CONTEMPORARY NEUROSURGERY
A BIWEEKLY PUBLICATION FOR CLINICAL NEUROSURGICAL CONTINUING MEDICAL EDUCATION
VOLUME 44 • NUMBER 12
August 30, 2022

From Outside to Inside: A Systematic Approach to the Head CT From the Emergency Department

CPT Michael R. Povlow, MD, Col Francis J. Cloran, MD, MS, and COL (Ret) Liem T. Bui-Mansfield, MD

Learning Objectives: *After participating in this CME activity, the neurosurgeon should be better able to:*
1. *Review indications for noncontrast head CT in the emergency setting.*
2. *Interpret radiology reports of common CT findings in patients with head injuries.*

The use of CT for the evaluation of head injuries and mental status changes is common. Radiologists must have a reliable system for evaluating a head CT scan, which will improve diagnostic accuracy and speed of interpretation. This article discusses a systematic approach to head CT interpretation and reviews some of the common pathologies seen in clinical practice.

The use of CT continues to increase, particularly in American emergency departments where one in seven patients has a CT performed.[1] One study determined that head CT scans account for approximately 50% of the total CT examinations ordered in U.S. emergency departments.[1] The most common reasons for the CT requests include traumatic head injuries, focal neurologic deficits, and new-onset headaches.[2] According to American College of Radiology practice parameters, these are all primary indications for head CT.[3] Given the clinical implications for directing further management (eg, the time-sensitive nature of interpreting head CTs for stroke assessment), this article describes a systematic approach for acute CT interpretation to improve diagnostic accuracy and to rapidly facilitate clinical care with the pressure of ever-increasing volume and turnaround time minimization (See Supplemental Digital Content 1, published online, http://links.lww.com/CDR/A0).

Approach

General Guidelines

Although axial images in the soft tissue kernel are a necessity for head CT imaging, sagittal and coronal reformats provide additional diagnostic information and troubleshooting capabilities for artifacts. Additional dedicated bone reformats in the axial plane are recommended for evaluation of the osseous structures. These are best viewed on picture archiving and communication system (PACS) workstations with the ability to change the window and

Dr. Povlow is a Resident, and Dr. Cloran is Diagnostic Radiology Program Director, SAUSHEC, Brooke Army Medical Center, 3551 Roger Brooke Dr, Fort Sam Houston, TX 78234; Dr. Cloran is Assistant Professor and Dr. Mansfield is Adjunct Professor, Department of Radiology, USUHS, Bethesda, Maryland; E-mail: michael.r.povlow.mil@mail.mil.

All authors, faculty, and staff have no relevant financial relationship with any ineligible organizations regarding this educational activity.

This article was published originally in *Contemporary Diagnostic Radiology* 2021;44(1):1-8.

Category: Neuroradiology, Trauma

Key Words: Brain, Head CT, Emergent CT

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the newsletter's website (www.CDRnewsletter.com).

Lippincott Continuing Medical Education Institute, Inc., is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Lippincott Continuing Medical Education Institute, Inc., designates this enduring material for a maximum of 2.0 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity. To earn CME credit, you must read the CME article and complete the quiz and evaluation on the enclosed form, answering at least 70% of the quiz questions correctly. This activity expires on August 29, 2025.

level. Many PACS platforms permit reformats at the workstation, which allow generation of other desired reformats such as three-dimensional (3D) surface-rendered reformations of the skull (eg, in the setting of pediatric head trauma where there is concern for fracture). At our institution, images are submitted to PACS in soft kernel in all three planes with 2.5-mm slice thickness at 2.5-mm increments whereas a dedicated bone kernel is submitted only in the axial plane planes with 2.5-mm slice thickness at 2.5-mm increments.

Subdural Window

Use of the subdural window, which has a wider range when compared with the brain window (eg, level 70, window 150), enables the delineation of small extra-axial hematomas, which may otherwise be masked by either beam hardening associated with the skull (Figure 1). Subtle extra-axial hematoma may be detected by closely assessing the extra-axial spaces along the inner table of the calvarium and the reflection of the falx and tentorium cerebelli. One should be able to trace the sulci and gyri leading to the edges of the inner calvarial table. If this is not seen, then pathology exists between the brain parenchyma and the skull, such as isoattenuating (subacute) blood products, until proven otherwise.

In the case of elderly patients, there is often some degree of parenchymal volume loss, which will show enlarged cerebrospinal fluid (CSF) attenuation extra-axial spaces between the skull and parenchyma; however, this should be relatively symmetric. When there is asymmetric enlargement of extra-axial CSF spaces, one should assess for mass effect on the brain parenchyma. If mass effect exists, one should consider entities such as chronic subdural hematoma, subdural hygroma, or an arachnoid cyst. In the absence of mass effect, one should look for signs of prior insult such as remote cerebral infarct.

