Midnight Radiology: Emergency CT of the Head

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Introduction

Computed tomography of the head has become an integral part of the emergency evaluation of a wide variety of conditions and chief complaints, including trauma, stroke, seizure, altered mental status, headache, and fever. In 2005, one in eight patients in one tertiary care Emergency Department underwent a head CT [1]. This review will detail important head CT findings, focusing on those which are most relevant to immediate patient management decisions. We'll cover pearls and pitfalls in CT head interpretation, including normal variants that may mimic disease, and subtle abnormalities that may have substantial implications for patient care. We'll provide a simple and rapid approach to head CT interpretation using the best available evidence. Even if you are not primarily interpreting head CT, the information provided here may help you to understand the radiologist's interpretation and make appropriate clinical decisions based on that information.

Emergency Physician Interpretation of Head CT

 The first CT scanner in clinical use was installed in 1971 and required nearly seven minutes per slice [2]. Emergency Physicians have interpreted head CT for two decades, and numerous studies have considered the accuracy of our interpretation compared with other specialists such as neurologists, radiologists, and neuroradiologists. Although overall, changes in patient management are reported to occur in less than one percent of cases based on discrepancies between a radiologist's interpretation of an imaging test and that of an Emergency Physician [3], in the case of head CT subtle differences may be more important. Emergency Physicians perform with only moderate accuracy, correctly identifying contraindications to TPA on head CT in the setting of acute stroke in about two-thirds of cases, compared with about 80 percent for radiologists and neurologists [4]. Of note, in the setting of ischemic stroke, only 17% of Emergency Physicians in one study were 100% sensitive in detecting hemorrhage, compared with 40% of neurologists and 52% of radiologists [4] – so significant improvements in physician education are needed, in all specialties. Practice does improve our performance – Emergency Physicians who routinely interpret head CT outperform their colleagues who don't [4]. Emergency Physicians have also been shown to improve their interpretation abilities after brief training and to retain that improvement for three months, using a simple mnemonic, "Blood Can Be Very Bad" ([see below](#)) [5]. A particular area of weakness for Emergency Physicians is the recognition of early findings of ischemic stroke [5,6], an area upon which this article will focus.

Must Emergency Physicians Really Interpret Head CT? What is the availability of radiology interpretation?

 The availability of radiology interpretation services varies substantially depending on geographic location, time of day, and community or academic setting. A survey of community Emergency Departments in the radiology literature found that 8% had no night radiology coverage, while 82% relied on teleradiology. 38% were able to consult radiologists at night for radiography questions, and 92% of CTs were read by radiologists "in time for patient care decisions". However, in low-volume Emergency Departments, under 10,000 visits per year, over 20% have no radiology coverage at night, and over 50% have only teleradiology for CT interpretation [7]. In the academic setting, two-thirds of plain-film radiology interpretations for clinical care are provided by Emergency Physicians during the day, and 79% on nights and weekends. In 21%, no radiology over-read is available before the patient

has left the department. 39% of academic EDs report that images are read within four hours on weekdays, and only 19% on nights and weekends. Specifically regarding CT interpretation, over 50% of academic Emergency Departments report dissatisfaction with turn-around times for reading and reporting CTs, although interpretation times are not available [8].

What about teleradiology?

Does teleradiology spell the end of any need for Emergency Physicians to interpret imaging studies independently? While teleradiology has the potential to shorten interpretation times and to provide twenty-four hour coverage to small Emergency Departments, this advantage has not yet materialized. A Yale study showed a 39.5 minute mean time for head CT interpretation, using an internationally-based teleradiologist. Of note, in the 19% of cases where there was disagreement in interpretation between the local radiologist and the teleradiologist, the teleradiologist was more often correct, in 65% of cases! But a 40 minute average belies the true range of delays to interpretation, which ranged from seven to 164 minutes [9]. Even assuming a 30 minute interpretation time, this represents a substantial portion of the potential diagnostic and therapeutic window for conditions such as ischemic stroke, where rapid and accurate diagnosis is essential. Head CT interpretation remains an area where immediate interpretation by the treating physician could influence patient outcomes.

Some CT basics

The importance of head CT interpretation is clear, so let's review some basic terminology, outline our approach to head CT, and then look at clinically relevant CT findings.

Types of head CTs - Commonly ordered head CTs include non-contrast and contrast head CT. A number of variations on non-contrast and contrast head CT can also be ordered. We'll briefly describe these differences, although this review will focus on non-contrast CT.

Non-contrast head CT - Non-contrast head CT, the most common protocol, provides information about hemorrhage, mass-effect, ventricular abnormalities such as hydrocephalus, cerebral edema, sinus abnormalities such as fluid opacification, and bone abnormalities such as fractures. Specific variations on non-contrast head CT, such as sinus CT, facial CT, or orbital CT, may provide more detail by providing thinner slices through the region of interest or by changing the patient's position in the scanner during image acquisition -- but general information about the face and sinuses can be gleaned from a generic non-contrast head CT.
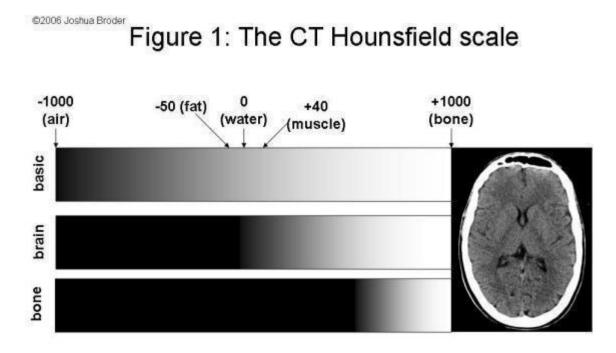
Contrast head CT - Contrast head CT is usually performed following a non-contrast CT, and the two are then compared. In a contrast head CT, intravenous contrast is injected, usually through an upper extremity IV. A time-delay is introduced to allow the venous contrast to pass to the brain. Depending on the delay between contrast injection and image acquisition, the resulting CT may be a CT cerebral arteriogram (CTA) or a CT cerebral venogram (CTV). The CT data may be reconstructed in any of several planes (typically axial, sagittal, or coronal) or even three dimensionally. Contrast CT is useful for depicting abnormal vascular structures such as aneurysms or AVMs, for demonstrating abnormal failure of filling of vascular structures, such as sagittal sinus thrombosis (akin to demonstrating a filling-defect in chest CT for pulmonary embolism – see the EMedHome.com article Midnight Radiology: Emergency CT of the Chest), and for demonstrating neoplastic, inflammatory, and infectious processes. A good example is so-called "ring-enhancement," the increased Hounsfield-density around abscesses, other infections such as toxoplasmosis, and tumors which occurs after IV contrast administration.

Hounsfield Units and Windows - As described in detail in an earlier EMedHome.com article (Midnight Radiology:

Emergency CT of the Abdomen), the color-scale assigned to CT images is named for one of the co-inventors of CT, Godfrey Hounsfield. Shifting the gray-scale to accentuate structures of a particular density is referred to as "windowing." For head CT, two window settings are important: bone windows and brain windows **(figures 1 and 2)**.

