Radiologists face the daily challenge of analyzing and interpreting a high volume of images in a timely manner. Minimizing errors, whether perceptual or cognitive in nature, is paramount for high-quality diagnostics and patient care. There are certain areas within the head encountered at routine brain imaging in which the interpreting radiologist is most prone to make perceptual errors. These areas, or “blind spots,” include the cerebral sulci, dural sinuses, orbits, cavernous sinuses, clivus, Meckel cave, brainstem, skull base, and parapharyngeal soft tissues. In addition, the use of an inappropriate window width and level for the evaluation of computed tomographic (CT) scans can be a virtual, rather than an anatomic, blind spot. The inclusion of a comprehensive checklist for evaluation of these blind spots as part of every brain imaging study is crucial for avoiding false-negative results. Knowledge of the anatomic features of these blind spots is also crucial, as well as familiarity with the normal CT and magnetic resonance imaging findings in these areas. In addition, the radiologist should be aware of possible interpretation pitfalls that may lead to false-positive results (eg, normal anatomic variants that may be mistaken for pathologic conditions). Finally, a well-developed differential diagnosis will help ensure correct interpretation and appropriate patient treatment.

**Abbreviations:** CSF = cerebrospinal fluid, FLAIR = fluid-attenuated inversion recovery, HIV = human immunodeficiency virus, ICA = internal carotid artery, SCC = squamous cell carcinoma

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Introduction

As the volume of cross-sectional imaging studies continues to increase, radiologists are faced with mounting challenges to comprehensive and accurate image interpretation. As with most other anatomic imaging, interpretation of routine brain imaging studies is predisposed to its own unique set of commonly missed pathologic conditions. In this article, we refer to the anatomic sites within the head that are most likely to harbor missed disease entities as “blind spots.” Pathologic findings are commonly missed in the cerebral sulci, dural sinuses, orbits, cavernous sinuses, clivus, Meckel cave, brainstem, skull base, and parapharyngeal soft tissues. We have found that, at our institution, missed diagnoses occur most frequently in the skull base, followed (in order of decreasing frequency) by the dural sinuses, Meckel cave, cavernous sinuses, parapharyngeal soft tissues, clivus, orbits, brainstem (particularly at computed tomography [CT]), and sulci.

Errors in interpretation can generally be categorized as either perceptual or cognitive in nature (1). Perceptual errors are those in which the radiologist does not see the abnormality, resulting in a false-negative interpretation. Cognitive errors, on the other hand, are those in which the abnormality is identified but the meaning or significance of the abnormality is not recognized. Cognitive errors can result in false-positive interpretation if, for example, a normal anatomic variant is mistaken for a pathologic condition. Conversely, they can result in true-positive interpretation but with misclassification if the radiologist arrives at the wrong conclusion or diagnosis due to misleading information, a lack of knowledge, or a limited differential diagnosis (2).

In the evaluation of the majority of blind spots described in this article, radiologists are prone to perceptual errors.

The reasons for false-negative interpretations vary greatly. Radiologists may miss pathologic conditions in blind spots due to incomplete or misleading clinical history, high study volume, prioritization of imaging findings on the basis of the patient’s clinical presentation, inappropriate or incomplete imaging protocols, or simple failure to continue the search following identification of an abnormality (ie, “satisfaction of search”).

In this article, we discuss and illustrate various blind spots within the head in terms of normal anatomy, potential interpretation pitfalls, and differential diagnosis. In addition, we discuss window width and level with respect to CT as a virtual, rather than anatomic, blind spot. We also present a comprehensive checklist that will help decrease the likelihood of interpretation errors at routine brain imaging.

Anatomic Sites

Cerebral Sulci

Normal Anatomy.—The cerebral sulci are grooves or infoldings on the surface of the brain that separate the gyri. During gestation, the sylvian fissure is the first fetal sulcus to appear (16–20 weeks), followed by the calcarine, parieto-occipital, and cingulate sulci at 20–22 weeks; the rolandic, interparietal, and superior temporal sulci by 25 weeks; and the precentral, postcentral, superior frontal, and middle temporal sulci at 24–28 weeks (3). The sulci are important neuroanatomic landmarks that define critical anatomy. For example, the rolandic sulcus, also known as the central sulcus, separates the primary motor cortex anteriorly from the primary sensory cortex posteriorly.

The pia mater adheres tightly to the brain and spinal cord and contains a network of blood vessels. It is separated from the overlying arachnoidea mater by the subarachnoid space, which contains cerebrospinal fluid (CSF) (4). The pia mater, subarachnoid space, and arachnoidea mater are indistinguishable at magnetic resonance (MR) imaging. Therefore, radiologists visually evaluate the character of the sulci together with the signal intensity of the subarachnoid space. The CSF within the sulci should always follow the signal intensity of water, which is hyperintense on T2-weighted MR images and fluid-attenuated inversion recovery (FLAIR) images.

Interpretation Pitfalls.—Because sulcal size varies with age, radiologists can sometimes run into problems with interpretation. The premature infant brain can be mistaken for lissencephaly if the postconception age of the child is unknown (3). In children aged 8–24 months, enlarged sulci and subarachnoid spaces with transiently enlarged head circumference out of proportion to other growth charting can be seen in idiopathic external hydrocephalus, a physiologic process
that could be mistaken for pathologic extraaxial fluid collections (5). Conversely, disease can be present even when the sulci appear normal, as in pseudotumor cerebri, in which the sulci are normal to slightly decreased in size despite increased intracranial pressure.

**Differential Diagnosis.**—The sulci are sensitive and important indicators of intracranial disease in that changes in brain volume are reflected in the size of the sulci. For example, in the setting of diffuse cerebral edema, the cerebral sulci are grossly narrowed, whereas in the setting of atrophy, the sulci are enlarged. With asymmetric sulcal narrowing, it must be determined if the sulci are narrowed due to local mass effect or because there is proteinaceous, infectious, or cellular material filling the sulci.

