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Cerebrovascular disease, with cerebral ischemia or infarction, is the most common disease affecting the brain. It is also the most common neurologic disease seen by the radiologist in daily practice. Cerebrovascular disease is an important health care problem, particularly in the older patient population. In the United States, it is the third leading cause of death after cancer and myocardial infarction. Half of the affected patients will have permanent neurologic deficits. There are more than 2 million survivors of cerebral infarction. Magnetic resonance imaging (MRI) is the modality that most completely characterizes cerebrovascular disease. MRI provides information regarding pathophysiology, anatomic location, and vascular patency.
CLINICAL CONSIDERATIONS

Stroke is a general term describing an acute neurologic insult, with a resulting permanent deficit, caused by a disease of the blood vessels. The term cerebrovascular accident (CVA) is synonymous with stroke. The clinical presentation of patients with stroke is variable and non-specific. Patients with a ruptured aneurysm, subdural hematoma, or hemorrhage into a tumor can present with stroke-like symptoms similar to those of cerebral ischemia or infarction. The role of the radiologist is to determine the cause of the symptoms in the individual patient.

In patients with ischemia or infarction, there is often a disparity between pathophysiology (the derangement of function seen in the disease) and clinical manifestations. Therefore, the accurate use of terminology describing ischemic disease is important. Cerebral ischemia and infarction describe pathophysiologic processes. Cerebral ischemia describes global or regional reduction of blood flow to the brain. Cerebral infarction occurs when the reduction of blood flow causes irreversible cellular damage (i.e., cell death). The clinical terminology describing ischemic neurologic events is based on clinical presentation and evolution. A transient ischemic attack (TIA) is a transient loss of neurologic function that resolves in 24 hours. Reversible ischemic neurologic deficit (RIND) indicates loss of neurologic function that resolves within 21 days. A progressing stroke or stroke in evolution describes a changing neurologic state. A completed stroke indicates a permanent and fixed neurologic deficit. A patient with cerebral infarction may present with any of these clinical states, even though permanent tissue damage has occurred. MRI in particular often detects subclinical cerebral ischemia and infarction.

PATHOPHYSIOLOGY

In the normal state, the brain receives 15% to 20% of the cardiac output, and the brain extracts 50% of the available oxygen and 10% of the available glucose for cerebral metabolism. After an ischemic event, the tissue oxygen concentration decreases more than the glucose concentration. Prolonged lack of oxygen reduces energy production, decreasing adenosine triphosphate (ATP) levels and building lactic acid levels. The sodium-potassium pump fueled by ATP fails, and as sodium moves into cells potassium leaks out. Tissue osmolality increases because of the continued presence of glucose. Water accumulates in cells because of the osmotic gradient and increased intracellular sodium. This process, in which fluid accumulates in the intracellular spaces, is called cytotoxic edema. Within 30 minutes after the insult, mitochondria are destroyed. Disruption of cytoplasmic and endothelial membranes follows. These pathophysiologic changes suggest that reversible ischemia occurs within the first hour, before disruption of the blood-brain barrier.

Disruption of the blood-brain barrier, which occurs by 6 hours, causes leakage of water and protein into the extracellular compartment. Reperfusion of the infarcted region can occur within the first 30 minutes by reestablishing the native circulation or by development of collaterals. The degree of reperfusion of the infarcted region determines the amount of fluid that enters the extracellular compartment. This increase in extracellular fluid is called vasogenic edema. The amount of vasogenic edema can progress with continued
reperfusion. The resulting mass effect causes compression of the adjacent microcirculation. There may be extension of the infarct by this process, with irreversible cellular damage (infarction) at the margins of the original ischemic region.

The mass effect caused by vasogenic edema progresses during the first 3 to 7 days, stabilizes during the second week, and begins to resolve by the third week. Blood-brain barrier disruption is commonly seen on imaging up to 8 weeks after the ischemic insult. The development of secondary hemorrhagic foci (i.e., petechial hemorrhage) occurs in up to 40% of cases, typically during the second week. These hemorrhages are usually clinically occult. Intraparenchymal hemorrhage can occasionally present clinically in the first few days, and in this situation the hemorrhage is commonly secondary to embolic infarction.

In the completed infarct, there is gliosis, loss of tissue, and associated focal atrophy. There may be residual cystic areas (i.e., macrocystic encephalomalacia) in the infarcted territory. If there has been associated hemorrhage, hemosiderin may be seen. Dystrophic calcification of the infarcted brain occurs rarely. In large supratentorial infarcts (and particularly those involving the motor cortex), anterograde degeneration of descending nerve pathways may be visualized and is called wallerian degeneration. MRI findings in wallerian degeneration include signal changes (gliosis) and loss of tissue volume. Changes can be noted in the posterior limb of the internal capsule, cerebral peduncles, anterior pons, and anterior medulla (where the fibers decussate).

**MRI PRINCIPLES (ISCHEMIC DISEASE)**

The prior description of pathophysiology provides a conceptual framework for understanding the appearance of cerebral ischemia and infarction on MRI. Before going into depth concerning the MRI appearance of ischemia and infarction, it is important to establish the terminology that is used regarding lesion dating. Unfortunately, there is no universal agreement regarding this terminology. The terminology presented here is one approach well accepted by both radiologists and neurologists. Hyperacute infarction is defined as that within the first 3 to 6 hours after onset of clinical symptoms. This is also the window of potential therapeutic reversibility with current treatment regimens. Acute infarction is defined as that within 6 to 24 hours after onset of symptoms. A TIA is defined as a sudden loss of neurologic function with complete recovery within 24 hours. If ischemia persists beyond 24 hours after onset of symptoms, the area of brain involved will be irreversibly injured and is unlikely to be rescued by reperfusion attempts. Subacute infarction is defined as that from 24 hours to 6 weeks. This time period is subdivided into early subacute (from 24 hours to 1 week) and late subacute (from 1 to 6 weeks). Chronic infarction is defined as that more than 6 weeks after clinical presentation.

Cerebral ischemia and infarction produce fluid changes in the intracellular and extracellular spaces, as previously described (i.e., cytotoxic and vasogenic edema). The sensitivity of MRI for detection of cerebral ischemia is high because of its ability to detect small changes in tissue water. Cytotoxic edema, which occurs very rapidly after the onset of symptoms, can be visualized directly on diffusion weighted scans (Fig.1). Diffusion imaging assesses the microscopic motion of water protons. The gradient magnetic fields used in
imaging are used to achieve sensitivity to diffusion, with both a longer duration and higher amplitude of the gradients increasing such sensitivity. Higher (faster) diffusion produces greater signal attenuation. Diffusion is restricted (slower) in acute ischemia, a result of the intracellular shift of water (cytotoxic edema). Acute infarcts are markedly hyperintense on diffusion-weighted scans, with corresponding low intensity on apparent diffusion coefficient (ADC) maps. Diffusion-weighted scans are also typically T2-weighted. Thus, without reference to a T2-weighted scan, it cannot be said with certainty whether high signal intensity on a diffusion scan represents restricted diffusion or a long T2. Clinical interpretation is aided by comparison with T2-weighted scans and reference to ADC maps. Cytotoxic edema (alone, without accompanying vasogenic edema) is high signal intensity on a diffusion-weighted scan, isointense on a T2-weighted scan (not detectable), and low intensity on an ADC map. Diffusion-weighted scans should be acquired when there is clinical suspicion of an acute or early subacute infarct. Some acute lesions will be visualized only by diffusion imaging. Such scans also permit the differentiation of acute and early subacute ischemia from chronic ischemic changes. Diffusion imaging permits detection of cerebral ischemia within minutes of onset. ADC values are initially low but progress with time to supranormal in irreversible ischemia. The transition from reduced to elevated ADC values is a current area of study; this change was reported by some investigators as early as 24 hours but by others not until 10 days after stroke onset.

Figure 1. Hyperacute left middle cerebral artery infarction demonstrating the utility of diffusion imaging. A, The T2-weighted axial scan is normal. B, The diffusion-weighted scan demonstrates abnormal high signal intensity because of the presence of cytotoxic edema. In very early infarcts, vasogenic edema is not present, and T2-weighted scans will appear normal. Diffusion or perfusion scans are necessary to diagnose these early infarcts.
Infarction of the left hemisphere secondary to internal carotid artery occlusion, illustrating the utility of perfusion imaging. (A) On fluid-attenuated inversion recovery scan abnormal high signal intensity caused by vasogenic edema is confined to the periventricular white matter. A first-pass perfusion study was performed immediately after bolus injection of a gadolinium chelate. On the cerebral blood volume (CBV) (B) and mean transit time (MTT) (C) calculated images, the entire left hemisphere is noted to be involved (with reduced CBV and delayed MTT).

Perfusion imaging is another major tool for the evaluation of brain ischemia; scan acquisition is recommended (in tandem with diffusion imaging) when acute or early subacute ischemia is suspected (Fig.2). The T2*, or susceptibility, effect of a gadolinium chelate is visualized on perfusion imaging during first pass of the contrast agent through the brain. Perfusion imaging thus requires rapid image acquisition during bolus contrast injection, the latter typically performed with a power injector. From the dynamic change in signal intensity during first pass of the contrast agent, cerebral blood volume (CBV) and mean transit time (MTT) calculated images (or "maps") are produced. CBV relates to the area under the time-concentration curve and MTT to the timing of arrival of contrast. In early ischemia, CBV is reduced and MTT prolonged.

Vasogenic edema forms later, after cytotoxic edema, in cerebral ischemia. Although vasogenic edema can be seen as early as 30 minutes after the onset of ischemia, typically changes are not noted until 4 to 6 hours. Findings on conventional MRI within the first 24 hours may be subtle; correct diagnosis relies on the use of diffusion and perfusion imaging (for detection of cytotoxic edema and perfusion deficits) or close inspection of conventional images supplemented with MRI angiography (Fig.3). Once fully established, vasogenic edema is clearly seen with conventional MRI techniques. The increased water content causes prolongation of both T1 and T2. Vasogenic edema thus has low signal intensity on T1-weighted scans and high signal intensity on T2-weighted scans. T2-weighted scans, however, are relied on in clinical practice for the visualization of vasogenic edema. Commonly used "T1-weighted" spin echo sequences (i.e., short time to repetition [TR] and short time to echo [TE]) do not have optimal T1 contrast. Such scans are only mildly T1-weighted. The abnormal low signal intensity on these scans (as a result of vasogenic edema) is less obvious than the abnormal high signal intensity on T2-weighted scans.
recovery sequences or three-dimensional gradient echo T1-weighted sequences (such as turbo-FLASH) are more heavily T1-weighted. With the latter types of scans, the abnormal low signal intensity resulting from vasogenic edema is much better visualized than with conventional T1-weighted spin echo scans.

Figure 3. Acute (24 h) left middle cerebral artery (MCA) infarction demonstrating the appearance of cytotoxic edema on conventional spin echo scans and the complementary role of magnetic resonance angiography (MRA). Subtle high signal intensity in the left MCA distribution on the T2-weighted scan (A) is indicative of early vasogenic edema. B, The thickening and increased prominence (visibility) of cortical gray matter (black arrows) on the T1-weighted scan is due to cytotoxic edema. These findings are subtle in distinction to those on diffusion imaging in early infarcts. C, The three-dimensional time-of-flight MRA exam reveals occlusion (white arrow) of the left MCA.

Clinical studies demonstrated the marked superiority of MRI compared with CT for the detection of cerebral ischemia and infarction, particularly within the first few days. Using diffusion and perfusion imaging, cerebral ischemia can be detected by MRI within minutes of onset. CT is positive for infarction in only 20% of patients within the first 6 hours and in 80% within the first 24 hours. MRI is also markedly superior to CT in detecting posterior fossa and brainstem infarcts. These regions are not obscured on MRI, unlike CT, by beam-hardening artifacts.

The intravenous administration of gadolinium chelates with extracellular distribution provides important ancillary information in brain infarction. Paramagnetic contrast agents decrease T1 relaxation times, increasing the signal intensity on T1-weighted images. Contrast enhancement of vessels supplying the infarct ("vascular," "intravascular," or "arterial" enhancement) is seen in more than half of all infarcts from 1 to 3 days after clinical presentation (Fig.4). Vascular enhancement is more common in cortical lesions and is rarely seen in noncortical gray or deep white matter infarcts. Vascular enhancement occurs when perfusion is absent (complete ischemia). Vascular enhancement dissipates and parenchymal enhancement develops as collateral flow is established. Meningeal enhancement, which is less common than vascular enhancement, can be seen adjacent to
large territorial infarcts from day 2 to day 6 (Fig. 5). In most cases, it is the adjacent dura that enhances. In some cases, the adjacent pia-arachnoid appears involved. Both vascular enhancement and meningeal enhancement are not seen after 1 week.

Parenchymal enhancement is consistently seen in late subacute infarcts and may persist for 8 weeks or more after clinical presentation (Fig. 6). Parenchymal enhancement occurs as a result of blood-brain barrier disruption. Scans should be not be taken immediately after contrast injection because parenchymal enhancement increases given a slight time delay. Lesion enhancement resulting from blood-brain barrier disruption will be substantially better on scans obtained 5 to 10 minutes postinjection as opposed to those obtained immediately after injection. MRI is slightly better than CT for the detection of abnormal contrast enhancement, partly because of the lack of beam-hardening artifact and the greater inherent sensitivity to the contrast agent.

Two types of parenchymal enhancement have been described: progressive enhancement and early or intense enhancement. In progressive enhancement, thin, faint enhancement is first seen at about 1 week near the margins of the lesion or the pial surface. The enhancement progresses over days and weeks to become thicker and more prominent, either in a gyriform pattern if cortical or uniform (solid) if noncortical. Progressive parenchymal enhancement, in both cortical and noncortical infarcts, typically lags behind (temporally) the changes on T2-weighted scans in both intensity and area of involvement. Early or intense enhancement is less common than progressive parenchymal enhancement. With early or intense enhancement, abnormal contrast enhancement is seen within 2 to 3 days of clinical presentation. The area involved equals or exceeds the size of the abnormality on T2-weighted scans in most cases. Clinical outcome in patients with early or intense enhancement includes reversible and persistent neurologic deficits. Early parenchymal enhancement is thought to occur in cases of incomplete ischemia, allowing for delivery of substantial contrast material to the ischemic tissue.

Figure 4. Intravascular contrast enhancement in an early subacute middle cerebral artery (MCA) infarction. A, Vasogenic edema is noted in the left MCA distribution on the T2-weighted scan. Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals enhancement of numerous vessels (intravascular enhancement) in the same region. This
finding is particularly striking when comparison is made to the normal right side, where no vessels are apparent postcontrast.

**Figure 5.** Meningeal contrast enhancement in an early subacute middle cerebral artery (MCA) infarction. A, A small amount of vasogenic edema is noted on the T2-weighted scan in the right MCA distribution. There is extensive sulcal effacement on the precontrast T1-weighted scan (B), indicative of a much larger lesion. Meningeal enhancement is present on the postcontrast T1-weighted scan (C) along the surface of this entire area. Meningeal enhancement, although not common, is important to recognize as such in early subacute infarction. This sign provides supportive evidence for the diagnosis of an infarct and should not be misinterpreted as suggesting a different cause.

**Figure 6.** Subacute middle cerebral artery (MCA) infarction demonstrating gyriform contrast enhancement. There is abnormal high signal intensity consistent with vasogenic edema on the T2-weighted scan (A) in the left MCA distribution (and putamen). Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals gyriform (parenchymal) enhancement in part of the infarct resulting from blood-brain barrier disruption.
In early subacute infarction, the MRI appearance is dominated by the presence of vasogenic edema. The latter is best seen on T2-weighted scans, with abnormal high signal intensity. At this time, the blood-brain barrier is usually still intact and parenchymal enhancement is lacking. In the late subacute phase, cerebral infarction continues to be characterized by increased signal intensity on T2-weighted images and moderately decreased signal intensity on precontrast T1-weighted images. MRI accurately defines the mass effect associated with infarction, which is often most pronounced in the early subacute phase. These findings include compression and effacement of the sulci or ventricular system and displacement of midline structures.

In the chronic phase (after 6 weeks), edema subsides and there is glial proliferation with brain shrinkage. Gliosis is accompanied by increased brain water content and appears as high signal intensity on T2-weighted images and low signal intensity on T1-weighted images. Focal atrophy is identified as enlargement of adjacent sulci or portions of the ventricular system. Cystic changes (e.g., macrocystic encephalomalacia), if present, are characterized by a fluid intensity that follows that of cerebrospinal fluid (Fig. 7). Typically, disruption of the blood-brain barrier, detected after intravenous gadolinium chelate injection, is not visualized beyond 8 weeks.

MRI is particularly sensitive to petechial hemorrhage, which commonly complicates infarction, especially in the subacute phase. Petechial hemorrhage or cortical hemorrhagic infarction (Fig. 8) is most commonly identified as high signal intensity on T1-weighted images because of methemoglobin (subacute stage). Acute and chronic petechial hemorrhage is also clearly depicted on MRI but has a distinct appearance compared with subacute blood. In the acute phase, cortical low signal intensity, resulting from the presence of deoxyhemoglobin, is seen on T2-weighted images (Fig. 9). This is outlined by subcortical vasogenic edema with high signal intensity. The cortical signal changes produced by deoxyhemoglobin are isointense with brain on T1-weighted images. In chronic hemorrhagic infarction, cortical low signal intensity is again seen on T2-weighted images. This is due, however, in the chronic phase to the presence of hemosiderin and ferritin. Because the susceptibility effects of deoxyhemoglobin, hemosiderin, and ferritin (which lead to low signal intensity on T2-weighted scans) are proportional to field strength, these findings are most pronounced at high field (1.5 T) and may not be detected at low field (0.5 T and below).

The cause of cerebral ischemia is often multifactorial. The efficiency of the heart, the integrity of vessels supplying the brain, and the state of the blood itself in supplying oxygen at the cellular level (e.g., oxygen-carrying capacity, viscosity, coagulability) are all contributory factors. Lesions of the vascular tree are commonly the dominant factor in the development of cerebrovascular insufficiency that leads to infarction.
Figure 7. Chronic middle cerebral artery (MCA) infarction. Normal brain has been replaced by cystic encephalomalacia, with high signal intensity on the T2-weighted scan (A) and low signal intensity on the T1-weighted scan (B) in the entire left MCA distribution. Ex vacuo dilatation of the left lateral ventricle is also present. C, Three-dimensional time-of-flight magnetic resonance angiography demonstrates the left MCA to be small and without peripheral branches. The patient was a 10-month-old infant with a history of a neonatal cerebrovascular accident.

Figure 8. Hemorrhagic (methemoglobin) left middle cerebral artery (MCA) infarction. An infarct in the left MCA distribution (anterior division) is easily recognized because of abnormal high signal intensity on the T2-weighted scan (A). The thin gyriform line of high signal intensity in the same region on the precontrast T1-weighted scan (B) corresponds to petechial hemorrhage, in the form of methemoglobin, within cortical gray matter. There
was marked gyriform enhancement postcontrast (image not shown), indicative of blood-brain barrier disruption in this late subacute infarct.