Bone windows - Bone windows are useful for evaluation in the setting of trauma. By shifting the gray scale to center on bone density, bone windows allow detection of abnormalities such as subtle fracture lines. At the same time, they sacrifice all detailed evaluation of structures less dense than bone (brain, CSF, blood vessels, air-containing structures) **(figures 1 and 2).**
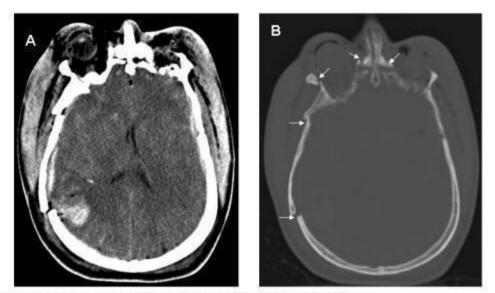
Brain windows - Brain windows are useful for evaluation of brain, other soft tissues such as hemorrhage, fluid-filled structures including blood vessels and ventricles, and air-filled spaces. The majority of our evaluations will be done using this window setting. On brain windows, bone and other dense or calcified structures (e.g. surgical clips, calcified pineal glands) all appear bright white, and internal detail of these high-density structures is lost **(figures 1 and 2).**

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Figure 1: The CT Hounsfield scale

The CT Hounsfield scale.

- Brain windows allow inspection of soft tissues, brain, hemorrhage, and CSF spaces. Detail of bone is obscured, although gross fractures may be seen. Note that on brain windows, CSF and air both will appear black.
- Bone windows give detailed information about fractures but obscure all soft tissue detail.

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Figure 2: Hounsfield Units and Windows

A single CT slice, shown in brain windows (A) and bone windows (B).

- Brain windows allow inspection of soft tissues, brain, hemorrhage, and CSF spaces. Detail of bone is obscured, although gross fractures may be seen.
- Bone windows give detailed information about fractures (small arrows) but obscure all soft tissue detail.
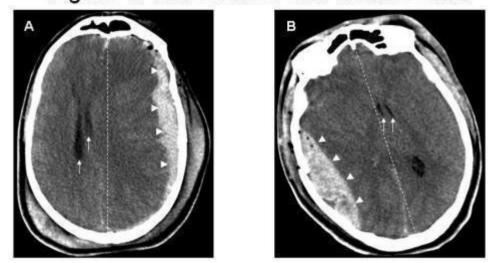
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Mass effect - The brain is normally a symmetrical structure. Mass effect refers to the distortion of the size or position of normal brain structures (including ventricles and sulci) when they are displaced by an abnormal structure or volume. This displacement may occur due to tumor, hemorrhage, edema, or obstruction of cerebrospinal fluid flow, to name but a few common causes. One measure of the severity of mass effect is the displacement of structures across the normal midline of the brain, called "midline shift." Modern PACS systems have convenient measurement tools that make this easy. Midline shift may have some prognostic value in determining the likelihood of regaining consciousness after surgical decompression; patients with significant shift, greater than 10mm, are more likely to benefit [10]. Patients with shift of 5mm or more are more likely to have neurologic deficits requiring long-term supervision [11]. Midline shift is also linked to probability of death after traumatic brain injury [12]. Published neurosurgical guidelines for surgical indications for brain lesions include midline shift as one of several parameters (as you'll see below), so recognizing and measuring midline shift is important **(figures 3 and 4).**

Figure 3: Mass effect and midline shift

The brain is normally a symmetrical structure. When a mass or hemorrhage deviates the normal position of an imaginary line dividing the brain, or pushes structures across this line, "midline shift" is present. The degree of midline shift may be more important than the exact etiology of the shift, since shift threatens to cause subfalcine herniation and may be an indication for surgery.

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Figure 4: Mass effect and midline shift

Two examples of mass effect and midline shift.

A. A large subdural hematoma (arrowheads) creates a mass effect and is displacing brain structures including the lateral ventricles (arrows) across the midline (dotted line).

B. A large epidural hematoma (arrowheads) creating mass effect. The lateral ventricles (arrows) are shifted across the midline (dotted line) to the patient's left.
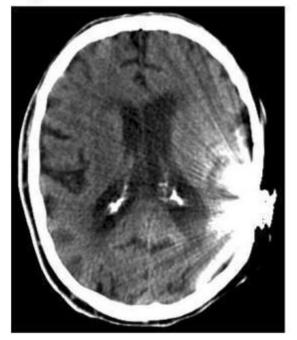
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Artifacts

Motion - Modern CT scanners acquire images at a very fast rate – a 64 slice CT can scan the entire brain in approximately 5 seconds [13].   As a consequence, CT is less subject to motion artifact than in the past, although significant patient motion may still render images uninterpretable. Just as in standard photographs, motion results in a blurry CT image.

Metallic streak artifact - Very dense objects create distortion on CT, called "streak artifact" or "beam-hardening artifact." Examples include implanted metallic devices, such as cochlear implants and dental fillings, or metallic foreign bodies such as bullets. These artifacts may make it difficult or impossible to identify pathologic changes in the region **(figure 5)**.

Figure 5: streak artifact

A metallic object (here, a cochlear implant) creates streak artifact which renders the patient's left hemisphere difficult to interpret. Nonetheless, important clinical information can be discerned, even from this imperfect scan. For example, no midline shift is visible.
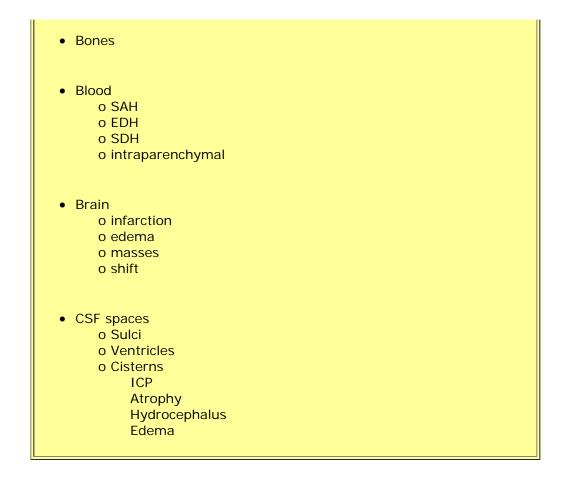
Click On Image to Enlarge

Approaching the Head CT

A systematic approach to head CT interpretation is essential to avoid missing important findings. Perron et al. found that a systematic approach based on a mnemonic improved interpretation accuracy among EM residents from 60 to almost 80% in a mere two hours, and this advantage was retained for three months [5]. Their mnemonic (**B**lood **C**an **B**e **V**ery **B**ad) concentrated on **b**leeding, cerebral **c**isterns, **b**ones, **v**entricles, and **b**rain, but performed relatively poorly for conveying information about infarction. We'll use a different mnemonic from the Perron group, adding information about air-spaces, consolidating the CSF spaces, and adding to the tools for recognizing infarction. Although a detailed understanding of neuroanatomy will improve your head CT interpretation, our mnemonic will avoid significant anatomic detail, as many clinical decisions don't require this level of sophistication.