The location of sulcal narrowing can also help the radiologist make the correct diagnosis. For example, widening of the medial frontal lobe sulci, temporal lobe sulci, and sylvian fissures is seen in the neurodegenerative disease known as frontotemporal dementia. Enlarged sulci in the cerebellum indicate cerebellar atrophy from a number of causes, including chronic alcohol use, antiepileptic therapy, hereditary spinocerebellar ataxia, and paraneoplastic syndrome. Focal sulcal enlargement could indicate encephalomalacia from underlying old cortical infarct, whereas focal sulcal effacement could indicate inflammation or edema of the underlying brain due to acute infarction or cerebritis.

In addition, hyperattenuating sulci at nonenhanced CT can indicate blood products within the adjacent subarachnoid space or chronic meningitis. At FLAIR imaging, hyperintensity within the sulci can indicate subarachnoid hemorrhage, proteinaceous material, infectious meningitis, carcinomatous meningitis, or sequelae of supplemental oxygen therapy (6,7). Contrast material–enhanced imaging is indicated when abnormal hyperintensity is seen within the sulci at FLAIR imaging. Contrast-enhanced T1-weighted imaging will show abnormal enhancement, a finding that helps confirm leptomeningeal disease, or lack of enhancement from causes such as blood, supplemental oxygen therapy, or increased protein (Fig 1).
Dural Sinuses

**Normal Anatomy.**—The dura mater, the outermost layer of the meninges, is composed of two distinct layers. The outer layer is tightly bound to the skull and also serves as the periostium of the inner table of the skull. The inner layer separates from the skull in specific areas to form the falx cerebri, tentorium cerebelli, diaphragma sellae, and falx cerebelli. The spaces formed between the layers of the dura mater serve as the dural venous sinuses, which receive blood from the superficial and deep cerebral venous systems.

The superior sagittal sinus forms at the root of the falx cerebri. The inferior sagittal sinus runs along the inferior free edge of the falx cerebri. The straight sinus lies between the falx cerebri, tentorium cerebelli, and falx cerebelli. It drains the inferior sagittal sinus as well as the deep venous system (internal cerebral veins and the vein of Galen) into the torcular Herophili. The torcular Herophili, or the confluence of sinuses, is located just below the occipital protuberance. It also receives blood from the superior sagittal sinus and the occipital sinus, an infrequently visualized sinus that forms at the root of the falx cerebelli. Blood then drains into the right and left transverse sinuses, which lie at the root of the tentorium cerebelli.

The transverse sinuses also receive blood from the superior petrosal sinuses, which run anteriorly along the petrous portion of the temporal bones. The sigmoid sinuses are the continuation of the transverse sinuses distal to the superior petrosal sinuses. At the jugular foramina, the sigmoid sinuses form the jugular bulbs (4).

**Interpretation Pitfalls.**—The dural sinuses are often difficult to evaluate without the intravenous administration of contrast material. Routine imaging protocols are usually not tailored to evaluate the venous structures. Thus, disease involving these sinuses can easily be missed, especially at routine nonenhanced brain CT.

However, radiologists can use several clues to help identify possible abnormalities within the dural sinuses. The sinuses are blood-filled structures and can be evaluated on the basis of their attenuation and morphologic features at CT. Increased attenuation may suggest thrombosis or hemoconcentration (Fig 2). Adjacent prominent vessels resulting in hyperattenuation outside the sinus are suggestive of dural arteriovenous fistulas. Complications of dural sinus thrombosis such as infarctions or hemorrhages within the brain that either do not conform to a specific arterial territory, are bilateral, or spare the cortex should also raise suspicion for dural sinus abnormalities.

At MR imaging, the dural sinuses should demonstrate normal flow voids. An acute thrombus in the sinus will be isointense at T1-weighted imaging, whereas a subacute thrombus will be hyperintense. However, care should be taken not to mistake slow-flowing venous blood, which can appear hyperintense at T1-weighted imaging, for thrombosis. A diagnostic clue to differentiating between these two entities is that thrombus will appear hyperintense in all imaging planes, whereas flow-related hyperintensity should persist only in vessels that are perpendicular to the imaging plane. T2-weighted images and gradient-echo images can be used together to determine if hypointensity within the sinus is due to acute thrombus (dark on both types of images) or flowing blood (dark on T2-weighted images and bright on gradient-echo images). Subacute thrombus will be hyperintense at T2-weighted imaging (4). One area that is especially susceptible to variable signal intensity at MR imaging is the sigmoid sinus near the jugular foramen. Contrast-enhanced imaging is required to accurately evaluate this area.

Time-of-flight MR angiographic images often allow only limited evaluation of the dural sinuses. Methemoglobin in subacute thrombus can appear hyperintense on these images, just as flowing blood would. Phase-contrast angiography is more likely to completely saturate out the thrombus and show only flowing blood. Furthermore, complete flow gaps can be seen in more than 30% of healthy patients at phase-contrast MR angiography, most often within a nondominant transverse sinus (8).

Ideally, intravenous contrast-enhanced time-of-flight MR venography or CT venography should be used to determine the presence of dural sinus thrombosis. With these modalities, a flow gap or filling defect within the dural sinus or failure of the entire sinus to enhance are suggestive of sinus thrombosis. A filling defect within the triangular superior sagittal sinus on axial images has been termed the empty delta sign. Traditionally, invasive angiographic procedures...
Figure 2. Dural sinus thrombosis in a 28-year-old man who presented with headache. The patient had no significant past medical history. (a) Routine axial nonenhanced brain CT scan shows asymmetric increased attenuation in the right transverse sinus (arrow). (b) Coronal contrast-enhanced T1-weighted MR image shows filling defects in the superior sagittal sinus ("empty delta sign") (top arrow) and right transverse sinus (bottom arrow), findings that are consistent with dural sinus thrombosis.