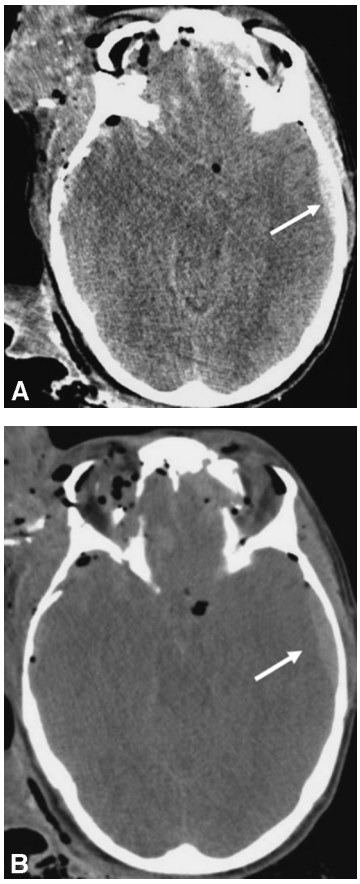

Figure 1. Unenhanced CT scans in brain (*A*) and subdural window (*B*) show severe trauma from a gunshot wound with pneumocephalus, fractures, SAH, and subdural hematoma. In the extra-axial space of the left temporal lobe, there is a lentiform-shaped region of acute epidural hematoma (*arrow*). This is seen to better advantage on subdural window, as it allows acute blood products to stand out from the adjacent bone.

When discriminating between acute extra-axial blood products, one should make every attempt to differentiate between an epidural and a subdural hematoma. Epidural hematomas are classically lentiform in shape, most often associated with fractures, and do not cross skull suture lines (unless fracture line involves the suture) (Figure 1). Morbidity of epidural hematoma is most often related to arterial

The continuing education activity in *Contemporary Neurosurgery* is intended for neurosurgeons, neurologists, neuroradiologists, and neuropathologists.

Contemporary Neurosurgery (ISSN 0163-2108) is published bi-weekly by Wolters Kluwer Health, Inc. at 14700 Citicorp Drive, Bldg 3, Hagerstown, MD 21742. **Customer Service: Phone (800) 638-3030, Fax (301) 223-2400, or E-mail customerservice@lww.com.** Visit our website at www.lww.com.

Wolters Kluwer

Publisher: Sharon Zinner

Subscription rates: *Individual*: US $891, international $1247. *Institutional*: US $1519, international $1569. *In training:* US $164, international $165. Single copies: $85. GST Registration Number: 895524239. Send bulk pricing requests to Publisher.

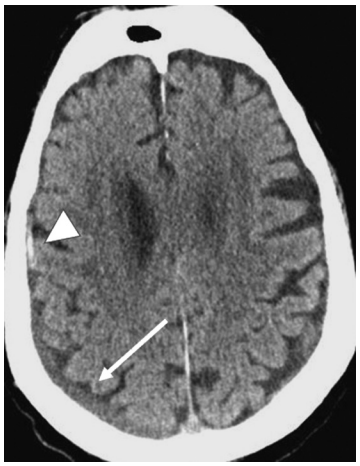

Figure 2. Unenhanced CT scan shows a right-sided, chronic subdural hematoma (*arrow*) with an additional focus of acute subdural hemorrhage (*arrowhead*). Note the difference in attenuation, which differentiates acute from chronic hemorrhage.

bleeding, which can rapidly accumulate. Subdural hematomas are crescentic and do not subtend the undersurface of the dura (Figure 2). The density of bone in the temporal lobes and the posterior fossa makes beam-hardening artifact a confounding issue, and the use of the subdural window and evaluating sagittal and coronal reformats may enable the radiologist to better identify extra-axial hemorrhage in these regions.

Brain Window

The radiologist should then use the narrower brain window. A setting of level 80 with window 40 HU is suggested. Again, one should inspect the borders of the inner skull, the falx, and tentorium assessing for blood products. Next, an assessment of the sulci and basal cisterns should be performed looking for subarachnoid hemorrhage (SAH) (Figure 3).

Once assessment for extra-axial hemorrhage is completed, one should address the white matter, identifying areas of low attenuation. The initial distinction should be whether areas of low attenuation are associated with mass effect. When there is an absence of mass effect or frank volume loss, the underlying etiology may be uncertain; however, it is typically not emergent (eg, encephalomalacia related to prior insult) and does not require immediate work-up. In neonates/infants, there are commonly regions of white matter hypoattenuation related

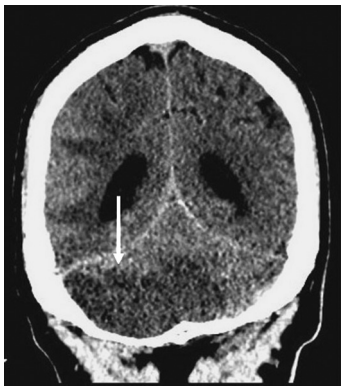

Figure 4. Unenhanced CT scan shows a large geographic region of hypoattenuation of the right cerebellum and vermis with loss of gray white matter differentiation (*arrow*). Findings are consistent with large territory acute posterior inferior cerebellar artery infarct.

to undermyelinated white matter, which typically radiate from the subcortical white matter, are symmetric, and should not be confused with white matter pathology. In the setting of white matter hypoattenuation with mass effect, many etiologies must be considered to include evolving parenchymal hematoma, infectious etiologies (eg, cerebritis, encephalitis, or abscess), neoplasm, ischemic infarct, or edema, which may be related to aforementioned etiologies.