**ARTERIAL VASCULAR TERRITORIES**

The arterial vascular territories of the brain are shown in Figure 10. The middle cerebral artery (MCA) supplies the majority of the lateral surface of the cerebrum, the insular cortex, and the anterior and lateral aspects of the temporal lobe. It is the most common vascular territory involved by infarction. The lenticulo-striate arteries originate from the M1 segment of the MCA and supply the basal ganglia and the anterior limb of the internal capsule. The sylvian triangle is composed of the MCA branches that loop over the insula deep in the sylvian fissure. Although MCA infarcts often involve a wedge-like section of brain, infarcts restricted to a small cortical distribution are not uncommon (Fig. 11).

The posterior cerebral artery (PCA) supplies the occipital lobe, the medial parietal lobe, and the medial temporal lobe (Figs. 3-12 to 3-14). PCA infarction follows MCA infarction in incidence. The thalamoperforating arteries arise from the P1 segment of the PCA and from the posterior communicating artery. These perforators supply the medial ventral thalamus and the posterior limb of the internal capsule.

The anterior cerebral artery (ACA) supplies the anterior two thirds of the medial cerebral surface, the corpus callosum, and 1 cm of superomedial brain over the convexity (Figs. 3-15 to 3-17). Of all cerebral hemispheric infarcts, ACA infarction is the least common and accounts for less than 3% of cases. The recurrent artery of Heubner originates from the A1 or A2 segment of the ACA and supplies the caudate head, the anterior limb of the internal capsule, and part of the putamen. Infarction of both the ACA and MCA territories occurs with thrombosis of the distal internal carotid artery in individuals with ineffective cervical collaterals or an incomplete circle of Willis (Fig. 18).

The anterior choroidal artery arises from the supraclinoid internal carotid artery. This vessel supplies the posterior limb of the internal capsule, portions of the thalamus, the caudate, the globus pallidus, and the cerebral peduncle.

In the posterior fossa, the posteroinferior cerebellar artery (PICA) supplies the retro-olivary medulla, the cerebellar tonsil, the inferior vermis, and the posterior lateral inferior cerebellum (Fig. 19). The anteroinferior cerebellar artery (AICA) supplies the anterolateral inferior cerebellum. Infarction restricted to the distribution of AICA is extremely rare. The superior cerebellar artery (SCA) supplies the superior cerebellum (Figs. 3-20 and 3-21). Cerebellar infarcts present with vertigo, nausea, poor balance, and dysarthria.
Figure 9. Hemorrhagic (deoxyhemoglobin) right middle cerebral artery (MCA) infarction. Abnormal high signal intensity is seen on the T2-weighted scan (A) in the distribution of right MCA (posterior division), compatible with an early subacute infarct. The patient presented with clinical symptoms 6 days before the magnetic resonance scan. Gyriform low signal intensity within the region of high signal intensity is due to the presence of petechial hemorrhage. B, The precontrast T1-weighted scan demonstrates substantial mass effect but adds little additional information in this instance.
Figure 10. Arterial vascular territories, in the axial (A-E) and coronal (F-J) planes. ACA anterior cerebral artery; ACh anterior choroidal artery; AICA anteroinferior cerebellar artery; BA perforating branches of the basilar artery; H recurrent artery of Heubner; LSA lenticulostriate artery; MCA middle cerebral artery; PCA posterior cerebral artery; PICA posteroinferior cerebellar artery; SCA superior cerebellar artery; WSCA watershed region supplied predominantly by the SCA.

Figure 11. Cortical infarction, with progression from the early to the late subacute stage. On magnetic resonance imaging (MRI) performed within 1 week after clinical presentation, vasogenic edema is noted in a small section of cortical gray matter (white arrow), with abnormal hyperintensity on the T2-weighted scan (A) and hypointensity on the postcontrast T1-weighted scan (B). The MRI examination was repeated 9 days
later, with the edema slightly less, as evaluated by the T2-weighted scan (C). Abnormal contrast enhancement (black arrow) is now noted on the postcontrast T1-weighted scan (D).

Figure 12. Early subacute posterior cerebral artery (PCA) infarction. The patient presented with a 2-day history of visual problems. Abnormal high signal intensity is noted in the right PCA distribution on the T2-weighted scan (A). The same area demonstrates subtle low signal intensity on the T1-weighted scan (B). C, Postcontrast, there is prominent intravascular enhancement in this region. This finding supports the leading diagnosis—cerebral infarction—and permits dating of the abnormality. Vascular enhancement is the earliest type of abnormal contrast enhancement identified on magnetic resonance imaging in cerebral infarction and is frequently seen in 1- to 3-day-old lesions.

Figure 13. Late subacute posterior cerebral artery (PCA) infarction. The magnetic resonance (MR) scan was obtained 19 days after clinical presentation. Precontrast T2- (A) and T1-weighted (B) scans are unremarkable, at least at first glance. C, Postcontrast, gyriform enhancement is noted in the right PCA distribution. Parenchymal enhancement occurs because of blood-brain barrier disruption, identifying brain damaged by cerebral ischemia. In the subacute time frame, as with computed tomography, there may be
sufficient resolution of vasogenic edema on MR imaging to render the lesion undetectable without intravenous contrast administration.

Figure 14. Chronic posterior cerebral artery (PCA) infarction. The patient, who has atrial fibrillation, presented clinically 2 years before the current magnetic resonance scan with confusion, unsteady gait, and difficulty reading. Cerebrospinal fluid signal intensity, consistent with cystic encephalomalacia, is noted in the distribution of the right PCA on both the T2- (A) and T1-weighted (B) scans.
Figure 15. Early subacute anterior cerebral artery infarction. There is abnormal high signal intensity in the genu and anterior body of the corpus callosum on the T2-weighted sagittal scan (A) (obtained just to the right of midline). Involvement of gray matter (with similar abnormal hyperintensity) in the anteromedial frontal lobe is also noted on both the sagittal (A) and axial (B) T2-weighted scans. The same medial strip of frontal lobe demonstrates sulcal effacement and abnormal hypointensity of cortical gray matter on the precontrast T1-weighted axial scan (C). D, Postcontrast, intravascular and meningeal enhancement is seen along the 1-cm strip of right frontal lobe adjacent to the midline. This is most prominent posteriorly.
Figure 16. Late subacute anterior cerebral artery (ACA) infarction. On the T2-weighted scan (A), abnormal high signal intensity is noted anterior to the left lateral ventricle and posterior to the right lateral ventricle. The latter finding relates to chronic ischemic changes previously documented in this patient.

Comparing the pre- (B) and postcontrast (C) axial T1-weighted scans, abnormal contrast enhancement is noted anteriorly, matching in position the lesion on the T2-weighted scan. On the coronal postcontrast T1-weighted scan (D), it is somewhat easier to recognize that the abnormal contrast enhancement lies within the ACA distribution. Enhancement is
present because of blood-brain barrier disruption in this subacute lesion. Compared with middle and posterior cerebral artery infarcts, ACA infarcts are much less common. Familiarity with the arterial distribution of the vessel and greater awareness of this entity make misdiagnosis less likely.

Figure 17. Chronic anterior cerebral artery (ACA) infarction. The posterior portion of the ACA territory on the left has abnormal hyperintensity on the T2-weighted scan (A) and hypointensity on the T1-weighted scan (B). C, The fluid-attenuated inversion recovery scan reveals the abnormality to be part gliosis (with high signal intensity) and part cystic
encephalomalacia (with low signal intensity). D, The sagittal T2-weighted scan just to the left of midline also clearly depicts the involvement of the posterior medial frontal lobe.

**THROMBOTIC INFARCTION**

Arterial thrombotic infarction occurs when the arterial lumen is narrowed significantly and blood clots form that occlude the artery. Degenerative atherosclerotic disease, inflammatory disease, or arterial dissection can cause arterial narrowing, although atherosclerotic disease is by far the most common cause. In atherosclerosis, there is degeneration of the intima and media of the arterial wall, with associated proliferation of these elements and lipid deposition. Atherosclerotic lesions or plaques occur at arterial branch points, which are the sites of greatest mechanical stress and turbulence. An atherosclerotic plaque slowly enlarges with time. A critical size is reached, and the surface of the plaque fissures and ulcerates. Platelets adhere to the irregular plaque surface and release prostaglandins, which promote deposition of additional platelet-fibrin plugs and clot on the plaque surface. Thrombosis then occurs, which results in arterial occlusion.

Atherosclerotic thrombotic infarction typically involves large arteries and causes major arterial branch distribution ischemic infarction. Atherosclerotic thrombosis most commonly involves the middle cerebral artery (50%), the internal carotid artery (25%), and the vertebrobasilar system (25%). The extent of infarction is determined by the location of obstruction (the more proximal the lesion, the less likely is the development of infarction), availability of collateral circulation, extent of occlusion, and state of the systemic circulation.

Atherosclerotic disease occurs more commonly in patients with hypertension, hypercholesterolemia, or hyperlipidemia and in those who smoke. Sex and race impact the distribution of lesions. In white men, atherosclerotic lesions predominate at the carotid bifurcation, at the carotid siphon, and in the vertebrobasilar system. White men also have a high incidence of vascular occlusive disease, hypertension, and hyperlipidemia. In women, blacks, and persons of Chinese or Japanese ancestry, atherosclerotic lesions predominate in the intracranial arteries. The common locations are the supracleinoid internal carotid arteries, the anterior, middle, and posterior cerebral arteries, and the vertebrobasilar branches supplying the cerebellum. These patients also have a high incidence of diabetes and hypertension.
Figure 18. Combined anterior cerebral artery (ACA) and middle cerebral artery (MCA) infarction, progressing from early subacute to chronic. Vasogenic edema causes abnormal hyperintensity in both the left ACA and MCA territories on the T2-weighted scan (A) at clinical presentation. B, The T1-weighted scan demonstrates sulcal effacement in the corresponding region, with moderate mass effect on the frontal horn of the lateral ventricle. The magnetic resonance (MR) scan was repeated 18 months later (C-F). At this time, three-dimensional time-of-flight MR angiography reveals collateral flow from the external carotid artery to the supraclinoid internal carotid artery via the ophthalmic artery (white arrow). D, The axial scan with intermediate T2-weighting reveals a mix of gliosis and encephalomalacia in the left ACA and MCA territories. The combined distribution of the two vessels is clearly depicted on the heavily T2-weighted scan (E) with abnormal hyperintensity and the T1-weighted scan (F) with abnormal hypointensity. Ex vacuo dilatation of the left lateral ventricle is also noted.

The MRI findings in thrombotic cerebral infarction are an area of increased water content, with high signal intensity on T2-weighted images and mild low signal intensity on T1-weighted images, that is strictly confined to a major arterial vascular distribution. The distribution is that of the occluded artery. Characteristically, thrombotic infarcts are sharply demarcated, wedge-shaped lesions that extend to the cortical surface. However, depending on the extent and location of the occlusion and the status of the collateral circulation, thrombotic infarcts can have a variety of configurations. Regardless, the signal
changes remain confined to a vascular distribution. For this reason, knowledge of the arterial territories of the brain is important.

The signal intensity characteristics of other brain abnormalities, in particular hyperacute hemorrhage, neoplastic disease, and inflammatory disease, can be similar to those of thrombotic infarction. Fortunately, additional findings on MRI assist in distinction of these entities. A hyperacute intraparenchymal hematoma is typically a round focal mass that is not localized to an arterial territory. Hematomas also have a characteristic temporal progression in signal intensity characteristics. Most thrombotic infarctions involve both gray and white matter. In contrast, neoplastic and inflammatory lesions (abscesses) are usually centered in the white matter. Neoplasms can on occasion extend to the cortex. The edema associated with a neoplasm extends diffusely into the adjacent white matter in finger-like projections, has ill-defined margins, and is unlikely to be restricted to an arterial distribution. Contrast enhancement adds further specificity to the MRI scan. A central enhancing mass is often seen with neoplastic and inflammatory disease. Contrast enhancement in cerebral infarction, although variable in type (depending on the age of the lesion), should conform to the wedge-shaped distribution of the arterial vessel. Despite these features, some lesions, particularly demyelinating disease, may be difficult to distinguish from bland thrombotic infarction.
Figure 19. Early subacute posteroinferior cerebellar artery (PICA) infarction. There is abnormal hyperintensity on the T2-weighted scan (A) and hypointensity on the T1-weighted scan (B) in the posteroinferior cerebellum. Cerebellar tissue anteriorly and laterally, the distribution of anteroinferior cerebellar artery, is
spared. C, The lesion is essentially unchanged postcontrast. The PICA distribution of this infarct is also well depicted in the sagittal plane (D) (precontrast, T1-weighted).

Figure 20. Early subacute superior cerebellar artery (SCA) infarction. A, The T2 weighted scan shows a wedge of vasogenic edema, with abnormal hyperintensity, in a portion of the left SCA territory. The scan plane is through the superior portion of the cerebellum and the occipital lobes. The outer edge of the lesion borders the tentorium. B, The postcontrast T1-weighted scan demonstrates subtle abnormal hypointensity in the same region but no abnormal enhancement. There is mild mass effect, causing slight compression of the fourth ventricle.
Figure 21. Late subacute superior cerebellar artery (SCA) infarction. The entire left SCA distribution is involved, with vasogenic edema noted on the T2-weighted scan (A) and parenchymal enhancement on the postcontrast T1-weighted scan (B). Although edema is present and the lesion is large, there is little mass effect, which would have resolved by this time in evolution of the lesion.

MRI also provides substantial information about vascular patency. The major cerebral arteries are consistently visualized as signal voids because of rapid blood flow on spin echo scans. The absence of a normal flow void in a major cerebral vessel is presumptive evidence of occlusion. Three-dimensional time-of-flight MRA also elegantly displays the arterial vasculature. MRA clearly depicts vessel occlusions, segmental narrowing, and routes of collateral flow.

EMBOLIC INFARCTION

In embolic cerebral infarction, the occlusive material originates from an area proximal to the occluded artery. Emboli most frequently arise from the heart or from atherosclerotic plaques involving the carotid bifurcation or vertebral arteries. The common causes of cardiac emboli include thrombi associated with myocardial infarction or cardiac arrhythmias, valvular disease (including prosthetic valves), bacterial or nonbacterial endocarditis, and atrial myxomas. The ulceration of atherosclerotic plaques produces cholesterol or calcific emboli. Rare embolic causes of infarction are nitrogen emboli from rapid decompensation, fat emboli from long bone fractures, and iatrogenic air emboli.

The location and temporal evolution of embolic infarction differ from thrombotic infarction. Embolic particles shower the intracranial cerebral circulation, often causing multiple peripheral infarcts in different major arterial distributions. Embolic occlusions frequently fragment and lyse between the first and fifth days, which re-establishes normal circulation. These findings differ from the relatively permanent occlusion of a single major vascular distribution with atherosclerotic thrombotic infarction. Fragmentation and lysis of embolic occlusion produces a higher perfusion pressure than that seen with simple occlusion (in which collateral vessels supply the circulation). There is also a loss of normal autoregulation of the cerebral vasculature, which can persist for several weeks. These factors produce hyperemia or luxury perfusion, with blood flow to the infarcted region greater than its metabolic requirements. This higher perfusion pressure can also cause hemorrhage into the infarct and conversion of a bland anemic infarct into a hemorrhagic one. This hemorrhage usually occurs between 6 hours and 2 weeks after the embolic event. Anticoagulant treatment of bland anemic infarcts can also result in hemorrhage.

Before lysis of the embolus, the MRI appearance of embolic infarction is similar to that of thrombotic infarction. However, in contrast to thrombotic infarctions, embolic infarctions are often multiple, may be located in more than one vascular distribution, and are approximately of the same age. After fragmentation of the embolus and the subsequent increase in perfusion pressure, a hemorrhagic infarction often develops. Most commonly, the hemorrhage in a hemorrhagic embolic infarction is petechial in nature and cortical in location. Occasionally, an intraparenchymal hematoma develops in the infarcted region.
Development of secondary hemorrhage is characteristic of embolic infarction but can be seen with thrombotic or hemodynamic infarction.

HEMODYNAMIC INFARCTION

Hemodynamic infarction occurs because of the failure of the heart to pump sufficient blood to oxygenate the brain. Common causes of hypoperfusion include cardiac failure, cardiac arrhythmias, and hypovolemia after blood loss. Patients may have concomitant systemic hypertension, a subcritical arterial stenosis, or even arterial occlusion that had been adequately perfused by collaterals. With development of systemic hypoperfusion and decreased perfusion pressure to the brain, areas of the brain that were adequately perfused are now underperfused, leading to cerebral ischemia or infarction. In many patients, this ischemic event occurs at night while they are asleep, probably because of a nocturnal reduction in blood pressure.

![Figure 22. Chronic hemodynamic infarction. A, Gliosis and encephalomalacia are seen on the T2-weighted scan at the junction of the right middle cerebral artery and posterior cerebral artery territories. B, The precontrast T1-weighted scan reveals petechial hemorrhage (methemoglobin) in the same watershed distribution.](image)

The areas of the brain most commonly involved in hemodynamic infarction are the watershed regions located at the margins of the major arterial distributions (Fig. 22). These regions are the terminal areas supplied by each major artery. They have the lowest perfusion pressure in that vascular distribution. Watershed areas are more prone to ischemic insults caused by systemic hypoperfusion. Knowledge of the arterial vascular territories is necessary to recognize these hemodynamic watershed infarcts. In the cerebral...
cortex, these watershed areas are located at the junctions of the regions supplied by the anterior, middle, and posterior cerebral arteries. The parieto-occipital watershed region is particularly susceptible to hemodynamic ischemic injury because this region is at the peripheral junction of the anterior, middle, and posterior cerebral arterial distributions. In the cerebellum, a watershed region exists at the junction of the territories of the superior cerebellar and inferior cerebellar arteries.

The MRI findings in hemodynamic infarction are increased tissue water content in the distribution of the watershed or border zones of the major arterial vascular distributions. Often the deep periventricular white matter is preferentially involved. White matter receives less blood flow than gray matter and is probably more susceptible to ischemia with a decrease in perfusion. Common locations of white matter hemodynamic infarctions are superior and lateral to the body and trigone of the lateral ventricles. The deep basal ganglia supplied by the lenticulostriate arteries can be similarly affected.

Trauma, with brain contusion and secondary ischemia, can lead to an imaging appearance similar to hemodynamic or thrombotic infarction. Awareness of this entity and access to clinical information is important for appropriate diagnosis (Fig. 23).

![Figure 23](image_url)

**Figure 23.** Cortical contusion. A young adult presents several days after a severe fall down a flight of stairs. There is subtle abnormal high signal intensity within the left frontal white matter on the T2-weighted scan (A). The precontrast T1-weighted scan (B) is unremarkable. C, Postcontrast, gyriform enhancement is noted in several locations, all within cortical gray matter of the frontal lobe. Contusion of the brain cortex has led to blood-brain barrier disruption, which better demonstrates (more so than vasogenic edema) the extent of injury in this case. It is important to note that gyriform contrast enhancement is not specific for infarction because of cerebrovascular disease and can occur in other situations such as trauma (in this instance).

**LACUNAR INFARCTION**

Lacunar infarcts or lacunes are small, deep cerebral infarcts involving the penetrating arteries that supply the basal ganglia, internal capsule, thalamus, and brainstem. These small arteries arise from the major cerebral arteries and include the lenticulostriate
branches of the anterior and middle cerebral arteries, the thalamoperforating branches of the posterior cerebral arteries, and the paramedian branches of the basilar artery. These penetrating arteries are small end arteries (100-500 μm in diameter) that are difficult to evaluate angiographically. Most of these arteries are unbranching single vessels with essentially no collateral circulation. For these anatomic reasons, deep lacunar infarcts typically are spherical in shape and range from 0.3 to 2.5 cm in diameter (Fig. 24). The larger lacunes typically result from more proximal obstructions.