Differential Diagnosis.—The most common abnormality involving the dural sinuses is thrombosis. When sinus thrombosis is being considered as a diagnosis, information from the patient's clinical history can be very helpful. This information includes risk factors such as hypercoagulable state, dehydration, pregnancy, oral contraceptive use, recent onset of headache, trauma, malignancy, malnutrition, or the presence of indwelling catheters. Imaging findings can also help in making the diagnosis and may include an adjacent infectious process (epidural abscess, mastoiditis, meningitis), neoplasm (meningioma compressing the sinus), the development of dural arteriovenous fistulas or collateral vessels (eg, medullary veins), or, as mentioned earlier, infarction or parenchymal hemorrhage.

However, it is important to include several other possibilities in the differential diagnosis. For example, a subdural hematoma or empyema adjacent to a dural sinus can mimic clot within the sinus. In addition, as described previously, arachnoid granulations and hypoplastic sinuses can mimic thrombosis.
Orbits

Normal Anatomy.—The osseous orbit is made up of many bones. The roof is formed by the orbital process of the frontal bone; the lateral wall, by the orbital surface of the zygomatic bone and the greater wing of the sphenoid bone; the floor, by the maxillary process of the zygomatic bone and the thin orbital plate of the maxillary bone; and the medial wall, by the frontal process of the maxillary bone, the lacrimal bone, the lamina papryacea of the ethmoid bone, and the lesser wing of the sphenoid bone (11).

Intraorbital contents include the globe, extraocular muscles, optic nerve and sheath complex, fat, arteries, nerves, veins, and the nasolacrimal apparatus including the lacrimal gland. The globes are round and symmetric. The ocular lens separates the smaller anterior segment, which includes the anterior and posterior chambers, from the larger posterior segment. Vitreous fluid filling the anterior and posterior chambers follows the signal intensity of water and is hyperintense on T2-weighted MR images and hypointense on T1-weighted images. The extraocular muscles demonstrate homogeneous enhancement on contrast-enhanced T1-weighted images and together form a “cone” extending from the globe to the orbital apex.

The optic nerve sheath complex is made up of the optic nerve and the surrounding pia mater, the subarachnoid CSF space, the arachnoidea mater, and the dural sheath. On coronal T2-weighted MR images, the components of the optic nerve sheath complex are easily visualized. Contrast-enhanced T1-weighted images demonstrate no enhancement of the optic nerve or dural sheath unless disease is present. Retrobulbar intraconal fat is present, and distortion of this fat can indicate infectious, inflammatory, or malignant changes within the orbit. The major artery supplying the orbit is the ophthalmic artery, which arises from the supraclinoid segment of the internal carotid artery (ICA). The superior ophthalmic vein courses between the optic nerve and the superior rectus muscle and drains into the cavernous sinus. The inferior ophthalmic vein lies adjacent to the inferior rectus muscle and also drains into the cavernous sinus.

The major sensory nerve innervating this area is the ophthalmic division of cranial nerve V, which carries sensation from the globe, lacrimal gland, conjunctiva, and eyelid (11). The major motor nerves include cranial nerve III, which innervates the medial rectus, superior rectus, inferior rectus, inferior oblique, and levator palpebrae muscles; cranial nerve IV, which innervates the superior oblique muscle; and cranial nerve VI, which innervates the lateral rectus muscle. The lacrimal gland is an exocrine secretory gland innervated by the greater superficial petrosal nerve, a branch of cranial nerve VII that is instrumental in tear production.

Interpretation Pitfalls.—Given the complexity of the orbit, an MR imaging protocol with a reduced field of view and pre- and postcontrast sequences is necessary for thorough evaluation. At MR imaging, distortion of the magnetic field can occur from paramagnetic cosmetics on the patient’s eyelid. Some iatrogenic causes of orbital disease can create confusion, such as intraocular gas injection for repair of retinal detachment if the clinical history is unknown (12). Lack of pertinent clinical history and high study
volume can lead to inattention to the orbits at routine brain CT, especially when the radiologist is trying to exclude life-threatening or time-sensitive imaging findings (Fig 3).

**Differential Diagnosis.**—When orbital disease is being considered, it is best to first consider problems that affect inherent structures. Diseases affecting the osseous orbit include dermoid and epidermoid cysts that occur near the sutures. Metastatic disease, fibrous dysplasia, traumatic fractures, Langerhans cell histiocytosis, and congenital abnormalities such as sphenoid wing dysplasia in neurofibromatosis type I can also affect the osseous orbit. Diseases affecting the extraocular muscles include rhabdomyosarcoma, thyroid orbitopathy, and idiopathic orbital inflammatory disease, also known as orbital pseudotumor.

Diseases affecting the optic nerve sheath complex include meningoia, perineuritis, glioma, optic neuritis, sarcoid tumor, and leukemia. Prominence of the subarachnoid space within the optic nerve sheath complex with or without flattening or indentation of the posterior globes is the radiologic equivalent of papilledema and implies increased intracranial pressure (13). Infections extending into the orbit include mucormycosis, aspergillosis, and postseptal peri orbital cellulites with or without abscess formation. Invading tumors or tumors associated with the lacrimal gland include benign mixed tumor (also known as pleomorphic adenoma), basal cell carcinoma, squamous cell carcinoma (SCC), melanoma, lymphoma, and minor salivary gland tumors.

Sjögren syndrome can also affect the lacrimal gland. In addition to lacrimal gland disease, other intraorbital masses include cavernous hemangioma, capillary hemangioma, lymphangioma, and varices. Intraorbital fat can be infiltrated by breast cancer metastases, which can cause fibrosis leading to enophthalmos. Other disease entities that can extend into the orbit and result in enophthalmos include mucoceles, encephalocoeles, sinonasal polyposis, and en plaque meningiomas of the greater wing of the sphenoid bone (14,15).

**Cavernous Sinuses**

**Normal Anatomy.**—The cavernous sinuses are multiseptate, extradural venous spaces that lie on either side of the sella turcica. Each multichannel venous structure completely encases the cavernous segment of the ICA. The cavernous sinuses communicate with each other via the intercavernous sinuses and receive drainage from the superior and inferior ophthalmic veins. Numerous other venous connections are made with the periclavicular venous plexus, superior petrosal sinus, inferior petrosal sinus, and pterygoid venous plexus (16).