A Mnemonic For Head CT: ABBBC

- Air-filled spaces
    - o Sinuses
    - o Mastoid air-cells
        - infections
        - fractures

- Bones

- Blood
    - o SAH
    - o EDH
    - o SDH
    - o intraparenchymal

- Brain
    - o infarction
    - o edema
    - o masses
    - o shift

- CSF spaces
    - o Sulci
    - o Ventricles
    - o Cisterns
        - ICP
        - Atrophy
        - Hydrocephalus
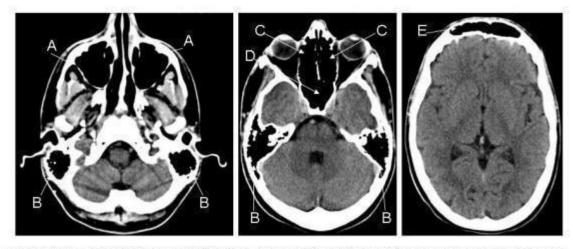   - Edema

The Mnemonic: ABBBC

A is for Air-spaces

Our mnemonic starts with A, for air-filled spaces in the head. The normal airspaces (frontal, maxillary, ethmoid, and sphenoid sinuses, plus the mastoid air-cells) appear black on either brain or bone windows, since air has the lowest Hounsfield density, negative 1000. The frontal, maxillary, ethmoid, and sphenoid sinuses are normally air-filled with no thickening of mucosa or air-fluid levels. The mastoid air cells are normally spongy bone filled with tiny pockets of air. If these airspaces become partially or totally opacified with fluid, this is easily recognized as a gray or white shade. Recognizing abnormalities of normally air-filled structures requires some basic knowledge of their normal location and configuration **(figure 6)**.
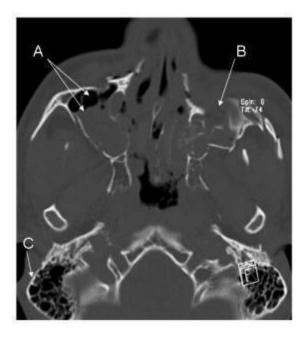
Figure 6: air-filled spaces

3 slices from a head CT, demonstrating the normal location and appearance of air-filled spaces when viewed on "brain windows." Air-filled spaces are normally black.

A. Maxillary sinuses
B. Mastoid air cells
C. Ethmoid sinuses
D. Sphenoid sinus
E. Frontal sinus

Click On Image to Enlarge

Sinus Trauma

 In trauma, fractures through the bony walls of sinuses result in bleeding into the sinus cavity. While the trauma patient remains in a supine position, this blood accumulates in the dependent portion of the sinus, forming an air-fluid level visible on CT. Previously existing sinus disease may be visible as circumferential sinus mucosal thickening, rather than as an air-fluid level. Inspect the sinuses carefully for air-fluid levels, as these may indicate occult fractures. In fact, in trauma, opacification of sinuses should be considered as evidence of fracture until proven otherwise, as the fracture itself may be hard to identify **(figure 7)**. The ethmoid sinuses are small and may be completely opacified by blood in the event of fracture. Opacified ethmoid sinuses should increase suspicion of a medial orbital blowout fracture and possible entrapment. Air-fluid levels in the maxillary sinus may be associated with inferior orbital blowout fractures, since the inferior wall of the orbit is the superior wall of the maxillary sinus. The frontal sinus is less easily fractured, as its anterior and posterior plates are thick and resistant to trauma. Fracture of the anterior wall of the frontal sinus is relatively less concerning, requiring plastic surgery or otolaryngology consultation. Fracture of the posterior wall of the frontal sinus is a potential neurosurgical emergency, due to communication of the sinus space with the CSF. Look for intracranial air whenever frontal sinus air-fluid levels are present and disruption of the posterior plate is suspected. When the mastoid air-cells are obliterated or opacified, suspect temporal bone fracture. The normal side is a useful comparison.

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Figure 7: fluid in normally air-filled spaces

Following trauma, fractures of the bony walls of sinus can result in bleeding. Blood pools in the dependent portion of air-filled spaces and results in an air-fluid level or sometimes in complete opacification of the normal air-space. Sometimes this is the only evidence of fracture. Without a history of trauma, fluid in an air-space may represent infection, such as sinusitis or mastoiditis.

Here, viewed on bone windows, the patient's right maxillary sinus (A) has an air (black) –fluid (gray) level. The left maxillary sinus (B) is badly fractured and completely opacified. The mastoid air cells (C) are normal and have no fluid, only thin bone trabeculae. In trauma, opacified mastoid air-cells suggest temporal bone fracture.

Click On Image to Enlarge

Sinus infections

In the absence of trauma, sinus mucosal thickening and air-fluid levels *may* be normal findings. They should not be used to make a diagnosis of bacterial sinusitis in the absence of strong clinical evidence, as they are nonspecific and may occur in allergic sinusitis or even asymptomatic patients. The mastoid air-cells are not normally fluid-filled, and in the presence of mastoid tenderness and erythema, their opacification on CT is evidence of mastoiditis.

Quick Tips: Sinuses and Mastoids

- Appearance: normally air-filled (black), no air-fluid levels
- Significance:
    - air-fluid levels or opacification in the setting of trauma may indicate fracture
    - in the absence of trauma, air-fluid levels or mucosal thickening may be *too* sensitive, and should not necessarily be equated to bacterial sinusitis in the absence of strong clinical evidence
    - mastoid opacification without trauma indicates mastoiditis

B is for Bones

The first "B" in our mnemonic is for bones. In trauma, bony fractures should be suspected, although they are often of less clinical significance than the underlying brain injury. To inspect for fractures, the PACS should be set to "bone windows," which will allow assessment of the internal structure of bone. In order to recognize fractures, normal suture lines should be identified. When a possible fracture is identified, inspect the opposite side for a similar finding, which suggests suture line if present **(figure 8)**. When a fracture is identified, look carefully for associated abnormalities. Inspect for any of the types of hemorrhage described below. Look for soft tissue swelling outside the calvarium overlying the fracture. On *brain* windows, inspect for air (black) within the calvarium (pneumocephalus, **figure 9**), which indicates an open fracture. Air may take the form of large amorphous collections abutting the calvarium, or small black spheres within hemorrhage associated with the fracture. To detect fractures through sinuses, look for associated air-fluid levels within sinuses, which in trauma are likely blood resulting from fracture (above).

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Figure 8: fractures

Fractures, viewed on bone windows. In A, multiple fractures are visible (arrows). The dotted line connecting to the contralateral side confirms that no sutures are present in these locations. In B, multiple displaced fractures are present (arrows demonstrate several). The patient's left maxillary sinus is badly comminuted. The associated air-fluid levels and opacification are additional clues.

Click On Image to Enlarge

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Figure 9: pneumocephalus

In this patient with multiple skull fractures, air (arrows) has entered the calvarium and is visible as black areas on brain windows. On this setting, CSF and air both appear black, so fine adjustment of the window setting may sometimes be necessary to confirm that this finding is air. In this case, the location, immediately adjacent to fractures, and the rounded shape, typical of air bubbles, are confirmatory.

Click On Image to Enlarge

Quick Tips: Fractures

- Appearance:
    - cortical defect, not in position of normal suture
    - air-fluid level in sinus
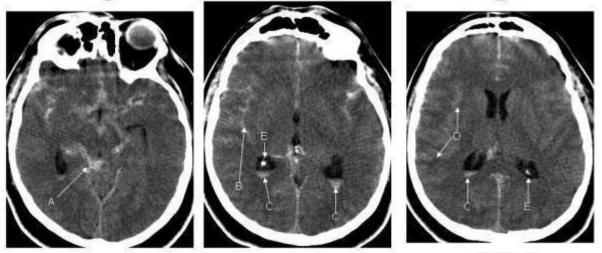

B is for Blood

The second "B" in our mnemonic is for blood. Hemorrhage can occur in any of several spaces within or around the brain. The shape and density of blood collections on CT depend on a number of factors, including the age of the blood products and the anatomic location. Unless otherwise stated, the descriptions in this section assume the use of "brain windows" during image interpretation. In general, recent hemorrhage appears as a bright white color on non-contrast CT. As time elapses, blood changes to a darker color, indicating lower density. This is likely due to a number of factors, including the absorption of water by hematoma. As a consequence, the sensitivity of CT to detect hemorrhage is thought to decline as time elapses from the moment of hemorrhage, although debate

continues about the accuracy of CT in dating blood [14].

