Primary Effects of CNS Trauma

Primary head injuries are defined as those that occur at the time of initial trauma even though they may not be immediately apparent on initial evaluation.

Head injury can be caused by direct or indirect trauma. Direct trauma involves a blow to the head and is usually caused by automobile collisions, falls, or injury inflicted by an object such as a hammer or baseball bat. Scalp lacerations, hematomas, and skull fractures are common. Associated intracranial damage ranges from none to severe.

Significant forces of acceleration/deceleration, linear translation, and rotational loading can be applied to the brain without direct head blows. Such indirect trauma is caused by angular kinematics and typically occurs in high-speed motor vehicle collisions (MVCs). Here the brain undergoes rapid deformation and distortion. Depending on the site and direction of the force applied, significant injury to the cortex, axons, penetrating blood vessels, and deep gray nuclei may occur. Severe brain injury can occur in the absence of skull fractures or visible scalp lesions.

We begin our discussion with a consideration of scalp and skull lesions as we work our way from the outside to the inside of the skull. We then delineate the spectrum of intracranial trauma, starting with extraaxial hemorrhages. We conclude this chapter with a detailed discussion of injuries to the brain parenchyma (e.g., cortical contusion, diffuse axonal injury, and the serious deep subcortical injuries).

Scalp and Skull Injuries

Scalp and skull injuries are common manifestations of cranial trauma. Although brain injury is usually the most immediate concern in managing traumatized patients, superficial lesions such as scalp swelling and focal hematoma can be helpful in identifying the location of direct head trauma. On occasion, these initially innocent-appearing “lumps and bumps” can become life-threatening. Before turning our attention to intracranial traumatic lesions, we therefore briefly review scalp and skull injuries, delineating their typical imaging findings and clinical significance.

Scalp Injuries

Scalp injuries include lacerations and hematomas. Scalp lacerations can occur in both penetrating and closed head injuries. Lacerations may extend partially or entirely through all five layers of the scalp (skin, subcutaneous fibrofatty tissue, galea aponeurotica, loose areolar connective tissue, and periosteum) to the skull (2-1).

Focal discontinuity, soft tissue swelling, and subcutaneous air are commonly identified in scalp lacerations. Scalp lacerations should be carefully evaluated
(2-1) Coronal graphic depicts normal layers of the scalp. Skin, subcutaneous fibrofatty tissue overlie the galea aponeurotica, loose areolar connective tissue. The pericranium is the periosteum of the skull and continues into and through sutures to merge with the periosteal layer of the dura. (2-2) NECT shows scalp laceration, hyperdense foreign bodies, and subgaleal air.

(2-3) Graphic shows the skull of a newborn, including the anterior fontanelle, coronal, metopic, sagittal sutures. Cephalohematoma is subperiosteal, limited by sutures. Subgaleal hematoma is under the scalp aponeurosis, not bounded by sutures. (2-4A) NECT scan in a newborn shows a small right and a large left parietal cephalohematoma. Neither crosses the sagittal suture.

(2-4B) Coronal scan in the same case shows the small right, large left-sided cephalohematomas. The elevated periosteum clearly separates the two blood collections. (2-4C) Sagittal scan reformatted from the axial data shows that the left parietal cephalohematoma does not cross the coronal suture.
Primary Effects of CNS Trauma

for the presence of any foreign bodies. If not removed during wound debridement, foreign bodies can be a potential source of substantial morbidity and are very important to identify on initial imaging studies. Wood fragments are often hypodense, whereas leaded glass, gravel, and metallic shards are variably hyperdense (2-2).

Scalp lacerations may or may not be associated with scalp hematomas. There are two distinctly different types of scalp hematomas: cephalohematomas and subgaleal hematomas. The former are usually of no clinical significance, whereas the latter can cause hypovolemia and hypotension.

Cephalohematomas are subperiosteal blood collections that lie in the potential space between the outer surface of the calvarium and the pericranium, which serves as the periosteum of the skull (2-3). The pericranium continues medially into cranial sutures and is anatomically contiguous with the outer (periosteal) layer of the dura.

Cephalohematomas are the extracranial equivalent of an intracranial epidural hematoma. Cephalohematomas do not cross suture lines and are typically unilateral. Because they are anatomically constrained by the tough fibrous periosteum and its insertions, cephalohematomas rarely attain large size.

Cephalohematomas occur in 1% of newborns and are more common following instrumented delivery. They are often diagnosed clinically but imaged only if they are unusually prominent or if intracranial injuries are suspected. NECT scans show a somewhat lens-shaped soft tissue mass that overlies a single bone (usually the parietal or occipital bone) (2-4). If more than one bone is affected, the two collections are separated by the intervening suture lines.

Complications from cephalohematoma are rare, and most resolve spontaneously over a few days or weeks. Occasionally the elevated periosteum at the periphery of a chronic cephalohematoma undergoes dystrophic calcification, creating a firm palpable mass.
Subgaleal hematomas are subaponeurotic collections and are common findings in traumatized patients of all ages. Here blood collects under the aponeurosis (the “galea”) of the occipitofrontalis muscle (2-5). Because a subgaleal hematoma lies deep to the scalp muscles and galea aponeurotica but external to the periosteum, it is not anatomically limited by suture lines.

Bleeding into the subgaleal space can be very extensive. Subgaleal hematomas are usually bilateral lesions that often spread diffusely around the entire calvaria. NECT scans show a heterogeneously hyperdense crescentic scalp mass that crosses one or more suture lines (2-6).

Most subgaleal hematomas resolve without treatment. In contrast to benign self-limited cephalohematomas, however, expanding subgaleal hematomas in infants and small children can cause significant blood loss.

Facial Injuries

Facial fractures are commonly overlooked on initial imaging (typically head CT scans). Important soft tissue markers can be identified that correlate with facial fractures and may merit a dedicated CT evaluation of the facial bones. These include peri orbital contusions and subconjunctival hemorrhage as well as lacerations of the lips, mouth, and nose.