Lacunar infarcts are commonly seen in patients older than 60 years with hypertension. Because this population is also prone to chronic small vessel disease, identification of small recent lacunar infarcts superimposed on chronic disease can be difficult. Diffusion imaging is extremely helpful in acute and early subacute infarcts in this regard. Contrast enhancement is likewise extremely helpful in identifying late subacute infarcts (Fig. 25).

The pathogenesis of lacunar infarction is as follows. Chronic hypertension causes degeneration of the tunica media (i.e., arteriosclerosis), with hyalin deposition in the artery wall that narrows the lumen. Plaque or thrombosis, called microatheroma, may subsequently occlude these vessels, particularly the larger vessels. The weakened tunica media also predisposes to the formation of microaneurysms, which can rupture, causing an intraparenchymal hematoma. A hypertensive hemorrhage or hypertensive hemorrhagic infarction has a characteristic location in the deep cerebral structures supplied by these deep penetrating arteries. Other uncommon causes of lacunar infarction include secondary arteritis caused by meningitis, microemboli, and arterial dissection.
Figure 24. Early subacute lacunar infarction involving the posterior limb of the left internal capsule. Vasogenic edema is noted (with abnormal high signal intensity) on the T2-weighted fast spin echo (A) and fluid-attenuated inversion recovery (B) scans. There is corresponding abnormal low signal intensity on the postcontrast T1-weighted scan (C). However, there is no abnormal contrast enhancement (with disruption of the blood-brain barrier yet to occur). The mean transit time (MTT) for the lesion is prolonged, as seen on a calculated MTT image (D) from a first-pass perfusion study. E, Diffusion weighted imaging and the apparent diffusion coefficient map (F) reveal the presence of cytotoxic edema, as would be anticipated in an infarct less than 1 week old.

Figure 25. Late subacute lacunar infarction involving the posterior limb of the right internal capsule. The patient is an elderly diabetic who presented with acute hemiparesis. The magnetic resonance exam was obtained 10 days after presentation, at which time the hemiparesis had resolved. Multiple high signal intensity abnormalities are noted bilaterally on the T2-weighted scan (A). The postcontrast T1-weighted scan (B) reveals punctate enhancement (arrow) in the posterior limb of the right internal capsule. This corresponds to a high signal intensity lesion on the T2-weighted scan. By identification of abnormal contrast enhancement, this subacute infarct can be differentiated from other chronic ischemic lesions, which are incidental to the patient’s current medical problems.

Lacunar infarction is often recognized by a distinctive clinical presentation. A pure motor stroke is the most common clinical syndrome, accounting for 30% to 60% of lacunar infarcts. A pure sensory stroke, combined sensorimotor stroke, ataxic hemiparesis, dysarthria (or "clumsy hand syndrome"), and brainstem syndromes are other characteristic clinical presentations of lacunar infarction. Patients with lacunar infarction often have a gradual progression of symptoms. An antecedent TIA occurs in approximately 25% of patients with lacunar infarction.
On MRI, lacunar infarcts appear as focal slitlike or ovoid areas of increased water content. They are high signal intensity on T2-weighted images and isointense to low signal intensity on T1-weighted images (Fig. 26). T2-weighted scans are more sensitive than T1-weighted scans for detection. In acute lacunar infarction, vasogenic edema may not be present; thus, diffusion-weighted scans are important for detection. Fluid-attenuated inversion recovery (FLAIR) scans are helpful in identifying small lacunes and differentiating them from spaces containing cerebrospinal fluid (CSF). If FLAIR is not an option, then spin echo scans with intermediate T2-weighting provide similar information. On either type of scan, lacunar infarcts appear as small high-signal intensity focal lesions and can be easily distinguished from the intermediate to low signal intensity of normal surrounding brain and CSF. MRI is much more sensitive than CT in detecting lacunar infarcts. Contrast enhancement of subacute lacunar infarcts, after intravenous gadolinium chelate administration, is consistently seen on MRI (Fig. 27). Enhancement occurs as a result of blood-brain-barrier disruption. Chronic lacunar infarcts are characterized by focal cavitation and a more pronounced decreased signal intensity on T1-weighted images than in the earlier stages of lacunar infarction. These chronic (cavitated) lacunar infarcts are isointense with CSF on all imaging sequences.

![Figure 26. Early subacute thalamic infarction. A, Two round lesions, with abnormal high signal intensity corresponding to vasogenic edema, are noted medially on the T2-weighted scan. The smaller lies in the right thalamus, the larger in the left thalamus. There is subtle low signal intensity in the corresponding areas on the T1-weighted precontrast scan (B). There was no abnormal contrast enhancement (not shown). Thalamic lesions are easily missed by inexperienced film readers, leading to the recommendation that the thalamus be visually checked for abnormalities on each scan.](image-url)
Penetrating vessels from the basilar artery and adjacent segments of the posterior cerebral arteries supply the brainstem. Infarcts involving the pons are most frequently small, unilateral, and sharply marginated at the midline. This location reflects the distribution of paramedian penetrating arteries, which consist of paired branches. Bilateral pontine infarcts do occur but are less common than unilateral infarcts. Lateral pontine infarction is extremely uncommon. The predominant finding on MRI in early subacute pontine infarction is vasogenic edema (Fig. 28). Contrast enhancement is consistently seen in late subacute pontine infarction (Fig. 29).
Figure 27. Late subacute lacunar (basal ganglia) infarction. On adjacent T2-weighted fast spin echo sections (A and B), abnormal high signal intensity is noted in the globus pallidus and body of the caudate nucleus on the right. Enhancement of both lesions is seen on the corresponding postcontrast T1-weighted sections (C and D). The use of intravenous contrast assists in lesion recognition (conspicuity) and in dating lesions. Involvement of both the globus pallidus and caudate nucleus is not uncommon and points to involvement of the lenticulostriate arteries. These small perforating vessels arise from the superior aspect of the proximal middle cerebral artery (M1 segment) and supply the globus pallidus, putamen, and caudate nuclei.

In the elderly population with arteriosclerotic disease, lateral medullary infarction (Wallenberg's syndrome) is not uncommonly encountered (Fig. 30). This lesion is not clearly seen on CT. It is important for the radiologist to be familiar with the MRI appearance of this lesion and for the medulla to be included in the routine search pattern. Otherwise, a lateral medullary infarct may go unrecognized. Clinical presentation includes long-tract signs (contralateral loss of pain and temperature sensation, ipsilateral ataxia, and Horner's syndrome) and involvement of cranial nerves V, VIII, IX, and X. Acute respiratory and cardiovascular complications can occur. In addition to the more common presentation resulting from thrombotic occlusion, lateral medullary infarction has also been reported after chiropractic neck manipulation. The latter occurs as a result of dissection of the vertebral artery near the atlantoaxial joint. The arteries supplying the lateral medulla typically arise from the distal vertebral artery but can originate from the PICA. Thus, lateral medullary infarction can accompany PICA infarction. Medial medullary infarction is less common than lateral medullary infarction. The clinical presentation of medial medullary infarction is that of contralateral hemiparesis, sparing the face.

Figure 28. Early subacute bilateral pontine infarction. The central portion of the pons has abnormal high signal intensity on the T2-weighted scan (A) and abnormal low signal intensity on the T1-weighted scan (B). Despite the lesion being bilateral, there is some indication of a straight border along the midline. A follow-up T1-weighted scan (C) performed 6 months later demonstrates cavitation of the lesion.
Figure 29. Late subacute pontine infarction. On the precontrast T2-weighted scan (A), an area of abnormal hyperintensity is noted in the left pons, with a sharp line of demarcation along the median raphe. The lesion enhances on the postcontrast T1-weighted scan (B). As with other lacunar infarcts, pontine infarcts will consistently demonstrate contrast enhancement after gadolinium chelate administration in the late subacute time period.
Figure 30. Lateral medullary infarction (early subacute). Abnormal hyperintensity is noted on the T2-weighted scan in the right lateral medulla (A). The T1-weighted scan (B) is grossly normal.

DILATED PERIVASCULAR SPACES

Dilated perivascular spaces (DPVSs) are invaginations of the subarachnoid (Virchow-Robin) space that surrounds vessels coursing through the brain. DPVSs are commonly found in the basal ganglia (Fig. 31) and in the periatral and supraventricular white matter (Fig. 32). A third common location is the midbrain (Fig. 33), at the junction of the substantia nigra and cerebral peduncle. DPVSs are small, round, or linear fluid collections that lie along the distribution of penetrating vessels and have signal intensity that strictly follows CSF. Because of their location and appearance, they can mimic lacunar infarction. Therefore, correlation of the anatomic MRI abnormality with clinical history is important.

DPVSs that involve the lenticulostriate arteries supplying the basal ganglia are commonly located adjacent to the lateral aspect of the anterior commissure. Pathologically, focal fluid intensities in the region of the inferior one third of the putamen invariably prove to be DPVSs, but lesions in the upper two thirds are commonly lacunar infarctions. DPVSs that surround middle cerebral artery branches located in the white matter of the centrum semiovale are seen with equal frequency in patients younger and older than 40 years. DPVSs in this location should be considered in the differential diagnosis of lacunar infarction, ischemic-gliotic white matter disease, and multiple sclerosis.
Figure 31. Dilated perivascular space (basal ganglia). A very large dilated perivascular space is noted, with cerebrospinal fluid (CSF) signal intensity on axial T2- (A) and T1-weighted (B) scans. This case illustrates the most common location for dilated perivascular spaces: within the inferior third of the basal ganglia and adjacent to the anterior commissure. On the sagittal T1-weighted scan (C), lenticulostriate vessels can be identified coursing superior from this CSF space.

DPVSs often have MRI characteristics that allow their differentiation from lacunar infarction and other small focal lesions. DPVSs are commonly tubular in shape, and lacunar infarcts are slitlike or ovoid. On sagittal or coronal images, the tubular configuration of DPVSs along the course of the penetrating arteries can often be appreciated. DPVSs strictly follow CSF characteristics on T1-weighted and T2-weighted images, although volume averaging of small lesions with adjacent brain can alter signal characteristics. Acute and subacute lacunar infarcts will be higher signal intensity than CSF on T1-weighted images, FLAIR, and spin echo scans with intermediate T2-weighting.
CHRONIC SMALL VESSEL DISEASE

Punctate or confluent areas of increased signal intensity are commonly seen in the white matter of older patients on T2-weighted scans (Fig. 34). The terms "small vessel disease" and "ischemic-gliotic disease" are used interchangeably. These hyperintense white matter foci can be seen in as many as 30% of asymptomatic patients older than 65 years. The majority of geriatric patients with cardiovascular risk factors and a history of completed stroke or ischemia (RIND or TIA) have hyperintense white matter foci. These lesions often create problems in diagnostic interpretation because of their prevalence, particularly in the asymptomatic patient, and their similarity to other lesions.

Figure 32. Periventricular (A-C) and high convexity (D-E) dilated perivascular spaces (DPVSs). After the basal ganglia, the next most common location for DPVSs is the white matter posterior and superior to the lateral ventricles. When adjacent to the trigones of the lateral ventricles (A, T2-weighted fast spin echo; B, T2-weighted fluid-attenuated inversion recovery; C, postcontrast T1-weighted), DPVSs are linear in shape on axial sections. In the high convexity white matter, they appear as small pinpoints on axial sections (D, T2-weighted fast spin echo; E, precontrast T1-weighted).
Figure 33. Midbrain dilated perivascular spaces. Another characteristic location for dilated perivascular spaces is the midbrain at the junction of the substantia nigra and cerebral peduncle. These may be unilateral or bilateral in location: the latter is illustrated here. Dilated perivascular spaces follow cerebrospinal fluid signal intensity on all pulse sequences, with high signal intensity on T2-weighted fast spin echo scans (A) and low signal intensity on T2-weighted fluid-attenuated inversion recovery (B) and T1-weighted spin echo (C) scans. Dilated perivascular spaces are, however, best visualized on fast spin echo T2-weighted scans.

Figure 34. Chronic small vessel ischemic disease. Multiple small foci with abnormal high signal intensity are noted in peripheral white matter on T2-weighted fast spin echo (A) and fluid-attenuated inversion recovery (B) scans. The same disease process also accounts for the hyperintensity immediately adjacent to ("capping") the frontal horns and surrounding the atria of the lateral ventricles.

Pathologic evidence, correlated with MRI, suggests that ischemia and infarction produce the majority of these lesions. One study found white matter atrophy and gliosis surrounding thickened vessels in the region of the hyperintense MRI white matter foci. The authors postulated that increased extracellular water is responsible for the increased signal intensity on T2-weighted scans. They also suggested the cause to be chronic, mild vascular
insufficiency rather than thrombotic or embolic occlusive infarction. True white matter infarction was commonly the cause of hyperintense MRI white matter foci. Central necrosis, axonal loss, and demyelination were found to be compatible with true infarction.

Because ischemia and infarction appear to be the predominant causes of hyperintense white matter foci in older patients, and because infarction often has a significant component of gliosis, we refer to these lesions as ischemic-gliotic disease and describe the degree of involvement as mild, moderate, or severe. In mild cases, there are a few scattered, small, hyperintense, white matter lesions. In severe cases, there can be confluent increased signal intensity in the white matter on T2 weighted scans. In moderate cases, the changes are intermediate in nature. In patients with diffuse white matter disease, diffusion imaging and postcontrast scans can be useful in distinguishing areas of acute and subacute infarction from chronic disease.

There are other, less common causes of hyperintense white matter lesions that should be recognized. Plaques of multiple sclerosis can occur with minimal clinical symptoms. In these subclinical cases, the lesions tend to be small and involve only the supratentorial white matter, sparing the brainstem and cerebellum. Brain cysts and congenital ventricular diverticula have increased signal intensity on T2-weighted images but are uncommon. These lesions characteristically border the ventricular system or subarachnoid space, have a smooth rounded configuration, and have CSF signal intensity on all pulse sequences. Occasionally, a cavitated infarct becomes cystic and displays similar signal intensity characteristics. Dilated perivascular spaces can also mimic other focal white matter lesions and lacunar infarcts. Binswanger's disease (subcortical arteriosclerotic encephalopathy) represents a distinct clinical entity with characteristic clinical findings in patients with hypertension, hydrocephalus, and dementia. These patients have rapid deterioration of their cognitive ability, gradual development of neurologic symptoms, and a lengthy clinical course with long plateau periods. MRI demonstrates focal or confluent white matter lesions on T2-weighted images. Hypertensive encephalopathy is an acute neurologic syndrome with the clinical presentation, including headache, somnolence, convulsions, and vomiting. T2-weighted images demonstrate hyperintense lesions in the white matter and cerebral cortex, particularly involving the occipital lobes. Reversibility of these lesions after treatment has been reported.

**ARTERITIS**

Cerebral arteritis can be classified as primary or secondary. In the primary form, the inflammatory process originates in the arteries. In the secondary form, the inflammatory process starts in the brain parenchyma or meninges, and the arteries are involved secondarily. Primary cerebral arteritis often presents with recurrent neurologic symptoms that may simulate multiple sclerosis. This disease tends to affect a younger age group than arteriosclerotic vascular disease. Primary cerebral arteritis is usually caused by systemic disorders. Causes include systemic lupus erythematosus (SLE), other collagen-vascular diseases, polyarteritis nodosa, giant cell arteritis, Behcet’s disease, and sarcoidosis.
Both white and gray matter involvement can be seen in SLE. There are two patterns of white matter involvement. One pattern consists of large, confluent areas of high signal intensity on T2-weighted images consistent with infarction (Fig. 35). The other pattern consists of small focal punctate white matter lesions, presumably corresponding to small microinfarcts. Lesions can also involve the gray matter. In some patients with gray matter involvement, clinical resolution may be accompanied by the resolution of these lesions on MRI.

Findings similar to SLE are seen in other vasculitides, including polyarteritis nodosa. Focal brainstem infarction has been described in Behcet’s disease. Some MRI findings help to differentiate arteritis from multiple sclerosis. Periventricular white matter involvement is less extensive and may be absent in primary cerebral arteritis. Multiple sclerosis is typically characterized by extensive, punctate periventricular white matter involvement (which is not symmetric from side to side). A lesion in a major cerebral artery vascular territory, or cortical involvement, favors a vascular disease process.

The cause of secondary cerebral arteritis is commonly meningitis. Bacterial or fungal organisms, including Mycobacterium tuberculosis, are common causes. A contrast-enhanced MRI should be performed to identify the location and extent of meningeal disease. T2-weighted scans demonstrate high-signal-intensity lesions compatible with ischemia or infarction in the vascular distribution involved by the meningeal process.

**VASOSPASM AND MIGRAINE**

Spasm of the intracranial arteries can be associated with subarachnoid hemorrhage or migraine headaches. Subarachnoid hemorrhage is commonly caused by a ruptured intracranial aneurysm (75% of cases). The arteries in the affected subarachnoid space can experience varying degrees of spasm, which may progress to complete occlusion. MRI demonstrates findings compatible with ischemia or infarction involving major arterial distributions or their watershed regions, corresponding to the distribution of the artery in spasm.

Migraine headaches are initiated by vasoconstriction of extracranial and intracranial arteries. This leads to ischemia, which produces neurologic deficits or an aura. Vasoconstriction is followed by vasodilatation, which produces the headache. CT and MRI findings consistent with ischemia or infarction have been described in these patients. MRI demonstrates focal lesions with increased signal intensity on T2-weighted images, predominantly involving the periventricular white matter but also involving the cortex. Corresponding hypointensity is seen in some lesions on T1-weighted images. Resolution of small focal lesions can be seen on MRI with time after resolution of symptoms.

Patients with the classic or common form of migraine, visual aura that responds to ergotamine followed by a unilateral throbbing headache, have focal periventricular lesions. Patients with neurologic deficits or complicated migraine have larger periventricular lesions and often have cortical lesions. Cortical lesions in general are associated with neurologic deficits.
ANOXIA AND CARBON MONOXIDE POISONING

Cerebral anoxia has many causes, including primary and secondary respiratory failure, drowning, and carbon monoxide poisoning. The cerebral ischemia or infarction that develops initially involves the regions of the brain in which the blood supply is most tenuous. The watershed regions of the cortex, periventricular white matter, and the basal ganglia are particularly prone to ischemic injury. In severe cases, the cortex, white matter, and basal ganglia can be diffusely involved (Fig. 36). Patients with irreversible injury demonstrate focal areas of necrosis or demyelination.

In children, the distribution of hypoxic-ischemic brain injury is related to the degree of development. In premature infants, the periventricular corona radiata is most predisposed to ischemic injury. These patients may later experience delayed myelination, periventricular leukomalacia (Fig. 37), cerebral atrophy, and hydrocephalus. In full-term infants and young children, the cortical and subcortical regions are most prone to infarction. The full-term infant and older child no longer have the collaterals between the meninges and cerebral arteries that protect the cortex as in the premature infant.

Figure 35. Systemic lupus erythematosus. A and B, T2-weighted scans reveal multiple bilateral parenchymal abnormalities. These lesions, which correspond to territorial infarcts, involve both gray and white matter in both the anterior and middle cerebral artery distributions.
Anoxic brain injury. A, At first glance, the T2-weighted scan appears normal. In retrospect, there is loss of the gray-white matter differentiation. Axial (B) and coronal (C) T1-weighted scans show reversal of the normal signal intensity relationship of gray and white matter. White matter has abnormal low signal intensity as a result of global vasogenic edema.