Subarachnoid hemorrhage

Subarachnoid hemorrhage (SAH) is blood within the subarachnoid space, which includes the sulci, Sylvian fissure, ventricles, and cisterns. Fresh SAH appears white, although the appearance varies depending on the ratio of blood to cerebrospinal fluid [15]. CT is believed to be greater than 95% sensitive for subarachnoid hemorrhage within the first 12 hours, but decline to 80% or less after 12 hours [16-18]. SAH may result from trauma or may occur spontaneously after rupture of an abnormal vascular structure such as an aneurysm. When looking for SAH, inspect the sulci for blood. In addition, common locations for SAH are the ventricles, Sylvian fissure, and cisterns surrounding the brainstem. Because subarachnoid blood may diffuse into adjacent regions, it may defy the guideline that hemorrhage and other abnormalities disturb normal brain symmetry. In other words, large amounts of subarachnoid hemorrhage, including hemorrhage into cisterns, may actually result in a symmetrical appearing head CT. Beware of this possibility when inspecting the brain for abnormalities. As time elapses from the moment of hemorrhage, blood will likely diffuse through the subarachnoid spaces, like a drop of food coloring dropped into a glass of water. Thus a bright white punctate finding on head CT is not likely to be SAH, especially hours after the onset of clinical symptoms. **Figure 10** shows several examples of SAH, involving different brain regions. Subarachnoid hemorrhage may be accompanied by other important changes, including hydrocephalus and cerebral edema, discussed below.

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Figure 10: subarachnoid hemorrhage

caudad ──────────────────→ cephalad

Diffuse subarachnoid hemorrhage. Note the presence of subarachnoid blood (bright white) filling the sulci, as well as extending into the cisterns, Sylvian fissures, and even lateral ventricles.

A blood in basilar cistern

B blood in Sylvian fissure

C blood in posterior horns of lateral ventricles

D blood in sulci

E extremely bright calcifications in the choroid plexus of the posterior horns of the lateral ventricles are common, normal findings – do not confuse these for hemorrhage. Note the similarity in density to bone of the calvarium.
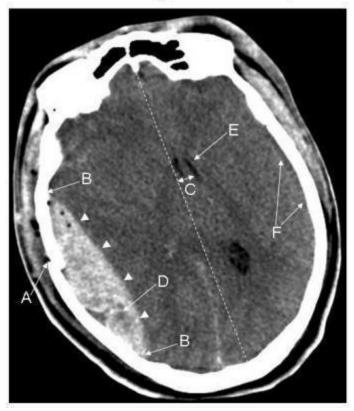
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Quick Tips: Subarachnoid Hemorrhage

- <u>Appearance:</u> White on brain windows
- <u>Location</u>: localized or diffuse
    - Sulci
    - Fissures
    - Cisterns
    - Ventricles
- <u>Shape</u>: assumes shape of surroundings
- <u>Pearl</u>: suspect increased ICP, look for signs of diffuse edema

Epidural hematoma

Epidural hematoma (EDH) is a collection of blood lying outside the dura mater, usually between the dura and calvarium. It is almost always a traumatic injury, resulting from injury to the middle meningeal artery. Because blood is extravasating from an artery under high pressure, rapid enlargement of the hematoma may occur, leading to significant mass effect and herniation. The common CT appearance is a biconvex disc or lens, collecting in the potential space between the calvarium and dura mater [19]. This shape occurs because the more superficial aspect of the EDH conforms to the curve of the calvarium, while the inner aspect expands and presses into the dura. The dura is usually tethered to the calvarium at sutures, so EDHs usually do not cross suture lines on CT. EDH may cross the midline, since there are no midline sutures in the frontal and occipital regions. The usual location of an EDH is temporal, although they occasionally occur in other locations. Transfalcine herniation may occur with epidural hematomas, so the midline of the brain should be carefully inspected on CT for midline shift or compression of the lateral ventricle. The "swirl sign", described as a bright white vortex or "swirl" within the EDH, has long been considered a finding of active bleeding and should be interpreted as a sign of continued expansion, although recent studies have questioned the prognostic significance of this finding [20-23]. **Figures 11 and 12** show several examples of epidural hematomas, with the classic findings described above. Interestingly, the volume of hematoma has not been shown to correlate with preoperative neurologic status or 6 month post-operative status [24].

Figure 11: epidural hematoma

This epidural hematoma (arrowheads) demonstrates several classic features:

• lens-like or biconvex disc shape
• temporal location, with associated temporal bone fracture (A)
• does not cross suture lines (B - expected location of suture, see also bone window small figure)
•mass effect with midline shift (C)
•swirl sign indicating active bleeding (D)
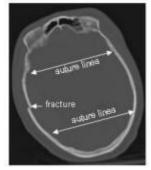•elevated ICP, with small ventricles (E) and no visible sulci (F)

__Click On Image to Enlarge__

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Figure 12: epidural hematoma

This epidural hematoma (arrowheads) demonstrates several classic features:

• lens-like or biconvex disc shape
• temporal location

• Although it appears small on this slice, the entire CT may reveal a different story. On this slice, sulci are absent, raising concern for elevated ICP.

Click On Image to Enlarge

Indications for surgery for EDH

Published criteria for surgical evacuation of an acute epidural hematoma include volume greater than 30cm^3 (regardless of GCS). Many PACS toolkits allow automated computation of volumes from 2D data sets by measuring the thickness of a structure or outlining it. For patients with GCS>8 and no focal deficit, EDH smaller than 30cm^3, less than 15mm thick, and with less than 5mm of midline shift can be managed nonoperatively. Anisocoria with GCS <9 is an indication for surgery, regardless of EDH size [25].

Quick Tips: Epidural Hematoma

- Appearance: Variable white to gray on brain windows
- Location: Usually temporal
- Shape: Biconvex disc or lens
- Pearl: does not cross suture lines
- White "swirl sign" means active bleeding

- <u>Significance</u>: May cause mass effect and herniation
  - Look for midline shift
  - Look for effacement of ventricles and sulci

Subdural hematoma

Subdural hematoma (SDH) is a collection of blood between the dura mater and the brain surface. Subdural hematomas usually occur from traumatic injury to bridging veins, although a history of trauma is not always found. Subdural hematomas may be self-limited in size due to the lower pressure of venous bleeding, but they can cause significant mass effect, resulting in herniation. They may also re-bleed after an initial delay, resulting in expansion. Moreover, they are frequently markers of significant head trauma, and patient outcomes may be compromised by associated diffuse axonal injury (see below) or edema. The typical CT appearance of subdural hematoma is a crescent, with the convex side facing the calvarium and the concave surface abutting the brain surface [16]. The shape of subdural hematomas results from their accumulation between dura and brain surface. There is no restriction of the extension of a SDH by sutures, as is seen in EDH, so SDH may cross suture lines. Moreover, each cerebral hemisphere is wrapped in its own dura, so SDH typically do not cross the midline but instead may continue to follow the brain surface into the inter-hemispheric fissure.

The color may vary depending on the age of the SDH. Fresh subdurals are typically brighter white (or lighter gray) than the adjacent brain. Older subdural hematomas, or acute hematomas in anemic patients, may become similar in density (isodense) to the adjacent brain and thus may be difficult to detect [26,27]. Clues to their presence include the obliteration of sulci on the brain surface, or mass effect resulting from the SDH. Still older subdural hematomas may become similar in density or color to the cerebrospinal fluid surrounding the brain, and thus may be difficult to recognize. Sometimes SDH are multi-colored or layered, indicating hemorrhage at multiple different times.