Cranial nerve III (within the oculomotor cistern), cranial nerve IV, the ophthalmic division of cranial nerve V (V1), and the maxillary division of cranial nerve V (V2) course superoinferiorly within the lateral dural wall. Cranial nerve VI is medially located within the cavernous sinus and courses closest to the cavernous segment of the ICA. At MR imaging, flow voids indicate patency of the ICA and are best visualized on T2-weighted images. At contrast-enhanced MR imaging, the cavernous sinuses enhance homogeneously, similar to the medially adjacent pituitary gland. There is a normal slight concavity to the lateral border of the cavernous sinus where the medial margin of the temporal pole abuts the sinus.

**Interpretation Pitfalls.**—The cavernous sinus is poorly evaluated without intravenous contrast material. Routine brain imaging is inadequate for viewing this small structure, and imaging with a reduced field of view as part of a dedicated orbit or sella turcica protocol is necessary for thorough evaluation. Oftentimes, a clinical history of cavernous sinus syndrome is what prompts the radiologist to make a more thorough search of this region (17). Without an appropriate clinical history to instigate further dedicated imaging, disease harbored in this blind spot will often go unnoticed.

**Differential Diagnosis.**—As expected, disease arises from structures in the cavernous sinus, with causes including but not limited to those of vascular, neural, and meningeal origin. Narrowed flow voids can indicate external compressive disease such as meningioma (Fig 4), lymphoma, or Tolossa-Hunt syndrome. However, the carotid artery can also appear narrowed and asymmetric in the setting of high-grade stenosis of the proximal cervical ICA. In addition, physiologic caliber asymmetries exist when there are circle of Willis variants, usually when a dominant ICA feeds not only the ipsilateral middle cerebral artery but also the bilateral anterior cerebral arteries via a patent

**Teaching Point**
Figure 4. Meningioma in a 45-year-old woman who presented with vision loss. (a) Time-of-flight MR angiogram of the brain obtained as part of a routine stroke protocol shows narrowing of the left cavernous and suprachiasmatic ICA segments (arrow). Persistent vision loss prompted further evaluation with dedicated contrast-enhanced orbital MR imaging. (b) Axial contrast-enhanced T1-weighted MR image shows abnormal convexity of the left cavernous sinus (arrow), the dural tail anterior to the left temporal pole (white arrowhead), and asymmetric decreased caliber of the left cavernous carotid artery (black arrowhead), findings that are consistent with a meningioma.

Anterior communicating artery due to an aplastic contralateral A1 segment (18). Enlarged fusiform or saccular flow voids within the cavernous sinus can represent aneurysms (Fig 5). Loss of flow voids can indicate arterial thrombosis and is best visualized on T2-weighted images (Fig 6).

Arterial thrombosis can occur from embolic sources, atherosclerotic disease, or dissection. Anomalous arteriovenous connections can occur in carotid cavernous fistulas.

Lack of enhancement of the cavernous sinus on contrast-enhanced T1-weighted MR images indicates sinus thrombosis. This pathologic condition can occur with coagulopathy or have infectious or malignant causes. In the immunocompromised patient with invasive fungal infection in the orbit or paranasal sinuses, careful inspection of the cavernous sinus for nonenhancing intracavernous material representing intracranial extension of disease is important for prognosis and treatment planning. Tumors can invade the cavernous sinus by extension, including pituitary adenoma, germinoma, craniopharyngioma, or metastases. Sarcoidosis can also involve the cavernous sinus.

Schwannoma and neurofibroma originate from neural structures and can occur along cranial nerves. In addition, perineural spread of tumor, especially lymphoma, SCC, adenoid cystic carcinoma, and mucoepidermoid carcinoma, can enter the cavernous sinus.

Clivus

Normal Anatomy.—Two bones, the basisphenoid and basioccipital bones, form the clivus. The clivus is a portion of the posterior cranial fossa and is the posterior limit of the middle cranial fossa. The sella turcica lies just anterosuperior to the clivus, and the foramina lacera lie just laterally on either side of the clivus. Posteriorly, the clivus slopes downward to the foramen magnum (19).

The clivus is best evaluated on sagittal T1-weighted MR images, which allow assessment of its marrow signal intensity and its contour. It is
Figure 5. Aneurysms in a 67-year-old woman who presented with headache. Routine MR imaging of the brain was performed after initial nonenhanced brain CT performed at another institution showed normal findings. (a) Axial T2-weighted MR image shows a bilateral increase in the number of flow voids in the cavernous sinuses (arrows). (b) Follow-up time-of-flight MR angiogram of the brain obtained as part of a stroke protocol for subsequent symptoms of transient ischemic attack shows bilateral large saccular aneurysms (arrows) arising from the cavernous segments of the ICAs.

Figure 6. Stenosis in a 58-year-old man who presented with symptoms of transient ischemic attack. (a) Axial T2-weighted MR image shows a flow void (arrow) in the left cavernous ICA segment. (b) Time-of-flight MR angiogram of the brain shows no flow in the left ICA or left middle cerebral artery (arrow), a finding that is consistent with chronic occlusion, as well as a flow void in the A1 segment of the left anterior cerebral artery, a finding that indicates high-grade stenosis.

Also important to evaluate the alignment of the clivus with the dens of the axis at the craniocervical junction. Traditionally, on conventional radiographs, a line is drawn along the posterior cortex of the dens and extended cranially. The distance between this line and the clivus should be less than 12 mm. At CT and MR imaging, this measurement must be made on an exact midline sagittal image to be reliable. The basion-dens distance between the inferior tip of the clivus and
mal marrow can demonstrate small amounts of enhancement, whereas tumor deposits within the clivus typically demonstrate avid enhancement. Reconversion of yellow marrow to red marrow can sometimes be difficult to distinguish from neoplastic involvement (21).

In addition to the signal intensity of clival marrow, the contour and margins of the clivus can help indicate tumor involvement, especially when there is bone erosion. Care must be taken to also evaluate nearby structures such as the cavernous sinus and cranial nerves for tumor involvement (22).