Holmgren et al. (2005) have proposed the mnemonic LIPS-N (lip laceration, intraoral laceration, periorbital contusion, subconjunctival hemorrhage, and nasal laceration) be used in conjunction with physical examination. If any of these is present, a traumatized patient should have a dedicated facial CT in addition to the standard head CT.

Skull Fractures

Noticing a scalp “bump” or hematoma on initial imaging in head trauma is important, as calvarial fractures rarely—if
Primary Effects of CNS Trauma

Ever—occur in the absence of overlying soft tissue swelling or scalp laceration. Skull fractures are present on initial CT scans in about two-thirds of patients with moderate head injury, although 25-35% of severely injured patients have no identifiable fracture even with thin-section bone reconstructions.

Skull fractures can be simple or comminuted, closed or open. In open fractures, skin laceration results in communication between the external environment and intracranial cavity. Infection risk is high in this type of fracture, as it is with fractures that cross the mastoids and paranasal sinuses.

Several types of acute skull fracture can be identified on imaging studies: linear, depressed, elevated, and diastatic fractures (2-7). Fractures can involve the calvaria, skull base, or both. Another type of skull fracture, a "growing" skull fracture, is a rare but important complication of skull trauma.

Linear Skull Fractures

A linear skull fracture is a sharply margined linear defect that typically involves both the inner and outer tables of the calvaria (2-8).

Most linear skull fractures are caused by relatively low-energy blunt trauma that is delivered over a relatively wide surface area. Linear skull fractures that extend into and widen a suture become diastatic fractures (see below). When multiple complex fractures are present, 3D shaded surface display (SSD) can be very helpful in depicting their anatomy and relationships to cranial sutures.

Patients with an isolated linear nondisplaced skull fracture (NDSF), no intracranial hemorrhage or pneumocephalus, normal neurologic examination, and absence of other injuries are at very low risk for delayed hemorrhage or other life-threatening complication. Hospitalization is not necessary for many children with NDSFs.
Depressed Skull Fractures

A depressed skull fracture is a fracture in which the fragments are displaced inward [2-9]. Comminution of the fracture fragments starts at the point of maximum impact and spreads centrifugally. Depressed fractures are most often caused by high-energy direct blows to a small surface with a blunt object (e.g., hammer, baseball bat, or metal pipe) [2-10].

Depressed skull fractures typically tear the underlying dura and arachnoid and are associated with cortical contusions and potential leakage of CSF into the subdural space. Fractures extending to a dural sinus or the jugular bulb are associated with venous sinus thrombosis in 40% of cases.

Elevated Skull Fractures

An elevated skull fracture—often combined with depressed fragments—is uncommon. Elevated fractures are usually caused by a long, sharp object (such as a machete or propeller) that fractures the calvaria, simultaneously lifting and rotating the fracture fragment [2-11].

Diastatic Skull Fractures

A diastatic skull fracture is a fracture that widens (“diastases” or “splits open”) a suture or synchondrosis. Diastatic skull fractures usually occur in association with a linear skull fracture that extends into an adjacent suture [2-12].

Traumatic diastasis of the sphenoooccipital, petrooccipital, and/or occipitomastoid synchondroses is common in children with severely comminuted central skull base fractures. As it typically does not ossify completely until the mid teens, the sphenoooccipital synchondrosis is the most common site.

“Growing” Skull Fractures

A “growing” skull fracture (GSF), also known as “posttraumatic leptomeningeal cyst” or “craniocerebral...
erosion,” is a rare lesion that occurs in just 0.3-0.5% of all skull fractures (2-13). Most patients with GSF are under 3 years of age.

GSFs develop in stages and slowly widen over time. In the first “prephase,” a skull fracture (typically a linear or comminuted fracture) lacerates the dura, and brain tissue or arachnoid membrane herniates through the torn dura. Stage I extends from the time of initial injury to just before the fracture enlarges. Early recognition and dural repair of stage I GSFs produce the best results.

Stage II is the early phase of GSF. Stage II lasts for approximately 2 months following initial fracture enlargement. At this stage, the bone defect is small, the skull deformity is relatively limited, and neurologic deficits are mild. Nevertheless, the entrapped tissue prevents normal fracture healing.

Stage III represents late-stage GSF and begins 2 months after the initial enlargement begins. During this stage, the bone defect becomes significantly larger. Brain tissue and CSF extend between the bony edges of the fracture through torn dura and arachnoid.

Patients with late-stage GSFs often present months or even years after head trauma. Stage III GSFs can cause pronounced skull deformities and progressive neurologic deficits if left untreated.

**Imaging**

**General Features.** Plain skull radiographs have no role in the modern evaluation of traumatic head injury. One-quarter of patients with fatal brain injuries have no skull fracture at autopsy. CT is fast, widely available, sensitive for both bone and brain injury, and the worldwide diagnostic standard of care for patients with head injuries. New generations of multislice CT scanners offer very short acquisition times with excellent spatial resolution.

Both bone and soft tissue reconstruction algorithms should be used when evaluating patients with head injuries. Soft tissue reconstructions should be viewed with both narrow (“brain”) and intermediate (“subdural”) windows. Coronal and sagittal reformatted images obtained using the axial source data are helpful additions.

Three-dimensional reconstruction and curved MIPs of the skull have been shown to improve fracture detection over the use of axial sections alone.

**CT Findings.** While fractures can involve any part of the calvaria or skull base, the middle cranial fossa is most susceptible because of its thin “squamous” bones and multiple foramina and fissures.

NECT scans demonstrate linear skull fractures as sharply margined lucent lines. Depressed fractures are typically comminuted and show inward implosion of fracture fragments (2-10). Elevated fractures show an elevated, rotated skull segment (2-11). Diastatic fractures appear as widened sutures or synchondroses (2-14) (2-15) and are usually associated with linear skull fractures.

Stage I “growing” fractures are difficult to detect on initial NECT scans, as scalp and contused brain are similar in density. Identifying torn dura with herniated brain tissue is similarly difficult although cranial ultrasound can be more helpful.