When considering causes of edema, there are two broad categories: vasogenic and cytotoxic. Cytotoxic edema is seen in the setting of cell death, such as acute ischemic infarct, where the sodium/potassium channels are no longer functioning leading to intracellular swelling with water and sodium.[4] Vasogenic edema is caused by disruption of the blood-brain barrier, leading to water accumulation within the extracellular spaces.[4] Cytotoxic edema will result in hypoattenuation of gray matter leading to loss of gray-white matter differentiation if the infarct is peripheral (Figure 4) and hypoattenuation of deep gray nuclei if central, whereas vasogenic edema will classically spare the cortex (Figure 5). With regard to symmetric hypoattenuation of the deep gray nuclei, one should consider hypoxic-ischemic injury as an etiology, but entities such as toxic exposures should also be considered (eg, carbon monoxide with bilateral globus pallidus hypoattenuation and methanol with bilateral putaminal hypoattenuation).

Then, one should briefly assess the major vessels, to include the arteries and the dural venous sinuses, to see whether any of them appears hyperdense, as this would

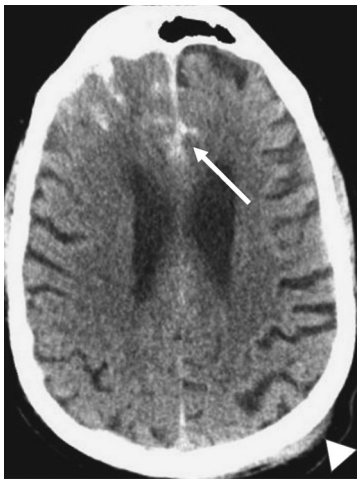

Figure 3. Unenhanced CT scan demonstrates acute hemorrhage interdigitating between the sulci of the right frontal lobe and along the falx cerebrum. This is consistent with acute SAH. The scalp hematoma over the left parietal region (*arrowhead*) is the site of impact, with the SAH opposite this location (*arrow*). This is the classic coup-contrecoup pattern.

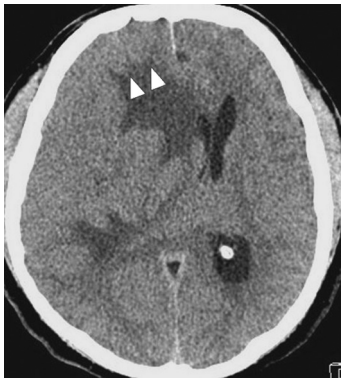

Figure 5. Unenhanced CT scan in brain window illustrates a right frontoparietal isoattenuating mass with surrounding vasogenic edema and mass effect causing leftward midline shift. Although the mass is difficult to see, the finding of mass effect plus vasogenic edema with sparing of cortex (*arrowheads*) is suspicious for an intracranial mass, which should lead to the recommendation of further imaging with MRI. Surgical pathology confirmed an atypical meningioma.

indicate intraluminal thrombus. A hyperdense middle cerebral artery (MCA) sign may be the only early noncontrast CT clue to a large MCA infarct. Note that this can occur in any vessel, so the circle of Willis, vertebrobasilar system, and dural venous sinuses should be assessed. Potential false positives of hyperdense vessel include beam-hardening artifact, increased hematocrit, arterial microcalcifications, and hypodense brain parenchyma in the setting of diffuse cerebral edema.

One should then evaluate CSF spaces and assess the size and symmetry of ventricles. Although ventricular enlargement can be seen in the setting of parenchymal volume loss (eg, related to age, medications, medical conditions, or prior insult such as infarct), one must assess for ventriculomegaly related to CSF outflow obstruction (ie, hydrocephalus). Communicating-type hydrocephalus such as normal pressure hydrocephalus (NPH) must be distinguished from noncommunicating hydrocephalus related to restriction of CSF outflow due to either mass effect or stricture. The prototypical type of communicating hydrocephalus can be considered NPH and most frequently afflicts the elderly population. This can be evaluated using the callosal angle, which in the setting of NPH should measure less than 90 degrees on coronal images at the level of the posterior commissure (Figure 6). In contradistinction, communicating hydrocephalus must be differentiated from noncommunicating hydrocephalus where lateral and third ventricular dilation may occur due to aqueductal stenosis or masses obstructing CSF outflow (Figure 7). Size and symmetry of the sulci and any layering intraventricular blood products should also be assessed (Figure 8). The septum pellucidum should line up in the midline (a line between the anterior and posterior falx can be used); if there is any shift seen of the septum pellucidum, one should thoroughly assess for causes of contributory mass effect (Figure 9).

One should then proceed to investigating the infratentorial region using the brain window and evaluate the basal cisterns and cerebellopontine angles, looking for any mass effect or blood products. Subtle asymmetric decrease of one basal cistern compared with the other should prompt a closer inspection for herniation. Classic patterns include transtentorial, subfalcine, and tonsillar herniation (Table 1).[5] The cerebellum and brainstem are often difficult to assess well on CT given the amount of artifact present, although any mass effect or broad geographic regions of low attenuation should raise concern (Figure 4).

