Imagine you are asked to create a list of the disorders of the brain, head, and neck that one might commonly expect to encounter at an emergency department (ED) and describe the typical imaging features. At first, this challenge seems straightforward enough. However, upon beginning the task, it soon becomes clear that almost every disorder within the realm of neuroradiology/head and neck radiology might at one time or another present as an acute emergency. Inclusion of certain diagnoses such as stroke, fractures, and epiglottitis is a must. Other diagnoses, such as oligodendroglioma or perhaps a slowly growing lesion, might seem less clear-cut. Ultimately, it is important to realize that a wide variety of processes will result in an alteration in mental status leading to an ED visit, with imaging playing a key role in diagnosis and appropriate management.

Upon admission, inpatient workups now occur on a 24/7 basis, with many complex examinations completed during the night shift. On-call radiologists (often residents or fellows) are expected to provide “wet readings” or complete interpretations for complex cases covering the full spectrum of medicine, pediatrics, surgery, and related subspecialties. It was not that many years ago that the radiologist was faced with a seemingly never-ending stack of plain films from the ED, inpatient wards, and intensive care units requiring rapid interpretations. This work was interrupted by an occasional computed tomography (CT) scan. In this new millennium, during a typical shift the radiologist must maintain a rapid pace to review thousands of cross-sectional CT and magnetic resonance images (MRI) with two-dimensional (2D) and three-dimensional (3D) reformats. For this reason, the majority of the discussion and most of the examples in this chapter are based on these modalities and the latest techniques.

The most daunting part of preparing this chapter was to boil down all of the disorders and details to a set of requisites. Division of this chapter into sections is not quite as neat as one might think. For example, it is not possible to separate the vascular system from discussion of the brain, head and neck, or spine, and the imaging methods applied to the extracranial vessels in the setting of stroke are similar to those used for blunt or penetrating trauma to the neck. One may therefore notice mention of similar techniques and findings in several places with examples appropriate to the context. All readers would do well to study the other volumes in the Requisites series (especially Neuroradiology, Musculoskeletal Imaging, and Pediatric Radiology), which cover this material in great detail. In this attempt at condensing so much material into one useful volume, important topics inevitably have been neglected. We hope that this volume can serve as a starting point for further study and become a valuable reference to on-call radiologists, emergency department physicians, and residents of both specialties.

### Intracranial Hemorrhage and Traumatic Brain Injury

Whether in the setting of head trauma, spontaneous development of headache, or alteration of mental status, the ability to diagnose intracranial hemorrhage (ICH) is of primary importance for all practitioners. These presentations are some of the most common indications for brain imaging in the emergency setting. Almost invariably, the requisition will read, “Rule out bleed.” An understanding of traumatic and nontraumatic causes of ICH, the usual workup, and recognition of ICH is therefore important and seems like a natural starting point. A discussion of the important types of mass effect resulting from ICH and traumatic brain injury is also included in this section. An understanding of hemorrhage and herniation syndromes is central to the discussion of other topics that follow, such as stroke and neoplasms.

The word *hemorrhage* has Greek origins: the prefix *haima-*, meaning “blood,” and the suffix *-rrhage*, meaning “to gush or burst forth.” Incidence of ICH is approximately 25 to 30 per 100,000 adults in the United States, with a higher incidence in elderly hypertensive patients. ICH is typically more common in the African American and Asian populations. Bleeding may take place within the substance of the brain (intraaxial) or along the surface of the brain (extraaxial). Intraaxial hemorrhage implies parenchymal hemorrhage located in the cerebrum, cerebellum, or brainstem. Extraaxial hemorrhages include epidural, subdural, and subarachnoid hemorrhages, and intraventricular hemorrhage can be considered in this group as well. Hemorrhages can lead to different types of brain herniation, from direct mass effect and associated edema or development of hydrocephalus, causing significant morbidity and mortality.
General Imaging Characteristics of Hemorrhage

The appearance of ICH on a CT scan can vary depending on the age of the hemorrhage and the hemoglobin level. The attenuation of blood is typically based on the protein content, of which hemoglobin contributes a major portion. Therefore, the appearance of hyperacute/acute blood is easily detected on a CT scan in patients with normal hemoglobin levels (approximately 15 g/dL) and typically appears as a hyperattenuating mass. This appearance is typical because, immediately after extravasation, clot formation occurs with a progressive increase in attenuation over 72 hours as a result of increased hemoglobin concentration and separation of low-density serum. On the other hand, in anemic patients with a hemoglobin level less than 10 g/dL, acute hemorrhage can appear isoattenuating to the brain and can make detection difficult. Subsequently, after breakdown and hemolysis, the attenuation of the clot decreases until it becomes nearly isoattenuating to cerebrospinal fluid (CSF) by approximately 2 months. In the emergency setting, one should be aware of the “swirl” sign with an unretracted clot that appears to be hypointensifying and resembles a whirlpool; this sign may indicate active bleeding and typically occurs in a posttraumatic setting. It is important to recognize this sign, because prompt surgical evacuation may be required. The amount of mass effect on nearby tissues will depend on the size and location of the hemorrhage, as well as the amount of secondary vasogenic edema that develops.

Use of an intravenous contrast agent usually is not necessary for CT detection of ICH. If a contrast agent is used, an intraxial hemorrhage can demonstrate an enhancing ring that is usually due to reactive changes and formation of a vascularized capsule, which typically occurs 5 to 7 days after the event and can last up to 6 months. Subacute and chronic extraxial hematomas also can demonstrate peripheral enhancement, usually because of reactive changes and formation of granulation tissue. Unexpected areas of enhancement should raise concern, because active bleeding can appear as contrast pooling. Refer to the section on aneurysms and vascular malformations in this chapter for a discussion of CT angiography in the setting of acute ICH.

MRI has greatly revolutionized the evaluation of ICH. The evolution of hemorrhage from the hyperacute to the chronic stage will have corresponding signal changes on T1-weighted images (T1WIs), T2-weighted images (T2WIs), fluid-attenuated inversion recovery (FLAIR) images, and gradient-echo sequences. These properties can assist in detection and understanding of the time course of the injury. Although it is beyond the scope of this chapter, a description of the physics of the signal characteristics of blood products on MRI is generally based on the paramagnetic effects of iron and the diamagnetic effects of protein in the hemoglobin molecule. The usual signal characteristics of hemorrhage and the general time course over which hemorrhages evolve are summarized in Table 1-1.

EXTRAAXIAL HEMORRHAGE

Extraaxial hemorrhage occurs within the cranial vault but outside of brain tissue. Hemorrhage can collect in the epidural, subdural, or subarachnoid spaces and may be traumatic or spontaneous. It is important to recognize these entities because of their potential for significant morbidity and mortality. Poor clinical outcomes are usually the result of mass effect from the hemorrhage, which can lead to herniation, increased intracranial pressure, and ischemia. Intraventricular hemorrhage will be considered with these other types of extracerebral hemorrhage.

Epidural Hemorrhage

*Epidural hematoma* is the term generally applied to a hemorrhage that forms between the inner table of the calvarium and the outer layer of the dura because of its mass-like behavior. More than 90% of epidural hematomas are associated with fractures in the temporoparietal, frontal, and parieto-occipital regions. CT is usually the most efficient method for evaluation of this type of hemorrhage. An epidural hematoma typically has a hyperdense, biconvex appearance. It may cross the midline but generally does not cross sutures (because the dura has its attachment at the sutures), although this might not hold true if a fracture disrupts the suture. Epidural hematomas usually have an arterial source, commonly a tear of the middle meningeal artery, and much less commonly (in less than 10% of cases) a tear of the middle meningeal vein, diploic vein, or venous sinus (Figs. 1-1 and 1-2). The classic clinical presentation describes a patient with a “lucid” interval, although the incidence of this finding varies from 5% to 50% in the literature. Prompt identification of an epidural hematoma is critical, because evacuation or early reevaluation may be required. Management is based on clinical status, and therefore alert and oriented patients with small hematomas may be safely observed. The timing of follow-up CT depends on the patient’s condition, but generally the first follow-up CT scan may be obtained after 6 to 8 hours and, if the patient is stable, follow-up may be extended to 24 hours or more afterward.

TABLE 1-1 Usual Magnetic Resonance Signal Characteristics of Hemorrhage

<table>
<thead>
<tr>
<th>Stage</th>
<th>Time</th>
<th>Component</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute</td>
<td>(0-12 h)</td>
<td>Oxyhemoglobin</td>
<td>Isointense</td>
<td>Hyperintense</td>
</tr>
<tr>
<td>Acute</td>
<td>(12 h-3 days)</td>
<td>Deoxyhemoglobin</td>
<td>Isointense</td>
<td>Hypointense</td>
</tr>
<tr>
<td>Early subacute</td>
<td>(3-7 days)</td>
<td>Methemoglobin (intracellular)</td>
<td>Hyperintense</td>
<td>Hypointense</td>
</tr>
<tr>
<td>Late subacute</td>
<td>(1 wk-1 mo)</td>
<td>Methemoglobin (extracellular)</td>
<td>Hyperintense</td>
<td>Hyperintense</td>
</tr>
<tr>
<td>Chronic</td>
<td>(&gt;1 mo)</td>
<td>Hemosiderin</td>
<td>Hypointense</td>
<td>Hypointense</td>
</tr>
</tbody>
</table>
Subdural Collections

Subdural hematoma (SDH) is the term generally applied to a hemorrhage that collects in the potential space between the inner layer of the dura and the arachnoid membrane. It is typically the result of trauma (e.g., motor vehicle collisions [MVCs], assaults, and falls, with the latter especially occurring in the elderly population). An SDH causes a tear of the bridging vein(s) and has a hyperattenuating, crescentic appearance overlying the cerebral hemisphere (Fig. 1-3). These hemorrhages can cross sutures and may track along the falx and tentorium but do not cross the midline. Inward displacement of the cortical vessels may be noted on a contrast-enhanced scan. SDHs have a high association with subarachnoid hemorrhage. Acute SDHs thicker than 2 cm that occur with other parenchymal injuries are associated with greater than 50%
mortality. As the SDH evolves to the subacute stage (within 5 days to 3 weeks) and then to the chronic stage (after more than 3 weeks), it decreases in attenuation, becoming isodense to the brain and finally to CSF. A subacute SDH can have a layered appearance as a result of separation of formed elements from serum. Subacute hemorrhages may be relatively inconspicuous when they are isodense, and therefore it is especially important to recognize signs of mass effect, such as sulcal effacement, asymmetry of lateral ventricles, and shift of midline structures, as well as sulci that do not extend to the skull (Fig. 1-4). Bilateral isoattenuating SDHs can be especially challenging because findings are symmetric. One should beware of bilateral isoattenuating SDHs, particularly in elderly patients who do not have generous sulci and ventricles. At this stage, the SDH should be conspicuous on MRI, especially on FLAIR sequences. A subacute SDH also may be very conspicuous on T1WIs because of the hyperintensity of methemoglobin.

Chronic subdural hematomas are collections that have been present for more than 3 weeks. Even a chronic hematoma may present in the emergency setting, such as

**FIGURE 1-2** An epidural hematoma and complications demonstrate on noncontrast CT. A, The “swirl” sign in this large epidural hematoma suggests continued bleeding. B, A pontine (Duret) hemorrhage (arrow) and effacement of the basal cisterns as a result of downward herniation. C, Uncal herniation (the arrow shows the margin of the left temporal lobe) and a resultant left posterior cerebral artery territory infarct. The brainstem is distorted and also abnormally hypodense. D, Infarcts in bilateral anterior cerebral, left middle cerebral, and left posterior cerebral artery territories as a result of herniations.
in a patient prone to repeated falls who is brought in because of a change in mental status. On both CT and MRI, these collections typically have a crescentic shape and may demonstrate enhancing septations and membranes surrounding the collection after administration of a contrast agent. Calcification of chronic SDH can occur and may be quite extensive (Fig. 1-5). Areas of hyperdensity within a larger hypodense SDH may indicate an acute component due to recurrent bleeding, termed an “acute on chronic subdural hematoma.” Mixed density collections also may be acute as a result of active bleeding or CSF accumulation as a result of tearing of the arachnoid membrane. A chronic SDH is usually isointense to CSF on both T1WIs and T2WIs, but the appearance can be variable depending on any recurrent bleeding within the collection. The FLAIR sequence is typically very sensitive for detection of chronic SDH as a result of hyperintensity based on protein content. Hemosiderin within the hematoma will cause a signal void because of the susceptibility effect, and “blooming” (i.e., the hematoma appears to be larger than its true size) will be noted on a gradient-echo sequence.

A subdural hygroma is another type of collection that is commonly thought to be synonymous with a chronic subdural hematoma. The actual definition of a hygroma is an accumulation of fluid due to a tear in the arachnoid membrane, usually by some type of trauma or from rapid ventricular decompression with associated accumulation of CSF within the subdural space. Many persons still use this term interchangeably with chronic subdural hematoma. CT demonstrates a fluid collection isodense to CSF in the subdural space. MRI can be useful in differentiating CSF from a chronic hematoma based on the imaging characteristics of the fluid on all sequences. Occasionally hygromas are difficult to differentiate from the prominence of the extraaxial CSF space associated with cerebral atrophy. The position of the cortical veins can be a helpful clue. In the presence of atrophy, the cortical veins are visible traversing the subarachnoid space, whereas...
with a hygroma, they are displaced inward along with the arachnoid membrane by the fluid in the subdural space.

**Subarachnoid Hemorrhage**

Subarachnoid hemorrhage (SAH) fills the space between the pia and the arachnoid membrane, outlining the sulci and basilar cisterns. SAH can be due to a variety of causes, including trauma, a ruptured aneurysm, hypertension, arteriovenous malformation, occult spinal vascular malformation, and hemorrhagic transformation of an ischemic infarction. SAH is often associated with overlying traumatic SDH. SAHs generally do not cause mass effect or focal regions of edema. However, in patients presenting with ominous signs on clinical grading scales, such as stupor or coma, diffuse cerebral edema may be evident. On CT, hypodensity is seen within the sulci and/or basilar cisterns (Figs. 1-6 and 1-7).

Although MRI may be as sensitive as CT for the detection of acute parenchymal hemorrhage and SAH, CT generally remains the modality of choice (and the imaging gold standard). The sensitivity of CT for the detection of SAH compared with CSF analysis can vary from up to 98% to 100% within 12 hours to approximately 85% to 90% after 24 hours of symptom onset. Other factors affecting sensitivity are the hemoglobin concentration and the size and location of the hemorrhage. CT is widely available, can be performed rapidly, and is relatively inexpensive. In several small studies, MRI has demonstrated sensitivity equivalent to CT for detection of acute parenchymal hemorrhage and SAH. In some cases of “CT-negative” (subacute) hemorrhage, MRI has shown greater sensitivity. However, results may be confounded by artifacts from CSF pulsations, an elevated level of protein (meningitis), or oxygen concentration (i.e., a high fraction of inspired oxygen) in CSF on FLAIR images and the presence of blood products from previous microhemorrhages on gradient-echo images.

**Intraventricular Hemorrhage**

In the adult population, intraventricular hemorrhage (IVH) is typically caused by trauma. It can result from extension of a parenchymal hemorrhage into the ventricles or from redistribution of SAH. Primary IVH is uncommon and is usually caused by a ruptured aneurysm, an intraventricular tumor, vascular malformation, or coagulopathy (Fig. 1-8). Large IVHs are quite conspicuous on CT or MRI. They may occupy a majority of the ventricle(s) and may result in hydrocephalus and increased intracranial pressure. Small amounts of IVH may be difficult to detect; one must check carefully for dependent densities within the atria and occipital horns of the lateral ventricles. Normal choroid plexus calcifications in the atria of lateral ventricles, in the fourth ventricle, and extending through the foramina of Luschka should not be mistaken for acute IVH.

Another less common type of extracerebral ICH that may present acutely is a pituitary hemorrhage, which is usually associated with pituitary apoplexy due to pituitary necrosis that may become hemorrhagic. Presenting symptoms may include headache, visual loss, ophthalmoplegia, nausea, and vomiting. Other causes of pituitary hemorrhage include tumors (e.g., macroadenoma and germinoma) and, less commonly, trauma.
Chapter 1 Traumatic and Nontraumatic Emergencies of the Brain, Head, and Neck

INTRAAXIAL HEMORRHAGE

The cause of intraaxial (parenchymal) hemorrhages can generally be categorized as spontaneous or traumatic. Traumatic causes include blunt injury from MVCs, assault, and penetrating injuries such as gunshot wounds. Intraaxial hemorrhages have many spontaneous causes, which are discussed in the section on hemorrhagic stroke.