Later-stage GSFs demonstrate a progressively widening and unhealing fracture. A lucent skull lesion with rounded, scalloped margins and beveled edges is typical (2-13). CSF and soft tissue are entrapped within the expanding fracture. Most GSFs are directly adjacent to posttraumatic encephalomalacia, so the underlying brain often appears hypodense.
**MR Findings.** MR is rarely used in the setting of acute head trauma because of high cost, limited availability, and lengthy time required. Compared with CT, bone detail is poor although parenchymal injuries are better seen. Adding T2* sequences, particularly SWI, is especially helpful in identifying hemorrhagic lesions.

In some cases, MR may be indicated for early detection of potentially treatable complications. A young child with neurologic deficits or seizures, a fracture larger than 4 millimeters, or a soft tissue mass extending through the fracture into the subgaleal space is at risk for developing a GSF. MR can demonstrate the dural tear and differentiate herniated brain from contused, edematous scalp.

**Angiography.** If a fracture crosses the site of a major vascular structure such as the carotid canal or a dural venous sinus (2-14), CT angiography is recommended. Sagittal, coronal, and MIP reconstructions help delineate the site and extent of vascular injuries.

Clival and skull base fractures are strongly associated with neurovascular trauma, and CTA should always be obtained in these cases (2-15). Cervical fracture dislocations, distraction injuries, and penetrating neck trauma also merit further investigation. Uncomplicated asymptomatic soft tissue injuries of the neck rarely result in significant vascular injury.

### SCALP AND SKULL INJURIES

**Scalp Injuries**
- Lacerations
  - ± Foreign bodies
- Cephalohematoma
  - Usually infants
  - Subperiosteal
  - Small, unilateral (limited by sutures)
- Subgaleal hematoma
  - Between galea, periosteum of skull
  - Circumferential, not limited by sutures
  - Can be very large, life-threatening

**Skull Fractures**
- Linear
  - Sharp lucent line
  - Can be extensive and widespread
- Depressed
  - Focal
  - Inwardly displaced fragments
  - Often lacerates dura-arachnoid
- Elevated
  - Rare
  - Fragmented rotated outward
- Diastatic
  - Typically associated with severe trauma
  - Usually caused by linear fracture that extends into suture
  - Widens, spreads apart suture or synchondrosis
- "Growing"
  - Rare
  - Usually in young children
  - Fracture lacerates dura-arachnoid
  - Brain/arachnoid herniates through torn dura
  - Trapped tissue prevents bone healing
  - CT: Rounded edges, scalloped margins of skull
  - MR: CSF ± brain

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(2-16) Graphic shows EDH, depressed skull fracture lacerating middle meningeal artery. Inset shows rapid bleeding, "swirl" sign.

(2-17A) Endocranial view shows temporal bone fracture crossing the middle meningeal artery groove. Note biconvex margins of EDH.

(2-17B) Dorsal view of the dura-covered brain shows the biconvex EDH on top of the dura. (Courtesy E. T. Hedley-Whyte, MD.)
Extraaxial Hemorrhages

Extraaxial hemorrhages and hematomas are common manifestations of head trauma. They can occur in any intracranial compartment, within any space (potential or actual), and between any layers of the cranial meninges. Only the subarachnoid spaces exist normally; all the other spaces are potential spaces and occur only under pathologic conditions.

**Epidural hematomas** arise between the inner table of the skull and outer (periosteal) layer of the dura. **Subdural hematomas** are located between the inner (meningeal) layer of the dura and the arachnoid. **Traumatic subarachnoid hemorrhage** is found within the sulci and subarachnoid cisterns, between the arachnoid and the pia.

To discuss extraaxial hemorrhages, we work our way from the outside to inside. We therefore begin this section with a discussion of epidural hematomas (both classic and variant), then move deeper inside the cranium to the more common subdural hematomas. We conclude with a consideration of traumatic subarachnoid hemorrhage.

**Arterial Epidural Hematoma**

Epidural hematomas (EDHs) are uncommon but potentially lethal complications of head trauma. If an EDH is promptly recognized and appropriately treated, mortality and morbidity can be minimized.

**Terminology**

An EDH is a collection of blood between the calvaria and outer (periosteal) layer of the dura.

**Etiology**

Most EDHs arise from direct trauma to the skull that lacerates an adjacent blood vessel (2-16). The vast majority (90%) are caused by arterial injury, most commonly to the middle meningeal artery. Approximately 10% of EDHs are venous, usually secondary to a fracture that crosses a dural venous sinus (see below).

**Pathology**

**Location.** Over 90% of EDHs are unilateral and supratentorial. Between 90-95% are found directly adjacent to a skull fracture. The squamous portion of the temporal bone is the most common site.

**Gross Pathology.** EDHs are biconvex in shape (2-17A). Adherence of the periosteal dura to the inner calvaria explains this typical configuration. As EDHs expand, they strip the dura away from the inner table of the skull, forming the classic lens-shaped hematoma (2-17B). Because the dura is especially tightly attached to sutures, EDHs in adults rarely cross suture lines (10% of EDHs in children do cross sutures, especially if a fracture traverses the suture or sutureal diastasis is present).

The typical gross or intraoperative appearance of an acute EDH is a dark purple (“currant jelly”) lentiform clot.

**Clinical Issues**

**Epidemiology.** EDHs are much less common than either traumatic subarachnoid hemorrhage (tSAH) or subdural hematoma. Although EDHs represent up to 10% of fatal injuries in autopsy series, they are found in only 1-4% of patients imaged for craniocerebral trauma.
(2-20A) Serial imaging demonstrates temporal evolution of a small nonoperated EDH. Initial NECT scan shows a hyperdense biconvex EDH.

(2-20B) Repeat scan 10 days later reveals that density of the EDH has decreased significantly.

(2-20C) Repeat study 6 weeks after trauma reveals that the EDH has resolved completely.

Demographics. EDHs are uncommon in infants and the elderly. Most are found in older children and young adults. The M:F ratio is 4:1.