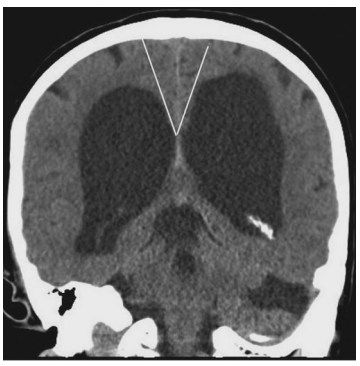

Figure 6. Unenhanced CT scan shows massive enlargement of the lateral ventricles. The angle of the ventricles on the coronal image at the level of the posterior commissure is less than 90 degrees. Findings are consistent with clinically known NPH.

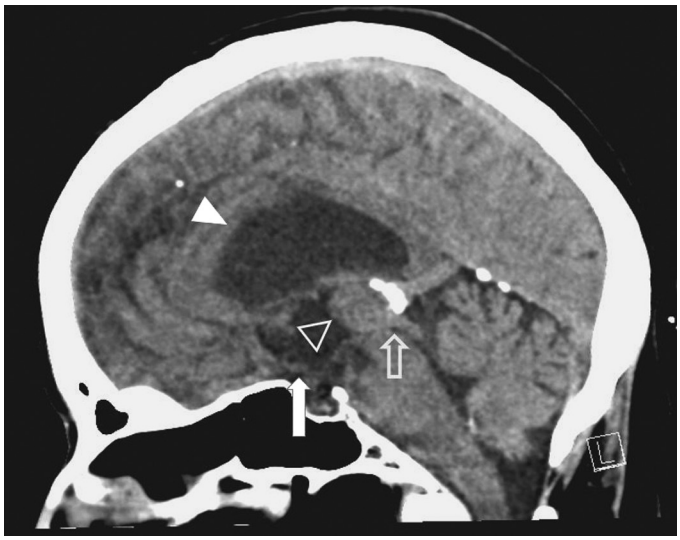

Figure 7. Unenhanced sagittal CT scan shows enlargement of the lateral ventricles (*solid arrowhead*) and third ventricle with bowing of the third ventricle floor (*solid arrow*). A soft tissue mass is seen within the pineal region, with thick calcifications at its posterior margin (*open arrowhead*) obstructing the cerebral aqueduct (*open arrow*). These findings are consistent with obstructive, noncommunicating hydrocephalus from a biopsy-proven pineocytoma.

Stroke Window

Now that the brain has been assessed in both the subdural and brain windows, one should investigate the brain parenchyma with the stroke window. Normal white matter has a mean attenuation of 29 HU and gray matter has a mean attenuation of 35 HU.[6] Given the similarities in attenuation values between them on CT imaging, the stroke window is important to accentuate their differences. Common settings include level of 32 with a window of 8 HU although the authors most frequently use level and a window of 40 and 40 HU. The purpose of these narrow setting is to assess for loss of gray-white matter differentiation concerning for an acute stroke (Figures 4 and 10). In MCA infarcts, the progressive hypoattenuation of the basal ganglia can make this structure indistinguishable from the adjacent white matter (ie, the "disappearing basal ganglia" sign) due to cytotoxic edema.[7] Another classic sign is the "insular ribbon" sign where the loss of gray-white differentiation is seen along the lateral margin of the insula.

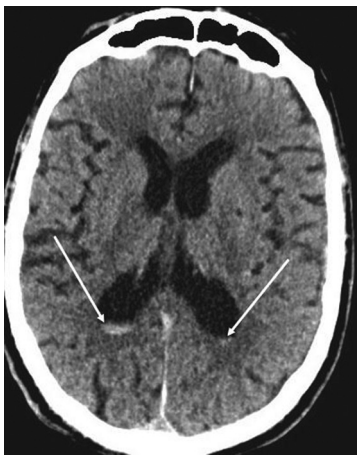

Figure 8. Unenhanced CT scan demonstrates acute intraventricular hemorrhage layering within both the right and left lateral ventricles (*arrows*) related to closed head injury.

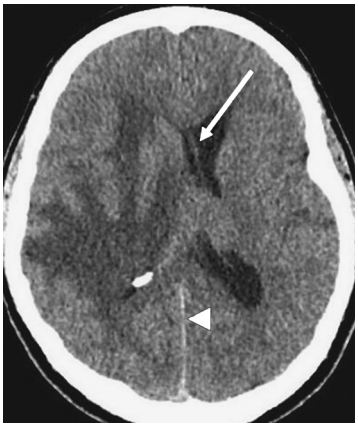

Figure 9. Unenhanced CT scan shows vasogenic edema with mass effect and midline shift. Note the septum pellucidum (*arrow*) is shifted leftward from the midline (*arrowhead*). The large region of hypoattenuation interdigitating between the cortical gray matter of the right parietal lobe is characteristic of vasogenic edema.