Contusion

Parenchymal contusions result from blunt trauma and can occur in the cortex or white matter. Their locations are typically at the site of greatest impact of brain on bone, including the anterior/inferior frontal lobes and the temporal lobes. They can be considered coup (occurring at the site of impact) or contrecoup (opposite the site of impact).

FIGURE 1-6 Subarachnoid hemorrhage from a ruptured aneurysm. A, Noncontrast computed tomography (CT) shows ill-defined hyperdense subarachnoid hemorrhage in the left Sylvian cistern (black arrow) and rim calcification in the wall of the aneurysm (white arrow). B, A volume-rendered image from CT angiography shows a large aneurysm (arrow) projecting above the lesser sphenoid wing. C, Reconstruction from a three-dimensional rotational digital subtraction angiogram shows the carotid-ophthalmic aneurysm to the best advantage.
impact) types. On CT, a contusion typically appears as an area of hyperdensity with a surrounding rim of hypodense edema. A parenchymal contusion can initially appear as a focal area of subtle hypodensity and may blossom on follow-up examination at 12 to 24 hours with development of an obvious central area of hyperdensity and a larger surrounding zone of hypodense edema (Fig. 1-9). On MRI, signal characteristics reflect the hemorrhagic and edematous components. Over time, the density and signal characteristics of the hemorrhage will evolve in a fashion similar to a spontaneous hemorrhage. Parenchymal hemorrhage due to penetrating trauma, such as from a gunshot wound or impalement, will follow the same general pattern of evolution.

**Diffuse Axonal Injury**

Diffuse axonal injury (DAI) is another type of traumatic brain injury that may present with parenchymal hemorrhages and is distinct from a parenchymal contusion. DAI is an injury to the axons caused by acceleration/deceleration injury with a rotational component (usually from an MVC or other blunt trauma to the head). Complete transection of axons may occur with injury to the associated capillaries, or partial disruption of the axons may occur. DAI lesions typically occur at the interfaces of gray and white matter in the cerebral hemispheres, the body and splenium of the corpus callosum, the midbrain, and the upper pons. Lesions also may be seen in the basal ganglia.
Patients sustaining DAI typically lose consciousness at the moment of impact. DAI may be suspected when the clinical examination is worse than expected based on the findings of an initial CT scan. Usually, the greater the number of lesions, the worse the prognosis. Persons who recover usually demonstrate lingering effects such as headaches and cognitive deficits. Initial CT scans in more than half of patients with DAI may be negative. CT findings include hypodense foci due to edema in areas of incomplete axonal disruption and hyperdense foci due to hemorrhage.
to petechial hemorrhage where complete transection of the axons and associated capillaries has occurred (Fig. 1-10). MRI is more sensitive than CT for detection of DAI. Approximately 30% of persons with negative CT findings will demonstrate abnormal findings on MRI. These findings include FLAIR and T2WI hyperintensities (edema) and gradient-echo hypointensities (hemorrhages) (Fig. 1-11). Lesions may appear hyperintense on diffusion-weighted images. It is estimated that more than 80% of DAI lesions are nonhemorrhagic. Generally, if imaging is repeated within 3 to 5 days, more lesions will become apparent as the process evolves.

A staging system for DAI based on locations of lesions on histopathology may be applied to MRI findings. Stage 1 is based on subcortical lesions in the frontal and temporal lobes. Stage 2 will also show lesions in the corpus callosum and lobar white matter, and stage 3 will have lesions in the midbrain and pons. Diffusion tensor imaging is well suited to the evaluation of white matter tracts and has been shown to be more sensitive than conventional MRI for detection of DAI and correlates more closely with clinical outcomes. Diffusion tensor imaging may be helpful for long-term diagnostic evaluation of patients with mild traumatic brain injury more so than in the initial emergency setting.

**BRAIN HERNIATIONS**

Brain herniation is a potentially devastating complication of increased intracranial pressure. The most common causes include ICHs, brain tumors, and cerebral edema from stroke or anoxic injury. To explain this concept, a common example from the literature describes the brain as being separated into multiple compartments within a rigid container. Any shift of the brain from one compartment to another is considered herniation. With shift of the brain, there can be mass effect on adjacent and contralateral parenchyma, the brainstem, major intracranial vessels, and cranial nerves. As a result, the feared complications of herniations include ischemic infarcts due to compression of the major intracranial vessels (commonly, the anterior and posterior cerebral arteries), cranial nerve...
palsies, and “brain death” due to compression and infarction of the brainstem. The major types of intracranial herniations include subfalcine, transtentorial, tonsillar herniation through the foramen magnum, extracranial herniation (through a defect in the skull), and, less commonly, transalar herniation. Once the complications of herniation have developed, it is often too late to intervene. Thus it is best to recognize the signs of impending herniation, when prompt neurosurgical intervention may avert disaster.

**Subfalcine Herniation**

Subfalcine herniation occurs as a result of displacement and impingement of the cingulate gyrus underneath the falk. It is usually caused by mass effect on the frontal lobe and is associated with ipsilateral lateral ventricle compression and obstruction of the foramen of Monro with dilatation of the contralateral ventricle (“trapped ventricle”). The degree of midline shift (not synonymous with subfalcine herniation) can be estimated by drawing a line between the anterior and posterior attachments of the falk and measuring the shift of the septum pellucidum relative to this line (see Fig. 1-3). Anterior cerebral artery territory infarct(s) may result from this type of herniation.

**Transtentorial Herniation**

Transtentorial herniations include two major types: descending transtentorial herniation (DTH) and ascending transtentorial herniation (ATH). An early DTH is known as uncal herniation, in which the uncus (i.e., the anterior portion of the parahippocampal gyrus) is displaced medially and occupies the ipsilateral suprasellar cistern. A later-stage DTH is caused by continued mass effect with displacement of the remainder of the medial temporal lobe through the tentorial incisura, which completely occupies the suprasellar cistern (along with the uncus) and causes enlargement of the ipsilateral and effacement of the contralateral ambient cisterns. This phenomenon occurs because, as there is marked shifting of brain in the supratentorial compartment, the brainstem shifts in the same direction. Occasionally, when marked mass effect is present, there can be compression of the contralateral cerebral peduncle against the tentorium, or “Kernohan’s notch,” which leads to ipsilateral motor weakness (this phenomenon may be a false localizing sign). Other imaging findings include a “trapped” temporal horn of the lateral ventricle contralateral to the side of the mass and Duret hemorrhages—that is, hemorrhages of the midbrain and pons caused by stretching and tearing of the arterial perforators. In cases of bilateral mass effect, displacement of both temporal lobes and midbrain can occur through the incisura, leading to effacement of the basilar cisterns bilaterally. Complications of this type of herniation include compression of the posterior cerebral artery and penetrating basal arteries with associated infarcts in these vascular distributions (see Fig. 1-2). In addition, compression of the oculomotor nerve (cranial nerve [CN] III) can occur with an associated palsy. ATH is less common and is caused by superior displacement of the cerebellum and brainstem through the incisura. ATH is usually due to mass effect in the posterior fossa (as from hemorrhage, tumor, or infarct), and imaging shows compression on the posterolateral midbrain with associated effacement of the ambient and quadrigeminal plate cisterns. Hydrocephalus is usually present as a result of obstruction at the level of the cerebral aqueduct of Sylvius.

**Tonsillar Herniation**

Tonsillar herniation is caused by downward displacement of the cerebellar tonsils through the foramen magnum into the spinal canal (generally by more than 5 mm). Imaging shows a peg-like configuration to the tonsils with obliteration of the CSF space in the foramen magnum (Fig. 1-12). Complications include obstructive hydrocephalus from compression of the fourth ventricle. Mild tonsillar ectopia, Chiari I malformations, and sagging tonsils due to intracranial hypotension should not be mistaken for acute tonsillar herniation, but it should be considered seriously when downward mass effect is expected based on brain edema, mass, or hemorrhage.

**Extracranial Herniation**

An extracranial herniation is the displacement of brain parenchyma through a cranial and dural defect that is usually caused by trauma or a craniectomy (usually performed to prevent downward herniation from acute cerebral edema). Complications may include infarction of the herniated brain tissue.

**Transalar Herniation**

Transalar herniation is uncommon and, by itself, does not cause symptoms. It is usually associated with subfalcine and transtentorial herniations. This type of herniation is caused by displacement of the temporal lobe anteriorly or of the frontal lobe posteriorly across the sphenoid wing. One should look for anterior or posterior displacement of the middle cerebral artery to identify this type of herniation.

In the setting of severe head trauma, many of these different types of injuries may coexist. The mechanism of injury should correspond with the degree of injury. In cases when the reported mechanism is mild, nonaccidental trauma (e.g., “Trauma X” and “shaken-baby syndrome”) should be considered. Infants, children, persons with mental or physical disabilities, and elderly persons are particularly at risk. Skull fractures, SAH, SDH, contusions, shear injuries, infarcts, vertebral compression fractures, and retinal hemorrhages constitute the usual neuroradiologic spectrum of abnormalities. Injuries of different ages, metaphyseal and rib fractures, and visceral injuries are other common findings of child abuse.

**ACUTE CEREBROVASCULAR DISORDERS**

Although acute cerebrovascular disorders usually do not occur as a result of trauma, they are treated with the same urgency as traumatic injuries or spontaneous ICH. According to the American Heart Association update for 2015, in the United States, approximately 800,000 strokes occur each year (on average, about one stroke occurs every 40 seconds, resulting in one death every 4 minutes). Almost 25% are recurrent, and 75% occur in persons older than
The 20% mortality rate is surpassed only by cardiac disease, cancer, and chronic lung disease. Stroke is the leading cause of severe, long-term disability and long-term care. Estimates of annual cost exceed $50 billion.

One clinical definition of stroke is a neurologic deficit caused by inadequate supply of oxygen to a region of the brain. Stroke can be due to a low flow state or rupture of a vessel and thus may be divided into ischemic and hemorrhagic varieties. The definition of stroke used for current clinical trials requires symptoms lasting more than 24 hours or imaging of an acute clinically relevant brain lesion in a patient with rapidly vanishing symptoms. In the past, a transient ischemic attack (TIA) implied resolution of the deficit within a 24-hour period. The current definition of TIA is a brief episode of neurologic dysfunction caused by a focal disturbance of brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of infarction. Estimates of the annual incidence of TIA in the United States vary from 200,000 to 500,000. The 1-year mortality rate after TIA has been reported to be 12%. Evidence of acute infarction may be identified by MRI (tissue-based case definition) in up to 30% of patients who meet the clinical criteria for a TIA. Semantics can be unclear when an abnormality is detected on imaging in the absence of symptoms.

**Hemorrhagic Stroke: Spontaneous Parenchymal Hemorrhage**

Approximately 10% to 15% of strokes present with an acute parenchymal hemorrhage. The most common cause is hypertension (Fig. 1-13). Coagulopathies, hematologic disorders including hypercoagulable states, amyloid angiopathy, drugs, vascular malformations and aneurysms, vasculitides, and tumors round out the usual list of causes. (Refer to the section on aneurysms and vascular malformations in this chapter for a discussion of CT angiography in the setting of acute ICH.) Hemorrhages resulting from use of illicit drugs and vascular malformations are commonly found in young adults (Fig. 1-14). Sickle
A parenchymal hemorrhage due to use of an illicit drug—Ecstasy (3,4 methylenedioxymethamphetamine). A, Computed tomography shows a hyperdense acute hemorrhage with minimal surrounding hypodense edema. B, A T1-weighted image shows iso- to mild hyperintensity with hypointense edema. Fluid-attenuated inversion recovery (C) and fat-suppressed T2-weighted gradient and spin echo (D) show hypointensity with surrounding hyperintense edema. E, Gradient echo shows a peripheral rim of signal loss and blooming. In summary, signal changes on T1- and T2-weighted images are consistent with deoxyhemoglobin, although the gradient echo suggests only a rim of deoxyhemoglobin.
The role of imaging in acute stroke diagnosis and management continues to evolve. Since the mid 1970s, unenhanced CT has been the first-line modality to determine the cause of acute neurologic deficits. CT can offer the chance to detect an ischemic infarct, generally in the middle cerebral artery territory, within 3 hours in up to one third of cases based on findings of subtle parenchymal hypodensity, loss of gray–white matter differentiation (including loss of the insular ribbon or margins of basal ganglia; Fig. 1-15), and effacement of sulci. A hyperdense vessel sign may indicate the presence of an acute thrombus and support the diagnosis. The sensitivity for detection of acute stroke has been shown to increase with the use of the “acute stroke” window and level settings (see Fig. 1-15). A very narrow window width of 8 Hounsfield units (HUs) and a level of 32 HUs (compared with 80 and 40 HUs, respectively) may increase the sensitivity of CT to approximately 70% without a loss of specificity.

CT is currently used to screen patients who may be considered for treatment with intravenous recombinant tissue plasminogen activator (rt-PA). This medication is currently approved by the Food and Drug Administration for use within 3 hours of onset based on guidelines from the National Institute of Neurologic Disorders and Stroke rt-PA trial. Since 2013, its use within 4.5 hours of stroke onset in selected patients has been endorsed in a joint clinical policy statement of the American College of Emergency Physicians, American Academy of Neurology, and Neurocritical Care Society. Beyond this time, the risk of ICH due to intravenous thrombolysis was shown to outweigh potential benefits. An association between larger stroke volumes (greater than one third of the middle cerebral artery territory) and reperfusion hemorrhage was initially reported.

This criterion for the use of 100 mL estimated infarct volume has commonly been applied in stroke trials. The Alberta Stroke Program early CT score, a 10-point topographic scoring system, was developed to try to more easily quantify initial stroke volumes. This score has been shown to correlate with the initial National Institutes of Health stroke score and is one piece of data that may be considered in determining patient management.

### Magnetic Resonance: Diffusion-Weighted Imaging

MRI with diffusion-weighted imaging (DWI), which became widely available in routine clinical practice in the late 1990s, offers significantly greater sensitivity and specificity for the detection of acute stroke (greater than 90% compared with approximately 60% for CT). Energy depletion will trigger a cascade that alters the internal cellular milieu, such as the glutamate excitotoxic pathway, in which reduced energy-dependent glutamate reuptake within the synaptic clefts results in the development of cytotoxic edema. The restriction of water molecule diffusion appears as hyperintensity on diffusion-weighted images. The diffusion “experiment” can be performed with a variety of rapid imaging techniques, such as echo planar imaging, and can acquire images of the entire brain in half a minute. This approach minimizes the effects of patient motion, which is especially important when the clinical presentation includes alteration of mental status. The apparent diffusion coefficient (ADC) value is a quantitative measure that may be calculated from the diffusion-weighted images. Because diffusion-weighted images rely on both diffusion and T2 effects, it is wise to confirm that the ADC values are indeed reduced before diagnosing an acute infarct. This confirmation will reduce the number of false-positive results due to the “T2 shine-through” effect from old infarcts (gliosis) or other T2 hyperintense processes such as vasogenic edema.

Acute ischemic infarcts appear as hyperintense regions on DWI (see Fig. 1-15) as quickly as 30 minutes after onset. Up to 100% sensitivity has been demonstrated in clinical studies. However, in routine practice, small lesions in the brainstem may not be perceived initially, only to be detected on a follow-up examination prompted by persistent symptoms. It is also possible that a region of ischemia (prior to a completed infarction) may not be detected on an initial imaging study, thus providing a false-negative result. False-positive results on DWI can be due to processes that mimic stroke and also cause diffusion restriction, such as certain neoplasms, multifocal metastatic disease, and abscesses. The presence or absence of associated findings on conventional MRI sequences—such as loss of gray–white matter differentiation on T1WIs and hyperintense edema on FLAIR and T2WIs—may help with diagnosis, although these signs may be inconspicuous for 6 to 12 hours after stroke onset. Blooming on gradient-echo sequences due to intravascular thrombus and loss of expected vascular flow voids are other useful clues.

Lacunar infarcts are generally less than 1 cm in diameter and are presumed to be due to occlusion of small perforating branches as a result of embolic, atheromatous, or thrombotic lesions. Lacunar infarcts occur most commonly in the basal ganglia, internal and external capsules, immediate periventricular white matter (corona radiata), and, less frequently, in the centrum semiovale. Occlusion of basilar artery perforators will result in lacunes in the brainstem. Diffusion imaging offers the ability to identify very small, acute infarcts even in the background of chronic white
matter disease and remote lacunes (Fig. 1-16). Although MRI is still considered a relatively expensive technique, it has the potential to reduce the number of unnecessary hospital admissions for recurrent small vessel infarcts in many patients. It also may assist in selecting the most appropriate pathway for patients with central embolic sources of infarcts based on the detection of infarcts in different vascular territories.

