (bright white) on cranial CT images. This is attributed to the fact that the globin molecule is relatively dense, and hence effectively absorbs x-ray beams. Acute blood is typically in the range of 50 to 100 HU. As the blood becomes older and the globin molecule breaks down, it will lose this hyperdense appearance, beginning at the periphery and working in centrally. On the CT scan, blood will become isodense with the brain at 1 to 2 weeks, depending on clot size, and will become hypodense with the brain at approximately 2 to 3 weeks (Fig. 69-6).

The precise localization of the blood is as important as identifying its presence (Fig. 69-7). Epidural hematomas, subdural hematomas, intraparenchymal hemorrhage, and subarachnoid hemorrhage each have a distinct appearance on the CT scan, as well as differing etiologies, complications, and associated conditions.

- **Epidural Hematoma**

Epidural hematoma most frequently appears as a lens-shaped (biconvex) collection of blood, usually over the brain convexity. An epidural hematoma will not cross a suture line, as the dura is tacked down in these areas. Epidural hematomas arise primarily (85%) from arterial laceration due to a direct blow, with the middle meningeal artery the most common source. A small proportion, however, come from other injured arteries and can even be venous in origin.

- **Subdural Hematoma**

Subdural hematoma appears as a sickle- or crescent-shaped collection of blood, usually over the cerebral
**CHAPTER 69 How to Read a Head CT Scan**

The “Blood Can Be Very Bad” Mnemonic

- **Blood**—Acute hemorrhage appears hyperdense (bright white) on CT. This is due to the fact that the globin molecule is relatively dense and hence effectively absorbs x-ray beams. As the blood becomes older and the globin breaks down, it loses this hyperdense appearance, beginning at the periphery. The precise localization of the blood is as important as identifying its presence.

- **Cisterns**—Cerebrospinal fluid collections jacketing the brain; the following four key cisterns must be examined for blood, asymmetry, and effacement (representing increased intracranial pressure):
  - **Circummesencephalic**—Cerebrospinal fluid ring around the midbrain; first to be effaced with increased intracranial pressure
  - **Suprasellar** (star-shaped)—Location of the circle of Willis; frequent site of aneurysmal subarachnoid hemorrhage
  - **Quadrigeminal**—W-shaped cistern at top of midbrain; effaced early by rostrocaudal herniation
  - **Sylvian**—Between temporal and frontal lobes; site of traumatic and distal mid-cerebral aneurysm and subarachnoid hemorrhage

- **Brain**—Examine for:
  - **Symmetry**—Sulcal pattern (gyri) well differentiated in adults and symmetric side-to-side.
  - **Gray-white differentiation**—Earliest sign of cerebrovascular aneurysm is loss of gray-white differentiation; metastatic lesions often found at gray-white border
  - **Shift**—Falx should be midline, with ventricles evenly spaced to the sides; can also have rostrocaudal shift, evidenced by loss of cisternal space; unilateral effacement of sulci signals increased pressure in one compartment; bilateral effacement signals global increased pressure
  - **Hyper-/hypodensity**—Increased density with blood, calcification, intravenous contrast media; decreased density with air/gas (pneumocephalus), fat, ischemia (cerebrovascular aneurysm), tumor

- **Ventricles**—Pathologic processes cause dilation (hydrocephalus) or compression/shift; hydrocephalus usually first evident in dilation of the temporal horns (normally small and slit-like); examiner must take in the “whole picture” to determine if the ventricles are enlarged due to lack of brain tissue or to increased cerebrospinal fluid pressure

- **Bone**—Highest density on CT scan; diagnosis of skull fracture can be confusing due to the presence of sutures in the skull; compare other side of skull for symmetry (suture) versus asymmetry (fracture); basilar skull fractures commonly found in petrous ridge (look for blood in mastoid air cells)

*Blood = blood, Can = cisterns, Be = brain, Very = ventricles, Bad = bone.

**FIGURE 69-6** CT scan appearance of central nervous system hemorrhage: **A**, acute; **B**, subacute; **C**, chronic.
convexity. Subdural hematomas can also be seen as isolated collections that appear in the interhemispheric fissures or along the tentorium. As opposed to epidural hematomas, subdural hematomas will cross suture lines, as there is no anatomic limitation to blood flow below the dura. A subdural hematoma can be either an acute lesion or a chronic one. While both occur primarily from disruption of surface and/or bridging vessels, the magnitude of impact damage is usually much higher in acute lesions. As such, they are frequently accompanied by severe brain injury, contributing to a much poorer overall prognosis than epidural hematoma.