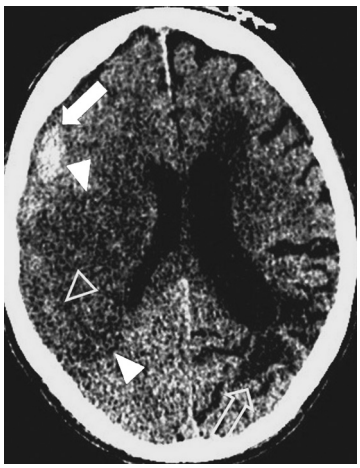

Figure 10. Unenhanced CT scan using a narrow stroke window (window/level 40:40). The large region of hypoattenuation of the right MCA territory (*solid arrowheads*), which is more conspicuous in the narrow stroke window, is consistent with an acute MCA infarct. There is also a small amount of adjacent acute subdural hematoma of the right frontal lobe (*solid arrow*). Note how the hypoattenuating region involves both the cortex and white matter (*open arrowhead*), which is characteristic of cytotoxic edema. Also seen in the left parieto-occipital lobe is a region of hypoattenuation (*open arrow*), which is near that of CSF with ex vacuo ventricular dilation; this is characteristic of encephalomalacia from remote infarct.

Bone Window

At this point, the brain has been well assessed on three different settings and the interpreter should now move to the bone reformat images. These use a high level and wide window for evaluation of the osseous structures. A suggested setting would be level of 400 with a window of 1800 HU. The obvious use of these windows is to assess for fractures, which can often be confused with sutures, so an understanding of where the normal sutures lie is critical. Sutures are generally symmetric, so if a linear lucency goes through the bone without a corresponding one on the contralateral side, this suggests a fracture. Additionally, the abnormal widening of a suture is consistent with a diastatic sutural fracture. Often a fracture may not be directly identified, although features such as pneumocephalus, mastoid, or paranasal sinus opacification may suggest this; in this case assessment with thinner slices and 3D volumetric imaging can provide increased sensitivity in fracture detection.[8] Whenever a fracture is identified, search carefully for an associated epidural hematoma or coup and contrecoup contusion of the brain. Also, fractures that involve the central skull base/carotid canal or subtend dural venous sinuses should prompt additional imaging with CT angiogram or venogram, respectively.

Table 1. Classic Patterns of Herniation[5]

Herniation Type	Description
Subfalcine	Cingulate gyrus is pushed across and under the falx cerebri
Downward transtentorial	Supratentorial mass effect causes downward displacement of the cerebrum into the posterior fossa through the tentorium cerebelli and exerts mass effect on the brainstem.
	Uncal herniation is a subtype where the medial temporal lobe is pushed under the tentorium cerebelli and can compress the midbrain
Upward transtentorial	Posterior fossa mass effect causes upward displacement of the cerebellum through the tentorium cerebelli
Tonsillar	The cerebellar tonsils herniate downward and compress the brainstem.

On bone windows, one should reassess areas of intracranial hyperattenuation, using bone windows, that were seen during the earlier evaluation to aid in differentiating parenchymal or extra-axial areas of calcification from hemorrhage. Assessment of the vasculature using bone windows may reveal calcifications of the intracranial arteries, which can be important in the detection of calcified embolus.

The mastoid air cells should be assessed for opacification, destruction, or fracture. In the setting of trauma, if a mastoid effusion is seen, this should prompt temporal bone CT to look for fractures; this should be done ideally with reformations from the initial head CT in the spirit of ALARA. In the absence of history of trauma, asymmetric mastoid opacification warrants close inspection of the nasopharynx to evaluate for an obstructing mass near the Eustachian tube.

Orbits/Sinuses

Sinuses are evaluated on both the soft tissue and bone windows. Sinus wall fractures are readily detected on bone windows. In the setting of trauma, sinus fractures are almost always accompanied by some degree of sinus opacification. Dedicated CT of the face should be considered in such cases to evaluate for underlying facial fractures. When a trauma has enough force to cause facial fractures, close inspection of the skull base is advised, as skull base fractures can lead to devastating consequences (eg, traumatic carotid injury). Although there is always a concern for facial fracture in the setting of sinus opacification, one must always consider that sinus opacification may be related to acute sinusitis in the absence of visible injury.

When visible, the orbits should be assessed, looking for any mass, hematoma, or fat stranding within the pre- and postseptal spaces. Any findings in the postseptal space can lead to devastating visual consequences, so clinicians need to be alerted, especially if unexpected. Additionally, directly assess the globes for abnormal morphology (eg, a malformed globe in the setting of rupture) and for their contents (eg, signs of vitreous hemorrhage or retinal detachment) (Figure 11); in the absence of trauma, abnormal