Indications for surgery for SDH

Published criteria for surgical evacuation of an acute subdural hematoma include thickness greater than 10mm or midline shift greater than 5mm, regardless of GCS. Surgery may be indicated with smaller SDH and lesser degrees of shift in patients with GCS less than 9, based on ICP, pupillary findings, and worsening GCS [28].
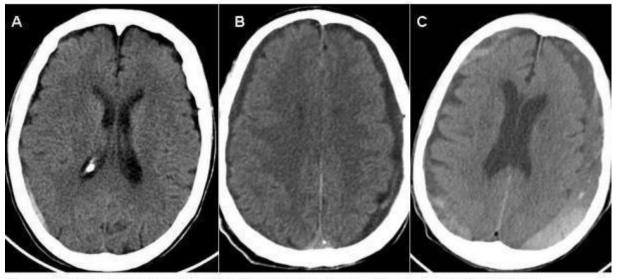
Figures 13 and 14 demonstrate some of the classic and less common findings of SDH.

Figure 13: subdural hematoma

This subdural hematoma (arrowheads) demonstrates several classic features:
- crescent shape
- crosses suture lines (A)
- mass effect with midline shift (B)
- elevated ICP, with small ventricles (C) and no visible sulci (D)

Click On Image to Enlarge

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Figure 14: subdural hematomas

Three variations of subdural hematoma. A, a small subdural hematoma. B, bilateral subdural hematomas, likely subacute as they are hypodense relative to brain. C, bilateral subdural hematomas with varying density, likely indicating hemorrhage at different times.

Click On Image to Enlarge

Quick Tips: Subdural Hematoma

- Appearance: Variable white to gray on brain windows
- Location: variable
- Shape: crescent shaped
- Pearl: may cross suture lines
- Significance: May cause mass effect and herniation
  - Look for midline shift
  - Associated with other brain injuries -- look for effacement of ventricles and sulci
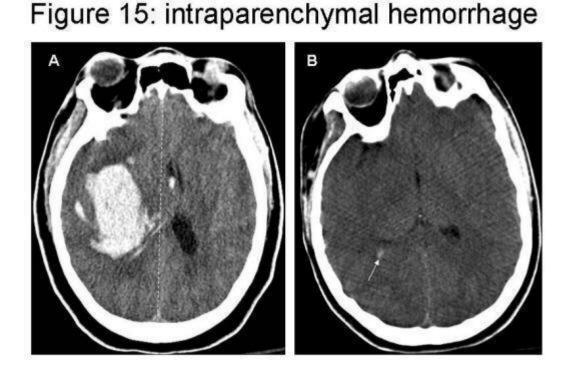- Surgical indications: 10mm thickness or 5mm midline shift

Intraparenchymal hemorrhage

Intraparenchymal hemorrhage, or hemorrhage within the substance of the brain matter, may occur in trauma or spontaneously, perhaps as a complication of hypertension. The appearance is generally bright white acutely. The

size may vary from punctate to catastrophically large, with associated mass effect and midline shift. Particularly for smaller punctate hemorrhages, care must be taken not to mistake hemorrhage for normal benign calcifications of the pineal gland, choroid plexus, and meninges, or vice versa. For these lesions, mass effect such as midline shift or ventricular effacement should be assessed. Signs of increased intracranial pressure should be identified.
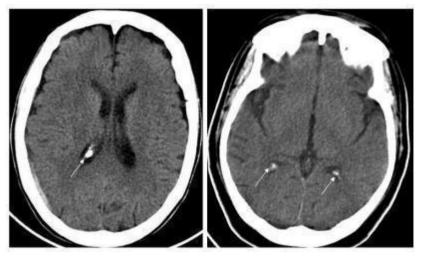
Figures 15 and 16 demonstrate common appearances of intraparenchymal hemorrhage and normal calcifications.

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Figure 15: intraparenchymal hemorrhage

Intraparenchymal hemorrhage. A, a large hemorrhage with midline shift and mass effect. B, a small amount of hemorrhage (arrow).

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Figure 16: choroid calcifications

Calcification in the choroid plexus(arrows) may resemble punctate intraparenchymal hemorrhage but is a common incidental finding. Clues are its bright white density, similar to bone of the calvarium, location in the posterior horn of the lateral ventricle, and frequent bilaterality.

Click On Image to Enlarge

Indications for surgery for parenchymal hemorrhage

Published criteria for surgical treatment of traumatic intraparenchymal hematoma are complex. They include size greater than 20cm^3 with midline shift greater than 5mm, and/or cisternal compression, if GCS is 6 to 8. Lesions greater than 50cm^3 in size should be managed operatively, regardless of GCS [24].

Quick Tips: Intraparenchymal Hemorrhage

- Appearance: usually bright white
- Location: variable
- Shape: rounded or irregular
- Significance: May cause mass effect and herniation
  - Look for midline shift
  - Associated with other brain injuries -- look for effacement of ventricles and sulci
- Don't be fooled by normal calcifications

B is for brain

The third "B" in our mnemonic is for brain. Brain abnormalities include neoplastic masses, abscesses, infarction, localized vasogenic edema, global brain edema, and diffuse axonal injury.
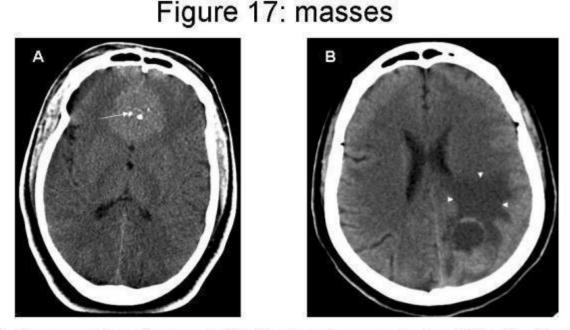
Masses

Masses are best delineated on CT with IV contrast, or with MRI. However, noncontrast CT can demonstrate a variety of masses if they are of sufficient size. In some cases, the mass itself may not be seen, but secondary findings such as mass effect or vasogenic edema may occur. IV contrast is useful in detecting masses because they are generally extremely vascular and thus enhance in the presence of contrast material.

On noncontrast CT, masses may be denser than surrounding brain if they are calcified. Examples are meningiomas, which are usually midline structures emanating from the falx cerebri.

Vasogenic edema

Malignant primary brain neoplasms or metastatic lesions often appear "hypodense" (darker, or grayer) compared with normal brain. This appearance is typical of localized vasogenic edema surrounding the lesion. Neoplasms often secrete vascular endothelial growth factor, resulting in the development of immature blood vessels that perfuse the tumor. These immature vessels have leaky endothelial junctions, allowing fluid to extravasate into the interstitium, causing vasogenic edema. Vasogenic edema must be differentiated from infarction, which may also cause a hypodense appearance. Vasogenic edema need not conform to a normal vascular territory within the brain, while hypodensity associated with ischemic stroke does. Vasogenic edema responds to treatment with dexamethasone, while other forms of cerebral edema such as traumatic edema do not [29].

Figure 17 shows examples of a meningioma with calcification, and a malignant tumor with vasogenic edema.

Figure 17: masses

A, a typical appearance for meningioma. Calcifications (arrow) are present and are distinguished from hemorrhage by their intense white color.