Chronic subdural hematoma, in contrast with acute subdural hematoma, usually follows a more benign course than acute subdural hematoma. Attributed to slow venous oozing after even a minor closed head injury, the clot can gradually accumulate, allowing the patient to compensate. As the clot is frequently encased in a fragile vascular membrane, however, these patients are at significant risk for re-bleeding as the result of additional minor trauma. The CT appearance of a chronic subdural hematoma depends on the length of time since the initial bleeding. A subdural hematoma that is isodense with brain can be very difficult to detect on CT, and in these cases contrast may highlight the surrounding vascular membrane.

- **Intraparenchymal Hemorrhage**

Cranial CT can reliably identify intraparenchymal (or intracerebral) hematomas as small as 5 mm. These appear as high-density areas on the CT scan, usually with much less mass effect than their apparent size would indicate. Traumatic intraparenchymal hematomas may be seen immediately following an injury, or they can appear in a delayed fashion, after there has been time for swelling. Additionally, contusions may enlarge and coalesce over first 2 to 4 days. Traumatic contusions most commonly occur in areas where sudden deceleration of the head causes the brain to impact on bony prominences (e.g., temporal, frontal, occipital poles).

In distinction to traumatic lesions, nontraumatic hemorrhagic lesions due to hypertensive disease are typically seen in elderly patients and occur most frequently in the basal ganglia region. Hemorrhage from such lesions may rupture into the ventricular space, with the additional finding of intraventricular hemorrhage on CT. Posterior fossa bleeding (e.g., cerebellar) may dissect into the brainstem (pons, cerebellar peduncles) or rupture into the fourth ventricle. Besides hypertensive etiologies, intraparenchymal hematomas can be caused by arteriovenous malformations, bleeding from or into a tumor, amyloid angiopathy, or aneurysms that happen to rupture into the substance of the brain rather than into the subarachnoid space.

- **Intraventricular Hemorrhage**

Intraventricular hemorrhage can be traumatic or secondary to intraparenchymal hemorrhage or subarachnoid hemorrhage with ventricular rupture. Identified as a white density in the normally black ventricular spaces, it is associated with a particularly poor outcome in cases of trauma (although this may be more of a marker than a causative issue). Hydrocephalus can be the end result regardless of the etiology. Cerebrospinal fluid (CSF) is produced in the lateral ventricles at a rate of 0.5 to 1 mL per minute, and this will occur regardless of the intraventricular pressure. A block at any point in the CSF pathway (lateral ventricles → foramen of Monro → 3rd ventricle → aqueduct of Sylvius → 4th ventricle → foramina of Luschka and Magendie → cisterns → arachnoid granulations) will result in hydrocephalus, with associated increased intracranial pressure and the ultimate potential for herniation.

- **Subarachnoid Hemorrhage**

Subarachnoid hemorrhage is defined as hemorrhage into any subarachnoid space that is normally filled with CSF (e.g., cistern, brain convexity). The hyper-density of blood in the subarachnoid space is frequently visible on CT imaging within minutes of the onset of hemorrhage (Fig. 69-8). Subarachnoid
hemorrhage is most commonly aneurysmal (75%-80%), but it can also occur with trauma, tumor, arteriovenous malformations and dural malformations. As a result of arachnoid granulations becoming plugged with red blood cells or their degradation products, hydrocephalus complicates approximately 20% of cases of subarachnoid hemorrhage.

The ability of a CT scanner to demonstrate subarachnoid hemorrhage depends on a number of factors, including the generation of scanner, the time since the initial bleeding, and the skill of the examiner. According to some studies, the CT scan is 95% to 98% sensitive for subarachnoid hemorrhage in the first 12 hours after the ictus. This sensitivity is reported to decrease as follows:

90%-95% at 24 hours
80% at 3 days
50% at 1 week
30% at 2 weeks

Extracranial Hemorrhage

The presence and significance of extracranial blood and soft-tissue swelling on CT imaging is often overlooked. The examiner should use this finding to lead to subtle fractures that can be identified in areas of maximal impact (and hence maximal soft-tissue swelling). This will also direct the examiner to search the underlying brain parenchyma in these areas for parenchymal contusions, as well as to areas opposite maximal impact to search for contrecoup injuries.