**Differential Diagnosis.**—Clival lesions can be benign or malignant. Benign abnormalities include fibrous dysplasia, marrow reconversion, pituitary macroadenoma with invasive features, meningioma, plasmacytoma, osteomyelitis, and Langerhans cell histiocytosis. Malignant lesions include metastases, nasopharyngeal carcinoma, chordoma, chondrosarcoma, multiple myeloma, and lymphoma (Fig 7). Lymphomatous involvement of the clivus is rarely primary in nature (22).

Atlanto-occipital dislocations are usually seen in the setting of trauma due to severe hyperextension and distraction. Associated brainstem and cranial nerve injury is usually present. On the other hand, atlanto-occipital subluxation can be seen with nontraumatic conditions such as rheumatoid arthritis or Down syndrome.

**Interpretation Pitfalls.**—The clivus as well as the calvaria are often overlooked in the interpretation of brain images. In some cases, the clivus can be the only site of abnormality, significantly affecting patient treatment if correctly identified. The most common abnormalities of the clivus are from neoplastic, hematologic, or traumatic processes. Identification of neoplastic involvement of or traumatic injury to the clivus is critical because they can be devastating for the patient if missed.

At MR imaging, the normal adult clival marrow can occasionally demonstrate foci of hypointensity on T1-weighted images, but these areas are usually iso- or hyperintense relative to the pons and contain foci of T1 hyperintensity (ie, normal yellow marrow). This normal finding must be distinguished from abnormal marrow signal intensity, such as with tumor infiltration, which is hypointense relative to the pons on T1-weighted images. Furthermore, normal marrow is usually isointense relative to the pons on T2-weighted images, whereas tumor involvement of the marrow is hyperintense relative to the pons. Following contrast material administration, normal marrow can demonstrate small amounts of enhancement, whereas tumor deposits within the clivus typically demonstrate avid enhancement.
Meckel Cave

Normal Anatomy.—The trigeminal nerve (cranial nerve V) exits the lateral pons and courses anteriorly through the prepontine cistern, over the petrous apex, and into the Meckel cave, where the sensory roots synapse in the gasserian ganglion. Cranial nerve V then trifurcates into V1, the ophthalmic division, which enters the orbit through the superior orbital fissure; V2, the maxillary division, which exits through the foramen rotundum; and V3, the mandibular division, which exits through the foramen ovale.

The Meckel cave is a dura-lined invagination into the posterolateral aspect of the cavernous sinus and is filled with CSF. The gasserian ganglion, also known as the trigeminal or semilunar ganglion, lies in the inferolateral portion of the Meckel cave. Just medial to the Meckel cave is the ICA within the cavernous sinus. The first two divisions of cranial nerve V, V1 and V2, exit the Meckel cave and course along the lateral wall of the cavernous sinus (4,19).

Interpretation Pitfalls.—The Meckel cave should follow CSF signal intensity with all MR imaging sequences (ie, hypointense on T1-weighted images and hyperintense on T2-weighted images) (23). At high-resolution postcontrast T1-weighted imaging, the only enhancing structures within the Meckel cave are the gasserian ganglion, which does not fill the entire cavity, and the dural lining, which should be thin (4). However, unless attention is specifically directed to this area, loss of this normal signal intensity is very easy to miss. Unfortunately, many of the lesions that infiltrate the Meckel cave are neoplastic, and a clinical history of focal trigeminal neuropathy is not always provided to direct the radiologist’s search for such disease.

The loss of normal CSF signal intensity within the Meckel cave can be due to intrinsic or extrinsic causes. Mass lesions arising from the sphenoid bone in the skull base can erode into this cavity and obliterate the CSF space. Similarly, mass lesions arising from the structures within the Meckel cave can fill the cavity. However, certain lesions such as schwannomas can have high signal intensity similar to that of CSF on T2-weighted images, thereby mimicking CSF. In such cases, it is important to look for secondary signs of mass lesions such as expansion or erosion of the cavity and increased nerve root caliber. In addition, such lesions manifest as contrast enhancement filling the cavity while the normal Meckel cave remains hypointense. If a lesion in this area spreads along the nerve roots, it can also expand or erode the exit routes, such as the superior orbital fissure, foramen rotundum, and foramen ovale (23).

Once disease is identified or suspected within the Meckel cave, the next step is to identify the neural ramifications. For example, sequelae of cranial nerve V denervation may be evident. The muscles of mastication, which are innervated by the motor roots of cranial nerve V (exiting through the foramen ovale), will appear hyperintense on T2-weighted images and can demonstrate contrast enhancement in the acute setting. In the setting of a chronic lesion, these muscles will be atrophic with hyperintense fatty infiltration on T1-weighted images (24).

Differential Diagnosis.—Lesions that infiltrate the Meckel cave commonly represent metastatic disease. Metastatic lesions can either be deposited hematogenously to the bones of the skull base, which then grow into this cavity, or spread to the Meckel cave via a perineural route (Fig 8). Meningeal and CSF seeding are also possible routes of metastatic infiltration of the Meckel cave. Of the divisions of cranial nerve V, V3 is the most common route of perineural tumor spread.

Vascular lesions can also cause imaging abnormalities in the region of the Meckel cave. For example, carotid artery aneurysms and arteriovenous malformations can extend into this adjacent cavity. Other mass lesions in the Meckel cave include trigeminal schwannomas, meningiomas, neurofibromas, neurosarcoma, pseudotumor, pituitary macroadenoma, epidermoid and dermoid cysts,
and lipomas. Infectious processes such as meningitis, neurocysticercosis, and herpetic neuritis can also involve the Meckel cave (23).

**Brainstem**

**Normal Anatomy.—**The brainstem arises embryologically from the mesencephalon (midbrain), metencephalon (pons and cerebellum), and myelencephalon (medulla). The midbrain is divided into the tegmentum anteriorly and the tectum posteriorly, with the cerebral aqueduct in between. The cerebral peduncles are located anteriorly. The CSF space immediately around the midbrain is called the mesencephalic cistern and contains the interpeduncular fossa and the ambient cisterns, which wrap around the peduncles laterally. The tectum contains the superior and inferior colliculi, which form the quadrigeminal plate. The CSF space posterior to the colliculi is the quadrigeminal plate cistern. Cranial nerves III and IV originate in the midbrain. The superior cerebellar peduncles are the white matter tracts that connect the midbrain to the cerebellum.