B, a mass with surrounding vasogenic edema (arrowheads). This form of edema is hypodense, like infarct, but does not correspond to a vascular territory. An abscess could have a similar appearance.

Click On Image to Enlarge

Abscesses

Abscesses may be visible on noncontrast CT as hypodense regions **(figure 17, B)**, occasionally with air within them. This appearance may be nonspecific, and a differential diagnosis including toxoplasmosis, mass with vasogenic edema, or CNS lymphoma may be considered, depending on the patient's clinical presentation. Abscesses, toxoplasmosis, and masses all may undergo "ring-enhancement," an increase in density around the lesion after administration of IV contrast. This reflects increased blood flow in the vicinity of the lesion, as well as leaky vascular structures which allow extravasation of contrast in the region.

Ischemic stroke and infarction

Ischemic stroke accounts for 85% of strokes [30]. It is potentially one of the most important indications for head CT, and is an area in which the interpretation of CT by Emergency Physicians might play the greatest role by shortening the time to diagnosis. One obvious reason is the three hour window for administration of intravenous tissue plasminogen activator – an intervention still fiercely debated in the Emergency Medicine community, and which has been reviewed elsewhere [31]. Understanding CT findings of acute ischemic stroke is important -- for those who do not believe in administration of TPA, they provide yet another argument against the treatment, while for those who would use TPA in select patients, they may allow more rational and safer patient selection. Apart from TPA administration, rapid diagnosis of ischemic stroke may allow the Emergency Physician to make better

informed decisions about patient management and disposition. If new stroke therapies such as intra-arterial thrombolysis and clot retrieval become validated and more widely available, rapid CT interpretation for ischemic stroke may become even more valuable.

A complex cascade of events leads to the evolving appearance of ischemic stroke on head CT.   Initially, at the moment of onset of cerebral ischemia, no abnormalities may be seen on head CT – thus this is one of the most difficult diagnoses for the Emergency Physician, as a CT may be normal in the presence of significant pathology. Studies have shown Emergency Physicians to be relatively poor at recognizing early ischemic changes, which we will review here.

How early does the non-contrast head CT indicate ischemic stroke?

Analysis of the NINDS data shows that early ischemic changes are quite common in ischemic stroke, occurring in 31% within 3 hours of stroke onset, in contrast to the widely held belief that ischemic strokes become visible on CT only after 6 hours [32,33]. Some findings may occur immediately, such as the hyperdense MCA sign, while other findings may require time to elapse, with the gradual failure of ATP-dependent ion pumps and resulting fluid shifts.

Early Ischemic CT changes within 3 hours, possibly altering management, from NINDS	
Any change	31%
Loss of GWMD	27%
Hypodensity	9%
Compression of CSF spaces	14%
Loss of GWMD >1/3 MCA	13%
Hypodensity> 1/3 MCA	2%
Compression of CSF spaces >1/3 MCA	9%
*GWMD = gray-white matter differentiation	

Hyperdense MCA sign

The hyperdense MCA sign is a finding of hyperacute stroke, indicating thrombotic occlusion of the proximal middle cerebral artery. This may be present on the initial noncontrast head CT *immediately following symptom onset*, since the finding does not require the failure of ion pumps and fluid shifts that lead to other ischemic changes on head CT. Because this lesion is associated with ischemia in the *entire MCA territory*, typically the patient with this finding will have profound hemiparesis or hemiplegia on the contralateral side, as well as other findings such as language impairment depending on the side of the lesion. In other words, this finding is not associated with mild or subtle strokes. In fact, the presence of a hyperdense MCA sign is an independent predictor of neurologic deterioration [34].

It may seem surprising that this vascular abnormality is visible on noncontrast head CT. As the name implies, the middle cerebral artery appears hyperdense (bright white) as compared with the normal side. A specific Hounsfield unit threshold of greater than 43 units has been recommended to avoid false positives [35]. Use your knowledge of

the location of the patient's neurological deficits to direct you to the likely side of the lesion, which will be on the contralateral side. Then use the normal symmetry of the brain to help you identify this abnormality. A related finding, the MCA "dot" sign, has been validated by angiography and found to be a very specific marker of branch occlusion of the MCA. This sign appears as a bright white dot in the sylvian fissure on the affected side [36].

Figure 18 shows the hyperdense MCA sign.

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Figure 18: hyperdense MCA sign

The hyperdense MCA sign (arrow) is a finding of hyperacute stroke and reflects thrombosis of the proximal middle cerebral artery.

Click On Image to Enlarge

Gray-white differentiation

Understanding this finding of stroke requires a brief and simple review of neuroanatomy. Gray matter is brain tissue without myelin – examples include the cerebral cortex. White matter is myelinated axons in brain tissue – rendered white by the high lipid content of the myelin sheath. You'll recall from the earlier discussion of Hounsfield units that lower density on CT means a darker color – low density fat appears a darker gray than does higher density water. Thus, the higher fat content of white matter makes it appear darker on CT. In other words, on a normal head CT, gray matter is white and white matter is gray. **Figure 19** shows the normal gray-white boundary.

Figure 19: gray-white matter

Normal gray-white matter differentiation. Myelinated areas (white matter) have a higher fat content than unmyelinated regions (gray matter). As a consequence, white matter is lower density and appears darker on CT. The dotted line on the patient's right outlines the border between gray and white matter. Trace this interface yourself on the patient's left.

When this interface becomes less discrete, due to ischemia, the CT finding is called loss of gray-white differentiation.

Click On Image to Enlarge

Loss of Gray-white differentiation

In an ischemic stroke, as brain tissue uses up ATP and is unable to replenish it, ATP-dependent ion pumps stop working. Ions equilibrate across membranes, and fluid shifts occur. Gray matter gains fluid, lowering its density (hypodensity), and as it does, its density becomes more similar to that of white matter. Since differences in density are the reason that these tissues look different on CT, their appearances become more similar, and it becomes more difficult to discern where gray matter ends and white matter begins. This change is called "loss of gray-white differentiation," and it is an early finding of ischemic stroke, occurring within 3 hours after onset of ischemia [32]. **Figure 20** shows an abnormal gray-white boundary.

Figure 20: gray-white matter

In this example, the gray-white differentiation is normal on the patient's right but is being lost on the patient's left. In fact, the patient is developing the frank hypodensity of ischemic stroke.

 Click On Image to Enlarge

Quick Tips: Ischemia

- Hyperdense MCA sign
- Loss of gray-white differentiation
  - Insular ribbon sign
  - Cortical sulcal effacement
- Ischemic focal hypoattenuation
  - MCA territory
  - basal ganglia
  - lentiform nucleus

Insular ribbon sign (loss of insular ribbon)

The insula (or insular cortex) is a thin ribbon of gray matter tissue which lies just deep to the lateral brain surface, separating the temporal lobe from the inferior parietal cortex. On CT, it is visible as the tissue layer lining the Sylvian fissure. This region is subject to early ischemic changes in the form of loss of gray-white differentiation, often called the insular ribbon sign or loss of the insular ribbon, as this area becomes less distinct.
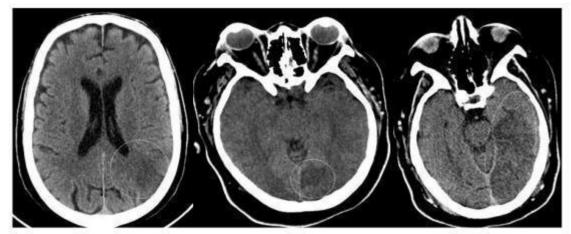
Hypodensity in ischemic stroke