Cisterns

Cisterns are potential spaces formed where there is a collection of CSF that is pooled as it works its way up to the superior sagittal sinus from the 4th ventricle. Of the numerous named cisterns (and some with multiple names), there are four key cisterns that the EP needs to be familiar with in order to identify increased intracranial pressure as well as the presence of blood in the subarachnoid space (Fig. 69-9). These cisterns are:

- Circummesencephalic—Hypodense CSF ring around the midbrain; most sensitive marker for increased intracranial pressure; will become effaced first with increased pressure and herniation syndromes.
- Suprasellar—Star-shaped hypodense space above the sella and pituitary; location of the circle of Willis, hence an excellent location for identifying aneurysmal subarachnoid hemorrhage.
- Quadrigeminal—W-shaped cistern at the top of the midbrain; can be a location for identifying traumatic subarachnoid hemorrhage, as well as an early marker of increased intracranial pressure and rostrocaudal herniation (Fig. 69-10).
- Sylvian—Bilateral CSF space located between the temporal and frontal lobes of the brain; another good location to identify subarachnoid hemorrhage, whether caused by trauma or aneurysm leak (particularly distal middle cerebral artery aneurysms).

Brain

Normal brain parenchyma has an inhomogeneous appearance where the gray and white matter interface. Cortical gray matter is denser than subcortical white matter; therefore the cortex will appear lighter on CT imaging. Given that many disease processes we are looking for in the ED are unilateral (e.g., cerebrovascular aneurysm, tumor, abscess), the clinician should be aware that there will normally be side-to-side symmetry on the scan. Similarly, the cortical gyral and sulcal pattern should be symmetric (Fig. 69-11). Besides symmetry, it is important to examine the brain parenchyma for:

- Gray-white differentiation—The earliest sign of an ischemic cerebrovascular aneurysm will be loss of gray-white differentiation. Tumors can also obscure this interface, particularly when there is associated edema (hypodensity).
- Shift—The falx should be midline, with ventricles evenly spaced to the sides. With rostrocaudal herniation the midline will be preserved, but this
SECTION VIII Injuries to Bones and Organs

FIGURE 69-9  Three important cerebrospinal fluid cisterns: A, cisterns viewed at high pontine level; B, cisterns viewed at level of cerebral peduncles; C, cisterns viewed at high midbrain level.

FIGURE 69-10  CT appearance of increased intracranial pressure: A, normal intracranial pressure; B, elevated intracranial pressure.
will usually be evidenced by loss of cisternal spaces. A unilateral effacement of sulci signals increased pressure in one compartment. Bilateral effacement signals global increased pressure.

- **Hyper/hypodensity**—Brain will take on increased density with blood, calcification, and intravenous contrast media. It will take on decreased density with air (pneumocephalus), water, fat, and ischemia (cerebrovascular accident). Tumor may result in either increased or decreased density on CT imaging, depending on tumor type and amount of associated water density (edema).

### Specific Brain Parenchymal Lesions

**Tumor.** Brain tumors usually appear as hypodense, poorly defined lesions on noncontrast CT scans. It is estimated that 70% to 80% of brain tumors will be apparent on plain scans without the use of an intravenous contrast agent. Calcification and hemorrhage associated with a tumor can cause it to have a hyperdense appearance. Tumors should be suspected on a noncontrast CT scan when significant edema is associated with an ill-defined mass. This vasogenic edema occurs because of a loss of integrity of the blood-brain barrier, allowing fluid to pass into the extracellular space. Edema, because of the increased water content, appears hypodense on the CT scan (Fig. 69-12).

Intravenous contrast material can help define brain tumors. Contrast media will leak through the incompetent blood-brain barrier into the extracellular space surrounding the mass lesion, resulting in a contrast-enhancing ring. Once a tumor is identified, the clinician should make some determination of the following information: location and size—intraaxial (within the brain parenchyma) or extraaxial; degree of edema and mass effect—for example, herniation may be impending due to swelling.