Presentation. The prototypical “lucid interval,” during which a traumatized patient has an initial brief loss of consciousness followed by an asymptomatic period of various length prior to onset of coma and/or neurologic deficit, occurs in only 50% of EDH cases. Headache, nausea, vomiting, symptoms of intracranial mass effect (e.g., pupil-involving third cranial nerve palsy) followed by somnolence and coma are common.

Natural History. Outcome depends on size and location of the hematoma, whether the EDH is arterial or venous, and whether there is active bleeding (see below). In the absence of other associated traumatic brain injuries, overall mortality rate with prompt recognition and appropriate treatment is under 5%.

Delayed development or enlargement of an EDH occurs in 10-15% of cases, usually within 24-36 hours following trauma.

Treatment Options. Many EDHs are now treated conservatively. Most traumatic EDHs are not surgical lesions at initial presentation, and the rate of conversion to surgery is low. Most venous and small classic hyperdense EDHs that do not exhibit a “swirl” sign and have minimal or no mass effect are managed conservatively with close clinical observation and follow-up imaging. Significant clinical predictors of EDH progression requiring conversion to surgical therapy are coagulopathy and younger age.

Imaging

General Features. EDHs, especially in adults, typically do not cross sutures unless a fracture with sutural diastasis is present. In children, 10% of EDHs cross suture lines, usually the coronal or sphenosquamous suture.

Look for other comorbid lesions such as “contre-coup” injuries, tSAH, and secondary brain herniations, all of which are common findings in patients with EDHs.

CT Findings. NECT scan is the procedure of choice for initial imaging in patients with head injury. Both soft tissue and bone reconstruction algorithms should be obtained. Multiplanar reconstructions are especially useful in identifying vertex EDHs, which may be difficult to detect if only axial images are obtained.

The classic imaging appearance of classic (arterial) EDHs is a hyperdense (60-90 HU) biconvex extraaxial collection. Presence of a hypodense component (“swirl” sign) is seen in about one-third of cases and indicates active, rapid bleeding with unretracted clot.

EDHs compress the underlying subarachnoid space and displace the cortex medially, “buckling” the gray-white matter interface inward.

Air in an EDH occurs in approximately 20% of cases and is usually—but not invariably—associated with a sinus or mastoid fracture.

Patients with mixed-density EDHs tend to present earlier than patients with hyperdense hematomas and have lower Glasgow Coma Scores (GCSs), larger hematoma volumes, and poorer prognosis.

Imaging findings associated with adverse clinical outcome are thickness > 1.5 cm, volume > 30 mL, pterional (lateral aspect of the middle cranial fossa) location, midline shift > 5 mm, and presence of a “swirl sign” within the hematoma on imaging.
MR Findings. Acute EDHs are typically isointense with underlying brain, especially on T1WI. The displaced dura can be identified as a displaced "black line" between the hematoma and the brain.

Angiography. DSA may show a lacerated middle meningeal artery with "tram-track" fistulization of contrast from the middle meningeal artery into the paired middle meningeal veins. Mass effect with displaced cortical arteries and veins is seen.

CLASSIC ACUTE EPIDURAL HEMATOMA

Terminology
- EDH = blood between skull, dura

Etiology
- Associated skull fracture in 90-95%
- Arterial 90%
  - Most often middle meningeal artery
- Venous 10%

Pathology
- Unilateral, supratentorial (> 90%)
- Dura stripped away from skull → biconvex hematoma
- Usually does not cross sutures (exception = children, 10%)
- Does cross sites of dural attachment

Clinical
- Rare (1-4% of head trauma)
- Older children, young adults most common
- M:F = 4:1
- Classic "lucid interval" in only 50%
- Delayed deterioration common
- Low mortality if recognized, treated
- Small EDHs
  - If minimal mass, no "swirl sign" often managed conservatively

Imaging
- Hyperdense lens-shaped
- "Swirl sign" (hypodensity) = rapid bleeding

Venous Epidural Hematoma

Not all EDHs are the same!! Venous EDHs are often smaller, are under lower pressure, and develop more slowly than their arterial counterparts. Most venous EDHs are caused by a skull fracture that crosses a dural venous sinus and therefore occur in the posterior fossa near the skull base (transverse/sigmoid sinus) (2-21) or the vertex of the brain (superior sagittal sinus). In contrast to their arterial counterparts, venous EDHs can "straddle" intracranial compartments, crossing both sutures and lines of dural attachment (2-22) and compressing or occluding the adjacent venous sinuses.

Venous EDHs can be subtle and easily overlooked. Coronal and sagittal reformatted images are key to the diagnosis and delineation of these variant EDHs (2-23). Several anatomic subtypes of venous EDHs, each with different treatment implications and prognosis, are recognized.

Vertex EDH

"Vertex" EDHs are rare. Usually caused by a linear or diastatic fracture that crosses the superior sagittal sinus, they often accumulate over hours or even days with slow, subtle onset of symptoms (2-24). "Vertex" hematomas can be subtle and are easily overlooked unless coronal and sagittal reformatted images are obtained.
**Anterior Temporal EDH**

Anterior temporal EDHs are a unique subgroup of hematomas that occur in the anterior tip of the middle cranial fossa. Anterior temporal EDHs are caused either by an isolated fracture of the adjacent greater sphenoid wing or by an isolated zygomaticomaxillary complex (“tripod”) facial fracture. The sphenoparietal dural venous sinus is injured as it curves medially along the undersurface of the lesser sphenoid wing, extravasating blood into the epidural space. Limited anatomically by the sphenotemporal suture laterally and the orbital fissure medially, anterior temporal EDHs remain stable in size and do not require surgical evacuation (2-25) (2-26).

**Clival EDH**

Clival EDHs usually develop after a hyperflexion or hyperextension injury to the neck and are possibly caused by stripping of the tectorial membrane from attachments to the clivus. Less commonly, they have been associated with basilar skull fractures that lacerate the clival dural venous plexus.