MRI is also valuable in the setting of neonatal hypoxic ischemic encephalopathy. Cranial ultrasonography and CT may be used to evaluate germinal matrix hemorrhages, periventricular leukomalacia, and hydrocephalus. Diffusion-weighted MRI is most sensitive for evaluating the different patterns of injury. In preterm infants subjected to mild hypotension, the periventricular regions are most often affected. With more severe hypotension, the basal ganglia, brainstem, and cerebellum may be involved. In full-term infants with mild hypotension, infarcts in the border zones between anterior and middle cerebral arteries or between middle and posterior cerebral arteries may result. Severe hypotension may result in infarcts of basal ganglia, hippocampi, corticospinal tracts, and sensorimotor cortex.

**FIGURE 1-15** A hyperacute infarct. A, Noncontrast computed tomography with a window/level of 80/40 shows a subtle decrease in density of the right insular cortex. B, The insular “ribbon” sign is more conspicuous with a stroke window/level of 40/40 (arrows). C, The infarct is much more conspicuous on a diffusion-weighted image. D, Time-of-flight magnetic resonance angiography shows attenuation of right middle cerebral artery distal branches.
Diffusion-weighted hyperintensity generally begins to decline after a few days, with the process of ADC pseudo-normalization usually taking place during the next few weeks. Final ADC values will vary based on the degree of gliosis or cavitation of the infarct. It should be noted that infarct development depends on the magnitude and duration of ischemia and the metabolic demands of the affected tissue. Although diffusion restriction due to ischemia almost always results in infarction, rare cases of spontaneous reversible diffusion abnormalities have been reported, as well as those occurring in the setting of thrombolytic therapy.

**Magnetic Resonance Angiography**

Noninvasive imaging of the vessels of the head and neck with MR angiography (MRA) based on time-of-flight or phase-contrast MRA techniques can be used to locate stenoses and occlusions in the extracranial and intracranial arterial systems (see Fig. 1-15). Gadolinium-enhanced MRA has become the standard of care at some institutions; this procedure requires consideration of renal function. Complete brain MRI and head and neck MRA examinations can be acquired in less than 30 minutes and have become the routine standard of care; they are often performed immediately or soon after completion of CT. It must be stressed...
that patient safety is a primary concern and therefore careful attention to screening for potential contraindications prior to MR scanning is a requisite at all times.

**Magnetic Resonance: Perfusion Imaging**

It became clear from imaging-based stroke trials that final infarct volumes were often larger than those identified by imaging at the time of admission. Advances in rapid scanning techniques soon led to the ability to obtain functional images of brain perfusion. By demonstrating an ischemic zone at the periphery of an acute infarct, salvageable tissue (the so-called penumbra) could be targeted with novel therapies. Dynamic gadolinium-enhanced T2* perfusion-weighted imaging (PWI) is one available technique that is based on the decrease in tissue signal intensity as a function of time during passage of a bolus of contrast material. Functional “maps” of different perfusion parameters may be calculated from the time-signal intensity curves obtained during a minute-long acquisition. Cerebral blood volume (CBV) and tissue mean transit time (MTT) can be estimated using different methods, most commonly deconvolution analysis. One limitation of MR-based techniques is that the blood volume estimate is a relative value. Cerebral blood flow (CBF) can be estimated by dividing CBV by MTT. A penumbra will be identified when a region of decreased CBF or prolonged MTT is larger than the infarct detected by DWI—a perfusion mismatch. Based on the extent of the mismatch, aggressive therapies may be pursued to limit the final infarct volume.

In some cases, the perfusion abnormality may exactly match the diffusion abnormality, and thus there is no penumbra. The final infarct volume is not expected to increase further. In other cases, when prompt reperfusion has occurred, such as from early vessel recanalization, the perfusion abnormality may be smaller than the diffusion abnormality. In both of these situations, the risk of aggressive treatment is probably not warranted. The potential of improved clinical outcomes from therapeutic strategies based on perfusion imaging may result from either salvage of tissue at risk or reduction of complications.

Arterial spin labeling (ASL) is an MR perfusion technique that does not require injection of a contrast agent. ASL uses paramagnetic labeling of water in blood flowing to the brain to produce measurements that are proportional to CBF. There are many varieties and technical limitations of ASL. Clinical utility will require determination of appropriate clinical perfusion thresholds. ASL is becoming increasingly available but is not yet used routinely at most centers.

**Computed Tomography: Perfusion Imaging**

The advent and wide availability of multidetector CT (MDCT) scanners has led to the use of CT angiography (CTA) and CT perfusion (CTP) imaging in the workup of suspected acute ischemic stroke. The ability to acquire a large 3D volume of data rapidly during administration of a bolus of contrast material combined with submillimeter spatial resolution results in CTA images that in many instances approach the diagnostic quality of more invasive digital subtraction angiograms. Large detector arrays and cine or shuttle modes of scanner operation allow for generous coverage of the brain during perfusion studies. Detector arrays with 64 rows are common now, and newer models with up to 320 rows are available. Analogous to PWI, CTP is based on measurement of tissue density as a function of time during a first pass of an intravenous contrast agent and commonly uses deconvolution analysis. In contrast to PWI, as a result of the proportionate increase in tissue attenuation due to iodine concentration, quantitative (rather than relative) estimation of CBV (and therefore CBF) can be obtained by CTP. Other parameters including time to maximum enhancement (Tmax) also may be measured.

CTP applied in the setting of acute stroke has been validated with clinical outcomes and follow-up imaging and also by comparison with DWI. Wintermark and colleagues initially proposed that regions with a CBV less than 2.5 mL/100 g could be considered the “core infarct” and regions with a CBF reduction of more than 34% be defined as the penumbra. These values were based on correspondence with initial DWI abnormality and final infarct size. Schaefer and colleagues later proposed other absolute values and the use of normalized CBV and CBF ratios to help distinguish ischemic tissue likely to become infarcted from that likely to survive after intraarterial recanalization therapy. Perfusion maps can be visually compared to determine if a penumbra is present but may underestimate or overestimate the extent of tissue at risk (Fig. 1-17). Software packages are available that can automatically segment the processed perfusion maps into core infarct, penumbra, and normal regions based on thresholding techniques.

Therapeutic trials of desmoteplase, a novel thrombolytic drug, including the Desmoteplase in Acute Ischemic Stroke (DIAS) and Dose Escalation study of Desmoteplase in Acute Ischemic Stroke (DEDAS) trials, were based on demonstration of a perfusion mismatch. Although clinical outcomes were not promising, these trials demonstrated the value of advanced imaging compared with unenhanced CT and use of a strict time limit alone.

Thrombolysis with intravenous rt-PA is limited by a short time window after onset of symptoms and a recanalization rate of less than 50%. Stroke specialists had hoped that an endovascular approach to treatment of severe strokes would be a valuable addition or alternative to intravenous therapy. The paradigm of acute stroke imaging has continued to evolve with clinical trials.

Over the course of several trials without promising results, including randomized, controlled trials of intraarterial treatment (Interventional Management of Stroke III [IMS III], Synthesis, and Mechanical Retrieval and Recanalization of Stroke Clots Using Embolectomy [MR RESCUE]), many lessons were learned. Improved outcomes were demonstrated with enrollment of patients with severe strokes, proof of proximal vessel occlusion, early initiation of treatment, and use of modern thrombectomy devices (i.e., retrievable stents). The recently published results of the Multicenter Randomized Clinical Trial of Endovascular Treatment of Acute Ischemic Stroke in the Netherlands (MR CLEAN), which required demonstration of a proximal vessel occlusion, ended the long drought of negative results for endovascular treatment (Fig. 1-18).

In the Extending the time for Thrombolysis in Emergency Neurological Deficits–Intra-Arterial (EXTEND-IA) trial, the imaging inclusion criteria were a proximal intracranial occlusion, a perfusion mismatch (CTP Tmax >6-second delay perfusion volume compared with CBF) or DWI...
FIGURE 1-17 Perfusion computed tomography (CT)—large mismatch. A, Noncontrast CT shows decreased density of the left caudate head and putamen. B, Cerebral blood flow (CBF) and mean transit time (MTT) abnormalities are much larger than core infarct (cerebral blood volume [CBV] abnormality). C, CT follow-up 1 week after conservative management shows that the final infarct volume closely matches the initial CBV abnormality. D, An initial CT angiogram showed abrupt occlusion of the left middle cerebral artery stem (arrow) but with filling of distal branches as a result of robust leptomeningeal collateral circulation.

FIGURE 1-18 Mechanical thrombectomy. A, An acute right middle cerebral artery occlusion. B, A Merci retriever is deployed distal to the thrombus. (Note that newer stent retrievers, such as the Solitaire revascularization device, have supplanted use of this device.) C, Recanalization of the right middle cerebral artery after clot retrieval.
infarct core), and infarct core lesion volume less than 70 mL. The study demonstrated that early thrombectomy with a stent retriever in this setting improved reperfusion, early neurologic recovery, and functional outcome.

Another consideration in penumbral imaging is based on assessment of collateral circulation by CTA, MRA, or digital subtraction angiography (DSA). Good collateral circulation has been associated with improved clinical outcomes after treatment even when reperfusion was achieved. Different methods of grading collaterals have been described based on single or multiphase CTA techniques. In the multicenter Endovascular Treatment for Small Core and Anterior Circulation Proximal Occlusion with Emphasis on Minimizing CT to Recanalization Times (ESCAPE) trial, imaging inclusion/exclusion criteria were based on CTA identification of the location of the occlusion, the extent of collateral vessels, and the extent of CTP mismatch (CBV and CBF). Rapid endovascular thrombectomy improved functional outcomes and reduced mortality in patients with acute strokes who had proximal vessel occlusion, a small infarct core, and moderate to good collateral circulation.

A recent mismatch-based penumbral imaging paradigm takes into consideration venous outflow based on MR susceptibility-weighted imaging (SWI). An increase in brain tissue demand for oxygen leads to an increase in concentration of deoxyhemoglobin in capillaries and veins and signal loss on SWI. Perfusion mismatch may be graded by the ratio of SWI abnormality to the DWI infarct core volume.

Thus current paradigms may be based on identification of tissue-based, collateral, or venous outflow–based mismatches. Continued outcomes-based research will be necessary to determine the best imaging protocol to identify the subset of patients who might benefit from aggressive treatment with a reduced risk of symptomatic hemorrhage. It should be noted that great variety exists in the application of these techniques in routine clinical practice. Some authors have proposed that CT and MR perfusion techniques should be limited to research patients and that clinical decisions regarding pursuit of endovascular treatment should be based on results of noncontrast CT, CTA, and DWI examinations.

**Hemorrhagic Transformation**

Hemorrhagic transformation of an ischemic infarct that is thought to result from reperfusion injury can be a dreaded complication of therapy or may happen spontaneously within hours or after a period of several weeks (Fig. 1-19). If a large hematoma is present at the time of initial imaging, it may not be possible to distinguish a primary parenchymal hemorrhage from hemorrhagic transformation. The blood products generally cause substantial artifacts on DWI. However, if the hemorrhage is confined within a larger zone of restricted diffusion, the cause may be clear. Petechial hemorrhages occur very commonly within an ischemic infarct, are best detected by gradient-echo imaging, and do not usually lead to increased morbidity.

**Cortical Laminar Necrosis**

A pattern of gyriform T1-weighted hyperintensity developing a week or two after an ischemic infarct may be attributed to cortical laminar necrosis (Fig. 1-20). It seems that gray matter is more vulnerable to ischemic necrosis than white matter (especially the third layer of the six cortical layers), and although the signal changes may lead one to diagnose hemorrhage or calcification, in one histologic study neither was detected. The exact cause of the T1
shortening is uncertain, but it may be due to high concentrations of proteins and macromolecules related to tissue necrosis.

Cerebral Venous Infarction and Sinus Thrombosis

An uncommon (with annual incidence estimates of less than 1 case per 100,000 population) but important cause of hemorrhage is cerebral venous thrombosis (Fig. 1-21), which may affect cortical veins and other portions of the superficial and deep venous drainage systems. Hypercoagulable states due to pregnancy and the postpartum period or oral contraceptive use, dehydration, regional infections, and trauma are relatively common. Common presenting symptoms include headache, seizure, and focal neurologic deficits. Fluctuating symptoms and intracranial hypertension are common as well. Bilateral parenchymal hemorrhages or infarcts that do not obey usual arterial territory borders can be clues to diagnosis. A working knowledge of the normal anatomy of the major venous structures and the common variants is necessary to avoid diagnostic pitfalls, especially false-positive results. On unenhanced CT, normal venous structures may appear denser than usual as a result of dehydration or elevated hematocrit, whereas thrombosis should appear hyperdense relative to arteries. A filling defect or occlusion may be detected on contrast-enhanced CT. CT venography can be performed with a thin-section, volumetric technique that allows the creation of 2D and 3D reconstructions. Normal and thrombosed venous sinuses take on many different appearances on MRI depending on scan parameters, flow velocity, and turbulence. Unexpected hyperintensity, loss of usual flow voids, and blooming on gradient-echo sequence are clues that flow-sensitive MR venography (MRV) should be performed. Time-of-flight MRV may be less sensitive than phase contrast technique because of shine-through of methemoglobin in a thrombosed sinus, simulating flow in the vessel. Contrast-enhanced MRV may be more sensitive, although enhancement of chronic thrombus can be misleading. Associated findings of edema, hemorrhage, or ischemic infarct may help one arrive at the correct diagnosis, but brain swelling without signal changes has been reported in up to approximately 40% of patients. Cavernous sinus thrombosis is discussed in relation to complex sinus and orbit infections in the section on head and neck imaging in this chapter. Prompt diagnosis of cerebral venous thrombosis is critical, because many of the parenchymal changes may be reversible. Systemic anticoagulation and local catheter-based thrombolytic, mechanical, or rheolytic (hydrodynamic) clot dissolution are treatment considerations. Intracranial hypertension and collateral formation leading to dural arteriovenous malformations are possible long-term sequelae.

Imaging of the cerebral venous sinuses is also often performed in the workup of patients with papilledema or suspected idiopathic intracranial hypertension (IIH). Many patients with IIH have venous hypertension and stenosis of either the dominant or both transverse sinuses. One retrospective study reported cure of IIH symptoms in 49 of 52 patients after transverse sinus stent placement.

ANEURYSMS, VASCULAR MALFORMATIONS, AND VASCULAR INJURIES

Aneurysms and cerebral vascular malformations present in various ways in the emergency setting. SAH resulting from a ruptured aneurysm and parenchymal hemorrhage related to an arteriovenous malformation are dramatic examples of disorders that may present with headache as the chief complaint. Presenting symptoms of nausea and vomiting are common with hemorrhages that arise in the posterior fossa. On routine noncontrast CT, large unruptured aneurysms may simulate other mass lesions and displace or compress adjacent structures. Arteriovenous malformations also may be conspicuous on routine CT based on abnormally enlarged feeding arteries and draining veins or internal calcifications (Fig. 1-22).

The traditional gold standard for diagnostic evaluation of vascular lesions, both spontaneous and traumatic, is DSA. The risk of major complication from this invasive procedure is low in experienced hands, and treatment (complete or partial) with endovascular techniques is possible for many types of aneurysms and other vascular lesions. That
being said, the constantly improving technology and clinical experience with CTA have led to a substantial decrease in the number of diagnostic angiograms. CTA is commonly applied in the setting of spine, facial, and skull base fractures. One study of CTA in 2004 in the setting of acute SAH reported a sensitivity of 89% and a specificity of 100% for detection of aneurysms. Dual-energy CT may provide benefits compared with standard techniques. A more recent study with 320-detector technique found overall sensitivity of 96%, specificity of 100%, and accuracy of 95%. Many centers have adopted immediate CTA in their protocol for the workup of spontaneous SAH (Fig. 1-23). If a ruptured aneurysm is detected by this method, detail may be adequate for treatment planning in certain situations. However, other investigators have offered the opinion that the gold standard of DSA (with 3D rotational angiography) provides greater sensitivity and should not yet be replaced by CTA. This opinion is based on reports of a 10% false-negative rate of CTA for aneurysm detection and the belief that the greater spatial resolution of DSA is necessary to accurately determine proper triage to surgery versus endovascular coiling. Both of these reasons support the use of DSA regardless of a negative or positive result from CTA. Given its availability and lack of invasive risks, CTA will probably continue to be used as a diagnostic tool in this setting.