The pons has a characteristic rounded shape anteriorly that is well seen on sagittal images. Cranial nerves V–VIII arise from the pons. Cranial nerves VII and VIII course anterolaterally in the cerebellopontine angle cistern to the internal auditory canal. The CSF space around the anterior pons is referred to as the prepontine cistern. The middle cerebellar peduncles connect the pons to the cerebellum and border the fourth ventricle.

The medulla is the site of origin of cranial nerves IX–XII. The pyramidal tracts are located anteriorly, and the olivary nuclei are located anterolaterally. Posterolaterally, the inferior cerebellar peduncles connect the medulla to the cerebellum. The medulla extends to the level of the foramen magnum (4).
Interpretation Pitfalls.—The brainstem is best evaluated at MR imaging, which allows visualization of anatomic detail such as white matter–gray matter differentiation. More important, however, unlike CT, MR imaging is not limited by beam-hardening artifact in the evaluation of the posterior fossa. The density of the bone at the skull base obscures much of the detail of the brainstem and cerebellum at CT. Subtle pathologic changes in the density of these structures can easily be masked by areas of hyper- or hypoattenuation caused by attenuation artifact or beam-hardening artifact. Adjustment of the window width and level may demonstrate areas of abnormal density that do not coincide with the pattern of beam-hardening artifact (Fig 9).

Evaluation of the brainstem with CT is highly dependent on indirect signs of disease. For example, mass effect has to be inferred from the results of evaluation of the basal cisterns for symmetry. Processes such as mass lesions or edema due to infarction may not be directly visible. Therefore, subtle clues such as effacement of the mesencephalic cistern should prompt further evaluation with MR imaging to determine the cause (Fig 10). If a mass lesion is suspected at CT, it is essential that contrast-enhanced MR imaging be performed for further evaluation (25). Moreover, pseudolesions such as the flocculus, a portion of the cerebellum that protrudes into the cerebellopontine angle, can mimic disease at CT and may prompt work-up in a healthy patient (4).
Figure 10. Left midbrain lesion in a 38-year-old HIV-positive man with toxoplasmosis who presented with altered mental status. (a) Routine axial nonenhanced brain CT scan shows subtle hypoattenuation in the midbrain with effacement of the left ambient cistern (arrows) due to enlargement of the left crus cerebri, findings that could easily be missed. (b) Axial FLAIR image shows marked vasogenic edema (arrow) due to an underlying lesion in the left midbrain. The lesion showed ring enhancement on contrast-enhanced images.

Differential Diagnosis.—Mass effect within the brainstem may be due to (a) vascular causes such as infarction, resulting in edema, hemorrhage, or vascular malformations; (b) neoplastic lesions such as gliomas or metastatic lesions; (c) traumatic injury with contusions or diffuse axonal injury; (d) infectious causes such as encephalitis, pyogenic abscesses, neurocysticercosis, or tuberculosis; or (e) infiltrating diseases such as sarcoidosis or Langerhans cell histiocytosis.

Certain lesions of the brainstem may result in little mass effect while changing the density of the parenchyma as their most prominent feature. Such lesions include small infarctions, progressive multifocal leukoencephalopathy, and demyelinating diseases such as multiple sclerosis or acute disseminated encephalomyelitis. Prominent perivascular spaces (often at the base of the cerebral peduncles) may also be visible at CT and should not be mistaken for disease.

Atrophy of portions of the brainstem can signify disease in other areas of the brain, such as with wallerian degeneration of the corticospinal tracts in the cerebral peduncles in the setting of an infarction involving the motor cortex (26). A prior infarction within the brainstem may also result in visible loss of tissue due to encephalomalacia. Certain neurodegenerative diseases such as progressive supranuclear palsy also manifest as brainstem atrophy.

Mass lesions and hemorrhage can be seen in the cisternal spaces around the brainstem. Focal lesions include meningiomas and schwannomas, particularly in the cerebellopontine angle (Fig 11); epidermoid cysts; arachnoid cysts; vascular aneurysms; lesions arising from the skull base such as chordoma, chondrosarcoma, or plasmacytoma; and metastatic lesions. Inflammatory or neoplastic infiltration of the basal meninges can also obliterate the basal cisterns.

Skull Base

Normal Anatomy.—Knowledge of basic anatomy is crucial to the evaluation of the skull base, which consists of five bones and apertures that transmit nerves, arteries, and veins. The five skull base bones include the occipital, temporal, sphenoid, frontal, and ethmoid bones. Table 1 lists the major skull base foramina and their transmitted contents (11).

Interpretation Pitfalls.—With current high-resolution multidetector CT technology and the ability to reformat images in all orthogonal views, the skull base foramina are visible. Occasional
Figure 11. Trigeminal schwannoma in a 45-year-old woman who presented with vague complaints of facial pain. (a) Routine axial nonenhanced brain CT scan shows an isodensity mass (arrows) in the preoptic cistern that obliterates the left cerebellopontine cistern, a subtle finding that can be difficult to identify. (b) On an axial contrast-enhanced T1-weighted MR image, the mass (arrow) is well marginated and is seen in the expected location of the fifth cranial nerve as it exits the lateral pons. The mass proved to be a trigeminal schwannoma.