Clival EDHs most often occur in children and present with multiple cranial neuropathies. The abducens nerve is the most commonly affected, followed by the glossopharyngeal and hypoglossal nerves. They are typically limited in size by the tight attachment of the dura to the basisphenoid and tectorial membrane (2-27).

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**VENOUS EPIDURAL HEMATOMA**

Not all EDHs are the same!
- Different etiologies in different anatomic locations
- Prognosis, treatment vary

Venous EDHs = 10% of all EDHs
- Skull fracture crosses dural venous sinus
  - Can cross sutures, dural attachments
- Often subtle, easily overlooked
  - Coronal, sagittal reformatted images key to diagnosis
- Usually accumulate slowly
- Can be limited in size; often treated conservatively

Subtypes
- **Vertex EDH**
  - Skull fracture crosses superior sagittal sinus (SSS)
  - SSS can be lacerated, compressed, thrombosed
  - Hematoma under low pressure, develops gradually
  - Slow onset of symptoms
  - May become large, cause significant mass effect
- **Anterior temporal EDH**
  - Sphenoid wing or zygomaticomaxillary fracture
  - Injures sphenoparietal venous sinus
  - Hematoma accumulates at anterior tip of middle cranial fossa
  - Limited anatomically (laterally by sphenotemporal suture, medially by orbital fissure)
  - Benign clinical course
- **Clival EDH**
  - Most common = child with neck injury
  - May cause multiple cranial neuropathies (CN VI most common)
  - Hyperdense collection under clival dura
  - Limited by tight attachment of dura to basisphenoid, tectorial membrane
  - Usually benign course, resolves spontaneously

Management of a clival EDH is dictated by severity and progression of the neurologic deficits and stability of the atlantoaxial joint. In patients with
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(2-25) Graphic depicts benign anterior temporal epidural hematoma. Fracture disrupts the sphenoparietal sinus. Low-pressure venous EDH is anatomically limited, medially by the orbital fissure and laterally by the sphenotemporal suture. (2-26A) Axial NECT in a 33y man with head trauma shows a biconvex anterior temporal acute epidural hematoma.

(2-26B) Axial bone CT in the same case shows a fracture through the right greater sphenoid wing. (2-26C) CT venogram in the same case shows a displaced, lacerated sphenoparietal sinus with contrast extravasation ("spot sign"). Note the EDH is limited medially by the orbital fissure. The patient was treated nonsurgically. The EDH showed no further enlargement and resolved completely.

(2-27A) Axial CTA in a child with craniovertebral junction trauma shows a small clival EDH. There was no evidence for vascular injury. (2-27B) Sagittal CTA reformatted from the axial source date nicely demonstrates the clival epidural hematoma.
Acute Subdural Hematoma

Acute subdural hematomas (aSDHs) are one of the leading causes of death and disability in patients with severe traumatic brain injury. SDHs are much more common than EDHs. Most do not occur as isolated injuries; the vast majority of SDHs are associated with traumatic subarachnoid hemorrhage (tSAH) as well as significant parenchymal injuries such as cortical contusions, brain lacerations, and diffuse axonal injuries.

Terminology

An aSDH is a collection of acute blood products that lies in or between the inner border cell layer of the dura and the arachnoid.

Etiology

Trauma is the most common cause of an aSDH. Both direct blows to the head and nonimpact injuries may result in formation of an aSDH. Tearing of bridging cortical veins as they cross the subdural space to enter a dural venous sinus (usually the superior sagittal sinus) is the most common etiology. Cortical vein lacerations can occur with either a skull fracture or the sudden changes in velocity and brain rotation that occur during nonimpact closed head injury.

Blood from ruptured vessels spreads quickly through the potential space between the dura and the arachnoid. Large SDHs may spread over an entire hemisphere, extending into the interhemispheric fissure and along the tentorium.

Tearing of cortical arteries from a skull fracture may also give rise to an aSDH. The arachnoid itself may also tear, creating a pathway for leakage of CSF into the subdural space, resulting in admixture of both blood and CSF.

Less common causes of an aSDH include aneurysm rupture, skull/dura-arachnoid metastases from vascular extracranial primary neoplasms, and spontaneous hemorrhage in patients with severe coagulopathy.

Rarely, an acute spontaneous SDH of arterial origin occurs in someone without any traumatic history or vascular anomaly. These patients usually have sudden serious disturbance of consciousness and have a poor outcome unless the aSDH is recognized and treated promptly.

Pathology

Gross Pathology. The gross appearance of an aSDH is that of a soft, purplish, “currant jelly” clot beneath a tense bulging dura. More than 95% are supratentorial. Most aSDHs spread diffusely over the affected hemisphere and are therefore typically crescent-shaped.

Clinical Issues

Epidemiology. An aSDH is the second most common extraaxial hematoma, exceeded only by tSAH. An aSDH is found in 10-20% of all patients with head injury and is observed in 30% of autopsied fatal injuries.
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Demographics. An aSDH may occur at any age from infancy to the elderly. There is no sex predilection.

Presentation. Even relatively minor head trauma, especially in elderly patients who are often anticoagulated, may result in an aSDH. In such patients, a definite history of trauma may be lacking. Clinical findings vary from none to loss of consciousness and coma. Most patients with aSDHs have low GCSs on admission. Delayed deterioration, especially in elderly anticoagulated patients, is common.

Natural History. An aSDH may remain stable, grow slowly, or rapidly increase in size, causing mass effect and secondary brain herniations. Prognosis varies with hematoma thickness, midline shift, and the presence of associated parenchymal injuries. An aSDH that is thicker than 2 centimeters correlates with poor outcome (35-90% mortality). An aSDH that occupies more than 10% of the total available intracranial volume is usually lethal.

Treatment Options. The majority of patients with small SDHs are initially treated conservatively with close clinical observation and follow-up imaging. Approximately 6-7% of these demonstrate an increase in SDH size over time and eventually require surgical intervention.

Patients with larger SDHs, a lesion located at the convexity, alcohol abuse, and repetitive falls are at the greatest risk for deterioration. Surveillance with follow-up CT scans is recommended until the SDH resolves or at least up to 5 weeks following the initial trauma.