Ischemic brain looks hypodense, or darker than normal brain in the same anatomic region. This change occurs for the same general reasons as does loss of gray-white differentiation. As neurons run out of ATP, cytotoxic and vasogenic edema occur. Ion gradients run back toward equilibrium, and water shifts into gray matter, making it less dense relative to normal tissue. The appearance of an infarct becomes progressively more hypodense over the first several days to weeks of an ischemic stroke. Again, this finding can occur as an early change within 3 hours of symptom onset [32].
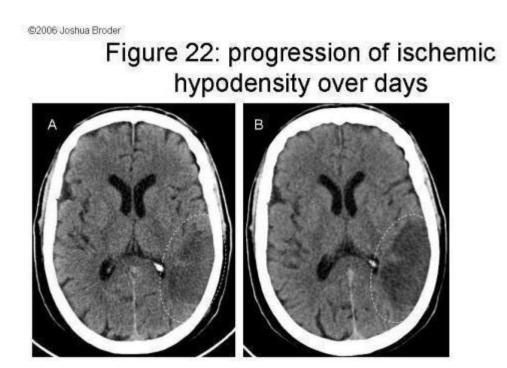
Figure 21 shows subtle examples of hypodensity. **Figure 22** shows the progressive hypodensity of an ischemic stroke over several days.

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Figure 21: early ischemic hypodensity

Three examples of subtle hypoattenuation in early stroke (dotted circles). Large areas of hypoattenuation predict an increased risk of hemorrhage and are relative contraindications for TPA.

Click On Image to Enlarge

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Figure 22: progression of ischemic hypodensity over days

A left MCA distribution stroke, day two (A) and day four (B). The exact rate at which infarctions appear likely depends on the degree of ischemia and collateral circulation.

Click On Image to Enlarge

Hypodensity of ischemic stroke versus vasogenic edema of masses

Hypodensity in an ischemic stroke follows a vascular distribution, whereas hypodensity due to vasogenic edema around a mass need not respect vascular territories.

Are ischemic changes a contraindication to TPA?

Early ischemic stroke findings were NOT used as exclusion criteria in the National Institute of Neurological Disorders and Stroke (NINDS) trial, which required only the absence of hemorrhage on initial head CT [37].   However, multiple studies following NINDS have shown an increased risk of intracranial hemorrhage, bad neurologic outcomes, and death in patients with early ischemic changes on head CT [38,39]. Ischemic changes are relative contraindications to TPA administration, and their presence may also suggest that greater than three hours have elapsed from symptom onset, in which case systemic TPA may be absolutely contraindicated. In addition, *the FDA, American Heart Association, and American Academy of Neurology specifically recommend against administering TPA if early signs of major infarction are present, due to increased risk of intracranial hemorrhage* [40,41,42]*.*   MCA infarction greater than 1/3 of the MCA territory predicts increased bleeding risk if TPA is given, and is poorly detected by radiologists, neurologists, and Emergency Physicians in past studies [6,43,44]. In addition, the greater the extent of ischemic changes on CT, the higher the risk of bleeding as demonstrated in the multinational ECASS II trial [39].

 The many findings of ischemic stroke may seem too much to hope to remember, and their clinical relevance may appear unclear. A few simple rules can make sense of this. First, a normal head CT is perhaps the most likely finding if the patient presents within 3 hours of symptom onset. In this setting, the most important job of the Emergency Physician in interpreting the head CT is to rule out hemorrhage. Second, in the presence of significant unilateral neurologic abnormalities, the hyperdense MCA sign should be sought. Third, early changes such as loss of gray-white differentiation and hypodensity should be identified, again using the patient's clinical symptoms to direct you to the likely abnormal side of the brain. These early changes may imply an earlier time of onset than suggested by the history, or a massive stroke in progress.

Cerebral Edema

Diffuse cerebral edema can result from many different pathological processes, including trauma, anoxic brain injury, carbon monoxide poisoning, and systemic fluid and electrolyte abnormalities. The appearance is therefore not diagnostic of the underlying etiology. As the brain swells, several changes occur. Cerebrospinal fluid spaces become collapsed as they give way to the increasing volume of solid brain tissue. As a result, the lateral ventricles become slit-like and ultimately become obliterated. In addition, the sulci become effaced as the gyri swell. The normal rim of CSF surrounding the brain disappears. The cisterns surrounding the brainstem become compressed, and risk of herniation rises. Moreover, as the intracranial pressure rises, the cerebral perfusion pressure falls, and global brain ischemia occurs. Just as with focal ischemia (stroke), ion pumps fail, and loss of gray-white differentiation occurs. **Figure 26** (see below) shows changes of diffuse cerebral edema.

Quick Tips: Cerebral Edema

- Loss of CSF-containing spaces
    - Ventricular effacement
    - Effacement of sulci
- Loss of gray-white differentiation
- Significance:
    - Increased ICP
    - Global brain ischemia from decreased cerebral perfusion pressure

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Diffuse Axonal Injury (DAI)

Diffuse axonal injury (DAI) is the widespread shearing of long axons that occurs as the result of deceleration injury. Common clinical scenarios include high speed motor vehicle collisions and falls from great height. This injury is not typical of blows to the head or penetrating brain injury. The CT appearance is nonspecific: normal in the hyperacute phase, often followed by cerebral edema over hours to days. Punctate intraparenchymal hemorrhage may occur as well. Often other traumatic brain injury will be evident, such as subdural or epidural hematoma. The prognosis is poor, and resolution of CT findings may not equate with clinical improvement. MRI is more diagnostic [45].

Normal findings which may simulate disease

Several common incidental findings may simulate disease. These include calcifications in the choroid plexus of the posterior horns of the lateral ventricles (recall that the choroid plexus secretes cerebrospinal fluid) (**figure 16**), and calcifications in the pineal gland [46]. These should not be confused with hemorrhage as they have a greater density (brighter white appearance) and a typical location. The significance of these findings is unknown, although choroid calcifications have been hypothesized to play a role in schizophrenia [47].

Frequently asked questions:

Why can two patients with the same CT findings have markedly different neurological exams?
Remember that CT offers a macroscopic snapshot in time of complex pathologic changes. It may be that a patient with severe neurologic impairment but a relative benign looking head CT will soon develop changes such as cerebral edema, due to neuronal injury that has already occurred or is ongoing. In other cases, a patient with a large subdural hematoma may appear surprisingly neurologically intact, while another patient with similar head CT findings is severely impaired. One explanation is the degree of diffuse axonal injury which may accompany abnormalities such as SDH. The patient with minimal deficits may have no DAI, while the patient with severe deficits may have severe DAI, which is not evident on CT. Another of many factors which may determine clinical status is the amount of cerebral atrophy which is present before the injury. Atrophy is loss of brain volume and is assoicated with a compensatory increase in the size of CSF-containing spaces, such as ventricles, cisterns, and sulci. When an injury occurs and cerebral edema or a space-occupying lesion such as a SDH occurs, the presence of atrophy **(figure 24,** below**)** may be protective by allowing room for expansion of the pathologic lesion without leading to herniation or precipitous rises in intracranial pressure.

C is for CSF-spaces

The final letter in our mnemonic, C, reminds us to inspect CSF spaces. *This is critical, even in cases where other pathology, such as intracranial hemorrhage, has already been found.* The CSF-spaces offer clues to intracranial pressure and may reveal a neurosurgical emergency. In addition, as discussed above, SAH may accumulate in CSF spaces including sulci, cisterns, and ventricles. Since the volume of the calvarium is fixed, as the size of one component of skull contents (brain, CSF, and blood) increase, the volume of other components must diminish.