MRI demonstrates increased signal intensity on T2-weighted images and isointense or low signal intensity on T1-weighted images in the ischemic or infarcted regions. In the infant, attention to imaging technique and scan interpretation are important to differentiate edema from the normal high water content of white matter at this age (Fig. 38). In the premature infant, ultrasonography may be a more useful modality for evaluating infarction. Increased iron deposition in infarcted regions in children who survive a severe ischemic-anoxic insult has been described. This iron deposition may be produced by disruption of normal axonal transport of brain iron by injury. It is more evident at higher field strengths and with gradient echo imaging.

Focal areas of ischemic necrosis are seen in carbon monoxide poisoning. Four types of lesions are described in pathologic studies: necrotic lesions of the globus pallidus, focal necrotic white matter lesions or confluent demyelination, spongy lesions in the cerebral cortex, and necrotic lesions of the hippocampus. Frequently, MRI demonstrates only abnormal high signal intensity in the globus pallidus bilaterally (on T2-weighted images). All four types of lesions can, however, be seen on MRI.
Figure 37. Periventricular leukomalacia. Abnormal increased signal intensity, resulting from gliosis, is noted on the T2-weighted scan in the periventricular white matter (A). The amount of periventricular white matter is also decreased, particularly in the periatrial region, as best seen on the T1-weighted scan (B). The patient is a 17-month-old infant with mild paralysis affecting the lower extremities (paraparesis).

**ARTERIAL DISSECTION**

Arterial dissection is often overlooked as a cause of cerebral ischemia or infarction. Arterial dissection may be caused by trauma, diseases intrinsic to the arterial wall, or local inflammatory disease, or it may have a spontaneous onset. Arteriography has been the best modality for diagnosing arterial dissection, but findings may be nonspecific. MRI is a sensitive and noninvasive method for identifying the hemorrhagic component of a dissection. MRI can provide a definitive diagnosis in patients with nonspecific arteriographic findings and is useful in monitoring the resolution of these lesions.

The temporal sequence of MRI changes in an arterial dissection with intramural hemorrhage is similar to that of an intraparenchymal hematoma. Hemosiderin is not deposited because the blood-brain barrier is not present. Subacute hemorrhagic dissection (containing extracellular methemoglobin) appears as a hyperintense lesion on T1- and T2-weighted images that expands the wall of the vessel and narrows its lumen. Axial images best demonstrate the intramural hemorrhage because the artery is visualized in cross-section. Sagittal images are difficult to interpret because of vascular tortuosity, volume averaging of the vessel, and the similarity of the linear, hyperintense intramural hematoma to an interstitial fat plane.
Figure 38. Neonatal infarction. A, On this T1-weighted scan of a 1-month-old infant, gray and white matter appear normal at first glance. In the neonate, before myelination, white matter will be of lower signal intensity than gray matter on T1-weighted scans. However, in this case, a focus of abnormal hyperintensity is seen in the right frontal lobe corresponding to hemorrhage (methemoglobin). Alerted by this finding, and looking more closely, it is noted that the gray matter mantle is too thin and that the gray-white matter contrast is accentuated (with white matter of too low signal intensity). Global infarction is confirmed on the follow-up scan (B) 1 month later, which demonstrates cystic encephalomalacia sparing only the immediate periventricular white matter.

An acute intramural hemorrhage may be difficult to diagnose because deoxyhemoglobin has low signal intensity on T2-weighted images, thus simulating a flow void. On T1-weighted images, an absence of the normal flow void indicates thrombosis. Phase images, gradient echo scans emphasizing flow, and time-of-flight MRA are useful in detecting the presence or absence of flowing these cases.

MOYAMOYA

Moyamoya is an ischemic vascular disease of unknown cause. There is progressive stenosis or occlusion of the supraclinoid segments of the internal carotid arteries. This is accompanied by the development of lenticulostriate and thalamoperforate collaterals. The proximal portions of the anterior, middle, and posterior cerebral arteries may also be involved. There is endothelial hyperplasia and fibrosis but no evidence of inflammatory disease. The disease usually develops during childhood, and children typically present with ischemic symptoms. Adults with the disease commonly present with subarachnoid or intracranial hemorrhage. There is an increased incidence of moyamoya in the Japanese population.
Angiography has been the procedure of choice in confirming the diagnosis. Arterial stenoses and occlusions and the vascular blush of the collaterals are characteristic of the disease. This vascular blush is called moyamoya, or "puff of smoke," in Japanese.

Characteristic MRI findings have been described for moyamoya. These include multiple bilateral infarctions involving the watershed regions of the carotid circulations, absence of the signal flow void in the supraclinoid internal carotid artery or middle cerebral artery, and visualization of the dilated collateral moyamoya vessels as multiple signal flow voids (Fig. 39).

**AMYLOID ANGIOPATHY**

Amyloid angiopathy is an uncommon cause of nonhypertensive hemorrhage in older patients. Amyloid deposits are identified in small and medium-sized arteries and arterioles in the cerebral cortex. The temporal, parietal, and occipital lobes are most frequently involved. In particular, the calcarine region of the occipital lobe is commonly involved. These pathologic findings probably reflects changes of aging. The amyloid deposition in the vessel wall presumably increases vessel fragility, which predisposes to rupture of the vessel and hemorrhage. The autopsy incidence of this disease is 40% in patients older than 70 years and 60% or greater in patients older than 80 years. Noncortical arteries of the brain are not involved. Cortical hemorrhages, which may extend into subcortical locations, suggest this disease in older patients. Subarachnoid hemorrhage is commonly an associated finding because of the peripheral location of the cortical hemorrhages.

![Figure 39. Moyamoya disease. An old right middle cerebral artery (MCA) infarct is noted on T2- (A) and T1-weighted (B) scans. The patient is only 27 years old. The perforating arteries feeding the basal ganglia appear prominent, particularly on the left, on the T1-weighted scan (which is postcontrast). C, Three-dimensional time-of-flight magnetic resonance angiography demonstrates occlusion of both internal carotid arteries just before their division into the anterior cerebral artery and MCA.](image)
VENOUS THROMBOSIS

Cerebral venous thrombosis may involve any of the cerebral veins, including the major venous sinuses, cortical veins, and deep veins. The clinical diagnosis of this disease is difficult because of nonspecific signs and symptoms. Because of the high incidence of morbidity and mortality, prompt recognition is important to improve patient outcome.

Cerebral venous thrombosis can be divided into two major etiologic categories: inflammatory and noninflammatory. Before the advent of antibiotics, inflammatory causes, particularly mastoid sinus disease, were common causes of cerebral veno-occlusive disease. Inflammatory causes are relatively uncommon today, but there are many noninflammatory causes. Venous thrombosis associated with pregnancy and the puerperium, trauma, dehydration, neoplasm, the use of oral contraceptives, or L-asparaginase therapy are today the most common causes.

MRI provides a sensitive, noninvasive means for evaluating cerebral venous thrombosis. There is an orderly temporal evolution of MRI findings. Initially, the absence of a normal flow void is seen on T1-weighted images. In this stage, the thrombus appears as intermediate signal intensity on T1-weighted images. On T2-weighted images, there is low signal intensity in the corresponding region. These findings are due to the presence of deoxyhemoglobin. The low signal intensity on T2-weighted images is more pronounced with increased field strength. A supportive finding is the identification of venous collaterals bypassing the obstruction. Later, the thrombus becomes high signal intensity, initially on T1-weighted images and subsequently on T2-weighted images. These findings are due to the formation of methemoglobin (Fig. 40). Long-term, the vessel can recanalize, and flow voids are again visualized.

Figure 40. Superior sagittal and transverse sinus thrombosis. On precontrast T2- (A) and T1-weighted (B) scans, the left transverse sinus has abnormal hyperintensity. This suggests occlusion (with the signal intensity caused by extracellular methemoglobin), which is supported by the lack of venous pulsation artifacts. Thrombosis is confirmed by visualization of the same signal intensity within the sinus on an orthogonal plane; the
sagittal T1-weighted scan (C) is chosen to illustrate this and to show occlusion of the superior sagittal sinus as well.

Slow flowing a normally patent vein can produce high or intermediate signal intensity and can have the appearance of a thrombus on a single sequence. Flow-related enhancement and even-echo rephasing must be recognized as such and identified as representing normal venous flow. However, a thrombus will maintain the same signal characteristics in any plane and on sequences done at different times. These features generally distinguish a thrombus from slow flow with high signal intensity. MR venography, using time-of-flight techniques, can also be helpful in diagnosis. Care should be exercised, however, to prevent the interpretation of a methemoglobin clot as representative of flow (on MR venography).

Venous thrombosis is often associated with infarction. Venous infarction can involve the cortex and underlying white matter. A common pattern seen with superior sagittal sinus thrombosis is multiple bilateral, parasagittal, high-convexity infarcts. Gyral enhancement is seen in subacute venous infarcts as a result of blood-brain-barrier disruption. Hemorrhage commonly accompanies venous infarction. Hemorrhagic venous infarction most often involves the cortex, often in a gyriform manner. Hemorrhage can also occur in the white matter with or without associated cortical hemorrhage.

References

1. Metwally, MYM: Textbook of neuroimaging, A CD-ROM publication, (Metwally, MYM editor) WEB-CD agency for electronic publication, version 12.1 April 2012
Cerebrovascular disease includes both structural vascular anomalies (aneurysms and vascular malformations) and ischemia. Vascular anomalies are discussed in this chapter. Cerebrovascular disease is often accompanied by intracranial hemorrhage. Knowledge of the appearance of hemorrhage on magnetic resonance imaging (MRI) is critical for scan interpretation, and this is discussed first. MRI is markedly more sensitive than computed tomography (CT) for the detection of cerebrovascular disease, including specifically hemorrhage, vascular anomalies, and ischemia. MRI often obviates the need for cerebral angiography, an invasive examination with accompanying increased risk.
MRI provides exquisite identification of intracranial hemorrhage. Understanding the appearance of hemorrhage requires knowledge of how the different forms of blood affect the local proton environment. Time to repetition (TR), time to echo (TE), type of imaging sequence, field strength, oxygen tension, hemodilution, rate of clot formation, and integrity of the blood-brain barrier (BBB) and the red blood cell membrane all affect the MRI appearance. Evolving hemorrhage follows an orderly progression of changes, although the exact timing of these changes is variable from patient to patient. To understand the effects of different forms of hemoglobin, proton relaxation enhancement must be considered. In each water molecule, there are two hydrogen atoms. The nucleus of each hydrogen atom is a single proton. This unpaired proton possesses angular momentum or spin, producing a magnetic moment. The latter is a vector quantity with direction and magnitude and defines a magnetic dipole (or, more simplistically, a tiny bar magnet). Proton-proton dipole-dipole interaction describes the behavior or interaction that occurs between the magnetic dipoles of different protons.

The human body is largely composed of water protons that are in constant motion (on a microscopic level). This motion is characterized by rotational, translational, and vibrational components. In pure water, T1 and T2 relaxation occurs by proton-proton dipole-dipole interactions. T1 is the characteristic time constant for spins to align with the external magnetic field. T2 is the characteristic time constant for loss of transverse magnetization or, equivalently, loss of phase coherence among spins. Water is a small molecule with a high frequency of motion compared with the Larmor (resonance) frequency used for imaging. In pure water there is inefficient T1 and T2 relaxation, resulting in long T1 and T2 relaxation times. A long T1 relaxation time yields low signal intensity on images with T1-weighting (short TR and short TE). A long T2 relaxation time yields high signal intensity on images with T2-weighting (long TR and long TE).

Many of the breakdown products of hemoglobin are paramagnetic substances, which have unpaired electrons. An unpaired electron is the dominant factor in a magnetic moment created by a proton (positive charge) and an electron (negative charge). An electron has a mass equal to 1/1000 of the mass of a proton. It thus has a magnetic moment 1000 times that of a proton. The addition of a paramagnetic substance to the water environment can change the predominant proton relaxation mechanism from a proton-proton dipole-dipole interaction to a proton-electron dipole-dipole interaction. For a proton-electron dipole-dipole interaction to occur, the proton must approach extremely close (within 0.3 nm) to the paramagnetic center. If this occurs, the frequency of motion of water decreases (the complex is bulkier), which yields a more efficient energy transfer (relaxation), shortening both T1 and T2. This process is called proton-electron dipole-dipole proton relaxation enhancement.

If a paramagnetic substance is confined within a red blood cell by the red blood cell membrane, the distribution in tissue will be heterogeneous. A high intracellular concentration of a paramagnetic substance causes local magnetic field inhomogeneity. The precession rate of water molecules (Larmor frequency) is proportional to the local field strength, and the local field strength varies with local magnetic inhomogeneity. Water protons diffusing through this local magnetic inhomogeneity precess at different rates and
lose coherence. These dephased protons cannot be refocused by the 180-degree spin echo (SE) pulse, and transverse phase coherence is lost. This causes a shorter transverse relaxation time (T2) without affecting T1. This process is called preferential T2 proton relaxation enhancement. T2 proton relaxation enhancement is proportional to the square of the magnetic field and, therefore, is more pronounced at higher field strengths. T2 proton relaxation enhancement is also proportional to the square of the concentration of the paramagnetic substance and is increased by lengthening the interecho interval (the time between the 180-degree refocusing pulses in a SE sequence). Lengthening the interecho interval allows the diffusing water molecules to encounter greater local magnetic inhomogeneity, which increases dephasing and further shortens T2.

Using knowledge about T2 proton relaxation enhancement, it is possible to predict the appearance of hemorrhage on SE and gradient echo sequences. The principal component of hemorrhage is hemoglobin, which occurs in several different forms, some of which are paramagnetic. As a hemorrhage ages, the hemoglobin molecule undergoes the following degradation pattern: oxyhemoglobin to deoxyhemoglobin to methemoglobin to hemosiderin.

Before describing each of these forms of hemoglobin, a brief discussion of MRI terminology is appropriate. Sequences with relative T1 weighting (short TR and short TE) are called T1-weighted images. Sequences with relative T2 weighting (long TR and long TE) are called T2-weighted images. Long TR, short TE sequences are called proton density images. Even though a sequence is called "T1-weighted" or "T2-weighted," the signal intensity derives from both T1 and T2 effects. Either effect may predominate and yield a particular signal intensity on a given image. Cranial lesions are described as hypointense (low signal intensity), isointense (signal intensity close to that of a reference tissue), or hyperintense (high signal intensity). Gray matter and white matter are typically used as reference tissues for signal intensity on T1 and T2-weighted images.

- **Oxyhemoglobin**

A simple intraparenchymal hemorrhage initially is composed of intact red blood cells containing oxygenated hemoglobin. Oxyhemoglobin contains iron in the ferrous state (Fe2⁺) and has no unpaired electrons. Oxyhemoglobin is thus not paramagnetic but rather diamagnetic. It has, for practical purposes, no magnetic moment and no proton relaxation enhancement. A hyperacute hematoma containing oxyhemoglobin exhibits long T1 and T2 relaxation times and is hypointense or isointense on T1-weighted images and high signal intensity on T2-weighted images. This is the expected MRI appearance of a protein-containing fluid. Oxyhemoglobin is often isointense with other intracranial mass lesions. Fortunately, oxyhemoglobin is quickly degraded in intra-axial hematomas, lasting only a few hours. It is thus uncommon to visualize oxyhemoglobin within an intraparenchymal bleed. However, the poor discrimination of oxyhemoglobin accounts for the limited ability of conventional SE sequences to detect acute subarachnoid hemorrhage.
**Deoxyhemoglobin**

In a few hours, the red blood cells become desaturated, and oxyhemoglobin is converted to deoxyhemoglobin. Iron remains in the ferrous state (Fe2⁺) but with four unpaired electrons, making deoxyhemoglobin paramagnetic. Intracellular deoxyhemoglobin is confined by the red blood cell membrane and is heterogeneously distributed. Water protons cannot approach within 0.3 nm of the paramagnetic center, probably because of a slight change in configuration of the hemoglobin molecule. Preferential T2 proton relaxation enhancement shortens T2 but not T1. As a result, intracellular deoxyhemoglobin is slightly hypointense or isointense on T1 weighted images and has low signal intensity on T2-weighted images (Fig. 1). The low signal intensity on T2-weighted images becomes more pronounced with increasing field strength, increased interecho interval, and greater amounts of the paramagnetic substance (deoxyhemoglobin).

**Methemoglobin**

Intracellular deoxyhemoglobin within a hemorrhage is oxidized to methemoglobin. This process depends on the partial pressure of oxygen. The rate of oxidation decreases substantially at very low or very high oxygen tensions. In certain circumstances, the formation of methemoglobin can thus be delayed. However, methemoglobin is usually seen by 2 days and persists for several weeks.

![Figure 1](image-url) **Figure 1.** Acute hematoma (deoxyhemoglobin). A large left frontal lobe mass is noted on precontrast T2-(A) and T1-weighted (B) scans. The mass is predominantly low signal intensity on the T2-weighted scan and intermediate to low signal intensity on the T1-weighted scan (consistent with deoxyhemoglobin). Increased signal intensity, representing vasogenic edema, is seen surrounding the low-signal-intensity bleed on the T2-weighted scan. There is substantial mass effect. The bleed extends into the left temporal lobe, a finding evident on close inspection of the sagittal T1-weighted scan (C).
In the formation of methemoglobin, the heme iron is oxidized to the ferric state (Fe\textsuperscript{3+}). Methemoglobin has five unpaired electrons and is highly paramagnetic. The molecular configuration of methemoglobin allows the water protons to approach within 0.3 nm of the protein's paramagnetic center. A proton-electron dipole-dipole interaction shortens both \( T_1 \) and \( T_2 \). The heterogeneous distribution of methemoglobin in the intracellular state accentuates \( T_2 \) relaxation (\( T_2 \) proton relaxation enhancement), causing intracellular methemoglobin to appear as high signal intensity on \( T_1 \)-weighted images and low signal intensity on \( T_2 \)-weighted images (Fig. 2).

Soon after methemoglobin forms within the red blood cell, glucose reserves become depleted, which causes a loss of red blood cell integrity and subsequent lysis. Extracellular (free) methemoglobin then accumulates in the hematoma. The distribution of methemoglobin is no longer heterogeneous (no longer partitioned by a red blood cell membrane), causing a loss of \( T_2 \) proton relaxation enhancement. With red blood cell lysis, extracellular methemoglobin produces proton-electron dipole-dipole proton relaxation enhancement, which decreases \( T_1 \). Extracellular methemoglobin is thus high signal intensity on \( T_1 \)-weighted images, like intracellular methemoglobin. The \( T_1 \) shortening and the effects of the high proton density of free methemoglobin overwhelm the \( T_2 \) shortening produced by the proton-electron dipole-dipole interaction. Thus, on \( T_1 \) and \( T_2 \)-weighted images, extracellular methemoglobin appears as high signal intensity. As methemoglobin is resorbed, a protein-containing fluid is formed, and actual prolongation of \( T_2 \) occurs, which also accounts for increased signal intensity on \( T_2 \)-weighted images. The signal intensity of extracellular methemoglobin on \( T_1 \)-weighted images is also affected by concentration (dilution). The signal intensity of free methemoglobin can vary from hyperintense to hypointense, depending on dilution, on a given \( T_1 \)-weighted image. As free methemoglobin is progressively diluted, its proton-electron dipole-dipole proton relaxation enhancement is lost. Its signal characteristics then approach those of cerebrospinal fluid (CSF).