Imaging

General Features. The classic finding of an aSDH is a supratentorial crescent-shaped extraaxial collection that displaces the gray-white matter interface medially. SDHs are typically more extensive than EDHs, easily spreading along the falx, tentorium, and around the anterior and middle fossa floors (2-29). SDHs may cross suture lines but generally do not cross dural attachments. Bilateral SDHs occur in 15% of cases. "Contre-coup" injuries such as contusion of the contralateral hemisphere are common.

Both standard soft tissue and intermediate ("subdural") windows as well as bone algorithm reconstructions should be used in all trauma patients, as small, subtle aSDHs can be obscured by the density of the overlying calvaria (2-30). Coronal and sagittal reformatted images using the axial source date are especially helpful in visualizing small ("smear") peritentorial and parafalcine aSDHs (2-31) (2-32).

CT Findings

NECT. Approximately 60% of aSDHs are hyperdense on NECT scans (2-29). Mixed-attenuation lesions are found in 40% of cases. Pockets of hypodensity within a larger hyperdense aSDH usually indicate rapid bleeding (2-33) (2-34). "Dots" or "lines" of CSF trapped within compressed, displaced sulci are often seen underlying an aSDH.

Mass effect with an aSDH is common and expected. Subfalcine herniation should be proportionate to the size of the subdural collection. However, if the difference between the midline shift and thickness of the hematoma is 3 mm or more, then mortality is very high. This discrepancy occurs when underlying cerebral edema is triggered by the traumatic event. Early recognition and aggressive treatment for potentially catastrophic brain swelling are essential (2-35).

In other cases, especially in patients with repeated head injury, severe brain swelling with unilateral hemisphere vascular engorgement occurs very
quickly. Here the mass effect is greatly disproportionate to the size of the SDH, which may be relatively small.

Occasionally, an aSDH is nearly isodense with the underlying cortex. This unusual appearance is found in extremely anemic patients (Hgb under 8-10 g/dL) and sometimes occurs in patients with coagulopathy. In rare cases, CSF leakage through a torn arachnoid may mix with—and dilute—the acute blood that collects in the subdural space.

**CECT.** CECT scans are helpful in detecting small isodense aSDHs. The normally enhancing cortical veins are displaced inward by the extraaxial fluid collection.

**Perfusion CT.** CT or xenon perfusion scans may demonstrate decreased cerebral blood flow (CBF) and low perfusion pressure, which is one of the reasons for the high mortality rate of patients with aSDHs. The cortex underlying an evacuated aSDH may show hyperemic changes with elevated rCBF values. Persisting hyperemia has been associated with poor outcome.

**MR Findings.** MR scans are rarely obtained in acutely brain-injured patients. In such cases, aSDHs appear isointense on T1WI and hypointense on T2WI. Signal intensity on FLAIR scans is usually iso- to hyperintense compared with CSF but hypointense compared with the adjacent brain. aSDHs are hypointense on T2* scans.

DWI shows heterogeneous signal within the hematoma but may show patchy foci of restricted diffusion in the cortex underlying the aSDH.

**Angiography.** CTA may be useful in visualizing a cortical vessel that is actively bleeding into the subdural space.

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**Images:**

1. **(2-33)** L Initial NECT in an anticoagulated male patient shows a small mixed-density SDH. (R) Scan 6 hours later shows expanding, actively bleeding aSDH. (2-34) NECT scan shows a 55y man with an actively hemorrhaging aSDH. Some clotted blood is present, but much of the hematoma consists of isodense unclotted hemorrhage.

2. **(2-35)** NECT shows a mixed-density 12-mm aSDH with a disproportionately large subfalcine herniation of the lateral ventricles (17 mm), indicating that diffuse holohemispheric brain swelling is present. Subfalcine herniation ≥ 3 mm portends a poor prognosis. (2-36) NECT scan in a very anemic patient shows an isodense aSDH. The aSDH is almost exactly the same density as the underlying cortex. The gray-white interface is displaced inward.

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Differential Diagnosis

In the setting of acute trauma, the major differential diagnosis is EDH. Shape is a helpful feature, as most aSDHs are crescentic, whereas EDHs are biconvex. EDHs are almost always associated with skull fracture; SDHs frequently occur in the absence of skull fracture. EDHs may cross sites of dural attachment; SDHs do not cross the falx or tentorium.

Subacute Subdural Hematoma

With time, subdural hematomas (SDHs) undergo organization, lysis, and neomembrane formation. Within 2-3 days, the initial soft, loosely organized clot of an acute SDH becomes organized. Breakdown of blood products and the formation of organizing granulation tissue change the imaging appearance of subacute and chronic SDHs.

Terminology

A subacute subdural hematoma (sSDH) is between several days and several weeks old.

Pathology

A collection of partially liquified clot with resolving blood products is surrounded on both sides by a “membrane” of organizing granulation tissue (2-37). The outermost membrane adheres to the dura and is typically thicker than the inner membrane, which abuts the thin, delicate arachnoid (2-38).

In some cases, repetitive hemorrhages of different ages arising from the friable granulation tissue may be present. In others, liquefaction of the hematoma over time produces serous blood-tinged fluid.

Clinical Issues

Epidemiology and Demographics. SDHs are common findings at imaging and autopsy. In contrast to acute SDHs, sSDHs show a distinct bimodal distribution with children and the elderly as the most commonly affected age groups.

Presentation. Clinical symptoms vary from asymptomatic to loss of consciousness and hemiparesis caused by sudden rehemorrhage into an sSDH. Headache and seizure are other common presentations.

Natural History and Treatment Options. Many sSDHs resolve spontaneously. In some cases, repeated hemorrhages may cause sudden enlargement and mass effect. Surgical drainage may be indicated if the sSDH is enlarging or becomes symptomatic.

Imaging

General Features. Imaging findings are related to hematoma age and the presence of encasing membranes. Evolution of an untreated, uncomplicated SDH follows a very predictable pattern on CT. Density of an extraaxial hematoma decreases approximately 1.5 HU/day (2-39). Therefore, an SDH will become nearly isodense with the underlying cerebral cortex within a few days following trauma.