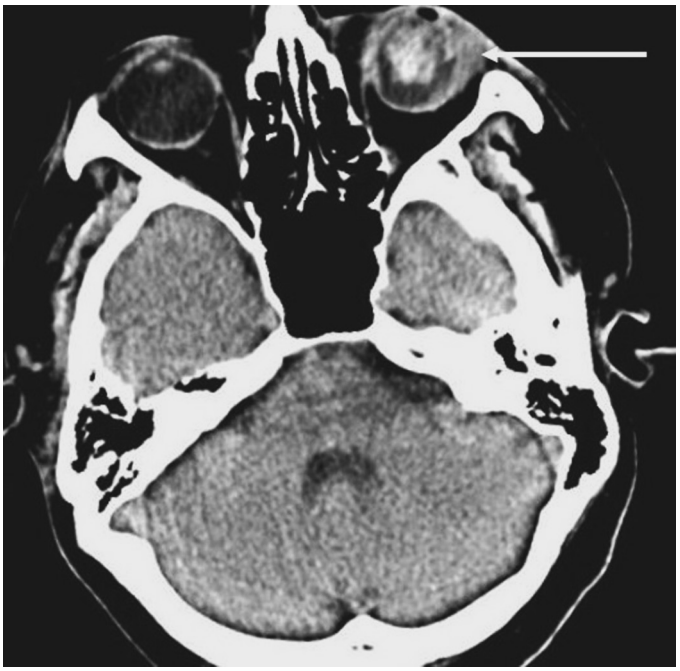

Figure 11. Unenhanced CT scan shows an open globe injury to the left eye with acute vitreous hemorrhage (*arrow*).

globe morphology can be seen in the setting of congenital disorders or remote prior ocular insult (eg, phthisis bulbi). Furthermore, a search for orbital foreign bodies, especially in the appropriate clinical setting, should be accomplished; metallic foreign bodies are usually easy to spot; however, wood can have a similar CT appearance as air, which may be overlooked.

Scalp/Soft Tissues

The soft tissues of the scalp and face should be assessed for contusion in the setting of trauma. In the presence of scalp hematoma, search carefully for associated skull fracture, intracranial hemorrhage, and coup or contrecoup brain contusion. Additionally, one should assess for skin thickening in areas where fat should normally be, as abnormal infiltrative thickening could indicate an occult scalp neoplasm. The soft tissues of the head and neck including at the stylomastoid foramen, retroantral fat, and nasopharynx should be evaluated for any asymmetry with soft tissue thickening, as abnormal soft tissue in this region could reflect infection, inflammation, or neoplasm.

Additional Planes and Scout Image

Additional planes are often helpful for localization and show certain anatomy better. The sagittal plane is useful for midline structures such as the pituitary gland. If the sella is empty, one should consider idiopathic intracranial hypertension. In the setting of a suprasellar mass, the optic chiasm must be investigated for mass effect, which can cause symptoms of vision loss. The coronal plane is also valuable for the assessment of subdural hemorrhage, which may layer along the tentorium cerebelli and be seen only as focal thickening. Finally, assess the scout image and evaluate for obvious cervical spine fractures, facial abnormalities, malpositioned support apparatus, or subtle skull fracture not seen on other images.

Common Abnormalities Found on Head CT

Trauma

Epidural hematomas are almost always related to traumatic injury and often have an associated temporal bone fracture. The etiology is most often from tearing of the middle meningeal artery. The appearance of these has been discussed earlier. The key imaging differentiator is that they do not cross suture lines so long as the suture line is intact. Because these are associated with arterial bleeds, the size can rapidly enlarge, which can cause mass effect leading to herniation; therefore, early diagnosis and recognition is imperative for the radiologist.

Subdural hematomas most often occur in the setting of trauma, although, in the elderly, only minimal trauma may be necessary. The etiology of these is generally through tearing of bridging cortical veins that pass within the subdural location to drain into the main venous sinuses. The appearance is often subtle and, as discussed earlier, using both the subdural and brain windows and the coronal plane will be helpful in their detection. Classic appearance is a crescent shape, which will be able to cross suture lines but usually unable to cross dural attachments. The density is dependent on timing. In the acute phase they are hyperdense to the surrounding brain parenchyma. As they age, they will begin to lose density. In the chronic phase they are hypoattenuating to the brain parenchyma (Figure 2). The most difficult time for detection is the subacute phase, where they will appear isoattenuating to the parenchyma. In this setting it is imperative to see the sulci and gyri extending to the inner calvarial table. If not, this may suggest a subacute subdural hematoma as the etiology. Hyperacute active hemorrhages show mixed density with regions of both decreased and increased attenuation where the hypoattenuating regions intermix with the hyperattenuating acute clotted blood; in this setting, hypoattenuating regions represent active bleeding.

SAH is caused by rupture of intracranial arteries and/or veins often seen in the setting of acute head trauma, although can also be seen after a ruptured circle of Willis aneurysm, with a chief symptom of worst headache of life.[9] Areas of hyperattenuation will be seen within the sulci and/or cisterns (Figure 3). If subarachnoid bleeding is found, the location may be helpful in differentiating aneurysmal versus nonaneurysmal (eg, most often traumatic). Even in the setting of trauma, one must consider the possibility of a ruptured aneurysm precipitating the trauma. When subarachnoid blood is seen within the sulci near regions of other fractures or intracranial contusion, this may suggest traumatic SAH as the cause. If the SAHs were seen in cisterns away from other regions of head trauma or in typical distributions for aneurysm, a ruptured aneurysm would be a leading concern prompting a CT head angiogram for further evaluation (Table 2).