In the setting of SAH, a negative result of a CTA or DSA examination often requires a repeat examination, depending on the pattern of hemorrhage. DSA offers evaluation of cerebral hemodynamics, which is important for the diagnosis of brain and dural vascular malformations. This type of detail is still not available with most current CTA techniques, but because of continued technical improvements, time-resolved (multiphase, four-dimensional) CTA is expected to be commonly available in the near future. On occasion, arteriovenous shunting may be inferred from the presence of dilated draining veins or from asymmetric opacification of the cavernous sinuses, although this shunting might just as well be due to normal physiologic variation. Evaluation of the smallest arteries is necessary for the evaluation of cerebral vasculitis. Improvements in spatial and temporal resolution and reconstruction techniques will certainly reduce the number of false-negative aneurysm hunts and increase the clinical utility of CTA for evaluation of arteriovenous malformations and vasculitis. Use of CTA in the setting of spontaneous parenchymal hemorrhage has become routine. Since the initial study of CTA in the setting of acute intracerebral hemorrhage that demonstrated that tiny enhancing foci (extravasation) within the hematoma (CTA spot sign) is an independent predictor of hematoma expansion, many other studies have evaluated this sign. An underlying vascular lesion may be found in up to 15% of patients with spontaneous ICH, potentially affecting acute management. Some authors recommend spot sign evaluation during CTP imaging. In addition to its clinical predictive value, the spot sign will continue to be of interest in trials of hemostatic therapy.

**CERVICOCEBRAL ARTERIAL INJURIES**

**Spontaneous Cervical Dissection**

Spontaneous cervical arterial dissection used to be considered a rarity, but since the 1980s, largely as a result of improvements in imaging, awareness of this cause of stroke has increased. It is estimated that up to 25% of strokes in young and middle-aged adults occur on this basis. Community-based studies have found that the annual incidence of internal carotid artery dissection is 3 per 100,000 per year, and that of vertebral artery dissection is 1.5 per 100,000 per year. A history of a trivial
Chapter 1 Traumatic and Nontraumatic Emergencies of the Brain, Head, and Neck

precipitating event, such as minor movement of the neck, is commonly reported. Chiropractic manipulation of the cervical spine has become recognized as a potential cause of dissection, but estimates of the rate of occurrence vary widely. Many different types of triggering events associated with hyperextension or rotation of the neck—including sport and recreational activities, painting of ceilings, coughing, sneezing, and vomiting—have been reported. A higher incidence in the autumn suggests inflammation or an excess amount of sneezing and coughing related to upper respiratory tract infections as predisposing factors. The effect of genetics has not yet been completely determined, but approximately half of patients show mild ultrastructural connective tissue alterations similar to Ehlers-Danlos syndrome. Traditional vascular risk factors have not been systematically studied, but atherosclerosis is generally not found in patients with spontaneous dissections. Migraine has been suggested as an independent risk factor.

**Traumatic Cervicocerebral Injuries**

The evaluation of vascular injuries of the neck and head has undergone a dramatic transformation as a result of the capabilities of helical and MDCT angiography. Since 2000, favorable results from the use of CTA in the setting of penetrating trauma have supported its clinical application on a routine basis. High sensitivity and specificity for the detection of vascular injuries have been reported in a number of studies. CTA has essentially replaced DSA, which is still the gold standard, as the initial screening modality of choice for penetrating neck injuries at many institutions. In many cases it has been used as a complementary technique to surgical exploration of the neck, but more recently, a normal CTA may avert the need for surgical exploration. CTA has been applied to the setting of blunt trauma with similar results and has resulted in a decrease in the number of DSA examinations performed. Abnormalities that may be detected include stenosis, occlusion, dissection, pseudoaneurysm formation, and contrast extravasation from vessel rupture. Vascular evaluation is generally limited to the common carotid, internal carotid, and vertebral and proximal branches of the external carotid arteries. With currently available equipment, submillimeter, subsecond imaging is possible, and even minor abnormalities of the distal external carotid branches are now being diagnosed prospectively. Nondiagnostic examinations may occur as a result of technical deficiencies such as extravascular contrast infiltration from intravenous catheters or patient motion. Use of a contrast test bolus or bolus tracking techniques helps reduce the number of poor-quality scans due to arrhythmias or compromised cardiac output. Because an intravenous contrast bolus may be impeded by transient compression of the left brachiocephalic vein as a result of pulsations of the great vessels, a right-sided antecubital injection site is preferred. Streak artifacts from dental fillings and hardware and beam-hardening effects also take their toll on image quality. Positive findings on CTA help guide therapeutic decisions toward medical, surgical, or endovascular intervention; however, well-defined pathways do not yet exist for most vascular injuries. It is the clinical expectation that with prompt diagnosis and implementation of antithrombotic or other vascular treatments, the incidence of stroke will be reduced. Because more subtle injuries will be detected with advances in CT technology, outcomes research will be necessary to help determine the most appropriate therapy. It is well known that a certain (small) percentage of patients will present with delayed formation and rupture of a posttraumatic pseudoaneurysm, yet recommendations for and timing of follow-up examination remain rather dubious (Fig. 1-24).

Dissection of the internal carotid and vertebral arteries may be the direct result of blunt trauma, with a reported incidence of less than 1% in some series. As might be expected, the risk of vascular injury in the setting of spine fracture is substantially higher. Another mechanism of carotid injury is intraoral trauma, such as from a fall with a pencil in the mouth or from iatrogenic causes. Basilar skull fracture, especially involving the carotid canal, is also included in this category in most studies of traumatic dissection. The indications for CTA continue to broaden. Because of the potential for devastating neurologic consequences of cerebrovascular injuries, some trauma centers follow a liberalized screening approach—including not only patients with symptoms referable to vascular injury but also asymptomatic patients undergoing CT for head and cervical spine trauma. In such a study reported by Biffl and colleagues in 2006, 5.4% of patients had blunt cerebrovascular injuries. In a 2-year retrospective review of patients at our center, 8% of 106 patients with fractures of the cervical spine and skull base (involving the foramen transversarium or near the carotid canal) had vascular injuries detected by CTA. Of 161 trauma patients without fractures who also underwent CTA, 2% had vascular injuries detected. Therefore the presence of a fracture yielded an odds ratio for vascular injury of approximately 4 to 1 in this population. False-positive and false-negative results are generally considered few, with the expectation that they will continue to decline as technology advances and experience with the technique increases.

Some authors propose mandatory imaging in the following instances: (1) arterial bleeding from the mouth, nose, ears, or wound; (2) expanding cerebral hematoma; (3) bruit in patients older than 50 years; (4) acute infarct; (5) unexplained neurologic defect or TIA; and (6) Horner syndrome or neck or head pain. In one angiographic study of asymptomatic patients with skull base fractures, up to 60% had abnormal angiogram findings. Other studies have reported significantly lower rates of serious injury. One recent series found a 4% incidence of traumatic carotid-cavernous fistula in patients with skull base fractures. Although prompt diagnosis and treatment of carotid-cavernous fistula are desirable, diagnosis at the time of admission is not as critical as it is for dissection with its inherent risk of stroke.

Whether performed for screening or based on symptoms, the workup and treatment of dissection continue to evolve. As with other vascular disorders, catheter angiography was once the only method available and is still used for confirmation if other studies are equivocal. The most common finding is a smoothly or mildly irregular tapered mid-cervical narrowing. Dissections that result in occlusion may show a “rat tail” or “flame-shaped” lumen, which may help distinguish other causes such as
thromboembolism or atherosclerotic disease from dissection (Fig. 1-25). Saccular or fusiform aneurysmal dilatation (pseudoaneurysm) also may be identified. The presence of an intimal flap or a false or double lumen is unusual in the cervicocerebral vessels. In the internal carotid artery, the dissection is typically found a few centimeters beyond the carotid bifurcation or a few centimeters below the skull base. The most common sites of vertebral artery dissection are at the entry into the C6 foramen transversarium and at the C1-C2 level.

Ultrasound assessment of cervical internal carotid and vertebral artery dissections is possible, but many pitfalls exist. The pathognomonic finding is a membrane in the longitudinal and axial views. In addition to the findings that may

![FIGURE 1-24 An arterial dissection resulting from blunt trauma. A, A volume-rendered image from a computed tomography (CT) angiogram shows a comminuted mandibular fracture (arrows). The internal jugular vein obscures the internal carotid artery. B, A 180-degree rotation and application of an anterior cutting plane reveal tapered contour of the internal carotid artery (arrows) as a result of dissection. C, Multiplanar reconstruction from a follow-up CT angiogram 1 week later shows development of a small pseudoaneurysm (arrow) with residual, mild, distal stenosis.](image)
be demonstrated by angiography, a thickened, hyperechoic vessel and high-resistance spectral waveform or absence of flow may be noted. However, Doppler analysis may be normal in cases of low-grade carotid stenosis, and ultrasound sensitivity of only 20% has been reported in such cases. Ultrasound evaluation of vertebral artery dissection is limited by the transverse processes of the cervical spine.

The combination of MRI and MRA is considered by many to be the preferred technique. MRA should be able to demonstrate the morphologic features of the vessel in a fashion similar to DSA. In addition, MRI may demonstrate an eccentrically located narrowed lumen, a crescentic or circumferential intramural hematoma, and an increase in the external artery diameter. Fat-suppressed T1WIs are recommended to improve sensitivity for detection of the intramural hematoma, and an increase in the eccentrically located narrowed lumen, a crescentic or circumferential intramural hematoma, and an increase in the external artery diameter. Fat-suppressed T1WIs are recommended to improve sensitivity for detection of the intramural hematoma (Fig. 1.26). However, in the acute setting, the clot should not be expected to appear hyperintense, because it may take a few days for conversion to methemoglobin to take place. The reported sensitivity and specificity for detection of carotid dissection are very high—approximately 95% and 99%, respectively. Sensitivity is lower for vertebral dissection (approximately 60%) because of the smaller size of the native vessel and the relatively high incidence of hypoplasia. One must remember that lack of flow-related signal due to slow flow in a vessel may simulate occlusion. These noninvasive techniques are very useful for follow-up of dissection as a result of the lack of ionizing radiation or the need for injection of a contrast agent.

CTA shares many of the advantages of MRA and provides higher spatial resolution. Another specific sign reported by CTA is the “target” sign, which is composed of a thickened wall and a narrowed eccentric lumen surrounded by a thin rim of contrast enhancement. CTA is certainly the fastest way to screen patients in the emergency setting and is often performed immediately after or at the time of imaging of the head and cervical spine.

CTA has also become the initial method for evaluation of stable patients with penetrating trauma to the neck, face, and head based on availability, efficiency, and similar profiles of sensitivity and specificity as in blunt trauma. The high sensitivity of CT for the detection of small amounts of contrast extravasation or air in the soft tissues helps to localize subtle injuries to major vessels that otherwise might not be noticed. An understanding of the expected trajectory of bullets or knives based on skin entry and exit wounds may increase the likelihood of the detection of injury. Detection of dissection, transection, pseudoaneurysm, and arteriovenous fistula formation can help guide treatment—that is, medical versus open surgical versus endovascular. Posttraumatic vasospasm may lead to a false-positive diagnosis of dissection or transection, appearing normal on immediate follow-up angiography. Definitive characterization of an injury in cases of segmental arterial narrowing or occlusion may be challenging in neck CTA, as well as in other areas, given the overlap in imaging findings for traumatic dissection, occlusion, and vasospasm. Retained bullets and shrapnel often cause substantial artifacts that limit the diagnostic value of CTA. DSA can usually overcome this limitation through the use of multiple oblique projections.

Spontaneous Intracranial Dissection

Intracranial arteries lack an external elastic lamina and have thinner media and adventitia compared with extracranial vessels, and thus intracranial dissections may behave differently. If the dissection occurs between the intima and media, then luminal stenosis or occlusion may lead to ischemia or infarct, similar to the extracranial setting. If the dissection plane is between the media and adventitia, then luminal stenosis or occlusion may occur. SAH is reported in approximately 20% of intracranial internal carotid dissections and more than 50% of intracranial vertebral dissections. The supraclinoid segment of the internal carotid artery and the segment of the vertebral artery near the posterior inferior cerebellar artery (V4) are the most common sites of intracranial dissection. The cause of intracranial dissection remains unknown, with some instances related to trauma or underlying connective tissue disorders (such as fibromuscular dysplasia, Marfan, and Ehlers-Danlos types). It may not be possible to differentiate traumatic stenosis or occlusion from atherosclerosis or thromboembolism by any imaging technique. The only truly diagnostic findings of dissection are the presence of a dissecting pseudoaneurysm and a double lumen.

Treatment options vary depending on stenosis versus pseudoaneurysm configuration and the presence of an infarct or SAH. Endovascular intervention with stent placement may be considered when medical therapy for stenosis has failed. Aneurysm coiling, proximal occlusion, and trapping of an abnormal segment are other possible techniques.

OTHER NONTRAUMATIC INTRACRANIAL EMERGENCIES

As with ischemic stroke and spontaneous hemorrhage, the indication for workup of other nontraumatic emergencies
affecting the brain will be based on an acute change in mental status or onset of headache, seizure, or a focal neurologic deficit. Delirium, or acute confusional state, is a common indication for brain imaging in concert with a search for other causes such as hypoxia, cerebral hypoperfusion, systemic or regional infection, intoxication, and other metabolic causes. Noncontrast head CT offers rapid noninvasive detection of lesions producing mass effect or brain edema. Spontaneous and subacute hemorrhages have been addressed in previous sections of this chapter. Other causes run the gamut of infection, inflammation, tumors, and other causes of encephalopathy. The following sections illustrate a few of the more commonly seen entities from these categories.

**Hydrocephalus**

Disturbance of the usual pattern of CSF flow or production/absorption may result in dilatation of the ventricular system. Hydrocephalus may occur acutely or may be of chronic duration, and the distinction between the two forms may not be entirely clear based on imaging alone. Hypodensities/T2-weighted hyperintensities in the periventricular white matter of the frontal and parietal regions may be a sign of interstitial edema—often described as transependymal flow of CSF—and may be seen with acute hydrocephalus. Chronically compensated hydrocephalus is less likely to demonstrate this finding. Terminology can be confusing; obstruction at the

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**FIGURE 1-26** Internal carotid artery dissection as seen on computed tomography (CT) and magnetic resonance (MR) angiogram. **A,** A CT angiogram source image shows a narrowed upper cervical right internal carotid artery lumen with an increase in the external caliber of the vessel (arrow). **B,** A fat-suppressed T1-weighted image shows corresponding narrowed flow void with a hyperintense eccentric intramural hematoma (arrow). **C,** A time-of-flight MR angiogram shows a narrowed segment of the right internal carotid artery (arrow).
level of the foramen of Monro, cerebral aqueduct of Sylvius, or foramina of Magendie and Luschka is considered noncommunicating hydrocephalus, whereas obstruction at the level of the arachnoid granulations is considered communicating hydrocephalus. One should always remember the value of prior examinations, because longstanding, compensated hydrocephalus is generally not a cause for alarm even if encountered in the emergency setting. Ventricular obstruction may develop as a result of SAH, IVH, intraventricular mass, aqueductal stenosis, and any lesion that may cause extrinsic mass effect. Analysis should include evaluation of basal cisterns and a search for possible complications that may result from herniation. Ideally, treatment of hydrocephalus will prevent such complications. Recurrence of symptoms due to ventricular shunt malfunction is a common problem that may result from catheter/tubing obstruction (intrinsic) or in the peritoneal cavity, disconnection, or migration (Fig. 1-27).

Classification is complicated further by normal pressure hydrocephalus. The cause of this disorder in which intracranial pressure is not elevated is unknown. In the setting of ventriculomegaly out of proportion to sulcal prominence, the presence of the classic triad of dementia, abnormal gait, and urinary incontinence can help to make this diagnosis. However, by the time the classic symptoms are evident, treatment may be ineffective. The fourth ventricle may be relatively spared, and periventricular edema may be absent in normal pressure hydrocephalus.