- **Hemosiderin and Ferritin**

Extracellular methemoglobin is oxidized to a series of compounds called hemichromes, which are degraded into hemosiderin. Hemosiderin is phagocytized and accumulates in the lysosomes of macrophages. Hemosiderin contains iron in the ferric state (Fe\textsuperscript{3+}) and is strongly paramagnetic. Hemosiderin is insoluble in water; therefore, no dipole-dipole interaction occurs. However, because hemosiderin has an inhomogeneous distribution, \( T_2 \) proton relaxation enhancement causes low signal intensity on \( T_2 \)-weighted images. This strong \( T_2 \) effect may be appreciated as slightly low signal intensity on \( T_1 \)-weighted images.

Hemosiderin can be distinguished from dense calcification on the basis of its \( T_2 \) proton relaxation enhancement effect. Hemosiderin is slightly low signal intensity on \( T_1 \)-weighted images, moderately low signal intensity on proton density weighted images, and very low signal intensity on \( T_2 \)-weighted images. Because \( T_2 \) proton relaxation enhancement is proportional to the square of field strength, these effects are most pronounced at higher field strengths. If fast \( T_2 \)-weighted images (high-speed radiofrequency [RF] refocused echo imaging) are used, the \( T_2 \) effect will be less. Calcium has no mobile protons and does not
change in signal intensity on T1-weighted, proton density, or T2-weighted images. However, calcium is sometimes mixed with hemosiderin, and the iron in the mixture produces a T2 proton relaxation enhancement effect.

Hemosiderin can persist indefinitely in a lesion with an intact blood-brain barrier (BBB) and is a landmark for identifying chronic hemorrhage. However, in lesions without an intact BBB, the hemosiderin-laden macrophages have access to the blood stream, and the hemosiderin is resorbed. The configuration of the hemosiderin rim can be an important feature in differentiating a simple intraparenchymal hematoma from intratumoral hemorrhage. In hemorrhage associated with neoplasm, hemosiderin deposition is discontinuous or in conspicuous because the BBB is not intact. In a simple intraparenchymal hematoma, the hemosiderin rim is well defined and continuous.

Figure 2. Subacute hematoma (intracellular methemoglobin rim). A left frontal lobe mass is noted on precontrast T2- (A) and T1-weighted (B) scans. The mass has a prominent low-signal-intensity rim on the T2-weighted scan, with a thick high-signal-intensity rim on the T1-weighted scan (findings consistent with intracellular methemoglobin). There is substantial surrounding vasogenic edema, best seen on the T2-weighted scan as high signal intensity. The presence of edema supports the conclusion, based on the signal intensity of blood products, that the bleed is recent. The bleed was a complication of aneurysm clipping.
SUMMARY

Table 1. The MRI biochemical stages of cerebral hematomas

<table>
<thead>
<tr>
<th>Biochemical substance</th>
<th>MRI changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxyhemoglobin</td>
<td>Oxyhemoglobin lacks unpaired electrons and thus clot signal is close to normal brain parenchyma - normal to slightly lower signal on T1-weighted images and slightly higher signal on T2-weighted images</td>
</tr>
<tr>
<td>Paramagnetic intracellular deoxyhemoglobin.</td>
<td>Because the deoxyhemoglobin within intact, clotted hypoxic red blood cells does not cause T1 shortening, the hematoma will have normal to slightly lower signal on T1-weighted MR images. The concentration of red blood cells with clot and the concentration of fibrin cause T2 shortening, with areas of very low signal on T2-weighted spin echo and T2 *-weighted gradient echo images</td>
</tr>
<tr>
<td>Paramagnetic intracellular methemoglobin.</td>
<td>Proton-electron dipole-dipole interactions between hydrogen atoms and the paramagnetic centers of methemoglobin will cause marked T1 shortening and very high signal intensity on T1-weighted images within the periphery of the hematoma. The intracellular methemoglobin will cause T2 shortening and very low signal on T2-weighted images.</td>
</tr>
<tr>
<td>Extracellular migration of methemoglobin.</td>
<td>MR will exhibit the persistent high signal of extracellular methemoglobin on T1- and T2-weighted images for up to a year. The peripheral rim of hemosiderin and ferritin has slightly low signal on T1- and marked low signal on T2-weighted images [20] from the susceptibility effect of hemosiderin within macrophage lysosomes.</td>
</tr>
<tr>
<td>Clot resorption begins from the periphery inward, and depending on the size of the hematoma, may vary from one to six weeks in duration. Necrotic tissue is sloughed and cystic cavities are formed over the next 6 to 12 months.</td>
<td>Focal atrophy is characterized by a decrease in the size of cortical gyri, with compensatory enlargement of cerebrospinal fluid spaces and dilatation of the adjacent ventricle. Cystic cavities are surrounded by gliosis and hemosiderin scarring.</td>
</tr>
<tr>
<td>Stage</td>
<td>Description</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------</td>
</tr>
<tr>
<td><strong>Hyperacute stage [0-12 Hr]</strong></td>
<td>Immediately after an intracerebral bleed, the liquefied mass in the brain substance contains oxyhemoglobin but no paramagnetic substances. Therefore, it looks like any other proteinaceous fluid collection.</td>
</tr>
<tr>
<td><strong>Acute stage [4Hr -3 days]</strong></td>
<td>Reduction in oxygen tension in the hematoma results in the formation of intracellular deoxyhemolobin and methemoglobin in intact red cells. These substances have a paramagnetic effect that produces T2 shortening. A thin rim of increased signal surrounding the hematoma on T2-weighted images represents edema.</td>
</tr>
<tr>
<td><strong>Subacute stage [3days-3 weeks]</strong></td>
<td>As red blood cells lyse, redistribution of methemoglobin into the extracellular space changes the effect of this paramagnetic substance to one of predominantly T1 shortening. The longer T2 results from (1) a combination of red blood cell lysis (T2 shortening disappears), (2) osmotic effects that draw fluid into the hematoma, and (3) the repetition times (TR) that are in general use for T2-weighted sequences, which are not sufficiently long to eliminate T1 contrast effects in the image.</td>
</tr>
<tr>
<td><strong>Chronic stage [3 weeks-3 months]</strong></td>
<td>Phagocytic cells invade the hematoma (starting at the outer rim and working inward), metabolizing the hemoglobin breakdown products and storing the iron as superparamagnetic hemosiderin and ferritin.</td>
</tr>
<tr>
<td>Stage</td>
<td>Blood Product Description</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Hyperacute stage [0-12 Hr]</strong></td>
<td>Oxyhemoglobin lacks unpaired electrons and thus clot signal is close to normal brain parenchyma-normal to slightly lower signal on TI-weighted images and slightly higher signal on T2-weighted images</td>
</tr>
<tr>
<td><strong>Acute stage [4Hr - 3 days]</strong></td>
<td>Deoxyhemoglobin within intact, clotted hypoxic red blood</td>
</tr>
<tr>
<td><strong>Early subacute stage [3 days-3 weeks]</strong></td>
<td>Strongly paramagnetic intracellular methemoglobin, TI shortening and very high signal intensity on TI-weighted images within the periphery of the hematoma</td>
</tr>
<tr>
<td><strong>Late subacute stage [3 days-3 weeks]</strong></td>
<td>Extracellular migration of ethemoglobin, MR will exhibit the persistent high signal of extracellular methemoglobin on TI - and T2-weighted images for up to a year</td>
</tr>
<tr>
<td><strong>Chronic stage [3 weeks-3 months]</strong></td>
<td>Focal atrophy is characterized by a decrease in the size of cortical gyri, with compensatory enlargement of cerebrospinal fluid spaces and dilatation of the adjacent ventricle. Cystic cavities are surrounded by gliosis and hemosiderin scarring.</td>
</tr>
</tbody>
</table>
Table 4. Effect of blood products on the MRI signal

<table>
<thead>
<tr>
<th>Phase</th>
<th>Time</th>
<th>Hemoglobin</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperacute</td>
<td>&lt;24 hours</td>
<td>Oxyhemoglobin (intracellular)</td>
<td>Iso or hypo</td>
<td>Hyper</td>
</tr>
<tr>
<td>Acute</td>
<td>1-3 days</td>
<td>Deoxyhemoglobin (intracellular)</td>
<td>Iso or hypo</td>
<td>Hypo</td>
</tr>
<tr>
<td>Early subacute</td>
<td>&gt;3 days</td>
<td>Methemoglobin (intracellular)</td>
<td>Hyper</td>
<td>Hypo</td>
</tr>
<tr>
<td>Late subacute</td>
<td>&gt;7 days</td>
<td>Methemoglobin (extracellular)</td>
<td>Hyper</td>
<td>Hyper</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt;14 days</td>
<td>Hemosiderin (extracellular)</td>
<td>Iso or hypo</td>
<td>Hypo</td>
</tr>
</tbody>
</table>

- **Intracerebral Hematoma**

The evolution of an intraparenchymal hematoma is depicted by a characteristic sequence of MRI changes. These changes depend on many factors, including size, compartmentalization, oxygen tension, and BBB integrity. Therefore, the staging of intracerebral hematomas is not rigid. For example, several components of hemoglobin can be seen concurrently within a large hematoma. Although the timing and appearance of these changes are variable, a temporal sequence of changes can be described that provides a conceptual framework for identifying the stages of an intracerebral hematoma. Four stages in the evolving hematoma can be described: hyperacute (first few hours), acute (first few hours to 2 days), subacute (2 days to 4 weeks), and chronic (more than 4 weeks).

In the hyperacute stage, an intraparenchymal hematoma is composed of a mixture of oxyhemoglobin and deoxyhemoglobin. The formation of deoxyhemoglobin depends on the local oxygen tension. For example, hemorrhagic cortical infarcts are in a high local oxygen environment (resulting from arterial perfusion), and the formation of deoxyhemoglobin may be retarded. However, in hemorrhagic venous infarction or in a large intraparenchymal hematoma, the oxygen tension is lower and deoxyhemoglobin predominates. In a hyperacute intraparenchymal hematoma in which oxyhemoglobin predominates, the lesion is hypointense or isointense relative to brain on T1-weighted images and hyperintense relative to brain on T2-weighted images. A hyperacute intraparenchymal hematoma containing oxyhemoglobin is indistinguishable from other.
intracranial mass lesions, and its signal may be isointense with CSF. Fortunately, the hyperacute stage is not commonly imaged. CT does not have this limitation and is excellent for the diagnosis of hyperacute bleeds.

Within the first few hours of the formation of a hematoma, oxyhemoglobin is converted into deoxyhemoglobin. An acute intraparenchymal hematoma, which contains deoxyhemoglobin within intact red blood cells, is slightly hypointense to isointense on T1-weighted images and hypointense T2-weighted images. The degree of hypointensity on T2-weighted images increases with the increasing field strength. Consequently, on low-field systems, an acute hematoma can be nearly isointense with brain on T2-weighted scans. Acute hemorrhage is surrounded by extracellular water, which initially is serum extruded by the retracting clot and later is edema. This increased water content causes a low-intensity margin on T1-weighted images and a high-intensity margin on T2-weighted images.

During the subacute stage, intracellular deoxyhemoglobin is oxidized to intracellular methemoglobin, a process that depends on the local oxygen tension. The formation of intracellular methemoglobin begins at the periphery of the hematoma, where the conditions for its formation are optimal, and progresses inward toward the center of the hematoma. The presence of intracellular methemoglobin results in a hyperintense periphery of the hematoma on T1-weighted images and a hypointense periphery on T2-weighted images, which progress centrally as deoxyhemoglobin is oxidized (see Fig. 2). On T1-weighted images, a subacute hematoma can have a low-intensity surrounding margin (edema), a hyperintense periphery (intracellular methemoglobin), and a hypointense or isointense center (intracellular deoxyhemoglobin). The corresponding appearance on T2 weighted images is a high-intensity surrounding margin, a low-intensity periphery, and a hypointense center of the hematoma. With time, the entire hematoma fills in (with intracellular methemoglobin) and has uniform high signal intensity on T1-weighted images (Fig. 3). After 1 week to 1 month, red blood cell lysis occurs, and intracellular methemoglobin becomes extracellular. Free methemoglobin has high signal intensity on both T1- and T2-weighted images. Because of dilutional effects, the signal in the central of the hematoma (with dilute free methemoglobin) can be low or isointense on T1-weighted images. At about the same time that methemoglobin becomes extracellular, the hematoma develops a peripheral rim of low intensity, which is more easily seen on T2-weighted images and corresponds to hemosiderin in macrophages. The formation of the hemosiderin rim requires an intact BBB. As the hematoma resorbs, the hemosiderin rim increases in thickness. The edema surrounding the hematoma, just beyond the hemosiderin rim, begins to resolve. After the surrounding edema has resolved, the hematoma (now late subacute in stage) is characterized by a low-intensity rim of hemosiderin and central high-intensity area of extracellular methemoglobin. This appearance is similar on T1 - weighted and T2-weighted images, except that the hemosiderin rim is more pronounced on T2-weighted images.

In the chronic stage (more than 4 weeks), the methemoglobin within the center of the hematoma is broken down and resorbed. As this occurs, the T1 shortening produced by the methemoglobin is lost. The remaining fluid contains some protein, without any iron, and is
isoointense with cerebrospinal fluid (CSF) (Fig. 4). This central fluid may be resorbed, leaving only a hemosiderin rim. Thus, a chronic hematoma can have several appearances. A chronic hematoma can have a center that is isointense or of high intensity (depending on whether methemoglobin is resorbed or present) with a low-intensity rim (hemosiderin). Alternatively, only a low-intensity hemosiderin cleft can be left, with complete resorption of any fluid.

- Cerebral edema associated with non-traumatic cerebral hemorrhage

Traditionally, ICH was believed to cause permanent brain injury directly by mass effect. However, the importance of hematoma-induced inflammatory response and edema as contributors to secondary neuronal damage has since been recognized.

At least three stages of edema development occur after ICH (Table 5). In the first stage, the hemorrhage dissects along the white matter tissue planes, infiltrating areas of intact brain. Within several hours, edema forms after clot retraction by consequent extrusion of osmotically active plasma proteins into the underlying white matter. The second stage occurs during the first 2 days and is characterized by a robust inflammatory response. In this stage, ongoing thrombin production activates by the coagulation cascade, complement system, and microglia. This attracts polymorphonuclear leukocytes and monocyte/macrophage cells, leading to up-regulation of numerous immunomediators that disrupt the blood-brain barrier and worsen the edema. A delayed third stage occurs subsequently, when red blood cell lysis leads to hemoglobin-induced neuronal toxicity. Perihematomal edema volume increases by approximately 75% during the first 24 hours after spontaneous ICH and has been implicated in the delayed mass effect that occurs in the second and third weeks after ICH.

Thrombin is an essential component of the coagulation cascade, which is activated in ICH. In low concentrations thrombin is necessary to achieve hemostasis. However, in high concentrations, thrombin induces apoptosis and early cytotoxic edema by a direct effect. Furthermore, it can activate the complement cascade and matrix metalloproteinases (MMP) which increase the permeability of the blood brain barrier.

Delayed brain edema has been attributed, at least in part, to iron and hemoglobin degradation. Hemoglobin is metabolized into iron, carbon monoxide, and biliverdin by heme oxygenase. Studies in animal models show that heme oxygenase inhibition attenuates perihematomal edema and reduces neuronal loss. Furthermore, intracerebral infusion of iron causes brain edema and aggravates thrombin-induced brain edema. In addition, iron induces lipid peroxidation generating reactive oxygen species (ROS), and deferoxamine, an iron chelator, has been shown to reduce edema after experimental ICH.
Table 5. Stages of edema after ICH

<table>
<thead>
<tr>
<th>First stage (hours)</th>
<th>Second stage (within first 2 days)</th>
<th>Third stage (after first 2 days)</th>
</tr>
</thead>
</table>
| • Clot retraction and extrusion of osmotically active proteins | • Activation of the coagulation cascade and thrombin synthesis  
• Complement activation  
• Perihematomal inflammation and leukocyte infiltration | • Hemoglobin induced neuronal toxicity |

- **Subdural Hematoma**

Subdural hematomas result from a venous injury, with blood lying outside the brain parenchyma between the dura and arachnoid. Like intraparenchymal hematomas, four stages in the evolution of subdural hematomas can be described: hyperacute, acute, subacute, and chronic.

Hyperacute subdural hematomas are composed of a mixture of oxyhemoglobin and deoxyhemoglobin and are hypo- to isointense to brain on T1-weighted images and hyperintense on T2-weighted images. An acute subdural hematoma is composed of deoxyhemoglobin in intact red blood cells, causing preferential T2 proton relaxation enhancement. An acute subdural hematoma is hypo- to isointense to brain on T1-weighted images and hypointense on T2-weighted images.
Figure 3. Subacute hematoma (intracellular methemoglobin). In this right frontal hemorrhage, the oxidation of deoxyhemoglobin to intracellular methemoglobin is nearly complete, resulting in almost uniform low signal intensity on the precontrast T2-weighted scan (A) and high signal intensity on the precontrast T1-weighted scan (B). There is a small rim of surrounding high signal intensity on the T2-weighted scan consistent with vasogenic edema, confirming that the hemorrhage is still relatively recent. With time, red blood cell lysis will result in the intracellular methemoglobin becoming extracellular in location, with the hematoma then high signal intensity on both T2- and T1-weighted scans. The patient was predisposed to an intracranial bleed as a result of severe vascular disease. This is reflected on the T2-weighted scan by the presence of chronic small vessel ischemic disease and several old lacunar infarcts.

In a subacute subdural hematoma (Fig. 5), intracellular deoxyhemoglobin is oxidized to methemoglobin, which is hyperintense on T1-weighted images and hypointense on T2-weighted images. By 2 weeks, red blood cell lysis results in free methemoglobin, causing hyperintensity on both T-1 and T2-weighted images. As methemoglobin is slowly broken down in the chronic phase, a subdural hematoma becomes intermediate in signal intensity between methemoglobin and CSF on T1-weighted images and high signal intensity but lower than CSF on T2-weighted images. These are the expected characteristics of a protein-containing extra-axial fluid collection. These characteristics distinguish a chronic subdural hematoma from the prominent CSF spaces seen with atrophy (Fig. 6).

In the subacute and chronic phases, the membrane delimiting a subdural hematoma may enhance with intravenous injection of a gadolinium chelate (see Fig. 6). Subdural hematomas may have a combination of acute and subacute chronic components, which
appear as fluid-fluid layers of the different forms of hemoglobin comprising the hematoma. Hemosiderin accumulation is typically absent in extra-axial fluid collections because of the lack of a BBB in the dura and access of hemosiderin-laden macrophages to the bloodstream. On rare occasions, hemosiderin can be identified in patients with recurrent bleeds into chronic subdural hematomas.

Figure 4. Chronic hematoma (hemosiderin rim) demonstrating the end result of a large basal ganglia hemorrhage. The hematoma has long since been resorbed, leaving a large cavity now filled with cerebrospinal fluid. This fluid collection has high signal intensity on the T2-weighted scan (A) and low signal intensity on the T1-weighted scan (B). The only direct evidence of previous hemorrhage is the low signal intensity (hemosiderin) rim, bordering the cavity, seen on the T2-weighted scan.
Figure 5. Subacute subdural hematoma. An extra-axial fluid collection is noted on the patient’s right side, which is principally hyperintense on the proton density weighted scan (A), hypointense on the T2-weighted scan (B), and hyperintense on the precontrast T1-weighted scan (C). The signal characteristics are consistent with intracellular methemoglobin.