CT Findings. sSDHs are typically crescent-shaped fluid collections that are iso- to slightly hypodense compared with the underlying cortex on NECT (2-40). Medial displacement of the gray-white interface (“buckling”) is often present, along with “dot-like” foci of CSF in the trapped, partially effaced sulci underlying the sSDH (2-41) (2-42). Mixed-density hemorrhages are common.
Trauma

(2-40) Axial NECT scan shows right sSDH that is isodense with the underlying cortex. The right GM-WM interface is displaced and buckled medially compared with normal left side.

(2-41) NECT scan in another patient shows bilateral “balanced” isodense subacute SDHs. Note that both GM-WM interfaces are inwardly displaced. A “dot” of CSF in the compressed subarachnoid space is seen under the left sSDH.

(2-42) NECT in elderly patient with sSDH, moderate cortical atrophy shows difference between nearly isodense SDH and CSF in underlying compressed subarachnoid space, sulci.

(2-43A) Axial T1WI in patient with a late-stage aSDH shows crescent-shaped hyperintense collection extending over entire surface of left hemisphere, gyral compression with almost obliterated sulci compared with normal right hemisphere.

(2-43B) T2* GRE scan shows some “blooming” in the sSDH. DWI shows the classic “double layer” appearance of an sSDH with hypointense rim on the inside and mildly hyperintense rim on the outside of the clot.
Bilateral sSDHs may be difficult to detect because of their "balanced" mass effect (2-41). Sulcal effacement with displaced gray-white matter interfaces is the typical appearance.

CECT scans show that the enhanced cortical veins are displaced medially. The encasing membranes, especially the thicker superficial layer, may enhance.

**MR Findings.** MR can be very helpful in identifying sSDHs, especially small lesions that are virtually isodense with underlying brain on CT scans.

Signal intensity varies with hematoma age but is less predictable than on CT, making precise "aging" of subdural collections more problematic. In general, early subacute SDHs are isointense with cortex on T1WI and hypointense on T2WI but gradually become more hyperintense as extracellular methemoglobin increases (2-43A). Most late-stage sSDHs are T1/T2 “bright-bright.” A linear T2 hypointensity representing the encasing membranes that surround the SDH is sometimes present.

FLAIR is the most sensitive standard sequence for detecting sSDH, as the collection is typically hyperintense (2-44). Because FLAIR signal intensity varies depending on the relative contribution of T1 and T2 effects, early sSDHs may initially appear hypointense due to their intrinsic T2 shortening.

T2* scans are also very sensitive, as sSDHs show distinct “blooming” (2-43B).

Signal intensity on DWI also varies with hematoma age. DWI commonly shows a crescentic high-intensity area with a low-intensity rim closer to the brain surface ("double layer" appearance) (2-43C). The low-intensity area corresponds to a mixture of resolved clot and CSF, whereas the high-intensity area correlates with solid clot.
T1 C+ scans demonstrate enhancing, thickened, encasing membranes (2-44D). The membrane surrounding an sSDH is usually thicker on the dural side of the collection. Delayed scans may show gradual “filling in” and increasing hyperintensity of the sSDH.

**Differential Diagnosis**

The major differential diagnosis of an sSDH is an isodense acute SDH. These are typically seen only in an extremely anemic or anticoagulated patient. A subdural effusion that follows surgery or meningitis or that occurs as a component of intracranial hypotension can also mimic an sSDH. A subdural hygroma is typically isodense/isointense with CSF and does not demonstrate enhancing, encapsulating membranes.

### Chronic/Mixed Subdural Hematoma

**Terminology**

A chronic subdural hematoma (cSDH) is an encapsulated collection of sanguineous or serosanguineous fluid confined within the subdural space. Recurrent hemorrhage(s) into a preexisting cSDH are common and produce a mixed-age or “acute on chronic” SDH (mSDH).

**Etiology**

With continued degradation of blood products, an SDH becomes progressively more liquified until it is largely serous fluid tinged with blood products (2-45). Rehemorrhage, either from vascularized encapsulating membranes or rupture of stretched cortical veins crossing the expanded subdural space, occurs in 5-10% of cSDHs and is considered “acute-on-chronic” SDH (2-46).

**Pathology**

**Gross Pathology.** Blood within the subdural space incites tissue reaction around its margins. Organization and resorption of the hematoma contained within the “membranes” of surrounding granulation tissue continue. These neomembranes have fragile, easily disrupted capillaries and easily rebleed, creating an mSDH. Multiple hemorrhages of different ages are common in mSDHs (2-47).

Eventually, most of the liquified clot in a cSDH is resorbed. Only a thickened dura-arachnoid layer remains with a few scattered pockets of old blood trapped between the inner and outer membranes.

**Clinical Issues**

**Epidemiology.** Unoperated, uncomplicated subacute SDHs eventually evolve into cSDHs. Approximately 5-10% will rehemorrhage, causing multiloculated mixed-age SDHs.

**Demographics.** Chronic SDHs may occur at any age. Mixed-age SDHs are much more common in elderly patients.

**Presentation.** Presentation varies from no/mild symptoms (e.g., headache) to sudden neurologic deterioration if a preexisting cSDH rehemorrhages.

**Natural History.** In the absence of repeated hemorrhages, cSDHs gradually resorb and largely resolve, leaving a residue of thickened dura-arachnoid that may persist for months or even years. Older patients, especially those with brain atrophy, are subject to repeated hemorrhages.

**Treatment Options.** If follow-up imaging of a subacute SDH shows expected resorption and regression of the cSDH, no surgery may be
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required. Surgical drainage with evacuation of the cSDH and resection of its encapsulating membranes is performed if significant mass effect or repeated hemorrhages cause neurologic complications.