A related disorder is IIH (pseudotumor cerebri), which may be due to poor CSF absorption such as from venous sinus thrombosis or stenosis. The incidence is highest in obese females of child-bearing age, and it may present with headache and visual loss due to papilledema. Although intracranial pressure may be severely elevated in this disorder, the ventricular system is generally small. Progressive visual loss may be an indication for emergent, fluoroscopic-guided, diagnostic, and therapeutic lumbar puncture. As described in the section on cerebral sinus thrombosis, CT or MR venography is commonly requested in this setting.

Colpocephaly is the term applied to enlarged occipital horns of the lateral ventricles as a result of a developmental structural abnormality of the brain, and it sometimes may be mistaken for acute hydrocephalus. Other congenital abnormalities such as agenesis of the corpus callosum will also present with abnormal ventricular configuration and should be recognized as one of the expected constellation of findings. Benign external hydrocephalus or benign extracerebral fluid collections are two of the terms commonly applied to the pattern of mild ventriculomegaly and generous subarachnoid spaces in neonates and infants. It is associated with macrocephaly and usually resolves spontaneously by 1 to 2 years of age.

Infections

Meningitis

Suspected meningitis is a very common cause for imaging, not necessarily for diagnosis, but as a precaution prior to performance of a lumbar puncture. In adults with suspected meningitis, clinical features can be used to identify those who are unlikely to have abnormal findings on CT of the head. However, many practitioners still rely on CT prior to lumbar puncture to exclude unsuspected mass effect and lesions that might result in rapid increases in intracranial pressure; this is especially true for patients who are immunocompromised or older than 60 years. Findings of the majority of examinations are normal, but the detection of findings such as brain edema possibly leading to herniation, hydrocephalus, or other complications will alter management. On FLAIR images, sulci may appear hyperintense as a result of proteinaceous exudates; however, SAH or a high concentration of inspired oxygen also may cause the same appearance. In the vast majority of cases, meningitis is aseptic (generally of viral origin, commonly enteroviruses) and is self-limited. Bacterial meningitis is more likely to result in severe disease, and in some cases the source of meningitis may be evident on imaging, such as a sinus or ear infection. On intravenous contrast-enhanced CT and MR examinations, diffuse enhancement within the sulci (leptomeningeal) may be detected. Imaging is also indicated for patients who do not respond to antibiotic treatment in the hope of detecting a drainable source, such as a subdural empyema or parenchymal abscess. Gadolinium-enhanced MR is generally preferred to CT because of its relatively higher soft tissue contrast. Neonatal meningitis due to Citrobacter species is another indication for imaging because brain abscess develops in 80% to 90% of cases. Meningoencephalitis (encephalomenigitis) is the term applied to brain parenchymal infection in association with meningitis. Other complications of meningitis include infarcts, venous thrombosis, subdural empyema...
or hygroma, and obstructive or communicating hydrocephalus (Fig. 1-28). Subdural empyema and epidural abscess are illustrated in the section on head and neck imaging.

Many other infectious agents may cause meningitis, including Lyme disease (*Borrelia*) and, especially in the immunocompromised setting, human immunodeficiency virus (HIV), toxoplasmosis, cryptococcosis, tuberculosis, syphilis, cytomegalovirus, and a variety of fungi (see Fig. 1-12).

Diffuse thickened enhancement of the dura is a sign of pachymeningitis, which can be due to carcinomatous, granulomatous, and noninfectious causes, including idiopathic intracranial hypotension (Fig. 1-29). Tuberculosis has a predilection for causing basal meningitis with resultant stroke due to involvement of arteries at the base of the brain.

**Brain Parenchymal Infection**

The term encephalitis refers to inflammation of the brain and is usually applied in the setting of viral infection.
Cerebritis is often used interchangeably; however, it should be reserved for the cerebrum with cerebellitis added when necessary. Usually the findings are nonspecific, with simultaneous involvement of various structures. A certain pattern may suggest a particular organism, such as the classic findings of T2WI hyperintensity, restricted diffusion, and minimal enhancement within the temporal lobe(s) and limbic system as a result of herpes simplex type 1 infection (Fig. 1-30). Nonspecific patchy T2 hyperintensities, variable diffusion, and enhancement characteristics may lead to a differential diagnosis including infarction, infiltrating neoplasm, status epilepticus, and toxic/metabolic causes. Rather than identify the exact agent, imaging findings may be able to suggest infection and exclude other possibilities.

**Abscess**

An abscess is a focal parenchymal infection due to bacteria, fungi, or parasites. The imaging characteristics reflect the phase of the infection as it evolves from early cerebritis to late cerebritis, then to early and finally late encapsulated stages. If diagnosed early, it often appears as a low-density (on CT), T2-weighted hyperintense region with faint enhancement and a mild amount of surrounding edema. Once in the capsule stage, a typical fluid collection with a thin rim of enhancement and larger amount of edema becomes evident (Fig. 1-31). Over time (with treatment), the lesion may decrease in size and develop a thicker rim of enhancement, and surrounding edema will wane. The rim is typically T2 hypointense and thinner along the deep margin, which may predispose cerebral lesions to rupture into the ventricular system, leading to ventriculitis and a dramatic clinical decline. A bacterial abscess typically shows diffusion-weighted hyperintensity, which may help to distinguish a bacterial abscess from a necrotic tumor. Lesions in the differential diagnosis other than neoplasm might include demyelination, subacute infarct, and subacute hematoma. In the setting of immune suppression, a fungal abscess should be considered. In the setting of acquired immunodeficiency syndrome (AIDS), the differential diagnosis for single or multiple ring-enhancing lesions includes toxoplasmosis versus lymphoma (Fig. 1-32). Also prevalent in the AIDS population are Mycobacterium tuberculosis and Cryptococcus, which can present as either meningitis or focal parenchymal lesions. To further complicate matters, another classic presentation of cryptococcosis is that of nonenhancing, gelatinous pseudocysts that distend the perivascular spaces. Single or multiple abscesses may develop from septic emboli as a result of endocarditis. Initially, these abscesses appear similar to other cardioembolic infarcts but in time develop relatively more surrounding edema and enhancement as a result of the inflammatory response (Fig. 1-33). If cardiac valve replacement is considered, screening for mycotic aneurysms may be requested prior to surgery.

Neurocysticercosis is an intracranial infection by the pork tapeworm, Taenia solium, which is endemic in many parts of the world. It is the leading cause of seizures worldwide and has a unique life cycle and imaging characteristics. After ingestion of contaminated food or water, larvae migrate from the gastrointestinal tract to the brain and skeletal muscle. Once intracranial, the larvae develop into
cysticerci, and these cysts may be located in the subarachnoid spaces, brain parenchyma, or ventricular system. The cysts then progress through four stages with distinct imaging features based on the lesion and the host response. In the vesicular stage, the lesions appear as thin-walled, fluid-filled cysts, possibly with mild rim enhancement, but without surrounding edema. Lesions are generally small (less than 1 cm), and often an eccentric scolex of a few millimeters can be detected. In the colloidal vesicular stage, in which the larva starts degenerating, a thicker rim of enhancement and surrounding edema develop. It is in this stage that the appearance is similar to that of any other brain abscess. Next is the granular nodular stage in which the cyst involutes, the wall thickens, the lesion begins to calcify, and edema decreases. In the final nodular calcified stage, only a small calcification persists and the edema resolves completely. Because lesions may progress at different rates, it is not uncommon to find more than one type of lesion. Identification of cysts in different compartments (parenchymal, subarachnoid, and intraventricular), the presence of a scolex, variable amounts of edema, and small calcifications in the same patient offer great sensitivity and specificity. It is not often that the radiologist can show such confidence in diagnosis, and therefore...
neurocysticercosis deserves special recognition (Fig. 1-34). One might not expect this infection to be common in the United States; however, because of travel and immigration, it presents fairly commonly in patients presenting to U.S. EDs with new or recurrent seizures.

**Tumors**

As with infection, it is not possible to review all of the myriad entities in this category. Within the scope of this chapter it is possible to provide only a limited framework and describe some of the more common lesions.

Metastases account for approximately half of all intracranial neoplasms in adults. They may present as multiple small enhancing lesions at the gray–white junction or as a single large lesion with extensive surrounding edema. Lesions are usually found in the cerebral hemispheres and may be solid, cystic, calcified, or hemorrhagic, and approximately 50% will present as a solitary lesion. The most common primary tumors are lung, breast, and melanoma.

**FIGURE 1-32** Toxoplasmosis. A, A fluid-attenuated inversion recovery image shows isointense lesions in the right basal ganglia and left parietal lobe with surrounding edema. B, A postgadolinium T1-weighted image shows faint rim enhancement of right basal ganglia lesion. C, A postgadolinium T1-weighted image shows a typical “target” sign in a left frontal lobe lesion.
Calcification may imply a tumor of mucinous origin (gastrointestinal tract), and hemorrhage may indicate hypervascular primaries such as choriocarcinoma, renal, or thyroid in addition to the other more common cancers. The World Health Organization classification of primary central nervous system neoplasms is generally based on either cell type of origin or other location (e.g., sellar). Knowledge of this classification system is important for accurate communication among clinicians from different subspecialties, especially pathology, neurooncology, and neurosurgery. In addition, the radiologist should be familiar with the typical imaging characteristics, locations, and demographics of the common lesions in each category. As in any field, expertise requires effort and experience. Although it may be very satisfying to correctly identify the tumor histology (or at least narrow the differential diagnosis to a select few), the major goal in the emergency setting is to recognize subtle lesions, bring attention to those that may soon result in significant complications, and provide reassurance when intervention is not indicated. Perhaps the first step in this process should be to try to determine whether a mass is intraaxial or extraaxial. Although not always clear, this

![Image 1-33] Septic emboli. Diffusion (left), fluid-attenuated inversion recovery (middle), and postgadolinium T1-weighted (right) images show multiple small lesions with surrounding edema and faint rim enhancement.

![Image 1-34] Neurocysticercosis. A, Computed tomography (left), fluid-attenuated inversion recovery (FLAIR) (middle), and postgadolinium T1-weighted (right) images demonstrate a cyst with scolex, thin rim of enhancement, and surrounding edema in the vesicular stage. B, FLAIR (left) and postgadolinium T1-weighted (right) images showing signal loss related to calcification, a small amount of edema, and rim enhancement in the granular nodular stage.
distinction helps guide the differential diagnosis toward the proper category. The location of the lesion (supratentorial vs. infratentorial) and relation to the skull base and cisterns (e.g., anterior or middle fossa, sellar/parasellar, pineal region, cerebellopontine angle cistern, or intraventricular), along with multiplicity, are also important details. Demographics and clinical presentation may be equally important. One must also be aware that the differential diagnosis for many primary neoplasms includes metastasis, as well as infection, infarct, demyelination, inflammatory conditions, and congenital/developmental anomalies. Although space limitations preclude even the most basic description of each of the intracranial tumors, a few examples are presented to illustrate common imaging features to be recognized during the workup of patients in the emergency setting (Figs. 1-35 to 1-39).

**Disorders of White Matter**

No review of nontraumatic emergencies could be complete without mention of the following set of loosely related disorders. The common bond may be that they do

![FIGURE 1-35 Hemangioblastoma. A, Computed tomography shows a fluid density mass in the cerebellum and surrounding edema resulting in compression of the fourth ventricle. B, On a fluid-attenuated inversion recovery image, the signal is relatively hyperintense to cerebrospinal fluid, suggesting protein content. C, A postgadolinium T1-weighted image shows a small enhancing mural nodule within the cystic mass. Note dilated lateral ventricles due to obstructive hydrocephalus.](image-url)
FIGURE 1-36 Craniopharyngioma. **A,** Coronal reformat of noncontrast computed tomography (CT) shows a large cystic sellar/suprasellar mass with peripheral calcifications. **B,** A volume-rendered image shows the cystic components of the lesion as a semitransparent surface. By varying the transparency of the mass, the remodeled sella is easily demonstrated. This 5-year-old girl was sent by her ophthalmologist for urgent CT because of worsening visual acuity.

FIGURE 1-37 Central neurocytoma. Noncontrast computed tomography (**left**), fluid-attenuated inversion recovery (**middle**), and postgadolinium T1-weighted (**right**) images show a large, enhancing intraventricular mass related to the septum pellucidum causing hydrocephalus. This 22-year-old woman presented to the emergency department with worsening headaches.

FIGURE 1-38 Glioblastoma multiforme (astrocytoma World Health Organization grade IV/IV). **A,** On noncontrast computed tomography the mass cannot be clearly separated from the edema. **B,** A postgadolinium T1-weighted image shows an irregular rim of enhancement of this mass that crosses the midline via the corpus callosum. This 50-year-old man presented with altered mental status.
Multiple Sclerosis

Patients presenting with a clinically isolated syndrome (first episode of neurologic dysfunction) often undergo imaging in the emergency setting mainly to exclude infarction or other processes. White matter lesions detected by CT or MRI may raise the suspicion of a demyelinating disorder. The diagnosis of multiple sclerosis (MS) can be complex, requiring dissemination of lesions in time and space and exclusion of other causes such as Lyme disease, vasculitis, neurosarcoidosis, lupus, and others. The McDonald Diagnostic Criteria for MS imaging (originally published in 2001, with revisions in 2005 and 2010) are a standardized set of diagnostic guidelines that are applied clinically and within clinical trials. These criteria are based on identifying lesions that are disseminated in space and time on the basis of number, location (periventricular, juxtacortical, infratentorial, and spinal cord), and enhancement. Classically, hypodense/T2 hyperintense lesions are located in a periventricular distribution, in the corpus callosum (callosal-septal interface), and also in the brainstem, cerebellum, and spinal cord. Transient disruption of the blood-brain barrier will lead to solid or rim enhancement of active plaques (Fig. 1-40). Active demyelination may appear hyperintense on DWI. Magnetization transfer and MR spectroscopy techniques may demonstrate abnormalities even in white matter that appears normal. Genetic and environmental factors and female to male ratios of almost 2 to 1 (adults) and greater than 5 to 1 (children) have been found. As the name implies, the rare tumefactive variety simulates a neoplasm, often leading to biopsy. Another autoimmune-mediated disorder that is generally monophasic is acute disseminated encephalomyelitis (ADEM), and it may be indistinguishable from MS on initial imaging (Fig. 1-41). Another presumably autoimmune disorder that mimics MS and ADEM is Susac syndrome. This rare syndrome has a clinical triad of encephalopathy, branch retinal artery occlusions, and hearing loss.

Progressive Multifocal Leukoencephalopathy

In the immunocompromised host (especially due to AIDS), nonenhancing T2 hyperintensities (often confluent) in the parietal and occipital lobes should raise suspicion of progressive multifocal leukoencephalopathy (Fig. 1-42). This demyelinating process due to infection with the John Cunningham (JC) virus was originally thought to affect only white matter, but cases with both gray and white matter lesions and variable enhancement have been reported. The progressive nature of the disorder is also less certain because of current, highly active, antiretroviral therapy (HAART) regimens. Progressive multifocal leukoencephalopathy (PML) has also been reported in patients with MS or psoriasis treated with immune-modulating medications such as natalizumab or dimethyl fumarate (Tecfidera).

A related disorder is central nervous system–immune reconstitution inflammatory syndrome (CNS-IRIS). In HIV-positive patients with severe immunosuppression that responds rapidly to the start of HAART therapy, an enhanced inflammatory response may lead to worsening neurologic
symptoms. In a patient with known PML, new enhancement of FLAIR hyperintense lesions, and development of interstitial edema, mass effect and restricted diffusion would support the diagnosis of PML-IRIS. Among the organisms most commonly associated with CNS-IRIS are the JC virus and Cryptococcus. Similarly, the inflammatory response could be directed toward other infections such as HIV, Mycobacteria, Toxoplasma, cytomegalovirus (CMV), varicella-zoster virus (VZV), and Candida with varied imaging findings. CNS-IRIS may develop in the absence of an infectious agent and even at long intervals after initiation of HAART. If the inflammation responds to steroid treatment, long-term outcome may be improved.

**Posterior Reversible Encephalopathy Syndrome**

Posterior reversible encephalopathy (and seizure) syndrome (PRES) has a number of common causes such as (pre)eclampsia, hypertensive crisis, lupus and other causes of nephropathy, and drug toxicity (including immunosuppressants and erythropoietin). In the workup of
new seizures, the classic features of T2 hyperintensity in a rather symmetric distribution within the occipital, parietal, and posterior frontal lobes should bring this diagnosis to mind (Fig. 1-43). Although white matter is primarily affected, gray matter structures in the basal ganglia, brainstem, and cerebellum also may be involved. Diffusion imaging is most helpful to differentiate this process from acute infarction; ADC values will be elevated in lesions of PRES. Small amounts of hemorrhage (and infarcts) may occur within the background of vasogenic edema resulting from abnormal vascular autoregulation. Differentiation from acute or subacute stroke is important in directing the search toward an offending agent rather than initiating the stroke clinical pathway.