MRI is very sensitive and superior to CT for detection of subacute and chronic subdural hematomas. Chronic extra-axial bleeds are low density on CT and may be indistinguishable from large CSF spaces seen with atrophy. Moreover, CT bone artifact can obscure small extra-axial fluid collections, even in the acute phase. In comparing the CTs and MRIs of a patient with a stable extra-axial fluid collection, the lesion will appear larger on MRI because of bone artifact and soft tissue windowing on CT. These factors tend to reduce the apparent size of the fluid collection on CT. The hyperintensity of methemoglobin in the subacute and chronic phases makes extra-axial hematomas readily identifiable on MRI.

- **Epidural Hematoma**

Epidural hematomas most often occur as a result of an arterial injury. Blood dissects between the calvarium and dura, producing a biconvex lentiform fluid collection (Fig. 7). Epidural hematomas follow the same pattern of evolution as subdural hematomas. An epidural hematoma can be distinguished from a subdural hematoma by its configuration and by the low-intensity fibrous dura that demarcates the margin of the hematoma from the brain parenchyma. However, in the acute phase, the low-intensity dura may not be visualized as a separate structure from the low-intensity hematoma (deoxyhemoglobin). The differentiation of an epidural hematoma from a subdural hematoma can be difficult if the configuration of the fluid collection is atypical.
Figure 6. Chronic subdural hematoma. A, The T2-weighted scan demonstrates an extra-axial fluid collection surrounding the left cerebral hemisphere. The fluid has high signal intensity on the T2-weighted scan and low signal intensity on the T1-weighted scan (B). The subdural fluid is, however, slightly higher in signal intensity on the T1-weighted scan than the cerebrospinal fluid of the lateral ventricles. This difference was more evident on the proton density weighted scan (not shown). The adjacent dura is thickened and enhances postcontrast (C).

Figure 7. Acute epidural hematoma. There is compression of the right cerebellum by an extra-axial soft tissue mass. On the T2-weighted scan (A), the mass has predominantly intermediate signal intensity, although a portion anteriorly has very low signal intensity. On the T1-weighted scan (B), most of the lesion is slightly low signal intensity, with a more anterior component of intermediate signal intensity. This suggests a fluid composition, with layering of different components. Computed tomography (not shown) demonstrated a right occipital fracture. The imaging appearance of an epidural hematoma is that of a biconvex, elliptical fluid collection. Because of its epidural location, the fluid collection can cross the midline (falx) or the tentorium, unlike subdural fluid. On the sagittal T1-weighted scan (C),
the lesion is noted to cross the attachment of the tentorium posteriorly and thus can be localized to the extradural (epidural) space. An epidural hematoma accumulates between the dura and the inner table of skull. This lesion is typically caused by a skull fracture with laceration of blood vessels. The most common vessel involved is the middle meningeal artery resulting from a temporal or parietal bone fracture. Less common is laceration of the transverse sinus (as in the case presented) caused by an occipital bone fracture.

- **Subarachnoid and Intraventricular Hemorrhage**

Subarachnoid hemorrhage, commonly secondary to rupture of an intracranial aneurysm or an arteriovenous malformation, is a potentially life-threatening event that requires prompt diagnosis and therapy. Hyperacute and acute subarachnoid hemorrhages are not well seen on conventional SE techniques. The conditions in the subarachnoid space are different from other intracranial locations, and the expected pattern of evolution of hemorrhage does not occur. The detection of subarachnoid hemorrhage on MRI requires the use of fluid attenuated inversion recovery (FLAIR) scan, a pulse sequence discussed later.

Acute subarachnoid hemorrhage occurs in, and is diluted by, CSF. This compartment has an average oxygen tension (PO2) of 43 mm Hg, with 72% of the hemoglobin in the saturated oxyhemoglobin state. Oxyhemoglobin has signal characteristics that are isointense to CSF and, therefore, not well seen on conventional MRI scans. The contribution of deoxyhemoglobin, which causes preferential T2 proton relaxation enhancement, to T2 shortening during this phase is negligible. Because T2 shortening is proportional to the square of the concentration of the paramagnetic compound, deoxyhemoglobin in a concentration of 28% (100% - 72%) contributes only 8% (0.282) to T2 shortening. The T2 shortening of deoxyhemoglobin is also masked by dilution with CSF and CSF pulsation artifacts. Therefore, it is not surprising that oxyhemoglobin and deoxyhemoglobin in acute subarachnoid hemorrhage are not well demonstrated by conventional SE techniques.

FLAIR images have been shown to be virtually 100% sensitive to acute subarachnoid hemorrhage. With this pulse sequence, CSF is attenuated and thus black. In acute subarachnoid hemorrhage, there is a small decrease in T1 caused by the higher protein content of the bloody CSF. This mild T1 shortening leads to hypertense CSF on FLAIR. One problem with FLAIR is the high-intensity CSF inflow artifacts in the basal cisterns, which may simulate subarachnoid hemorrhage. This artifact is markedly lessened by the use of a FLAIR sequence in which the thickness of the 180-degree inverting RF pulse has been slightly increased.

In subacute subarachnoid hemorrhage, characteristic signal intensity changes can often be identified on T1 and T2-weighted images corresponding to methemoglobin within thrombus (Fig. 8). In rare instances, deoxyhemoglobin within thrombus in the acute phase is visualized. In chronic or recurrent subarachnoid hemorrhage, hemosiderin deposition can occur in a subpial location, which is called "superficial hemosiderosis" or "superficial siderosis." A thin rim of marked hypointensity on T2-weighted images lines the
parenchymal surface in superficial siderosis. This condition can be caused by hemorrhage from vascular abnormalities, intracranial tumors, ependymoma of the conus medullaris, or neonatal hemorrhage. Occasionally, patients develop hearing loss with involvement of cranial nerve VIII, other cranial nerve abnormalities, and cerebellar ataxia.

Figure 8. Subacute subarachnoid hemorrhage (intracellular methemoglobin). Clotted blood, containing methemoglobin, is well visualized in the interpeduncular cistern because of its low signal intensity on the T2-weighted scan (A) and high signal intensity (black arrow) on the T1-weighted scan (B). The bleed is 4 days old. C, The sagittal precontrast T1-weighted scan reveals clotted blood, with high signal intensity, in the pontine, interpeduncular, and chiasmatic cisterns. A small amount of intraventricular blood is also present, layering posteriorly in the occipital horns. Subtle intraventricular hemorrhage can be missed without close inspection of the ventricles. In this instance, the blood is best detected in the ventricles on the T2-weighted scan, with low signal intensity (white arrows).

Intraventricular hemorrhage is much like subarachnoid hemorrhage in signal characteristics and temporal evolution. Like subarachnoid hemorrhage and unlike intraparenchymal, subdural, or epidural hemorrhage, intraventricular hemorrhage mixes with CSF in an environment with high oxygen tension. Oxidative denaturation of hemoglobin to methemoglobin is delayed. Substantial amounts of methemoglobin, with high signal intensity on T1-weighted scans (Fig. 9), are not formed for several days.

- **Gradient Echo Imaging in Hemorrhage**

In gradient echo imaging, a reduced flip angle RF pulse and a subsequent applied gradient that refocus the echo are used rather than the 90-degree RF pulse and 180-degree refocusing pulse used in routine SE imaging. Gradient echo imaging is particularly sensitive to the magnetic susceptibility effects of paramagnetic substances.

Magnetic susceptibility is defined as the ratio of the induced magnetic field to the main magnetic field. Magnetic susceptibility occurs when substances are induced to form their own weak magnetic field under the influence of an externally applied field. Three classes of substances exhibit this type of magnetic behavior: paramagnetic, superparamagnetic, and
ferromagnetic substances. Superparamagnetic and ferromagnetic substances can acquire large magnetic moments, even if exposed to very weak external magnetic fields. Ferromagnetic substances, unlike paramagnetic and superparamagnetic substances, retain their magnetism after the external magnetic field is removed.

Figure 9. Intraventricular hemorrhage (methemoglobin). Axial (A) and sagittal (B) T1-weighted scans demonstrate a high-signal-intensity (methemoglobin) blood clot filling the right lateral ventricle. The clot was also high signal intensity and thus not well distinguished from cerebrospinal fluid on the T2-weighted scan (not shown). The signal characteristics are compatible with extracellular methemoglobin. The clot was approximately 2 weeks old.

Paramagnetic substances have a high degree of magnetic susceptibility. Because many of the degradation products of hemoglobin (deoxyhemoglobin, methemoglobin, and hemosiderin) are paramagnetic, they are visualized with increased sensitivity on gradient echo imaging compared with SE imaging. Small amounts of these paramagnetic hemoglobin compounds can be detected with gradient echo imaging that may not be visualized with standard SE imaging or CT. For example, small cortical petechial hemorrhages or small amounts of residual hemosiderin from old hemorrhagic angiomas can be identified as areas of focal signal loss on gradient echo imaging. However, these susceptibility effects can also overwhelm other signal characteristics of a lesion and obscure important diagnostic features. For example, the central high-intensity area in a cavernous angioma (cavernoma) (a distinguishing characteristic feature) can be obscured by the susceptibility effects of the hemosiderin rim. Furthermore, the boundary between deoxyhemoglobin or intracellular methemoglobin and surrounding brain appears as a hypointense rim on gradient echo imaging, which can obscure identification of a
hemosiderin rim. Identification of the rim is important in evaluating hemorrhagic intracranial tumors, which typically have an incomplete surrounding hemosiderin rim because of the lack of an intact BBB.

**VASCULAR ANOMALIES**

Cerebrovascular anomalies can be divided into two major categories: intracranial aneurysms and vascular malformations. Clinical presentation of patients with cerebrovascular anomalies is variable. However, these patients not uncommonly present with acute intracranial hemorrhage, either secondary to subarachnoid hemorrhage or an intraparenchymal hematoma. In acute cases, detection of subarachnoid hemorrhage is critical, and the MRI exam must include a FLAIR sequence. MRI is very accurate for characterizing intracranial vascular disease. MRI has also replaced CT as the screening modality for detecting vascular malformations and intracranial aneurysms and associated mural thrombi.

Vascular malformations are congenital developmental abnormalities of the vascular system. Cerebrovascular malformations are divided into four major pathologic categories: arteriovenous malformation (AVM), venous angioma, capillary telangiectasia, and cavernous angioma (cavernoma). AVMs and venous angiomas can be routinely visualized with angiography, CT, and MRI. Capillary telangiectasias and cavernous angiomas (cavernomas) are not directly visualized with current imaging modalities but are detected because of hemorrhage associated with the lesion. Thrombosed AVMs and venous angiomas may also be detected only by the presence of hemorrhage if their abnormal vessels become obliterated. These vascular malformations detected by the presence of recurrent or chronic hemorrhage, in which the vascular anomaly is not visualized angiographically, are called occult cerebrovascular malformations (OCVMs). Based on current imaging studies, the majority of vascular malformations are divided into three major radiologic groups: AVM, venous angioma, and OCVM.

- **Aneurysm**

An aneurysm is a focal dilatation of a vessel. Aneurysms can be characterized by their configuration as fusiform (a spindle-shaped dilatation of a vessel) or saccular (a sharply circumscribed, spherical sac). Fusiform aneurysms are commonly secondary to arteriosclerosis and often involve the basilar artery and intracranial carotid arteries. The vast majority of aneurysms are saccular (berry aneurysms) and are thought to be congenital. They commonly arise at arterial branch points and are secondary to an inherent defect in the tunica media (Fig. 10). Mycotic infection, trauma, and neoplasm cause fewer than 5% of all aneurysms. These aneurysms typically occur at peripheral locations rather than branch points.

In the general population, the incidence of intracranial aneurysms is approximately 3%. The common congenital saccular aneurysm involves the anterior carotid circulation in 85% to 90% of cases and the posterior basilar circulation in 10% to 15% of cases. In the anterior circulation, the most common locations of saccular aneurysms are the anterior
communicating artery (30%), the posterior communicating artery (25%), and the middle cerebral artery bifurcation/trifurcation (20%) (Fig. 11). In the posterior circulation, the basilar artery trunk and bifurcation (10%) (Fig. 12) and the vertebral-posterior inferior cerebellar artery (3%) are the most common sites. In 20% of patients, multiple aneurysms are identified at angiography.

A ruptured intracranial aneurysm is the most common cause (75%) of subarachnoid hemorrhage. Vascular malformations account for 5% of cases. Patients with a ruptured aneurysm commonly present with acute onset of a severe headache that may progress to coma. In the patient with a suspected acute subarachnoid hemorrhage caused by a ruptured aneurysm, CT still remains the screening modality of choice. CT readily visualizes acute hemorrhage as high density, and CT is more easily performed in the uncooperative patient.

MRI is often initially performed in patients without intracranial hemorrhage and in those with intracranial hemorrhage and atypical symptoms. In these patients, conventional MRI (without the use of magnetic resonance [MR] angiography) often demonstrates the aneurysm (Fig. 13). In approximately 20% of aneurysms that bleed, there is an associated intraparenchymal hematoma. Beyond the hyperacute stage, MRI exquisitely demonstrates intraparenchymal hematomas, and their location can suggest the diagnosis of a ruptured aneurysm. For example, an intraparenchymal hematoma adjacent to the anterior interhemispheric fissure suggests a ruptured anterior communicating artery aneurysm, and an intraparenchymal hematoma adjacent to the sylvian cistern suggests a ruptured middle cerebral artery aneurysm. In patients with known aneurysms, MRI can identify the bleeding site by demonstrating subacute hemorrhage adjacent to the aneurysm. Subacute or chronic subarachnoid hemorrhage resulting from a ruptured aneurysm is also clearly identified by MRI.
Figure 10. Ophthalmic artery aneurysm. On precontrast T2-(A) and T1-weighted (B) scans a small round signal void (arrow) is identified anterior to the supraclinoid segment of the left internal carotid artery. There is enhancement of the lesion rim on the axial T1-weighted image postcontrast (C). D, A maximum intensity projection image from the three-dimensional time-of-flight magnetic resonance angiography examination reveals a small aneurysm just medial and anterior to the extracavernous intracranial segment of the left internal carotid artery.

Figure 11. Middle cerebral artery bifurcation aneurysm. An abnormal low-signal-intensity flow void (large black arrow) is noted on the precontrast T2-weighted scan (A). The pulsation artifact emanating from this structure anteriorly and posteriorly (small black
and white arrows) in the phase encoding direction confirms that the structure is vascular in nature. The lesion has paradoxical high signal intensity, again because of the flow phenomenon, on the precontrast T1-weighted scan (B). Also noted are chronic cavitated infarcts bilaterally: a small lacuna on the right and a larger hemosiderin-lined lesion on the left. Three-dimensional time-of-flight magnetic resonance angiography (C) confirms the presence of an aneurysm, at the middle cerebral artery bifurcation and approximately 1 cm in diameter. Also noted are multiple focal vessel stenoses.

Figure 12. Basilar artery aneurysm. An oval flow void is identified on the precontrast T2-weighted scan (A) in the prepontine cistern at the expected location of the basilar tip. B, A single slice from the three-dimensional time-of-flight magnetic resonance angiogram depicts the structure as high signal intensity and thus confirms it as a vascular structure. C, The maximum intensity projection image reconstructed from the three-dimensional examination, the image depicted in B being one of many slices in this data set, depicts a moderate-sized aneurysm arising from the tip of the basilar artery.
Figure 13. Left middle cerebral artery (MCA) aneurysm detected by conventional planar magnetic resonance imaging. A, The T2-weighted scan is unremarkable. On the precontrast T1-weighted scan (B), a question of abnormal hyperintensity, just posterior to the left MCA trifurcation, is raised. On the postcontrast axial (C) and coronal (D) T1-weighted scans, enhancement of a small aneurysm (arrow) is seen, permitting detection. Slow flow within this berry aneurysm leads to marked contrast enhancement after intravenous gadolinium chelate administration. The lumen of the aneurysm is thus well depicted.

A unique feature of MRI is its ability to detect vascular flow, particularly in the arterial system. The appearance of flow on conventional (planar) MRI is presented first followed by a discussion of MRA. High-velocity flowing arteries or veins appears commonly as a flow void on MRI. This high-velocity signal loss occurs when protons in flowing blood do not remain within the selected slice long enough to acquire both the 90- and 180-degree pulses used to produce an SE. Saccular aneurysms appear as regions of flow void with a typical configuration and location. Pulsation artifact, propagating in the phase-encoding direction, is another supporting finding seen with pulsatile flowing patent aneurysms. Pulsation artifacts are more pronounced after contrast administration because of the increased signal within the vascular space.
Depending on the imaging parameters selected and the effects of turbulence and rephasing, slow-flowing blood within a vessel or an aneurysm can have high or mixed signal intensity rather than a flow void. Flow-related enhancement and even echo rephasing are two processes that cause increased signal intensity within vascular structures. Such phenomena should be recognized as possible pitfalls in the diagnosis of intracranial aneurysms. Another less common pitfall in the diagnosis of basilar artery aneurysms is CSF pulsation artifact in the prepontine cistern, which can simulate a basilar artery aneurysm. This artifact is more pronounced with increased slice thickness.

Small aneurysms are well depicted using 3D time-of-flight (TOF) MRA. This is particularly true for aneurysms at arterial branch points (Fig. 14). Diagnostic interpretation of MRA studies should be based on review of both the original thin-section axial images and the maximum intensity projection (MIP) images derived from this source data. The spatial resolution of current 3D TOF MRA is slightly better than 1 1 1 mm, permitting detection of aneurysms as small as 2 to 3 mm. Aneurysms smaller than 3 mm are thought not to bleed and thus are of little clinical concern. On occasion, particularly with internal carotid artery lesions, the aneurysm neck may not be visualized. The use of targeted reconstruction, shorter TEs, and smaller voxels can substantially improve the quality of 3D TOF MRA exams. MRA should be considered complementary to conventional planar MRI scans, with the recommendation that both be acquired.

![Image of right middle cerebral artery bifurcation aneurysm detected on three-dimensional time-of-flight magnetic resonance angiography (MRA). The image presented is a maximum intensity projection derived from the thin-section axial three-dimensional MRA examination. Although aneurysms can be detected on conventional magnetic resonance imaging, as shown in FIGURE 13, MRA is far more sensitive for detecting small aneurysms.](image-url)
lesions and should be performed in all patients being evaluated for a possible intracranial aneurysm.

Conventional MRI routinely visualizes large intracranial aneurysms (1.0–2.5 cm in diameter) and giant aneurysms, which are defined as aneurysms larger than 2.5 cm in diameter (Fig. 15). These aneurysms commonly present with symptoms related to mass effect rather than subarachnoid hemorrhage. Large and giant aneurysms are often partially thrombosed and can be confused with an intraparenchymal hematoma. MRI provides an elegant, noninvasive method for diagnosing partially thrombosed giant intracranial aneurysms (Fig. 16). MRI is superior to CT and angiography in characterizing this type of aneurysm.