Stroke

Acute infarcts can be ischemic, hemorrhagic, or ischemic with hemorrhagic transformation. Imaging is an important step to delineate them, as management is different. Hemorrhagic strokes account for approximately 10% to 15% of all

Table 2. Classic Location of Subarachnoid Hemorrhage (SAH) in Relation to Location of Saccular Aneurysm

SAH Location	Saccular Aneurysm Location
Interhemispheric fissure	Anterior communicating artery
Suprasellar cistern	Posterior communicating artery
Sylvian fissure, temporal lobe sulci	Middle cerebral artery
Cerebellopontine angle cistern and cisterna magna	Posterior inferior cerebellar artery
Interpeduncular cistern	Basilar tip
Associated intraventricular hemorrhage	
Third ventricle	Anterior or posterior communicating artery
Fourth ventricle	Posterior inferior cerebellar artery

strokes,[10] and infarcts with hemorrhage are a contraindication to thrombolytic therapy. Hemorrhagic infarcts are commonly found in the basal ganglia and cerebellum in the setting of hypertension. Other nonneoplastic etiologies for intraparenchymal hemorrhage are diverse and include but are not limited to amyloid angiopathy (elderly patient in lobar distribution), diffuse axonal injury (trauma patient with hemorrhage within subcortical white matter, corpus callosum, or brainstem), arteriovenous malformations, or cortical vein/venous sinus thrombosis (young patient without history of trauma). In arriving to a limited differential diagnosis for an intraparenchymal hemorrhage, the age, location, and history clearly become essential.

Ischemic strokes are far more common and often are not readily apparent on initial head CT, as findings are not seen in the hyperacute phase. When they are apparent, there will classically be a geographic region of cytotoxic edema manifesting as a moderately hypoattenuating region with loss of differentiation between the white and gray matter and associated mass effect in the acute setting (Figures 4 and 10). Locations are dependent on the vascular territory identified with MCA infarcts occurring most commonly.[11] Differentiating between an acute stroke versus an old infarct can sometimes be challenging. Acute infarcts are characterized by cytotoxic edema with moderate hypoattenuation whereas chronic infarcts demonstrate encephalomalacia and appear near that of CSF density and often with associated volume loss.[12]

Infection

In the emergency setting, infection should always be a consideration for any patient with symptoms of mental status changes or headaches.

Herpes encephalitis is the most common cause of sporadic viral encephalitis and has a classic location of involvement of the anterior and medial temporal lobes where one should look for hypoattenuation.[13] However, if no findings are seen on initial CT, this should not exclude the diagnosis, as they can be difficult to visualize before fifth day of infection.[13] Other infections such as cerebritis and abscess may present with vasogenic edema but are difficult to characterize on noncontrast CT. If high clinical suspicion, either a contrast-enhanced CT or MRI would be a more appropriate study. Meningitis may present with hydrocephalus but is without specific noncontrast head CT findings.

Conclusion

A systematic approach to head CT interpretation is necessary in any setting but most critical in the emergency department where rapid assessment is critical for optimal patient outcome. An outside-to-inside approach using various window and level settings optimizes detection of salient findings. A final dedicated approach to extracranial structures ensures that other pertinent abnormalities are addressed. Consistency to any approach is the most important factor when reviewing a study; however, a stepwise approach such as this will maximize both efficiency and accuracy in patient care.

References

1. Larson DB, Johnson LW, Schnell BM, et al. National trends in CT use in the emergency department: 1995-2007. *Radiology*. 2011;258(1):164-173.
2. Sharp AL, Nagaraj G, Rippberger EJ, et al. Computed tomography use for adults with head injury: describing likely avoidable emergency department imaging based on the Canadian CT head rule. *Acad Emerg Med*. 2017;24(1):22-30.
3. American College of Radiology. ACR-ASNR-SPR Practice Parameter for the Performance of Computed Tomography (CT) of the Brain. https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CT-Brain.pdf. Published 2015.
4. Michinaga S, Koyama Y. Pathogenesis of brain edema and investigation into anti-edema drugs. *Int J Mol Sci*. 2015;16(5):9949-9975. doi:10.3390/ijms16059949.
5. Riveros Gilardi B, Muñoz López JI, Hernández Villegas AC, et al. Types of cerebral herniation and their imaging features. *Radiographics*. 2019;39(6):1598-1610. doi:10.1148/rg.2019190018.
6. Weinstein MA, Duchesneau PM, Macintyre WJ. White and gray matter of the brain differentiated by computed tomography. *Radiology*. 1977;122(3):699-702.
7. Saenz RC. The disappearing basal ganglia sign. *Radiology*. 2005;234(1):242-243.
8. Orman G, Wagner MW, Seeburg D, et al. Pediatric skull fracture diagnosis: should 3D CT reconstructions be added as routine imaging? *J Neurosurg Pediatr*. 2015;16(4):426-431. doi:10.3171/2015.3.PEDS1553.
9. van Gijn J, Rinkel GJE. Subarachnoid haemorrhage: diagnosis, causes and management. *Brain*. 2001;124(2):249-278.
10. Vymazal J, Rulseh AM, Keller J, et al. Comparison of CT and MR imaging in ischemic stroke. *Insights Into Imaging*. 2012;3(6):619-627.
11. Ng YS, Stein J, Ning M, et al. Comparison of clinical characteristics and functional outcomes in ischemic stroke in different vascular territories. *Stroke*. 2007;38:2309-2314.
12. Srinivasan A, Goyal M, Al Azri F, et al. State-of-the-art imaging of acute stroke. *Radiographics*. 2006;26(suppl 1):S75-S95. doi:10.1148/rg.26si065501.
13. Zimmerman RD, Russell EJ, Leeds NE, et al. CT in the early diagnosis of herpes simplex encephalitis. *AJR Am J Roentgenol*. 1980;134(1):61-66. doi:10.2214/ajr.134.1.61.