Imaging

General Features. cSDHs have a spectrum of imaging appearances. Uncomplicated cSDHs show relatively homogeneous density/signal intensity with slight gravity-dependent gradation of their contents ("hematocrit effect").

mSDHs with acute hemorrhage into a preexisting cSDH show a hematocrit level with distinct layering of the old (top) and new (bottom) hemorrhages. Sometimes, septated pockets that contain hemorrhages of different ages form. Dependent layering of blood within the loculated collections may appear quite bizarre.

Extremely old, longstanding cSDHs with virtually complete resorption of all liquid contents are seen as pachymeningopathies with diffuse dura-arachnoid thickening.

CT Findings

NECT. A hypodense crescentic fluid collection extending over the surface of one or both cerebral hemispheres is the classic finding in cSDH. Uncomplicated cSDHs approach CSF in density (2-48). The hematocrit effect creates a slight gradation in density that increases from top to bottom.

Trabecular or loculated cSDHs show internal septations, often with evidence of repeated hemorrhages (2-49). With age, the encapsulating membranes surrounding the cSDH become thickened and may appear moderately hyperdense. Eventually, some cSDHs show peripheral calcifications that persist for many years. In rare cases, a cSDH may densely calcify or even ossify, a condition aptly termed "armored brain" (2-50).

CECT. The encapsulating membranes around a cSDH contain fragile neocapillaries that lack endothelial tight junctions. Therefore, the membranes show strong enhancement following contrast administration.

MR Findings. As with all intracranial hematomas, signal intensity of a cSDH or mSDH is quite variable and depends on age of the blood products. On T1 scans, uncomplicated cSDHs are typically iso- to slightly hyperintense compared with CSF (2-51A). Depending on the stage of evolution, cSDHs are iso- to hypointense compared with CSF on T2 scans.

Most cSDHs are hyperintense on FLAIR (2-51B) and may show "blooming" on T2* scans if subacute-chronic blood clots are still present. In about one-quarter of all cases, superficial siderosis can be identified over the gyri underlying a cSDH.

The encapsulating membranes of a cSDH enhance following contrast administration. Typically, the outer layer is thicker than the inner layer (2-51C) (2-51D) (2-52).

Uncomplicated cSDHs do not restrict on DWI. With cSDHs, a "double layer" effect—a crescent of hyperintensity medial to a nonrestricting fluid collection—indicates acute rehemorrhage.

Differential Diagnosis

An mSDH is difficult to mistake for anything else. In older patients, a small uncomplicated cSDH may be difficult to distinguish from simple brain atrophy with enlarged bifrontal CSF spaces. However, cSDHs exhibit mass effect; they flatten the underlying gyri, often extending around the entire
Trauma

(2-51A) Axial T1WI shows a right-sided cSDH. The collection is slightly hyperintense compared with CSF. (2-51B) Axial FLAIR in the same case shows that the cSDH is hyperintense relative to CSF.

(2-51C) T1 C+ FS in the same case shows that the outer membrane is thick and enhances uniformly. The inner membrane is thin, almost inapparent. (2-51D) Coronal T1 C+ in the same case shows the thick outer and thin inner membrane of the cSDH.

(2-52) Autopsy shows different aged cSDHs. Thickened dura-arachnoid residual clot is contained within a thin inner, thick outer membrane. (2-53) NECT in a 77y man with headaches 10 days following trauma shows bilateral hypodense collections. These are measured CSF-like subdural hygromas, caused by CSF extravasating through a tear in the arachnoid.
hemisphere and into the interhemispheric fissure. The increased extraaxial spaces in patients with cerebral atrophy are predominantly frontal and temporal.

A traumatic subdural hygroma is an accumulation of CSF in the subdural space after head injury, probably secondary to an arachnoid tear. Subdural hygromas are sometimes detected within the first 24 hours after trauma; however, the mean time for appearance is 9 days after injury.

A classic uncomplicated subdural hygroma is a hypodense, CSF-like, crescentic extraaxial collection that consists purely of CSF, has no blood products, lacks encapsulating membranes, and shows no enhancement following contrast administration (2-53). CSF leakage into the subdural space is also present in the vast majority of patients with cSDH. Therefore, many—if not most—cSDHs contain a mixture of both CSF and blood products.

A subdural effusion is an accumulation of clear fluid over the cerebral convexities or in the interhemispheric fissure. Subdural effusions are generally complications of meningitis; a history of prior infection, not trauma, is typical.

A subdural empyema (SDE) is a hypodense extraaxial fluid collection that contains pus. Most SDEs are secondary to sinusitis or mastoiditis, have strongly enhancing membranes, and often coexist with findings of meningitis. A typical SDE restricts strongly and uniformly on DWI.

**Traumatic Subarachnoid Hemorrhage**

Traumatic subarachnoid hemorrhage (tSAH) is found in virtually all cases of moderate to severe head trauma. Indeed, trauma—not ruptured saccular aneurysm—is the most common cause of intracranial SAH.

**Etiology**

tSAH can occur with both direct trauma to the skull and nonimpact closed head injury. Tearing of cortical arteries and veins, rupture of contusions and lacerations into the contiguous subarachnoid space, and choroid plexus bleeds with intraventricular hemorrhage may all result in blood collecting within the subarachnoid cisterns. Less commonly, tSAH arises from major vessel lacerations or dissections, with or without basilar skull fractures.

Although tSAH occasionally occurs in isolation, it is usually accompanied by other manifestations of brain injury. Subtle tSAH may be the only clue on initial imaging studies that more serious injuries lurk beneath the surface.

**Pathology**

**Location.** tSAHs are predominantly found in the perisylvian regions, in the anteroinferior frontal and temporal sulci, and over the hemispheric convexities (2-54). In very severe cases, tSAH spreads over most of the brain. In mild cases, blood collects in a single sulcus or the dependent portion of the interpeduncular Fossa. Rarely, Terson syndrome (intraocular hemorrhage) is associated with tSAH.

**Gross Pathology.** With the exception of location and associated parenchymal injuries, the gross appearance of tSAH is similar to that of aneurysmal SAH (aSAH). Curvilinear foci of bright red blood collect in cisterns and surface sulci (2-55).