The MRI findings in partially thrombosed large or giant intracranial aneurysms include a flow void in the residual patent lumen of the aneurysm, with an adjacent high-signal-intensity rim. The rim is high intensity on both T1- and T2-weighted images and corresponds to extracellular methemoglobin. This finding contrasts with the formation of methemoglobin in an intraparenchymal hematoma, in which methemoglobin first forms at the periphery rather than centrally. Mixed, laminated signal intensity surrounds the high-signal-intensity methemoglobin rim, which represents different stages of organized clot in the thrombosed portion of the aneurysm. Perianeurysmal hemorrhage and adjacent edema within the brain may occur and can be distinguished from the aneurysm itself. Hemorrhage is typically high signal intensity on T1-weighted images and either hypo- or hyperintensity on T2-weighted images because of the presence of intracellular or extracellular methemoglobin. Edema within the adjacent brain is hypointense on T1-weighted images and hyperintense on T2-weighted images.

MRI can readily demonstrate complete thrombosis of large aneurysms. An old, organized thrombus will have a signal that is isointense with soft tissue or protein-containing fluid. Other soft tissue masses, including neoplasms, can have similar appearances and should be considered in the differential diagnosis. Patency or thrombosis of adjacent major intracranial vessels can be determined using MRA or, on conventional scans, by the presence or absence of arterial flow voids. MRI is also useful in evaluating aneurysm thrombosis after embolization.

MRI is contraindicated in the evaluation of the postsurgical patient with a ferromagnetic aneurysm clip. However, currently, most aneurysm clips are nonferromagnetic, and patients with these clips can be successfully imaged. Extreme care should be exercised in this area because at least one patient with a ferromagnetic clip is known to have died after MRI. This occurred despite a program at the site involved designed to differentiate ferromagnetic and nonferromagnetic clips.
Figure 15. Giant intracranial aneurysm of the left internal carotid artery. On the axial T2-weighted scan (A), a large predominantly low signal intensity mass is seen in the suprasellar region. The lesion is isointense with brain on the axial T1-weighted scan (B). C, Post-contrast, enhancement is marked and homogeneous on the axial scan. On the coronal precontrast T1-weighted scan (D), the lesion is predominantly low signal intensity. The variation of signal intensity with plane of acquisition (compare with B) is consistent with flow phenomena. On the coronal postcontrast scan (E), the intensity of the lesion is mixed, with much of the signal lost because of pulsation. A faint pulsation artifact can be identified in D, extending right to left across the scan. This artifact (arrows) is greatest on the postcontrast coronal scan (E), extending right to left and encompassing the entire height of the lesion. The imaging appearance of giant aneurysms on magnetic resonance imaging can be complex because of the presence of both flowing blood and thrombus (which may be layered). In the current case, there is no evidence of thrombus. The presence of pulsation artifacts, often accentuated on postcontrast scans, offers a clue to the nature of the lesion.

- **Vertebrobasilar Dolichoectasia**

A dolichoectatic vessel is one that is both too long (elongated) and too large (distended). Basilar artery elongation is present, by strict criteria, when the artery lies lateral to either the clivus or dorsum sellae or terminates above the suprasellar cistern. A basilar artery
larger than 4.5 mm in diameter is defined as ectatic (too large). The term "fusiform aneurysm" has, unfortunately, been used interchangeably in the scientific literature with dolichoectatic change and ectasia, all referring to diffuse tortuous enlargement and elongation of an artery. Dolichoectasia occurs with greatest frequency in the vertebrobasilar system (Fig. 17) but may also involve the intracranial internal carotid and middle cerebral arteries. A contour deformity of the pons resulting from basilar artery ectasia is a not uncommon incidental finding on MRI in the elderly population. Traction or displacement of cranial nerves can, however, lead to symptoms. Depending on the segment of the basilar artery involved, cranial nerve II, III, VI, VII, or VIII can be affected. The lower cranial nerves can be affected with vertebral artery involvement.

Symptomatic vertebrobasilar dolichoectasia exists in two different patient populations: those with isolated cranial nerve involvement and those with multiple neurologic deficits. The latter population includes patients with combinations of cranial nerve deficits (resulting from compression) and central nervous system deficits (resulting from compression or ischemia). A tortuous, Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals enhancement in only the more anterior and medial portions of the lesion (white arrow). Three-dimensional time-of-flight magnetic resonance angiography depicts a patent lumen within the mass corresponding in position to that suggested by the pulsation artifact and contrast enhancement. The majority of this giant aneurysm of the cavernous and distal petrous carotid artery is thrombosed. Only a crescent of residual lumen remains. The precontrast scans are misleading because the clotted portion of the aneurysm has very low signal intensity on the T2-weighted scan and intermediate to low signal intensity on the T1-weighted scan. but normal-caliber, basilar artery is more likely to produce isolated cranial nerve involvement, whereas ectasia is more likely to cause multiple deficits of either compressive or ischemic cause.
Figure 16. Partially thrombosed giant intracranial aneurysm. A large low-signal-intensity lesion is noted on the spin echo scan with intermediate T2-weighting (A) in the region of the left cavernous sinus. A pulsation artifact (black arrows) is seen extending in the phase encoding direction posteriorly from the lesion but originating from only the more medial portion.

- **Arteriovenous Malformation**

AVMs are the most common type of vascular malformation, occurring in approximately 0.1% of the general population. The clinical presentation is variable and includes headaches, seizures, neurologic deficits, and symptoms related to hemorrhage.
Occasionally, AVMs are discovered as incidental findings during the evaluation of an unrelated problem. All age groups are affected; most patients present with symptoms between the third and fourth decades.

AVMs occur throughout the central nervous system and are characterized pathologically by a direct communication between the arterial and venous circulations, without an intervening capillary bed. Intracranial AVMs are most commonly supratentorial (80%) and involve the peripheral branches of the middle cerebral artery. Angiographically, AVMs consist of a tangled nidus of dilated vessels supplied by enlarged tortuous feeding arteries and draining veins. Most commonly, the arterial supply of AVMs is pial, arising from the cerebral or cerebellar arteries. In some AVMs, there is a mixed pial-dural or dural blood supply. Half of the infratentorial lesions and approximately 20% of the supratentorial lesions have a dural component to their blood supply. Aneurysms are associated with the feeding arteries of AVMs in approximately 10% of patients.

SE MRI accurately defines the vascular channels forming AVMs (Fig. 18). Typically, the arteriovenous shunting is so rapid that most of the vessels appear as flow voids rather than with increased signal (Figs. 2-18 and 2-19). The latter is seen in slow flow and many normal veins. As with intracranial aneurysms, pulsation artifacts may be seen with AVMs. Pulsation artifacts become more pronounced on contrast-enhanced images. Feeding arteries are often easy to identify (because of location and dilatation). Draining veins can be identified by their caliber (larger than the arteries) and drainage into deep or cortical veins. After the administration of intravenous contrast, many of the larger vessels involved will show prominent enhancement (Fig. 20). However, this effect is variable from patient to patient depending on flow-rates and the pulse sequence used. Three-dimensional TOF MRA can be diagnostically useful in demonstrating feeding arteries, the nidus, and draining veins (Fig. 21). MRA has several problems, including signal void in tortuous feeding vessels (as a result of complex flow), nonvisualization of some draining veins (resulting from spin saturation), and difficulty in differentiation of flow from blood clot (methemoglobin). Conventional MRI accurately depicts and stages (in regard to age) intraparenchymal hematomas associated with AVMs (Fig. 22). MRI, unlike CT, is also very sensitive for the detection of superficial siderosis related to chronic subarachnoid hemorrhage. Superficial siderosis is frequently associated with vascular malformations.
Figure 17. Vertebrobasilar dolichoectasia. A and B, Precontrast T1-weighted axial scans reveal the vertebral and basilar arteries to be large in diameter, with the former causing a
deformity of the medulla and the latter a deformity of the pons. C and D, Postcontrast, the vertebral and basilar arteries demonstrate uniform enhancement and are thus more readily identified. E and F, On coronal postcontrast T1-weighted scans, the elongation of the vertebrobasilar system is clearly evident, with the basilar artery coursing lateral to the clivus and terminating above the suprasellar cistern.

Figure 18. Arteriovenous malformation, depiction on T2-weighted scans (A-C) and three-dimensional time-of-flight (TOF) magnetic resonance angiography (MRA). At the lower two anatomic levels (A and B), the T2-weighted scans reveal multiple enlarged draining veins (including the vein of Galen) as well as enlargement of the anterior and middle cerebral arteries. At the highest anatomic level shown (C), the scan reveals a large heterogeneous mass in the expected location of the right basal ganglia and thalamus. The mass consists of innumerable serpiginous structures, most with low signal intensity because of fast blood flow. D, The maximum intensity projection from the three-dimensional TOF MRA exam shows the branches of the right middle cerebral artery to be enlarged and draping around the vascular malformation. Several enlarged draining veins are also visualized.
Figure 19. Perimesencephalic cistern arteriovenous malformation depicted on conventional spin echo scans. On proton density (A), T2-weighted (B), and T1-weighted (C) precontrast scans, a cluster of abnormal vessels is seen posterior and to the left of the pons, compressing the cerebral aqueduct. The vessels are low signal intensity on all sequences because of fast flow. The occipital horn of the lateral ventricle is dilated as a result of chronic compensated obstructive hydrocephalus.

Figure 20. Arteriovenous malformation (AVM). On the T2-weighted scan (A), a large lesion is noted in the left frontal lobe; mixed high and low signal intensity is suggestive of flow. Tubular-like signal voids are present on the precontrast T1-weighted scan (B). C, Postcontrast, a large enhancing nidus is identified. Also seen is enhancement of multiple large draining veins. AVMs are well depicted on conventional planar spin echo magnetic resonance images because of flow phenomena. On precontrast scans, multiple serpiginous structures can be identified, most with low signal intensity because of rapid flow. After intravenous contrast administration, enhancement can be noted in areas of slower flow, particularly within draining veins.

In contrast to congenital AVMs, pure dural-based AVMs are often secondary to trauma or inflammatory disease. These lesions drain into the venous sinuses or cortical veins and commonly have associated intracranial or subarachnoid hemorrhage. Planar MRI, without the use of MRA, can have difficulty in detecting lesions adjacent to the inner table of the
skull because vascular flow and cortical bone both appear as signal voids. CT also has difficulty in detecting such lesions. Hemorrhage complicating these lesions is clearly seen.

AVMs can have calcified components; CT is more sensitive in detecting these than MRI. Dense calcification has no mobile protons and appears as a signal void on MRI scan, which can be confused with flowing blood. In difficult cases in which accurate characterization of calcification, blood flow, and hemorrhagic components is desired, gradient echo techniques may be used as a helpful adjunct to SE imaging. On these sequences, flowing blood generally has increased signal intensity and can be distinguished from calcification or hemosiderin. Occasionally, however, flowing blood can have low signal intensity on gradient echo imaging as a result of turbulent, in plane, or very slow flow, and this potential pitfall should be recognized. Gliosis, edema, or ischemia can involve the brain adjacent to an AVM. These parenchymal changes are best detected as abnormal high signal intensity on T2-weighted images. Although this high signal intensity is nonspecific, the absence of a soft tissue mass favors a benign process. AVMs also typically do not have substantial associated mass effect (unless accompanied by parenchymal hemorrhage).

Figure 21. Thalamic arteriovenous malformation (AVM), best visualized on three-dimensional time-of-flight (TOF) magnetic resonance angiography (MRA). A, The precontrast T2-weighted scan reveals abnormal iron deposition (with low signal intensity) in the right thalamus, along the border of the right lateral ventricle, and surrounding an old lacunar infarct in the right putamen. B, The precontrast T1-weighted scan reveals slight ex vacuo dilatation of the right lateral ventricle. C, The maximum intensity projection image from the three-dimensional TOF MRA reveals an abnormal tangle of vessels (AVM, arrow) just to the right of the midline and medial to the right posterior cerebral artery. Viewing the T2-weighted scan in retrospect, several abnormal vessels can be identified because of their flow voids medial to the hemosiderin staining along the margin of the lateral ventricle.
Figure 22. Small frontal lobe arteriovenous malformation (AVM) presenting clinically with intraparenchymal hemorrhage in a pediatric patient. T2- (A) and T1-weighted (B) precontrast scans reveal a large left frontal mass with marked hypointensity on the T2-weighted image and hyperintensity centrally on the T1-weighted image (the signal characteristics of intracellular methemoglobin). Surrounding cerebral edema, with high signal intensity, is well depicted on the T2-weighted scan. Just lateral and anterior to the primary lesion, a second smaller serpiginous lesion is noted (black arrow, A). This has low signal intensity on both T2- and T1-weighted precontrast scans. The same area (white arrow) enhances on the postcontrast T1-weighted scan (C). As with larger lesions, this small AVM is characterized by flow voids precontrast and enhancement postcontrast. Prospective identification on precontrast scans is difficult because of the lesion's small size and the large adjacent hematoma. The AVM was confirmed by x-ray angiography.

Included in the spectrum of AVMs is a rare congenital anomaly, the vein of Galen aneurysm. Dilatation of the vein of Galen occurs if there is a downstream venous obstruction and increased flow through the vein of Galen secondary to an arteriovenous shunt. Hydrocephalus often develops. Infants usually present with cardiac failure, and older children present with hydrocephalus and increased intracranial pressure. MRI is useful in defining the anatomic extent of the abnormality and in evaluating blood flow patterns or thrombus within the aneurysm. Preoperative angiography remains essential because precise identification of the feeding arteries is necessary.

Multiple intracranial AVMs may be seen in patients with Wyburn-Mason's syndrome and Osler-Weber- Rendu disease. Wyburn-Mason's syndrome is rare and consists of a midbrain AVM, a facial cutaneous nevus in the distribution of the trigeminal nerve, and a retinal angioma ipsilateral to the facial nevus. MRI can noninvasively evaluate the retinal and midbrain components of this syndrome.

- **Venous Angioma**

Venous angiomas are vascular malformations involving only the venous side of the circulation. They occur throughout the central nervous system but are most common in the
frontal lobes and posterior fossa (Fig. 23). Patients with venous angiomas are most often asymptomatic. Although it was previously believed that venous angiomas had a high propensity to bleed, this is now generally regarded as an incidental finding. Venous angiomas consist of a group of dilated medullary venous tributaries, often arranged in a radial "spoke-wheel" pattern, draining into a large vein. This large transparenchymal vein drains into a venous sinus, a cortical vein, or a subependymal ventricular vein.

On SE MRI, venous angiomas appear as tubular flow voids with a radial configuration in the white matter. The enlarged draining vein and its site of drainage are often also visualized. Both T1- and T2-weighted images are used to detect venous angiomas; T2-weighted sequences are often more sensitive compared with precontrast T1-weighted scans. Because of slow venous flow, which can cause increased intravascular signal, these lesions may be inapparent (isointense to adjacent brain) on some imaging sequences. In particular, periventricular lesions can be difficult to identify on precontrast scans alone. Venous angiomas are best visualized on contrast-enhanced scans (Fig. 24).

Included in the spectrum of venous malformations is the Sturge-Weber syndrome (encephalotrigeminal angiomatosis). This syndrome consists of a cutaneous facial nevus (port-wine stain), usually in the ophthalmic distribution of the trigeminal nerve, ipsilateral leptomeningeal angiomatosis, and ipsilateral cortical atrophy with linear cortical gyral calcifications in a tram-track configuration. Dilated deep venous collaterals provide abnormal drainage. The leptomeningeal angiomatosis displays marked contrast enhancement. Gradient echo imaging can be useful for depiction of the cortical gyral calcifications.

Figure 23. Infratentorial venous angioma. Precontrast T2- (A) and T1-weighted (B) scans reveal a linear, tubular flow void within the right cerebellar hemisphere. This is better seen on the T1-weighted scan, in which there is also a suggestion of feeding branches. There is no surrounding edema or associated parenchymal abnormality. C, The postcontrast T1-weighted scan reveals intense enhancement of the lesion (black arrow), with improved visualization of both the caput of dilated medullary veins and the large central draining vein.
Figure 24. Supratentorial venous angioma. Two small round lesions with decreased signal intensity are noted in the right frontal lobe on the first (A) and second (B) echoes of the T2-weighted scan. These two lesions have intermediate signal intensity on the precontrast T1-weighted scan (C). The signal characteristics are compatible with hemosiderin. Abnormal contrast enhancement of numerous tiny veins and a solitary large draining vein (arrow) is identified in the right frontal lobe on the axial T1-weighted postcontrast scan (D). The solitary draining vein extends to the midline and was noted on other images (not shown) to drain into the superior sagittal sinus.
A cavernous angioma (cavernous malformation or cavernous hemangioma) is a collection of endothelial-lined vascular spaces with no intervening brain parenchyma between these vessels. These lesions occur throughout the central nervous system but are more common in a supratentorial, subcortical location. Cavernous angiomas are multiple in as many as 33% of cases. These lesions are usually asymptomatic, but some patients present with seizures. There are two forms of cavernous angioma: sporadic and familial. The familial form has a high incidence of multiple lesions, is autosomal dominant in transmission, and appears to have an increased frequency in Mexican American families. Cavernous angiomas display a well-defined low-signal-intensity border, caused by hemosiderin deposition, on T2-weighted images (Fig. 25). Gradient echo imaging, using sequences with high sensitivity to T2* (susceptibility), often reveal more lesions than conventional imaging (in patients with multiple lesions). The internal architecture of cavernous angiomas is complex because of repeated hemorrhage.

Multiple hyperintense, and often hypointense, round areas are seen separated by low signal intensity septations on both T1- and T2-weighted images. Because of the presence of large vascular spaces within the lesion, cavernous angiomas enhance after administration of intravenous contrast.

Capillary telangiectasias (capillary angiomas)

are small, solitary lesions frequently found in the pons. In contrast to cavernous angiomas, capillary telangiectasia consists of dilated capillaries with intervening brain parenchyma between the vessels. Most of these lesions are asymptomatic clinically but can occasionally be associated with hemorrhage.
• **Occult Cerebrovascular Malformations**

Any of the four previously described pathologic entities comprising cerebrovascular malformations can be categorized as an OCVM if the lesion is angiographically occult. MRI has become the primary screening modality for detection because of its exquisite ability to visualize the components of hemorrhage.

After an acute hemorrhage, OCVMs may be difficult to distinguish from an intraparenchymal hematoma, particularly those caused by neoplasm. Beyond this acute stage, OCVMs can be distinguished from hematomas of other causes by their continuous hemosiderin rim, absence of parenchymal mass effect or edema, location of the lesion, and expected temporal evolution of hemorrhage in a simple hematoma.

The detection of very small OCVMs by MRI depends on identifying the characteristic circumferential low-intensity hemosiderin rim. High-field MRI and gradient echo techniques are more sensitive in detecting hemosiderin and, therefore, OCVMs. Routine SE MRI may miss small OCVMs that can be detected by CT because of the presence of small focal calcifications.

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**References**


INTRODUCTION

Since the early 1980s, magnetic resonance imaging (MRI) has been the technique of choice for visualizing white matter lesions in the brain. This is especially true for the plaques found in multiple sclerosis (MS). Careful review of scans and the use of pattern recognition are critical for differential diagnosis. Although sensitive to disease, MRI cannot always provide a specific diagnosis. Clinical presentation is then critical for disease differentiation.