Table 1
Skull Base Foramina and Their Transmitted Contents

<table>
<thead>
<tr>
<th>Aperture</th>
<th>Contents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optic canal</td>
<td>CN II</td>
</tr>
<tr>
<td>Superior orbital fissure</td>
<td>CN III, IV, V1, VI; superior ophthalmic vein</td>
</tr>
<tr>
<td>Foramen rotundum</td>
<td>CN V2</td>
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<tr>
<td>Foramen ovale</td>
<td>CN V3</td>
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<tr>
<td>Foramen spinosum</td>
<td>Middle meningeal artery</td>
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<tr>
<td>Foramen lacerum</td>
<td>Meningeal branches of the ascending pharyngeal artery</td>
</tr>
<tr>
<td>Vidian canal</td>
<td>Vidian artery and nerve</td>
</tr>
<tr>
<td>Internal auditory canal</td>
<td>CN VII, VIII</td>
</tr>
<tr>
<td>Jugular foramen pars nervosa</td>
<td>CN IX</td>
</tr>
<tr>
<td>Jugular foramen pars vascularis</td>
<td>CN X, XI; internal jugular vein</td>
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<tr>
<td>Carotid canal</td>
<td>ICA</td>
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<tr>
<td>Stylomastoid foramen</td>
<td>CN VII</td>
</tr>
<tr>
<td>Hypoglossal canal</td>
<td>CN XII</td>
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<tr>
<td>Foramen magnum</td>
<td>Medulla</td>
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</table>

Note.—CN = cranial nerve.

variants occur, such as absence of the foramen spinosum when there is a persistent stapedial artery (27). Fibrous dysplasia enlarges the diploic marrow space and can lead to foraminal narrowing with consequent clinical neuropathy. Errors are usually made due to lack of attention to or knowledge of normal anatomy. Evaluating the skull base with bone windows as well as soft-tissue windows is mandatory.
Differential Diagnosis.—Myriad diseases can arise from inherent bone, nerve, or vascular structures or can be hematogenously deposited as metastatic disease. Although it is common knowledge that the posterior fossa is less well seen at CT than at MR imaging, careful attention should be paid to all structures when clinical symptoms are referable to this area (eg, vertigo, ataxia, hearing loss, nausea) with use of both soft-tissue and bone windows (Fig 12). Bone lesions can include entities such as traumatic fracture, sclerotic or lytic metastases, cartilaginous or osseous tumors, and fibrous dysplasia. Vascular lesions include pseudoaneurysms and arteriovenous malformations. Neural diseases can include schwannomas, neurofibromatosis, and plexiform neurofibromas. Other tumors include glomus tumors, lymphoma, melanoma, and minor salivary gland tumors. Infections include entities such as mastoid disease, tuberculosis, petrous apicitis, cholesterol granuloma, and cholesteatoma.

Parapharyngeal Soft Tissues

Normal Anatomy.—Routine brain imaging often incidentally includes portions of the neck. Consequently, knowledge of the anatomy of the parapharyngeal soft tissues and spaces is crucial in the identification of disease that may arise in these areas.

The nasal cavity and nasopharynx, the oropharynx and oral cavity, and often, portions of the hypopharynx (the space between the epiglottis and the pharyngoesophageal junction) are visible at brain CT and MR imaging. These mucosal spaces should appear symmetric, a finding that is particularly important for the pharyngeal recess (fossa of Rosenmuller), which is located just posterior to the torus tubarius in the nasopharynx.

Surrounding the mucosal spaces of the head and neck are the parapharyngeal soft tissues, which are divided by fascial planes into discrete spaces: the parapharyngeal space (normally containing fat), carotid space, masticator space, retropharyngeal space, and perivertebral space. In addition, the salivary glands (usually the parotid glands) can be at least partially visualized at routine brain imaging.

Lesions can be localized to one of the deep spaces of the neck if the anatomic relationship between the spaces is understood. For example, a lesion in the masticator space displaces the fat in the parapharyngeal space posteromedially. A lesion arising from the deep lobe of the parotid gland displaces the parapharyngeal fat anteromedially. Anterior displacement of the fat...
is suggestive of a carotid space lesion. Posterior displacement of the carotid space (indicated by the position of the carotid artery and internal jugular vein) or parapharyngeal fat completely surrounding a lesion implies a parapharyngeal space lesion. Posterolateral displacement of the parapharyngeal fat indicates a mucosal space lesion, whereas anterolateral displacement suggests a retropharyngeal space lesion (19).

The lymph nodes in the neck have been categorized into seven regions or levels. Of these, levels I and II and portions of levels III and V may be seen at brain imaging. Level I denotes submental and submandibular lymph nodes. Along the jugulodigastric chain, level II nodes are located above the hyoid bone and level III nodes are located between the hyoid bone and the cricoid cartilage. The jugulodigastric level II lymph node (node of Rouviere) is located next to the posterior belly of the digastric muscle. It is important to distinguish this node from the muscle tissue that extends anteromedially to ultimately form the anterior belly of the digastric muscle. Level V nodes are posterior to the posterior border of the sternocleidomastoid muscle (4).

**Interpretation Pitfalls.**—The most likely reason that disease in the parapharyngeal soft tissues is missed at brain imaging is simply the radiologist’s failure to look at these areas. As mentioned earlier, in the neck, the presence of asymmetry is the key to identifying disease. For example, in the pharyngeal mucosal space at the level of the torus tubarius and the fossa of Rosenmuller is the nasopharynx, which can harbor nasopharyngeal carcinoma that may only be suggested by slight asymmetry of these structures. Similarly, at the floor of the mouth, subtle asymmetry of the muscles and fat planes can suggest disease such as SCC or floor-of-mouth abscess.

Adenoidal tissue in the high nasopharynx is another potential indicator of disease. Normally, the adenoids regress by 30 years of age. Lymphoma, infection, and human immunodeficiency virus (HIV) can cause abnormal hypertrophy of the adenoids that can easily be missed (Fig 7). Similarly, lymphadenopathy may at times be the only imaging abnormality and could signify occult neoplasm, as with the node of Rouviere (Fig 13).

**Differential Diagnosis.**—Neoplasms arising from the mucosal spaces of the head and neck are usually SCC. In turn, lymphadenopathy in the neck of an adult is most often due to metastatic spread of SCC. However, other differential considerations in lymphadenopathy include reactive nodes, lymphoma, supplicative lymph nodes, and metastatic disease from other primary neoplasms such as thyroid malignancies.