Sulci

Sulci, the CSF spaces between the undulating gyri of the brain surface, should appear black on brain windows. Normal sulci are visible but not prominent, and a thin ribbon of CSF should outline the entire brain. In cases of cerebral edema, the sulci may be completely effaced as the brain swells. In hydrocephalus, the volume of brain remains fixed but ventricles increase in size, leading to compression of the sulci. In cases of cerebral atrophy, loss of brain tissue volume leads to a compensatory increase in the size of sulci **(figures 23-26)**.

Ventricles/Hydrocephalus

Hydrocephalus is an important finding for Emergency Physicians, because of its potential as a neurosurgical emergency. Untreated, hydrocephalus can result in tonsillar herniation, brainstem compression, and respiratory arrest [48]. In general, as hydrocephalus becomes severe, the lateral ventricles become significantly enlarged. Because the volume of the calvarium is fixed, and solid brain tissue is essentially incompressible, as the ventricles

expand, other CSF spaces become compressed – consequently the sulci become effaced. In contrast, in the patient with atrophy, the ventricles may appear dilated but sulci appear similarly enlarged. Comparison with a prior head CT is always valuable in assessing for hydrocephalus, because ventricular size alone is a relatively poor predictor of ICP [49].

A variety of CT criteria for acute and chronic hydrocephalus have been described (see quick tips). **Figure 25** shows CT findings of hydrocephalus.
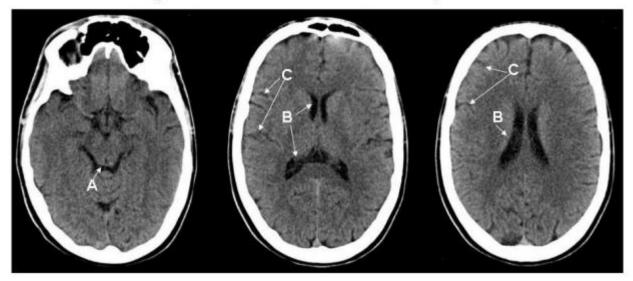
Quick Tips: Acute Hydrocephalus

- Dilated lateral ventricles
- Effaced sulci
- Both temporal horns >2mm
- Sylvian and interhemispheric fissures effaced
- Ratio largest width of frontal horns: internal diameter from inner table to inner table >0.5
- Ratio of largest width of frontal horns to maximum biparietal diameter >30%
- Periventricular low density due to transependymal absorption
- Ballooning of frontal horns of lateral ventricles and third ventricle, aka "Mickey Mouse" ventricles – aqueductal obstruction
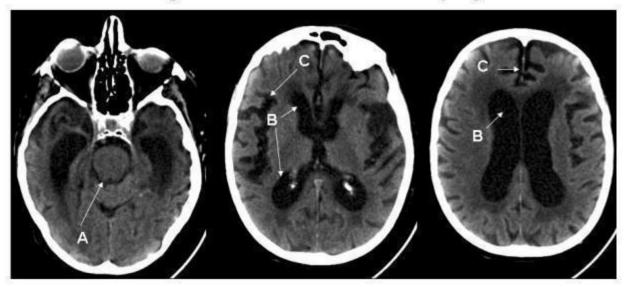
Atrophy

In cerebral atrophy, loss of brain volume results in relatively symmetrical increase in size of sulci and ventricles. The basilar cisterns should also remain patent **(figure 24)**.
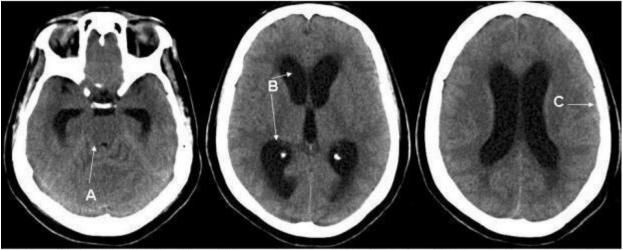
Figure 23: normal CSF spaces

Normal CSF spaces. The basilar cisterns (A, arrow) are open (sometimes informally called the "smile sign"). The lateral ventricles are open and not enlarged (B). Sulci (C) are visible but not prominent.

Click On Image to Enlarge

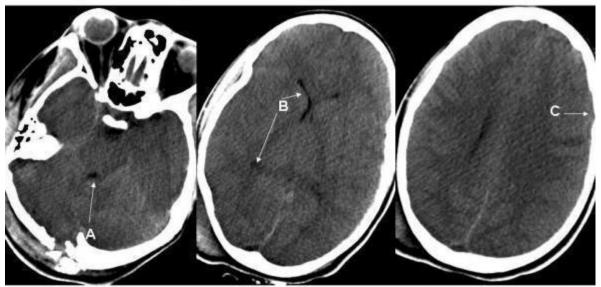# Figure 24: cerebral atrophy

Cerebral atrophy. The basilar cisterns (A, arrow) are open (sometimes informally called the "smile sign"). The lateral ventricles are enlarged (B), but sulci (C) are equally prominent.

Click On Image to Enlarge

Figure 25: hydrocephalus

Hydrocephalus. The basilar cisterns (A, arrow) is effaced. The lateral ventricles are enlarged (B), and sulci (C) are effaced.

 Click On Image to Enlarge

Figure 26: cerebral edema

Cerebral edema. The basilar cisterns (A, arrow) is effaced. The lateral ventricles are slit-like (B), and sulci (C) are effaced.

Click On Image to Enlarge

CT findings of elevated intracranial pressure

Following trauma, a variety of CT findings may indicate elevated intracranial pressure, though none are completely predictive. Findings that suggest increased ICP are decreased ventricle size, decreased basilar cistern size, effacement of sulci, degree of transfalcine herniation (midline shift), and loss of gray-white matter differentiation [50].

Assessment of CSF spaces and ICP

Condition	Sulci	Ventricles	Cisterns
Atrophy	enlarged	enlarged	enlarged
Hydrocephalus	effaced	enlarged	effaced
Cerebral edema	effaced	effaced	effaced

What CT findings are contraindications to lumbar puncture?

Before performing lumbar puncture, confirm that findings of midline shift or elevated ICP are not present. The midline should be midline, the sulci should be evident, and the cisterns should be open. Ventricle size should not be excessive, particularly when compared to sulci.

Quick Tips: increased ICP after trauma

- Ventricle size
  - Slit-like or none
- Basilar cistern size
  - Mildly effaced or effaced
- Sulci size
  - Effaced or none visible
- Transfalcine herniation (midline shift)
- Loss of gray-white matter differentiation

Summary

When interpreting a head CT, the Emergency Physician can use clinical information to frame important questions. Is trauma known or suspected? If so, are fractures present? Is the underlying brain injured? Is bleeding present? Is the intracranial pressure elevated? Is mass effect present? Are there signs of impending herniation? Is lumbar puncture contraindicated? Are there signs of cerebral infarction? Is focal cerebral edema present, such as vasogenic edema? Is diffuse cerebral edema present?

The mnemonic presented above will help to prevent you from forgetting to assess for clinically important findings. In a given scenario, you may choose to skip "A" or "B$_1$," selecting neurosurgical emergencies such as hemorrhage, ischemic stroke findings, or evidence of elevated ICP as your first priorities. Even if you choose not to interpret CT yourself, a better understanding of CT abnormalities and limitations may assist your clinical care of patients.

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