**Toxic Encephalopathy**

Whether due to accidental or intentional exposure/ingestion, a variety of well-known toxins may cause encephalopathy. Symmetric bilateral hypodensities/T2 hyperintensities in the globus pallidus are the hallmark of hypoxic damage due to carbon monoxide poisoning; hemispheric white matter also may be involved. Methanol toxicity typically affects the putamen and can be hemorrhagic. In addition to cerebellar degeneration, chronic alcoholism may lead to the development of Wernicke encephalopathy. It can also be the result of thiamine deficiency and a variety of other less common systemic conditions, presenting with lesions of the hypothalamus, mammillary bodies, dorsal medial thalamus, and periaqueductal gray matter.

**FIGURE 1-41** Acute disseminated encephalomyelitis. A, A fluid-attenuated inversion recovery image shows nonspecific periventricular and subcortical white matter hyperintensities. A diffusion-weighted image (B) and apparent diffusion coefficient map (C) show central areas of restricted diffusion and surrounding vasogenic edema (“T2 shine-through”). This acute demyelinating process mimics acute stroke. The distribution of the lesions is not typical of an acute infarct.
Ischemic and hemorrhagic complications related to use of illicit drugs have already been discussed. Toxic encephalopathy may also result from drug abuse. The pattern of white matter abnormality that results from inhalation of heroin vapor (known as “chasing the dragon”) can be quite dramatic (Fig. 1-44). Encephalopathy may also be iatrogenic and related to chemotherapy (e.g., methotrexate) or radiation therapy and generally affects the periventricular white matter in a diffuse fashion. Another potentially iatrogenic disorder may result from the rapid correction of hyponatremia, leading to the classic central pontine or extrapontine varieties of demyelination (myelinolysis).

**HEAD AND NECK TRAUMA**

According to statistics from the 2014 American College of Surgeons National Trauma Data Bank survey (based on more than 6 million records from the United States and Canada), injuries resulting from MVCs accounted for 27% of all cases. Other cases were due to falls (42%), being struck (7%), and gunshot wounds (4%). Bicyclists were involved in 2% of cases. The largest percentage of fatalities (almost half) were due to falls, followed by MVCs and firearms. A large percentage of the injuries involve the head, face, and neck. Diagnostic imaging plays an important role in decision making for immediate patient management, as well as surveillance for potential long-term complications. Osseous injuries including facial, skull, and skull base fractures and soft tissue injuries to orbits, vessels, the airway, and the pharynx should be considered in the routine workup performed in the settings of blunt and penetrating trauma.

Acute traumatic injuries in the head and neck region are best evaluated by CT. At our institution, we use 64-row detector helical scanners with 0.625-mm detector size. Depending on the protocol, images may be prospectively processed as contiguous 1.25-mm and 5-mm sections for review of the brain, 1.25-mm sections for the facial bones, calvarium, and cervical spine, or 0.625-mm sections of the skull base. Images are processed using both soft tissue and bone algorithms, and coronal and sagittal reformats are routinely created. Images through any region can be retrospectively processed at 0.625-mm section thickness if finer detail is necessary based on a review of the initial images. Three-dimensional volume-rendered reconstructions are often created to present an overview for referring physicians and for surgical planning. Noncontrast head CT, followed immediately by CTA of the neck and head performed with 0.625-mm contiguous sections during bolus intravenous injection of low-osmolar, nonionic contrast material, will allow for simultaneous screening for vascular injuries.

**Skull Fractures**

In the past, the workup of head trauma included skull radiographs, but those days are gone. The use of CT has become the standard of care for rapid assessment of the skull and intracranial contents. In the early days of CT, it was estimated that more than half of skull fractures were not detected because they were oriented almost parallel to the imaging plane and therefore volume averaged with adjacent normal bone. With recent decreases in section thickness and use of the multidetector-row technique, this
limitation has effectively been overcome (Fig. 1-45). Careful review of millimeter or submillimeter thickness sections and 3D volume-rendered images and familiarity with normal sutural anatomy and variants may be necessary to accurately detect fractures. The radiologic evaluation should start with the scalp, because soft tissue swelling and scalp hematoma are very helpful indicators of possible underlying fracture. Skull fractures may be linear or comminuted, and either type may be depressed. Diastasis is the term applied to separation of linear fracture fragments or sutures. Diastatic fractures occur more commonly in children. If the arachnoid membrane gets trapped within a diastatic fracture, a “growing fracture” (leptomeningeal cyst) may develop over time.

Bullet wounds may leave behind a wake of comminuted bone and metallic fragments and burst fractures at the point of exit. Identification of a fracture should lead to the dedicated search for other important findings, such as ICH. Fractures through the skull base may lead to other complications such as vascular or cranial nerve injuries or CSF leak. The presence of intracranial air should prompt the search for fracture through an aerated paranasal sinus or pneumatized portion of the temporal bone, because antibiotic therapy may be indicated for prophylaxis against

**Figure 1-44** Toxic encephalopathy. A, A fluid-attenuated inversion recovery image shows symmetric, confluent, primarily white matter hyperintensities. B, A postgadolinium T1-weighted image shows minimal enhancement. The key to the diagnosis is the association with inhalation of heroin vapor—“chasing the dragon.”

**Figure 1-45** Skull fracture. A, Nondisplaced skull fracture is occult on routine 5-mm-thick section due to volume averaging. Fracture is most conspicuous on 0.625-mm section although with an increase in noise. B, Volume-rendered image shows fracture is nearly parallel to axial imaging plane.
meningitis. One should not be alarmed by all bubbles of air, because reflux into the cavernous and other dural sinuses often occurs after insertion of intravenous catheters and injections of saline solution, medications, or contrast material with lax technique. The presence of fluid within the sphenoid sinuses, middle ear cavities, or mastoid air cells should also prompt a search for fractures through these regions. Temporal bone fractures may result in damage to the facial nerve, ossicles, and otic capsule. Screening for potential vascular injury in the setting of fracture of the central skull base may be accomplished with CTA, which was discussed in the section on cerebrovascular emergencies (see also Fig. 1-54).

Maxillofacial Fractures

MDCT has also revolutionized the workup of facial fractures. Once within the realm of plain film radiography, these injuries now are almost exclusively evaluated with CT. The general principles of fracture detection are often taken for granted but warrant quick mention. An unexpected linear lucency, the disappearance or displacement of a normal structure, and a double density from abnormal overlap of adjacent structures are classic radiographic findings of facial fractures. A cortical defect, separation of a suture, and presence of subcutaneous, orbital, or periorbital air are findings that apply equally to radiographic and CT examinations. Likewise, soft tissue swelling and fluid levels in the paranasal sinuses are equally important clues to recognize, although these clues are less specific and may occur without fractures. Whereas in the past several CT scans were necessary to evaluate the brain and face (including both thin-section axial and direct coronal scans), complete evaluation of the head and facial skeleton now can be performed with a single exposure. As an example, our 64-detector scanner offers the prospective creation of routine 5-mm-thick contiguous sections processed with a soft tissue algorithm to evaluate the brain and 0.625- or 1.25-mm-thick contiguous sections processed with a bone algorithm for evaluation of the face and skull. The thinner sections are routinely reformatted in coronal and sagittal planes, and, if desired, 3D volume-rendered images may be created at the CT console or other workstation. Depending on clinical examination findings, imaging of the head may begin at the level of the dental occlusal plane or include the chin and continue to the vertex. This method improves detection and characterization of facial fractures and reduces the amount of additional radiation exposure and scan time. Comprehension of the fracture may be improved by illustrating its relationship to normal anatomic landmarks. This technique helps the radiologist apply the most appropriate terminology or classification and it helps the surgeon to determine the need for operative reduction and fixation, usually with titanium plates and screws. Sometimes an implant is necessary to repair a large defect, as in the orbital floor.

The facial skeleton can be considered to be composed of several vertical and horizontal buttresses of thickened bone that support the functions of the face, eyes, mouth, and airway. The buttresses are linked to each other and act as attachments of the face to the calvarium and skull base. The individual components of this system and the sutures are important anatomic landmarks for the radiologist and the surgeon but are beyond the scope and space limitations of this chapter. A brief overview of common facial fractures and associated injuries follows.

Orbital Blow-Out Fractures

Isolated trauma to the orbit, a common cause of ED visits, plays a role in many cases of head trauma. Nontraumatic orbital emergencies are discussed later in this chapter. Fractures and injuries to the orbital soft tissues come in many different varieties. A blow to the eye causes an increase in intraorbital pressure, which is transmitted to the thin walls of the orbit. Blow-out fractures may involve the orbital floor, the medial wall, or both; the orbital rim should not be involved (Fig. 1-46). Even though the medial wall (lamina papyracea) is thinner, the network of ethmoid sinus septations acts as a support, and therefore
isolated orbital floor fractures are more common. In up to about half of floor fractures, medial wall fractures also occur. Orbital emphysema and herniation of orbital contents are possible associated findings. Coronal images are very useful to assess the degree of displacement of fracture fragments. Sagittal reformatted images help define the anteroposterior extent of orbital floor fractures. Fragment size and displacement help determine conservative versus operative management. Numbness of the upper cheek due to injury to the infraorbital nerve and diplopia may be presenting symptoms. Persistent diplopia and enophthalmos are possible long-term complications. Fractures that affect the orbital apex may be surgical emergencies. A bone fragment or hematoma can compress the optic nerve, leading to acute decrease in visual acuity. An afferent papillary defect should also prompt careful review of this region with bone and soft tissue algorithms and display settings. Although extraocular muscle entrapment is a clinical diagnosis, certain imaging features may offer guidance to the surgeon. If the inferior rectus muscle is displaced and does not retain its normal flattened shape on coronal images, then the fascial sling of the globe may be disrupted. Herniation of the muscle into the maxillary sinus can occur in the absence of a visible defect in the floor, implying that a “trapdoor” fragment has sprung back into place, and clearly, prompt repair will be necessary. Hematomas within the orbital soft tissues may occur in association with fractures, including retrobulbar and subperiosteal locations.

Although orbital decompression results from the blowout fracture mechanism, up to 25% have an associated ocular injury. Imaging findings can signal the need for an ophthalmologic examination that may be overlooked in the setting of multitrauma. A ruptured globe, caused by either blunt or penetrating trauma, is small, often with a flattened contour. Hemorrhage within the eye appears relatively hyperdense and can occur in several different compartments, including subretinal, subchoroidal, and subhyaloid. The distinction between these compartments can often be made based on shape and limits of the hemorrhage but is best made by physical examination. Dislocation of the lens, whether partial or complete, is not usually a subtle finding but can be easily overlooked if it is not included in the usual search pattern. Rupture of the capsule of the lens results in lens edema, appearing as a decrease in attenuation of this usually high density (proteinaceous) structure. Intraorbital and intracranial foreign bodies may be detected by CT; however, certain materials, such as wood, are better detected by ultrasound or MRI. Calcifications of the trochlear apparatus, the sclera, and optic drusen are common and should not be misinterpreted as foreign bodies. The shrunken, calcified globe (phthisis bulbi) resulting from past trauma or infection should not be mistaken for an acute injury. Likewise, intraocular silicone oil or gas and scleral buckles (both high- and low-density silicone) for treatment of retinal detachments, as well as other devices (such as aqueous shunts for treatment of glaucoma), also should be properly recognized and not become causes of concern.

Orbital roof fracture is an uncommon injury that may extend to the frontal or ethmoid sinuses. This fracture is best appreciated with coronal and sagittal reformatted images. With a “blow-in” type of fracture, as from pressure phenomenon due to a gunshot wound to the head, progressive herniation of the frontal lobe into the orbit may result from cerebral edema. A blow-out fracture of the orbital roof is an uncommon occurrence.

**Nasal Fractures**

Although nasal fractures commonly occur in isolation, they are also often associated with other fractures of the midface. Nasal fractures account for approximately half of all facial fractures. Radiographs occasionally may be requested for isolated nasal trauma, and therefore one should be aware that fractures are usually oriented perpendicular to the nasal bridge and will cross the normal nasomaxillary suture and groove for the nasociliary nerve. These fractures can be of cosmetic importance or may result in airway obstruction. Attention must be directed to the nasal septum because treatment of a septal hematoma might be necessary to avoid possible complications of ischemic necrosis, saddle deformity, and abscess formation.

As the name implies, naso-orbitoethmoid fractures are more complex and involve the nasal bones, as well as the central upper midface. Impaction of the nose and disruption of the medial canthal regions, medial orbital walls, and ethmoid sinuses will result in widening of the intercanthal distance and the need for repair. Involvement of the nasofrontal duct or nasolacrimal canal may lead to significant morbidity and also may require surgical attention to reduce the chance of frontal sinus mucocele development.

**Zygomaticomaxillary Complex Fractures**

The zygoma has four suture attachments, two to the skull and two to the maxilla. The zygoma is the second most commonly fractured facial bone. Isolated arch fractures account for a minority of zygomatic fractures and can be easily demonstrated with the traditional submentovertex view (“bucket handle”) radiograph. More commonly, 3D volume-rendered images from an MDCT dataset are used to arrive at the same impression. This injury may require repair for cosmetic reasons or because impaction of fracture fragments against the coronoid process of the mandible or temporalis muscle may result in trismus.

The more common injury to the zygoma involves the three major attachments (zygomaticomaxillary, zygomaticofrontal, and zygomaticosphenoid) and therefore has acquired the term ***tripod fracture***. The preferred term may be zygomaticomaxillary complex (ZMC) fracture because it is more aptly considered a quadriodiped fracture as a result of involvement of the posteriorly located zygomaticotemporal attachment. Specific components include the inferior orbital rim, orbital floor, and lateral wall, as well as the attachments to the skull base (sphenoid and temporal bones). Impaction and rotation of a large fracture fragment will result in significant deformity of the cheek and orbit (Fig. 1-47). Fractures of the pterygoid plates or greater wing of the sphenoid may sometimes be seen with severe ZMC fractures.
Le Fort Fractures

The Le Fort classification of midface fractures applies to separations of the maxilla from the skull base. The three-tier classification was developed by Le Fort in 1901 based on cadaveric experiments. These fractures rarely occur in their pure forms, and although originally they were described as being bilateral, common usage allows for unilateral and combined bilateral types. The Le Fort I fracture involves the maxillary antra, crossing midline above the hard palate. If it is unilateral, a sagittally oriented fracture of the palate will be present. Fractures of the pterygoid plates may occur, although some persons consider this to be a requirement by definition. With this injury, the hard palate will be allowed to separate from the remainder of the face, and it often is diagnosed by physical examination. Le Fort II fractures involve both maxillary antra and inferior orbital rims crossing at the nasion (either at the nasofrontal junction or the frontal process of the maxilla). The maxilla will be separated from the remainder of the face. In Le Fort III fractures, also termed craniofacial dissociations, the inferior orbital rims are intact but the lateral orbital walls and zygomatic arches are fractured, allowing separation of the face from the remainder of the skull. Variations and combinations of Le Fort and ZMC fractures are common (Fig. 1-48).

FIGURE 1-47 Zygomaticomaxillary complex fracture. Composite images show fractures of the left maxillary sinus anterior wall (white arrow) and posterolateral wall (double arrows), orbital floor (arrowhead), and inferior orbital rim (double arrowheads). Volume-rendered images demonstrate diastasis of the zygomaticofrontal suture (black arrow) and degree of impaction of the malar eminence.

FIGURE 1-48 Le Fort fractures. A volume-rendered image from an unrestrained driver involved in a rollover motor vehicle collision shows multiple displaced fractures. Components: red dots, Le Fort I; yellow dots, Le Fort II; and blue dots, unilateral Le Fort III. Orange dots may be considered the zygomaticomaxillary complex. The arrow shows a fracture of the mandibular symphysis.
Chapter 1 Traumatic and Nontraumatic Emergencies of the Brain, Head, and Neck

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Smash Fractures

The term facial smash is a general term applied to severely comminuted fractures of the facial bones that generally occur in association with fractures of the calvarium. ICH and traumatic brain injury occur commonly with these types of fractures. The frontal type commonly involves the anterior and posterior walls of the frontal sinus (Fig. 1-49). The naso-orbitoethmoid complex can be considered in this category. Central skull base types include fractures of the sphenoid bone. Vascular injuries related to skull base fractures are discussed in the sections on temporal bone fractures and cerebrovascular emergencies.