Other neoplasms in the incidentally imaged portions of the head and neck may arise from the parotid gland (pleomorphic adenoma, Warthin tumor, mucoepidermoid carcinoma), neural tissue (paraganglioma, neurofibroma, schwannoma), muscles (rhabdomyosarcoma), and nasopharynx (juvenile nasopharyngeal angiofibroma). Congenital lesions include Tornwaldt cysts, ectopic thyroid tissue, hemangiomas, lymphangiomas, vascular malformations, dermoid cysts, branchial cleft cysts, and thyroglossal duct cysts. Inflammatory
Figure 14. Arteriovenous malformation in a 25-year-old woman who presented with headache. (a) Routine axial nonenhanced brain CT scan shows subtle clustered hyperattenuating areas in the right parietal lobe (arrow). (b) On the same image viewed with a different window width and level, the clustered areas (arrow) are hyperattenuating relative to the surrounding parenchyma. (c) Axial contrast-enhanced T1-weighted MR image shows a tangle of engorged vessels with a prominent draining vein (arrow), findings that are consistent with an arteriovenous malformation.

lesions include abscess, adenitis, Castleman disease, lymphoepithelial lesions of the parotid gland, sialadenitis, and odontogenic disease.

Window Width and Level

Normal Anatomy
The human eye can differentiate only a limited number of shades of gray (28). At digital CT, one can designate which part of an image matrix appears white and which part black. The numeric range between white and black that one establishes is termed the window width. The center of this numeric range is called the window level. Standard windows such as soft-tissue, bone, lung, liver, subdural, and brain windows are often preprogrammed into picture archiving and communication systems for ease of use when volumes of CT scans are being viewed. Radiologists routinely view the same volume of images with multiple windows to increase the conspicuity of disease. Institutional norms may vary, but at our institution our preprogrammed window width/level settings are as follows: normal brain, 80/35; bone, 1500/450; subdural, 31/35; and soft tissue, 426/55.

Interpretation Pitfalls
Problems can arise when the radiologist makes a window too narrow or too wide, thereby losing valuable diagnostic information. With the high volume of images that can be created with multidetector CT, quick-working radiologists can fail to look at each study with a variety of windows, consequently missing important pathologic findings. For example, small congenital lipomas along the interhemispheric falx, which are commonly seen at routine nonenhanced brain CT, could be

Table 2
Comprehensive Interpretation Checklist for Routine Brain Imaging

<table>
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<th>Clinical history</th>
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<td>Sulci*</td>
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<tr>
<td>Ventricles</td>
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<tr>
<td>Midline structures</td>
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<tr>
<td>Parenchymal symmetry (adjust window width and level*)</td>
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<tr>
<td>Basal cisterns</td>
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<tr>
<td>Cavernous sinus*</td>
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<tr>
<td>Meckel cave*</td>
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<td>Dural sinuses*</td>
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<tr>
<td>Symmetry and density of posterior fossa</td>
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<td>Bones (bone window)</td>
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<tr>
<td>Skull base foramina*</td>
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<td>Clivus*</td>
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Note.—Contrast-enhanced images should be acquired if infection or malignancy is being considered. *Potential blind spot.
brain imaging in which the interpreting radiologist is most prone to make perceptual errors. In other words, these are the areas that are most likely to be overlooked if the radiologist uses a targeted approach in reviewing brain images. These areas include the sulci, dural sinuses, orbits, cavernous sinuses, clivus, Meckel cave, brainstem (at CT), skull base, and parapharyngeal soft tissues. In addition, the use of an inappropriate window width and level for the evaluation of CT scans can be problematic.

The crucial first step in avoiding false-negative results is remembering to evaluate these blind spots with use of a comprehensive checklist for every brain imaging study (Table 2). Knowledge of the anatomy in these areas is also crucial, as is an understanding of the normal

Differential Diagnosis
Varying the window width and level can improve detection of parenchymal disease (Fig 14). It can also increase the conspicuity of dense intracranial arteries in the setting of hyperacute stroke from intraarterial thrombus (29). In the setting of acute stroke, varying the window width and level manually can increase the radiologist’s detection rate at nonenhanced brain CT (Fig 15) (30).

Conclusions
The blind spots we have reviewed in this article are areas within the head encountered at routine brain imaging in which the interpreting radiologist is most prone to make perceptual errors. In other words, these are the areas that are most likely to be overlooked if the radiologist uses a targeted approach in reviewing brain images.
imaging findings seen at CT and with different MR imaging sequences. In addition, in evaluating these blind spots, it is important to be aware of possible interpretation pitfalls that may lead to false-positive results (eg, normal anatomic variants that may be mistaken for pathologic conditions). Finally, a well-developed differential diagnosis will help ensure correct interpretation and appropriate patient treatment.

References

Blind Spots at Brain Imaging

Simin Bahrami, MD and Catherine M. Yim, MD

Errors in interpretation can generally be categorized as either perceptual or cognitive in nature. Perceptual errors are those in which the radiologist does not see the abnormality, resulting in a false-negative interpretation. Cognitive errors, on the other hand, are those in which the abnormality is identified but the meaning or significance of the abnormality is not recognized.

At FLAIR imaging, hyperintensity within the sulci can indicate subarachnoid hemorrhage, proteinaceous material, infectious meningitis, carcinomatous meningitis, or sequelae of supplemental oxygen therapy. Contrast material--enhanced imaging is indicated when abnormal hyperintensity is seen within the sulci at FLAIR imaging.

The cavernous sinus is poorly evaluated without intravenous contrast material. Routine brain imaging is inadequate for viewing this small structure, and imaging with a reduced field of view as part of a dedicated orbit or sella turcica protocol is necessary for thorough evaluation.

The density of the bone at the skull base obscures much of the detail of the brainstem and cerebellum at CT. Subtle pathologic changes in the density of these structures can easily be masked by areas of hyper- or hypointensity caused by attenuation artifact or beam-hardening artifact.

The crucial first step in avoiding false-negative results is remembering to evaluate these blind spots with use of a comprehensive checklist for every brain imaging study (Table 2).

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