To earn CME credit, you must read the CME article and complete the quiz and evaluation on the enclosed form, answering at least 70% of the quiz questions correctly. **Select the best answer and use a blue or black pen to completely fill in the corresponding box on the enclosed answer form.** Please indicate any name and address changes directly on the answer form. If your name and address do not appear on the answer form, please print that information in the blank space at the top left of the page. Make a photocopy of the completed answer form for your own files and mail the original answer form in the enclosed postage-paid business reply envelope.Your answer form must be received by Lippincott CME Institute, Inc., by **August 29, 2025.** Only two entries will be considered for credit. For more information, call (800) 638-3030.

Online quiz instructions: To take the quiz online, **log on to your account** at **www.contempneurosurg.com**, and click on the "CME" tab at the top of the page. Then click on "Access the CME activity for this newsletter," which will take you to the log-in page for **http://cme.lww.com**. Enter your *username* and *password*. Follow the instructions on the site. You may print your official certificate ***immediately.*** Please note: Lippincott CME Institute, Inc., **will not** mail certificates to online participants. **Online quizzes expire on the due date**.

The American Association of Neurological Surgeons (AANS) manually tracks *AMA PRA Category 1 Credits*™ earned from neurosurgery activities not sponsored or joint-sponsored by the AANS. As a service to AANS members, Lippincott CME Institute will continue to provide the AANS a monthly listing of their participants and the CME credits they earned so that AANS members do not have to send their individual certificates to the AANS for tracking.

1. Subdural is the *best* window to assess subtle peripheral extra-axial intracranial hemorrhage to limit artifact.

   True or False?

2. Epidural hematomas are almost always related to traumatic injury and often have an associated temporal bone fracture.

   True or False?

3. On CT, active hemorrhage will display CSF attenuation.

   True or False?

4. The middle cerebral artery is the *most* common location for an acute ischemic infarct.

   True or False?

5. Because of beam-hardening artifact, acute subdural hemorrhage is often difficult to detect in the temporal lobes.

   True or False?

6. Mass effect with midline shift is typically evaluated by assessing the location of the septum pellucidum.

   True or False?

7. A patient presents with recent trauma from a motor vehicle collision. The only finding on the noncontrast head CT is opacification of the maxillary sinus, with a few small locules of gas. The presumed cause of this finding is an occult fracture.

   True or False?

8. The disappearing basal ganglia sign is described in the setting of an intracranial mass.

   True or False?

9. A patient presents to the emergency department with no history of trauma. Noncontrast head CT is performed, which shows acute SAH within the basal cisterns. The next imaging step should be a head CT angiogram.

   True or False?

10. Figure 12 is a noncontrast head CT scan of a patient with a history of fall and altered mental status. The *most* likely diagnosis is an acute infarct.

    True or False?

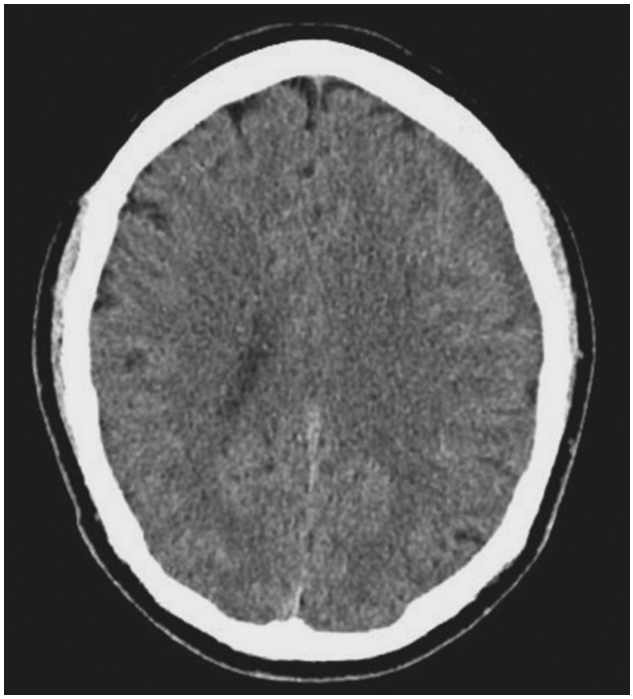

Figure 12.