Mandibular Trauma

Fractures of the mandible are common and are classified by location—alveolar, symphyseal, parasymphyseal, body, angle, ramus, subcondylar (neck), or condylar. The mandible may be considered a ring structure, but with some flexibility due to the temporomandibular joints. Therefore, one, two, or more fractures may occur depending on the magnitude and direction of the applied force.

Bilateral temporomandibular joint dislocation without fracture is an uncommon injury resulting from a blow to the symphysis. The mandibular condyles should normally be seated symmetrically in the glenoid fossae. On occasion, the condyles may be anteriorly displaced, simply a reflection of normal open mouth position at the time of image acquisition. However, in a true dislocation, the glenoid fossae are empty, with condyles positioned anterior to the glenoid tubercles and the jaw locked in the open position.

If the styloid process is disrupted in the setting of mandibular fracture, air may be seen in the infratemporal fossa, possibly related to oral or Eustachian tube laceration. Mandibular fractures may be associated with hematomas within the masticator space.

Airway compromise may result from bilateral parasymphyseal fractures because the symphysis becomes a free fragment, allowing the tongue to obstruct the oral cavity. Fractures across the mandibular canal may cause injury to the inferior alveolar nerve (branch of mandibular division of the trigeminal nerve) and result in paresthesia of the chin. Fractures affecting the teeth are considered open and require antibiotic prophylaxis. Fractured or avulsed teeth can pose a risk for aspiration, particularly in the unresponsive patient. A dedicated search for radiopaque foreign bodies in the airway on all other imaging studies is therefore necessary. Muscular forces may act favorably or unfavorably, resulting in nondisplaced or displaced fractures, respectively. Displacement may occur in either the vertical or horizontal direction. Reduction with maxillomandibular fixation or open reduction and internal fixation may be required to restore occlusion and allow proper masticatory function, speech, and facial contour. As with other facial fractures, multiplanar reconstruction and 3D volume-rendered images are helpful in comprehending the injury, especially the orientation of fracture fragments.

FIGURE 1-49 Smash fracture. A, A volume-rendered image shows the complexity of a comminuted frontal smash fracture that involves the frontal sinuses and both orbits. B, A fluid-attenuated inversion recovery image months later shows residual signal abnormality involving the gyrus rectus of both frontal lobes as a result of prior contusions.
and relationships to the coronoid processes and condyles, and they aid in surgical planning (Figs. 1-50 and 1-51).

**Temporal Bone Fractures**

Temporal bone fracture should always be suspected when opacification of the mastoid air cells and/or middle ear compartments is present in the trauma setting. Slice thickness of 1 mm or less may be necessary to clearly identify and fully characterize the fracture. Temporal bone fracture usually occurs as a result of blunt head injury and is classically characterized as either longitudinally or transversely oriented relative to the long axis of the petrous bone. The other components of the temporal bone, namely, the mastoid, styloid, squamous, and tympanic, can be involved as well. Temporomandibular joint dislocation and styloid process fracture are discussed with mandibular fractures in the section on facial trauma. In practice, however, fractures through this region tend to be complex, and more typically than not they demonstrate both longitudinal and transverse fracture characteristics (Fig. 1-52). Furthermore, characterization of the fracture plane does not provide much prognostic insight. It is now recognized that it is more important to characterize whether the otic capsule (the bony housing of the inner ear structures including cochlea and semicircular canals) and internal auditory canal are involved and whether adjacent critical vascular structures (the internal carotid artery and sigmoid sinus) are at risk for injury.

Temporal bone fractures parallel to the long axis of the petrous bone are more common than transversely oriented fractures (Fig. 1-53). Fracture components can secondarily involve the external auditory canal, tympanic cavity, and squamosal portions of the temporal bone. Blood and fracture fragments in the external auditory canal and tympanic cavity and ossicular disruption can account for the common clinical presentation of conductive hearing loss. Longitudinally oriented fracture planes tend to extend anteriorly toward the Eustachian tube and middle cranial fossa, avoiding the bony labyrinth (anterior subtype). The glenoid fossa can be involved, and disruption of the middle meningeal artery can result in a concomitant epidural hematoma. Less often the fracture plane may extend posteriorly behind the bony labyrinth to involve the jugular foramen and posterior fossa (posterior subtype). In these instances, careful attention is required to evaluate for involvement of foramen lacerum or the sphenoid bone, which put the traversing internal carotid artery at risk for traumatic injury. If the fracture plane approximates or involves the carotid canal, CTA or conventional angiography can be performed to evaluate for possible internal carotid artery injury at the skull base (Fig. 1-54).

Injury to the facial nerve is not uncommon in the setting of temporal bone fractures. A careful search for fractures along the entire course of the facial nerve from the internal auditory canal to the stylomastoid foramen is necessary. Focal swelling or a hematoma developing within an otherwise intact facial nerve may manifest clinically as partial facial weakness in a delayed rather than immediate fashion. This presentation is associated with better long-term prognosis for facial nerve function because the nerve is not disrupted.

Transversely oriented fractures are perpendicular to the long axis of the petrous bone and can be further classified into medial and lateral subtypes, depending on the position of the fracture plane relative to the arcuate eminence. The medial subtype transgresses at or medial to the lateral-most aspect of the internal auditory canal, whereas the lateral subtype transgresses the bony labyrinth. As with longitudinal fractures, careful attention must be directed to the relationship of the fracture plane to the carotid canal, because injury to the internal carotid artery can be seen in this setting. Both transverse subtypes can be associated with acute and permanent sensorineural

**FIGURE 1-50 Mandibular fractures.** A, Axial and reformatted coronal computed tomography images show bilateral mandibular subcondylar fractures (arrows) and fracture of right ramus (arrowheads). B, Volume-rendered images provide a clear overview of the fractures.
hearing loss as a result of acute transection of the vestibulocochlear nerve. Facial nerve paralysis, if present, is also immediate and complete because of transection of the facial nerve.

Given the complex anatomy of the temporal bone, normal sutures and channels often can be mistaken for fracture planes. Normal petro-occipital, temporo-occipital, and occipitomastoid sutures can demonstrate irregular, coarse margins, raising concern for possible fracture (Fig. 1-55). Diastasis of sutures (equivalent to a fracture) can be seen without apparent fracture. Intrinsic sutures to the temporal bone itself, including tympanosquamous, tympanomastoid, and petrotympanic sutures, also can be confused with acute fractures (Fig. 1-56). Small channels in the middle and inner ear such as the cochlear and vestibular aqueducts, singular canal, and subarcuate fossa also can convey an appearance similar to fracture planes. An understanding of normal temporal bone anatomy is essential to avoid these errors. Symmetry, lack of opacification of mastoid air cells and middle ear compartments,
absence of overlying soft tissue swelling, and the clinical context also can provide reassurance that no fracture is present.

In addition to varied severity of facial weakness and conductive and sensorineural hearing loss, complex temporal bone fractures can have long-term complications. Squamous epithelial proliferation along fracture planes can result in cholesteatoma without concurrent mastoid disease. CSF leak can result from disruption of the epitympanic roof; this frank communication of the intracranial CSF space and middle ear can present as otorrhea if the tympanic membrane is disrupted or as rhinorrhea if the tympanic membrane is intact. Although otorrhea and rhinorrhea may resolve in the early posttraumatic setting, persistent leakage must be surgically managed because of the increased risk of meningitis. Cephalocele is also possible if the bony defect is large enough. A posttraumatic perilymphatic fistula between the inner and middle ear can occur with disruption of the oval or round windows or fractures through the otic capsule and may present with hearing loss and vertiginous symptoms. Perilymphatic fistulas are difficult to demonstrate radiographically but can be suspected based on clinical manifestations.

Airway and Pharyngeal Injuries

Acute trauma to the airway and pharynx typically occurs in the setting of penetrating trauma (stab wounds and gunshot wounds) or can be iatrogenic (as with traumatic intubation). Injury to the aerodigestive tract should be suspected in the setting of unexplained gas in the soft tissues adjacent to the airway or pharynx. It is important to keep in mind that lack of associated soft tissue gas does not exclude injury to these structures. Soft tissue gas adjacent to the aerodigestive tract could be a result of superior tracking of pneumomediastinum and pneumothorax from a penetrating chest wound or could be related to the tract of the penetrating object itself. Tracheal diverticula are often seen at the thoracic inlet level on the right and represent a common normal variant. The presence of air along the aerodigestive tract can be idiopathic or related to asthmatic pneumomediastinum. Correlation with clinical presentation is necessary to avoid unnecessary imaging studies.

If an injury is suspected, endoscopic evaluation with direct visual inspection is essential. A single contrast radiographic swallow study with a water-soluble contrast agent may be helpful in the initial evaluation of a hypopharyngeal injury; however, in a patient with potential concomitant airway injury or poor airway control, it may not be safe to perform such an examination given the risk of aspiration and possible complication of chemical pneumonitis.

Injuries to the airway may be accompanied by injury to the larynx and vocal cords. The presence of a laryngeal hematoma and vocal cord injury should be suspected if asymmetry is present in these regions when comparing right and left (Fig. 1-57). Patients with such an injury typically present with hoarseness soon after the acute event. Again, if such an injury is suspected, direct visualization by endoscopy is indicated. It should be remembered that patient phonation or phase of respiration can result in apposition of the vocal cords, creating an appearance of airway obstruction. Lack of associated soft tissue swelling and clinical correlation can help in minimizing the false-positive diagnosis of laryngeal injury in these cases.

INFECTIONS

Infections in the head and neck region are commonly seen in the ED setting. Usually the diagnosis is clear based on the physical examination and history. The role of imaging is to define the extent of infection and to provide information that is critical to immediate patient management.
Peritonsillar Abscess

Pharyngitis is typically a clinical diagnosis, but imaging may be performed upon clinical suspicion of complications. Imaging findings of tonsillar or peritonsillar soft tissue asymmetric enlargement with adjacent fat stranding may signal inflammation. A peritonsillar abscess, often seen in pediatric and immunocompromised patients, is a peripherally enhancing fluid collection formed in the potential space between the fibrous capsule of the tonsil and pharyngeal constrictor muscles. The patient typically presents with dysphagia, odynophagia, ear pain, and trismus after improvement of acute symptoms of tonsillitis. Infection extending beyond the pharyngeal constrictor muscles into the parapharyngeal space may cause mycotic aneurysms and thrombophlebitis. A discrete abscess identified by imaging may require immediate incision and drainage. Associated reactive lymphadenopathy is usually present.

Retropharyngeal Infection

Spread of pharyngeal infection to the medial or lateral (Rouvière) retropharyngeal nodes may lead to cellulitis or development of suppurrative adenitis (an enlarged, rim-enhancing
node). Associated edema of the retropharyngeal soft tissues is quite common, noted by expansion and diffusely increased density of the retropharyngeal fat. Extracapsular spread of nodal infection may result in a true retropharyngeal abscess typically identified as a rim-enhancing bow-tie-shaped fluid collection. Retropharyngeal space infections may quickly spread into the mediastinum (Fig. 1-58), which is a serious complication with high mortality and morbidity. Immediate surgical drainage of a retropharyngeal abscess may be indicated. Imaging plays an important role in determining the indication and access route for surgical drainage. Vascular complications of infection include spasm, arteritis, and mycotic aneurysm of the internal carotid artery and thrombophlebitis of the internal jugular vein (Lemierre syndrome). The latter condition was more common in the preantibiotic era, with rare cases still encountered in the present day. On postcontrast CT or MRI, a filling defect in the internal jugular vein with enhancement of the vessel wall can be demonstrated. Early diagnosis is critical, because the untreated thrombus can propagate systemically, resulting in septicemia and pulmonary emboli/infarcts.

In the setting of peritonsillar and retropharyngeal infections, careful examination of the airway is vital. Prophylactic intubation should be considered if the airway is significantly compromised.

**Prevertebral Infection/Inflammation**

Prevertebral collections in the neck typically occur in the setting of discitis and osteomyelitis of the cervical spine (Fig. 1-59). An associated prevertebral abscess can progress in size to potentially compromise the airway. In these cases, urgent intubation and surgical drainage may have to be considered. Retropharyngeal inflammation often occurs as a result of prevertebral space infection. Identification of the primary site of infection is important for appropriate patient management.

**Salivary Gland Disorders**

Inflammation of the submandibular or parotid glands demonstrated by glandular enlargement and associated surrounding fat stranding could be related to distal obstruction of Wharton or Stensen ducts, respectively, often as a result of obstructing calculus. Of the salivary glands, the submandibular gland is most prone to sialolithiasis, in part because of stasis from the descending then ascending course of its duct and the relatively more alkaline nature of its glandular secretions. Bacterial and viral infections can also result in salivary gland inflammation. Bacterial inflammation is most commonly seen in debilitated patients or neonates, caused by localized *Staphylococcus aureus* infection, and is typically unilateral. Viral inflammation occurs in children, tends
FIGURE 1-58 Retropharyngeal abscess. A, An abscess (arrow) is demonstrated within the lateral retropharyngeal node (of Rouvière) in a young patient presenting with painful swallowing. Note the associated smaller caliber of the cervical left internal carotid artery as a result of vasospasm (arrowhead), which is common in pediatric patients with an intranodal abscess. B, Sagittal reformat demonstrates associated retropharyngeal fluid extending to the C5 level (arrow). A lack of peripheral enhancement suggested that this fluid was reactive, rather than a discrete abscess. Because the patient’s condition did not improve with intravenous antibiotics, incision and drainage were required.

FIGURE 1-59 Discitis/osteomyelitis resulting from intravenous drug use. A, A sagittal fat-suppressed T2-weighted image shows abnormal signal hyperintensity in the C5-6 disk and adjacent vertebral bodies and a large associated prevertebral process (arrow). B, A postgadolinium axial T1-weighted image shows enhancement of prevertebral phlegmon (arrowhead) and circumferential epidural extension (arrow) resulting in spinal cord compression.

to be related to systemic infection, most commonly mumps, and is usually bilateral. The inflamed gland is enlarged and hyperenhancing, with stranding of adjacent fat reflecting cellulitis, or it may contain a discrete abscess (Fig. 1-60). Enlarged heterogeneous parotid glands with solid and cystic changes may reflect lymphoepithelial lesions seen in the setting of HIV disease. Similar lesions can be seen in Sjögren syndrome and other autoimmune disorders. In the chronic phase of this condition, atrophy of the parotid gland with parenchymal calcifications can be seen.

**Thyroid-Related Disorders**

When inflamed, the thyroid gland can appear diffusely enlarged or relatively hypoechoic, and it may demonstrate an associated fluid collection. Suppurative thyroiditis is
rare because of its iodine content. In such cases, pyriform sinus fistula, or third or fourth branchial pouch anomaly, should be considered as an underlying cause. Most cases involve the left lobe of the thyroid. Such patients require a barium swallow test after completion of antibiotic therapy to identify the fistulous tract.

A thyroglossal duct cyst is a remnant of the thyroglossal duct and can be found in a midline or paramidline location between the foramen cecum at the base of the tongue and the thyroid bed. The cyst is classically embedded within the strap muscles, which helps distinguish this lesion from other midline neck masses, such as dermoid and epidermoid cysts. Although thyroglossal duct cysts are found most commonly at the level of the hyoid bone, they also can be suprathyroid or infrathyroid. If the cystic lesion exhibits peripheral rim enhancement, superinfection should be suspected in the appropriate clinical setting. The cyst is usually lined by respiratory or squamous epithelium and occasionally contains thyroid tissue, which carries the potential for development of malignancy, most commonly papillary carcinoma. For this reason, any enhancing soft tissue within a thyroglossal duct cyst should be reported, and surgical excision should be strongly considered.

**Branchial Cleft Cysts**

Branchial cleft cysts are congenital epithelial-lined cystic lesions in the neck originating anywhere from the level of the mandible (first branchial cleft) to the supravacular region (fourth branchial cleft). These cysts can become superinfected and present as an acutely enlarging neck mass. The second branchial cleft cyst is most common, present in the submandibular region, anteromedial to the sternocleidomastoid muscle and anterolateral to the carotid vessels (Fig. 1-61). An infected branchial cleft cyst will demonstrate peripheral rim enhancement. Necrotic infectious and metastatic lymphadenopathy may present similarly and should be considered in the differential diagnosis, particularly in adult patients.