**Abscess.** Brain abscess will appear as an ill-defined hypodensity on non-contrast CT scan. A variable amount of edema is usually associated with such lesions and, like tumors, they will frequently demonstrate ring-enhancement with the addition of an intravenous contrast agent.

**Ischemic Infarction.** Strokes are classified as either hemorrhagic or nonhemorrhagic. Nonhemorrhagic infarctions can be seen as early as 2 to 3 hours following ictus, but most will not begin to be clearly evident on the CT scan for 12 to 24 hours. The earliest change seen in areas of ischemia is loss of gray-white differentiation, due to influx of water into the metabolically active gray matter. With the loss of blood flow, the energy-dependent cellular ion pumps fail, and the movement of ions such as sodium and potassium is no longer regulated. By osmotic forces, water follows the ions into the cells, where they cease metabolic activity. Because gray matter is metabolically more active than white matter, the gray cells are affected first, become water-filled, and take on the CT appearance of white matter. This loss of gray-white differentiation can initially be a subtle finding but ultimately will become evident and will usually be maximal between days 3 and 5 (Fig. 69-13).

Any vascular distribution can be affected by ischemic lesions (e.g., middle cerebral artery aneurysm, posterior inferior cerebellar artery). One specialized type of stroke frequently identified on CT imaging is
hydrocephalus is first evident in dilation of the temporal horns, which are normally small with a slit-like morphology. When examining the ventricular system for hydrocephalus, the clinician needs to take in the entire picture of the brain, as ventricles can be large for reasons other than increased pressure (e.g., atrophy). If the ventricles are large, the clinician should investigate whether other CSF spaces in the brain are large (e.g., sulci, cisterns). In this case, it is likely that this enlargement is the result of brain volume loss rather than the increased ventricle size. Conversely, if the ventricles are large, but the brain appears “tight” with sulcal effacement and loss of sulcal space, then the likelihood of hydrocephalus is high. The clinician also should look for evidence of increased intracranial pressure (e.g., cisternal effacement).

**Bone**

As demonstrated earlier, bone has the highest density on the CT scan (+1000 HU). Because of this, depressed or comminuted skull fractures can usually be easily identified on the CT scan; however, small linear (nondepressed) skull fractures and fractures of the skull base may be more difficult to find (Fig. 69-15). Also, making the diagnosis of a skull fracture can be confusing due to the presence of sutures in the skull.

Fractures may occur at any portion of the bony skull. The presence of a skull fracture should increase the index of suspicion for intracranial injury. If intracranial air is seen on a CT scan, this indicates that the skull and dura have been violated at some point (Fig. 69-16). Basilar skull fractures are most commonly found in the petrous ridge (the dense pyramidal-shaped portion of the temporal bone). Due to the density of this bone, the fracture line may not be easily identified in this area. The clinician should not only search for such a fracture line but should also pay close attention to the normally aerated mastoid air cells that are contained within this bone. Any blood in the mastoid air cells means that a skull base fracture is likely. Analogous to the mastoid air cells, the maxillary, ethmoid, and sphenoid sinuses should

**Ventricles**

Pathologic processes can cause either dilation (hydrocephalus) or compression/shift of the ventricular system (Fig. 69-14). Additionally, hemorrhage can occur into any of the ventricles, resulting in the potential for obstruction of flow and resulting hydrocephalus. The term “communicating hydrocephalus” is used when there is free CSF egress from the ventricular system, with a blockage at the level of the arachnoid granulations. The term *noncommunicating hydrocephalus* is used if there is obstruction anywhere along the course of flow from the lateral ventricles through egress from the 4th ventricle. Hydrocephalus frequently is first evident in dilation of the temporal horns, which are normally small with a slit-like morphology.
be visible and aerated; the presence of fluid in any of these sinuses in the setting of trauma should raise suspicion of a skull fracture. In nontraumatic cases, fluid in the mastoids may indicate mastoiditis, and fluid in the sinuses may indicate sinusitis.

**Summary**

Cranial CT is integral to the practice of emergency medicine and is used on a daily basis to make important, time-critical decisions that directly impact the care of ED patients. An important tenet in the use of cranial CT is that accurate interpretation is required to make good clinical decisions. Cranial CT interpretation is a skill, like ECG interpretation, that can be learned through education, practice, and repetition.

**REFERENCES**