![Salivary gland inflammation](image)

**FIGURE 1-60** Salivary gland inflammation. 
A, In this patient with acute right facial swelling, computed tomography coronal reformat demonstrates an enlarged, hyperenhancing, right submandibular gland (arrow) with stranding of the subcutaneous fat. B, An axial image with bone window/level settings confirms a sialolith (arrow) within the gland.

![Second branchial cleft cyst](image)

**FIGURE 1-61** Second branchial cleft cyst. This second branchial cleft cyst is in the classic location, that is, the submandibular region, anteromedial to the sternocleidomastoid muscle and anterolateral to the carotid vessels.
Superficial Abscesses

Multiple bilateral superficial neck abscesses can be seen in intravenous drug abusers or “skin poppers.” Occasionally, retained fragments of needles may be present in the soft tissues. After routine operations on the neck, fluid collections may develop in the superficial or deep soft tissues. In these cases, careful evaluation of adjacent vascular structures is warranted to assess for potential injury (pseudoaneurysm, dissection, thrombosis, or occlusion) or extension of inflammation (Fig. 1-62).

Lymphadenopathy

Cervical lymphadenopathy can present clinically as diffuse neck swelling and may be related to an inflammatory process or underlying neoplasm. The presence of necrosis...
within normal-sized or enlarged cervical lymph nodes can be due to intranodal abscess formation but should raise suspicion for metastatic disease, especially from squamous cell carcinoma. If it is metastatic, the primary tumor typically arises from the base of the tongue, tonsils, nasopharynx, and pyriform sinus or from papillary thyroid cancer. This form of thyroid cancer often affects relatively young women. Certainly, necrotic adenopathy can be seen in the setting of infection such as tuberculosis and atypical mycobacterial infections such as Mycobacterium avium-intracellulare complex, particularly in immunocompromised patients. Large conglomerations of necrotic lymph nodes can be seen in these settings. Careful evaluation of the lung apices for an inflammatory process is necessary, because this finding can support the diagnosis of tuberculous adenitis. Imaging can play an important role in raising the possibility of tuberculosis in patients not suspected of having this disease. In such a case, the referring service should be notified immediately, because isolation may be required. Interestingly, calcifications related to tuberculosis are not typically found in the cervical lymph nodes.

Croup

In the pediatric patient population, laryngotracheobronchitis, or “croup,” presents clinically with a characteristic cough. The condition usually occurs in children younger than 3 years and is thought to be most often caused by parainfluenza virus, although other respiratory viruses and Mycoplasma have also been implicated. Croup is the most common pediatric infection causing stridor, accounting for approximately 15% of clinic and ED visits for pediatric respiratory infections. The cough is a consequence of subglottic edema and manifests classically on plain radiographs with symmetric, subglottic narrowing, the so-called “steeple” sign (Fig. 1-63). Coronal CT reformats can now replicate this appearance, although cross-sectional imaging is typically not necessary for this particular indication.

Epiglottitis

Epiglottitis is now less commonly seen in the pediatric ED setting as a result of childhood immunization against the offending agent, Haemophilus influenzae. However, epiglottitis can occur at any age, and recently an increase in incidence has occurred in the adult population, which is often underappreciated. Patients who have not been immunized can present with acute airway compromise and may require urgent intubation. On a lateral radiograph, the classic “thumb” (or “thumbprint”) sign is produced by the thickened, inﬂamed epiglottis (Fig. 1-64). The clinical picture and radiographic imaging are usually so characteristic that cross-sectional imaging is generally unnecessary. CT imaging can provide greater diagnostic detail and can demonstrate the inﬂamed epiglottis along with symmetrically thickened and inﬂamed aryepiglottic and pharyngoepiglottic folds. However, management should be based on clinical and radiographic findings because obtaining a CT examination might delay proper treatment.

Angioedema

Although angioedema is not due to infection, it is included in this section on airway disorders. It can be hereditary or acquired and may be idiopathic, manifesting as acute narrowing of the airway, possibly resulting in airway compromise. Angioedema due to allergic reactions is often associated with urticaria. Although clinical diagnosis should be straightforward, CT imaging can be useful for further evaluation. Imaging can show diffuse mucosal and submucosal swelling involving the hypopharynx and larynx, with infiltration of the subcutaneous fat and deep tissue planes of the neck.

Sinus and Orbital Infections

Uncomplicated acute sinusitis is typically clinically diagnosed and managed. On imaging, findings of acute sinusitis are not very specific. An air–fluid level within a sinus can suggest acute sinusitis; however, lack of this finding does not mean that a patient does not have acute sinusitis. Occasionally, hyperdense opaciﬁcation in the paranasal sinuses can be seen; the hyperdensity can reﬂect hemorrhage related to trauma, trapped proteinaceous debris, or fungal infection. Fungal infection is an important diagnostic consideration, because steroids probably should be incorporated into the treatment regimen if the cause is related to allergic disease (Fig. 1-65).

When advanced sinus disease is of concern, imaging plays a more deﬁnitive role. Complicated sinus disease should be clinically suspected if the patient presents with visual changes, altered mental status, or seizures. Initial imaging for workup of complicated sinusitis can begin with CT. At our institution, axial images through the facial bones are routinely acquired at 1.25-mm intervals with coronal and sagittal reformats using bone and soft tissue algorithms. If

![FIGURE 1-63 Croup. In this patient who presented acutely with a barking cough, a frontal radiograph of the neck demonstrates the classic “steeple” sign (arrow) representing concentric subglottic edema.](image)
concern exists regarding intracranial or orbital extension of infection, contrast-enhanced CT using similar imaging parameters can provide additional critical information. If clinical concern exists regarding intracranial extension, the entire brain should be imaged. Because complicated sinus infection usually warrants close follow-up imaging, MRI is the preferred imaging modality to minimize radiation exposure, especially in the pediatric setting. Typical brain MRI protocol includes sagittal T1-weighted, axial FLAIR, fat-suppressed T2-weighted, gradient echo, and diffusion-weighted images, followed by multiplanar postcontrast T1WIs of the brain. If orbital extension is of clinical concern, high spatial resolution T2WIs with and without fat suppression through the orbits and axial and coronal pre- and postcontrast T1WIs can provide excellent diagnostic detail.

CT is particularly useful in establishing the integrity of paranasal sinus walls and can effectively demonstrate areas of bony dehiscence. Intracranial or orbital spread of infection can occur directly through areas of bony dehiscence or indirectly by perivascular extension, most commonly as a result of frontal or ethmoid sinus disease. Perivascular spread more commonly occurs in the pediatric setting. Intracranial extension is usually seen in the setting of frontal sinus infection, with involvement of the anterior cranial fossa and frontal lobes (Fig. 1-66). Imaging can demonstrate associated dural enhancement, epidural or subdural collections, and meningoencephalitis with abscess formation. The patient may have concomitant swelling of the overlying soft tissues of the forehead, known as Pott’s puffy tumor (Fig. 1-67). This finding does not imply neoplastic involvement but rather describes osteomyelitis with extracranial soft tissue abscess formation. The presence of pachymeningeal enhancement does not necessarily imply that brain parenchyma is involved and may only reflect dural reaction. Epidural and subdural collections can be

FIGURE 1-64 Epiglottitis. In this young child who presented with difficulty breathing, a lateral neck radiograph demonstrates the classic “thumbprint” sign (arrow) of the inflamed epiglottis.

FIGURE 1-65 Sinusitis. A, Mucosal thickening in the left maxillary sinus is accompanied by an air–fluid level due to acute sinusitis in this patient presenting with sinus pressure, pain, and headache. B, In a different patient, coronal reformat in soft tissue window/level settings demonstrates hyperdense material in most of the paranasal sinuses as a result of allergic fungal sinusitis.
FIGURE 1-66 Sinusitis—subdural empyema and infarct. A, An axial T2-weighted image shows proteinaceous material within the frontal sinuses. B, A postgadolinium coronal T1-weighted image demonstrates an associated subdural abscess and leptomeningeal enhancement.

FIGURE 1-67 Sinusitis—Pott’s puffy tumor and epidural abscess. A, An axial computed tomography image shows opacification of the frontal sinuses and overlying soft tissue swelling. A tract from the anterior table of the frontal sinus (arrow) and adjacent fluid collection (arrowhead) are shown. B, An axial T2-weighted image demonstrates abnormal hyperintensity of marrow in the frontal bone (arrowheads) and overlying soft tissue swelling, including small scalp fluid collection (arrow). C, A postgadolinium sagittal T1-weighted image shows an epidural abscess (arrow) and fluid-filled paranasal sinuses with inflamed mucosa (arrowhead).
demonstrated on either CT or MRI as extraaxial fluid, possibly compressing the subjacent brain parenchyma. Subdural collections can cross sutures and generally maintain a crescentic shape, whereas epidural collections are restricted by sutures and may have a biconvex shape. Whereas a reactive subdural effusion may be associated with smooth pachymeningeal enhancement, an infected subdural or epidural collection can demonstrate restricted diffusion and thickened, more irregular dural enhancement on MRI. Leptomeningeal involvement is demonstrated as curvilinear enhancement extending into sulci. This manifestation may be accompanied by FLAIR hyperintensity in a corresponding distribution, which is a sensitive but nonspecific finding. Parenchymal involvement can be demonstrated by FLAIR hyperintensity and enhancement. Frank abscess formation can be demonstrated by CT or MRI as a peripherally enhancing fluid collection. Restricted diffusion in the collection can support this impression.

Although posterior ethmoid disease can also lead to intracranial involvement, more commonly, untreated ethmoid sinusitis can extend into the medial aspect of the orbit (Fig. 1-68). Infection can spread in a subperiosteal...
fashion along the medial wall of the orbit, appearing as an elliptical phlegmon or fluid collection, and may cause displacement of extraocular muscles. Without treatment, the phlegmon or abscess can then break through the periosteum and extend directly into the orbit and into the extraocular muscles. Depending on the size of the inflammatory process, displacement of extraocular muscles and the globe can occur, along with distortion, stretching, and compression of the optic nerve. Such findings constitute an ophthalmologic emergency, because prolonged mass effect on the optic nerve can result in permanent blindness.

Other possible critical complications of orbital infection include extension of infection and involvement of the contents of the orbital apex (CN II), superior orbital fissure (CNs III, IV, V1, and VI and the superior ophthalmic vein), and inferior orbital fissure (V2, infraorbital nerve). Idiopathic inflammation involving the cavernous sinus, known as Tolosa-Hunt syndrome, can manifest clinically as palsies of the cranial nerves that traverse the cavernous sinus, namely, CNs III, IV, V1, V2, and VI.

Venous sinus thrombosis can result from ethmoid or sphenoid sinus or orbital infection, and careful attention to the cavernous sinuses on postcontrast images is necessary. Facial soft tissue, dental, and ear infections are other possible causes. Direct signs of septic cavernous sinus thrombosis, also termed cavernous sinus thrombophlebitis (CST), include an enlarged sinus with a convex border and a single large filling defect or multiple irregular filling defects. Normal neural structures and intracavernous fat deposits should not be mistaken as true filling defects. Indirect signs include dilation of the superior ophthalmic vein, exophthalmos, soft tissue edema, thrombi in the superior ophthalmic vein, superior and inferior petrosal, or sigmoid sinuses, and a decrease in the caliber of the internal carotid artery. Thin-section, contrast-enhanced CT with coronal reformats may be equivalent to MRI for the detection of these findings. However, once venous sinus thrombosis is suspected, MRI of the brain may confirm the diagnosis and evaluate for possible complications of meningitis, subdural empyema, cerebritis, and even pituitary necrosis. Postcontrast T1-weighted coronal and axial images are generally sufficient to make the diagnosis. Contrast-enhanced MR venography also may offer support in diagnosis. (Phase contrast MR venography is generally limited for this evaluation. Contrast-enhanced CT [CT venography] and MRI with thin slices are generally more helpful to diagnose CST.) Prior to the antibiotic era, CST was almost always fatal. In the modern era, mortality in the range of 20% to 30% may still be expected. In addition to antibiotics, early institution of anticoagulant therapy may reduce mortality. Morbidity may result from cranial nerve dysfunction including blindness, pituitary insufficiency, and hemiparesis.

**Ear Infections**

Much like uncomplicated paranasal sinus disease, external and middle ear infections can be diagnosed and followed up clinically. Occasionally acute and chronic otomastoiditis can be picked up on imaging and should be reported, because these conditions may not be suspected clinically (Fig. 1-69). If clinical concern exists regarding a more complicated infectious process, CT and MRI can be very useful adjuncts in diagnosis. CT imaging of the temporal bone includes the entire auditory system. At our institution this imaging is performed with overlapping 0.625-mm-thick sections with coronal and sagittal reformats in bone and soft tissue algorithms. If clinical concern exists regarding intracranial

![FIGURE 1-69 Mastoiditis. A, Opacification of mastoid air cells without bony changes in a patient with symptoms of acute mastoiditis. B, Opacification of the middle ear with sclerotic changes of the mastoid bone in a patient with a history of chronic otomastoiditis.](image-url)
involvement, contrast-enhanced CT using the same imaging parameters can provide additional diagnostic detail. MRI can be particularly useful in the setting of cranial nerve involvement. Our technique includes high spatial resolution T2WI and pre- and postcontrast axial and coronal images from the level of the orbits to the base of the brain, in addition to routine brain imaging.

Malignant otitis externa, which is typically seen in diabetic and immunocompromised patients, is a rare but serious complication of external ear infection. The inflammation can quickly progress to involve the entire ear, external auditory canal, and middle and inner ear structures. Evaluation of the stylomastoid foramen for extension of inflammation with fat stranding is important, particularly in patients with facial nerve symptoms. Associated osteomyelitis is seen as a locally aggressive destructive process of the temporal bone. Urgent treatment is indicated, usually with surgical debridement, because intracranial extension of infection can develop rapidly. Cranial nerve involvement from untreated middle ear infection can occur secondarily by spread of inflammation into the cavernous sinus, typically involving CN V within Meckel’s cave and CN VI within the cavernous sinus (Gradenigo syndrome) (Fig. 1-70). Additional cranial nerve involvement can quickly ensue. Intracranial extension can result from dehiscence of the temporal bone, with direct spread into the middle cranial fossa. As with complicated paranasal sinus infection, extension into the middle cranial fossa can result in epidural abscess, subdural empyema, meningoencephalitis, or brain abscess. Transverse sinus,
sigmoid sinus, and jugular vein thrombosis are other possible complications (Fig. 1-71).

**Complicated Dental Disease**

Periapical lucencies around individually infected teeth are often incidentally detected on routine CT images of the mandible and maxilla performed for other reasons. Occasionally the infection can progress to the medullary bone, eventually breaking through the overlying buccal or lingual cortex. In these cases, this sinus tract acts as a conduit for spread of infection into the adjacent soft tissues (Fig. 1-72). With time, an abscess can develop, and the patient may present to the ED with pain, swelling, and an elevated white blood cell count. Fluid collections in the face therefore should be carefully evaluated for a possible dental source, because the soft tissue infection will not be cured by drainage alone. Rapid progression of infection into other spaces, including the orbit and intracranial compartment, is a serious and potentially life-threatening complication. Ludwig angina is characterized by extensive facial swelling and hardened cellulitis centered in the submandibular space, classically bilateral, related to periodontal disease (Fig. 1-73).

In the case of a suspected dental-related facial abscess, maxillofacial CT with thin (1.25-mm) axial sections and coronal
and sagittal reformats in bone and soft tissue algorithms should be performed. Intravenous contrast-enhanced examination increases the sensitivity for detection of an abscess based on peripheral rim enhancement. CT is particularly useful in identifying areas of cortical disruption of the mandible or maxilla, whereas MRI may identify the fluid collection but could easily miss the source of the infection, namely, the affected tooth, because of artifacts inherent to MRI.

**SUMMARY: HEAD AND NECK**

A wide variety of disease may present as emergencies in the head and neck region. Although many conditions can be diagnosed and managed clinically, imaging plays an important role in diagnosis and management when more complicated disease and acute traumatic injuries are encountered. A clear understanding of the history and physical findings based on communication between the radiologist and referring clinician will guide selection of the most appropriate imaging modality and protocol. As with all other parts of the body, a working knowledge of the anatomy and variants, as well as the disorders specific to the region, is necessary to provide excellent patient care.

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**SUGGESTED READINGS: BRAIN**


A comprehensive list of references related to traumatic and nontraumatic emergencies of the brain, head, and neck is provided.
Suggested Readings: Head and Neck


Hopfer RA, Salemy S, Sze RW. Diagnosis of midface fractures with CT: what the surgeon needs to know. Radiographics. 2006;26:783–793.