Other disease entities can easily be confused on MRI with MS. In MS, periventricular changes are typically punctate and asymmetric in distribution (from side to side). Postmortem studies have confirmed that the white matter abnormalities demonstrated by MRI correspond to MS plaques. Edema associated with acute lesions and gliosis with
chronic lesions permit visualization. Demyelination by itself does not contribute significantly to alterations in proton density or relaxation, and thus is not directly visualized with conventional imaging techniques. In chronic small vessel ischemic disease, which can mimic MS, periventricular changes are often milder and smoother in contour. However, the white matter changes found in some patients closely resemble those of advanced MS.

In very ill, uncooperative patients with intracranial infection, computed tomography (CT) can be superior to MRI because of its shorter imaging times. However, MRI offers the advantage of direct, high-resolution, multiplanar imaging with superior sensitivity to inflammatory change. In a mature abscess, the capsule can often be differentiated from inner debris and surrounding edema on unenhanced MRI. CT offers advantages in detecting calcifications, such as those associated with chronic infections (e.g., cysticercosis) and end-stage congenital infection. However, MRI is more sensitive to parenchymal hemorrhage regardless of stage. MRI can detect hemorrhage long after CT scans become normal, allowing more complete characterization of certain infectious diseases. With meningeal disease, enhanced MRI is more sensitive than enhanced CT. In the encephalitides (e.g., herpes simplex type 1), MRI can also reveal widespread abnormalities simply not seen on CT.

MRI is favored in almost all instances over CT for the evaluation of patients with suspected white matter disease or intracranial infection. CT should be considered only if the detection of calcifications is important for diagnosis. MRI's strength lies in its superior demonstration of soft tissue abnormality because of its ability to gauge tissue water. In common with CT, differential diagnosis is largely based on the pattern of disease involvement.

**WHITE MATTER DISEASE**

- **Multiple Sclerosis**

MS is characterized clinically by multiple neurologic episodes separated in time. Two thirds of patients are female. The disease progresses in a relentless stepwise fashion, marked by exacerbations and remissions. MS is highly variable in its course. A study from the Mayo Clinic documented that 75% of patients were alive 25 years after onset, 55% without significant disability. McAlpine's scale, based on clinical criteria, defines definite MS as that with characteristic transient neurologic symptoms and one or more documented relapses. Probable MS is defined as that with one or more attacks of disease and clinical evidence in the first attack of multiple lesions. Possible MS is defined as that with a similar history to probable disease but with a paucity of findings or unusual features. Dictation of films should avoid use of this terminology (definite, probable, or possible MS), a standard in the practice of neurology and based on clinical criteria alone. MRI is extremely sensitive for the detection of MS plaques in the brain and spinal cord. However, clinical assessment continues to be crucial for appropriate diagnosis.
The diagnosis of MS by MRI hinges on pattern recognition. Most lesions are small, 1 to 5 mm in diameter. The most common location of MS plaques is in the periventricular region, particularly adjacent to the superolateral angles of the lateral ventricles (Fig.1). There is often a marked asymmetry in lesion distribution (comparing lesions in the right and left hemispheres), a factor distinguishing it from ischemic disease, which is often encountered in the elderly patient. Other common locations for lesions include the centrum semiovale, atrial trigone, occipital horns, forceps major and minor, colliculi, and temporal horns (Fig.2). Approximately 30% of patients demonstrate brainstem and cerebellar lesions; the middle cerebellar peduncles are a preferred location. Corpus callosum involvement by MS is common (Fig.3). Thirty percent of patients demonstrate focal lesions in this location, a percentage established both by imaging studies and pathologic exam. Callosal lesions with a flat border along the ependymal surface of the ventricles and otherwise a round or oval shape are relatively specific for MS. These are best visualized on sagittal images. Focal or diffuse atrophy of the corpus callosum is seen in 40% of patients. Thinning of the corpus callosum results from general cerebral atrophy and accompanying wallerian degeneration. Changes in the corpus callosum are most prominent in patients with long-standing and extensive disease. Although MS is commonly thought of as a white matter disease, 5% to 10% of plaques occur in gray matter. These can be seen in the cortex and in the basal ganglia. There are many pitfalls in the MRI diagnosis of MS. Differentiation from other clinical entities that mimic MS on MRI depends on pattern recognition and correlation with clinical history.
Figure 1. Multiple sclerosis (characteristic lesion locations). T2-weighted scans from two patients, one man and one woman, both 38 years old, are presented. Each patient had intermittent weakness and numbness of the upper and lower extremities as well as problems with balance. Multiple punctate high-signal-intensity lesions are noted, located predominantly in the white matter. Lesions can be identified in the medulla (A), in the pons and middle cerebellar peduncle (B), adjacent to the temporal horn (C, arrow), and in the white matter immediately adjacent to the lateral ventricles (D and E). Only the periventricular and supraventricular lesions were clearly seen on the T1-weighted images (not shown). In both patients, there was no abnormal enhancement noted on the postcontrast exam (not shown).

Fast spin echo scans with moderate T2-weighting and fluid-attenuated inversion recovery (FLAIR) scans with heavy T2-weighting are preferred for visualization of MS plaques in the brain. Both techniques depict lesions as high-signal-intensity foci, contrasting well against a background of intermediate to low-signal-intensity brain and cerebrospinal fluid (CSF). Conventional heavily T2-weighted scans should not be used. These fail to detect some MS lesions because of their proximity to high-signal-intensity CSF. The primary plane for imaging is axial. This choice is often supplemented by T2-weighted scans in the sagittal and coronal planes. The use of thin sections, 5 mm or less, is critical for lesion detection, minimizing partial volume effects.

MS plaques are characterized by prolonged T1 and T2 relaxation times and increased proton density. MS plaques are low signal intensity on T1-weighted scans and high signal intensity on T2-weighted scans. T1-weighted scans are poor for lesion visualization, unless heavily T1-weighted FLAIR scans are used. Even with these, only lesions entirely circumscribed by normal white matter are well seen. T1-weighted scans, regardless of technique, are insensitive to lesions adjacent to the ventricles or gray matter, because of the lack of contrast with these structures. T2-weighted scans, which depict plaques as high-signal intensity foci compared with adjacent normal brain, are preferred for lesion detection. For visualization of brainstem and cerebellar lesions, compensation by software techniques (such as gradient moment nulling) for CSF motion is important. MRI is markedly more sensitive than CT for detection of lesions, regardless of location. CT detects only larger lesions. Less severe disease is undetected by CT, with the scan appearing normal.
Figure 2. Multiple sclerosis (other characteristic lesion locations and imaging appearances). T2-weighted scans are shown from a 32-year-old white woman with a 10-year history of disability. The patient initially presented with fatigue and unsteadiness. Clinical exacerbation of disease led to two previous hospital admissions. Ataxia of all extremities was noted 3 years before the current admission; the patient became wheelchair bound 1 year later. The patient now presents with increasing numbness of the extremities and urinary incontinence. However, neurologic exam does not reveal evidence of a new focal brain lesion. Lesions (which are predominantly punctate in configuration) are noted in the right cerebellar hemisphere (A), in the left pons and superior colliculus (B), and immediately adjacent to the lateral ventricles (C), with asymmetry of disease involvement when comparing the right and left hemispheres. Because of the large number of plaques immediately adjacent to the lateral ventricles, the disease appears somewhat confluent in this region. Other scans (not shown) revealed mild diffuse cortical atrophy and thinning of the corpus callosum. No abnormal enhancement was noted on postcontrast T1-weighted scans (not shown).
Figure 3. Multiple sclerosis (involvement of the corpus callosum). T2-weighted scans are shown from an 18-year-old woman with new onset of left lower extremity paresthesia, which progressed to include the left upper extremity. A few days later, abnormal sensation developed in the right lower extremity. A and B, At the level of the lateral ventricles, at least four periventricular lesions (white arrows) are seen. The lesions lie medial to the lateral ventricle and thus lie within the corpus callosum. On a sagittal scan with intermediate T2-weighting (C), the larger of the callosal lesions is well seen (black arrow). On the postcontrast exam (not shown), several other larger lesions were noted to enhance along with a cord lesion at C2.

Acute MS lesions tend to be large, greater than 1 cm, with indistinct margins. Well-demarcated, small punctate lesions are much more common, and for the most part correspond to chronic (quiescent) disease. Lesions larger than 1 cm can represent confluent plaques of different ages or clinically active disease. Both acute and chronic lesions have high signal intensity on T2-weighted scans. In acute disease, this corresponds to edema. In chronic disease, this corresponds to gliosis. Demyelination per se does not contribute significantly to the change in relaxation time.

MS plaques are also not necessarily homogeneous in appearance. The border of a lesion can, on occasion, be differentiated from the center on precontrast scans, an appearance more common with acute plaques. A thin line of moderately high signal intensity (T1 shortening) can be seen at the edge of some MS lesions on T1-weighted images. Postcontrast, this line corresponds to the edge of the enhancing region. On tissue pathology, an accumulation of myelin breakdown products is found in this region at the edge of active lesions.

The vast majority of MS plaques remain unchanged on follow-up MR scans. However, new lesions are often observed with the apparent resolution of older lesions. Confluent abnormalities in the periventricular region correlate with long-standing disease. Periventricular disease, when severe, has a characteristic irregular, "lumpybumpy" outer margin. This feature can be useful to distinguish MS from small vessel ischemic disease. The latter typically has a smooth outer margin in the immediate periventricular region. Involvement of the periventricular white matter in MS is also often markedly asymmetric, when the left hemisphere is compared with the right.

MRI is commonly used to assess disease activity and the effectiveness of medical therapy. Patients with more severe disease have a larger number of plaques and more confluent white matter disease. Thinning of the corpus callosum and generalized parenchymal atrophy are also seen in long-standing disease. Many of the lesions depicted by MRI are clinically silent. Consequently, MRI is more sensitive for detecting disease and demonstrating disease activity than physical examination. Studies with experimental allergic encephalomyelitis (EAE), an animal model of demyelinating disease, have advanced substantially our knowledge of imaging-pathologic correlation.

With regard to contrast administration, it is the minority of patients with MS who demonstrate enhancing lesions. The majority of lesions visualized on MRI are chronic in
nature and thus do not enhance. Results from clinical trials reveal that contrast enhancement is more sensitive than clinical exam in detecting active disease. Enhancement after contrast administration is a consistent finding in new lesions. MS is a dynamic disease; lesions demonstrate dramatic changes during longitudinal study. Lesion enhancement is best seen on scans obtained within 5 to 10 minutes after contrast injection. Lesion enhancement is transient, persisting for fewer than 4 weeks in most cases. Some lesions demonstrate punctate enhancement (Fig.4) and others ring enhancement (Fig.5). Evolution in appearance, over days to weeks, from punctate to ring-like enhancement, has also been observed. Serial scans reveal some lesions reverting to normal signal intensity on T2-weighted images, suggesting resolution of transient inflammatory changes.

MS plaques can also be visualized in the cervical and thoracic spinal cord. These lesions often do not respect gray-white matter boundaries, nor do they follow specific fiber tracks. Lesions are often elongated, paralleling the axis of the cord, and are more common dorsally and laterally within the cord. Before the advent of MRI, spinal cord lesions were rarely demonstrated radiologically. Cervical lesions are detected more commonly by MRI than thoracic lesions, a finding that may be related to technique. Imaging of the thoracic cord is still inferior to that of the cervical cord because of differences in coil design and problems caused by respiratory and cardiac motion. T2-weighted imaging in both the sagittal and axial planes is recommended to confirm the presence of lesions. Although lesions can be demonstrated in the cervical and thoracic cord, brain MRI is advocated (in addition to spine imaging) for the evaluation of patients with primarily spinal cord symptoms. As an imaging modality, MRI is more sensitive for the detection of brain lesions in MS than spinal cord lesions. Furthermore, the demonstration of characteristic periventricular plaques can confirm the diagnosis of MS, whereas spinal imaging may reveal only one or two nonspecific lesions.

![Image of brain MRI scans](image_url)

**Figure 4.** Multiple sclerosis (active disease). Bilateral punctate high-signal-intensity white matter lesions are noted in the periventricular white matter and in the body of the corpus callosum on the T2-weighted scan (A). These findings are consistent with the diagnosis of multiple sclerosis. B, The precontrast T1-weighted exam identifies only a few of these
abnormalities. C, Postcontrast, several lesions demonstrate abnormal enhancement, signifying active disease. Contrast enhancement plays a specific role in multiple sclerosis for the demonstration of active lesions and for monitoring response to therapy.

Figure 5. Multiple sclerosis (MS) mimicking metastatic disease. On the sagittal heavily T2-weighted fast spin echo scan (A), multiple periventricular high-signal-intensity abnormalities are noted. Some involve the corpus callosum and have a broad base along the border of the lateral ventricle. The distribution of the lesions in the periventricular white matter is confirmed on the axial scan with intermediate T2-weighting (B). On the corresponding postcontrast T1-weighted scan (C), many of the lesions demonstrate ring enhancement. Focusing on the postcontrast exam alone, the multiplicity of lesions and ring enhancement could lead to an incorrect diagnosis of metastatic disease. The knowledge that MS plaques can demonstrate ring enhancement, together with recognition of the characteristic location of these lesions, leads to the proper diagnosis. The availability of pertinent clinical history is also paramount to film interpretation.

- **Optic Neuritis**

For the study of patients with optic neuritis, both a screening examination of the brain and an examination focusing on the optic nerves are recommended. The actual demonstration of optic nerve lesions can be difficult, demanding attention to imaging technique. With good technique, optic nerve lesions are seen in more than 90% of symptomatic patients. However, visual evoked potentials remain more sensitive for isolated optic nerve lesions. Disseminated areas of demyelination in the brain can also be observed in patients with optic neuritis in a pattern similar to MS. The frequency with which patients with isolated optic neuritis subsequently acquire MS remains controversial.

The use of fat suppression is particularly important for the study of the optic nerves. Surrounding orbital fat impedes recognition of optic nerve lesions because of chemical-shift artifact and loss of lesion contrast. T2-weighted scans with fat suppression reveal nerve enlargement and edema. Postcontrast T1-weighted scans with fat suppression show abnormal contrast enhancement of the nerve.
**Small Vessel Ischemic Disease**

Patchy white matter lesions, or small vessel ischemic disease, common in elderly patients and those with cerebrovascular disease, must be differentiated on MRI from MS. The lesions can be periventricular in location or situated more peripherally (Fig. 6). Involvement in the two hemispheres is usually relatively symmetric (Fig. 7). This is different from MS, in which involvement is often markedly asymmetric. When periventricular in location, the exterior margin of the involved region is often relatively smooth, providing another key for differentiation from MS.

Twenty percent to 30% of elderly patients in good general medical health demonstrate patchy white matter lesions on brain MRI. These correspond on postmortem study to areas of gliosis and demyelination, presumably caused by chronic vascular insufficiency. Larger lesions may demonstrate necrosis, axonal loss, and demyelination, thereby representing true infarcts. These lesions and those of frank infarction account for the majority of focal white matter lesions seen on MRI in the elderly population. CT commonly fails to reveal these abnormalities. The patchy white matter lesions seen in the elderly population should be distinguished from focal gliosis and encephalomalacia surrounding ventricular shunts.

In most patients, some degree of periventricular hyperintensity can be recognized on MRI. A fine line of high signal intensity adjacent to the ventricular system, often more prominent surrounding the frontal horns, should be considered a normal finding and not indicative of demyelinating disease or hydrocephalus. This pattern must be distinguished from that seen with transependymal flowing obstructive hydrocephalus (Fig. 8).

![Figure 6. Small vessel ischemic disease with predominantly punctate lesions. The patient is a 72-year-old man with multiple medical problems. Numerous foci of increased signal intensity are present in the white matter (primarily the subcortical white matter, a distinguishing factor from multiple sclerosis) on the first (A) and second (B) echoes of the axial T2-weighted scan. The lesions are not clearly seen on the axial T1-weighted scan (C). Note the poor gray-white matter contrast on both the T1- and T2-weighted images. There was no abnormal contrast enhancement (not shown).](image-url)
Figure 7. Small vessel ischemic disease, a mixture of punctate, and less well-defined white matter lesions. Multiple foci of abnormal high signal intensity are noted on the T2-weighted scan (A) in the subcortical and periventricular white matter. The abnormal areas correspond pathologically to necrosis, infarction, demyelination, and astroglial proliferation. The lesions adjacent to cerebrospinal fluid are better seen on the fluid-attenuated inversion recovery scan (B). Note that the involvement is very symmetric, from side to side, one distinction from the typical imaging presentation with multiple sclerosis. The lesions are poorly visualized on the T1-weighted scan (C) and do not demonstrate enhancement on the postcontrast scan (D).
Figure 8. Transependymal cerebrospinal fluid (CSF) flow. There is dilatation of the lateral ventricles on the intermediate (A) and heavily (B) T2-weighted scans. The patient is a 5-year-old girl who received radiation therapy for a brainstem glioma (not shown). A thick, smooth rim of periventricular white matter hyperintensity is identified surrounding the lateral ventricles, best seen on the scan with intermediate T2-weighting (A). This involves only the periventricular white matter and does not extend into the basal ganglia. Ventricular size and periventricular signal intensity were normal on the axial T2-weighted scan (C) performed 45 days earlier. At that time, there was no obstruction to CSF flow. The brainstem lesion subsequently hemorrhaged, enlarging and obstructing CSF outflow.

- **Systemic Lupus Erythematosus**

As with most other injuries to the brain, MRI demonstrates high sensitivity to the lesions of systemic lupus erythematosus (SLE). Patients with SLE demonstrate a broad range of disease involvement, from perivascular microinfarctions to discrete cerebral infarction. Partial or complete resolution of gray matter lesions can be seen on follow-up exams. The wedge shape of lesions in many patients and involvement of both gray and white matter assist in differentiation from MS. MRI is an important modality for detecting the extent of cerebral injury in SLE; CT is much less sensitive.

- **Hypoxemic Injury**

Hypoxemic (subnormal oxygenation of arterial blood) injury to the brain can be the result of decreased concentration of functional hemoglobin (anemic hypoxia), hypoperfusion (ischemic hypoxia), or defective oxygenation (hypoxic hypoxia). Causes include carbon monoxide poisoning, cardiopulmonary arrest (Fig.9), and near-drowning. All can produce irreversible brain damage. The white matter diseases discussed previously should not be confused with ischemic damage resulting from hypoxemia. Cortical gray matter, basal ganglia, and deep white matter are commonly involved. Care should be exercised in interpreting scans in the infant, when the question of hypoxic injury is raised, because of the normal prolonged T1 and T2 values of immature (nonmyelinated) white matter (Fig. 10).
Figure 9. Hypoxemic injury (infarction). A, Abnormal high signal intensity is noted bilaterally on the T2-weighted scan in the putamen, globus pallidus, and caudate nuclei. There is also patchy increased signal intensity in cortical gray matter. This is most prominent on the patient's left side, in the watershed regions between the anterior and middle cerebral artery territories, and between the middle and posterior cerebral artery territories. Findings are similar, but less evident, with abnormal low signal intensity on the T1-weighted scan (B). The patient presented for imaging several days after respiratory arrest.

The brain is not affected uniformly in hypoxemic injury. Gray matter (neurons) is more vulnerable than white matter; watershed zones between arterial circulations are particularly vulnerable. Highly susceptible regions include the hippocampus, cerebral cortex, cerebellum, caudate, and putamen. The globus pallidus, thalamus, hemispheric white matter, and brainstem are less susceptible but may also be involved.

- **Periventricular Leukomalacia**

Periventricular leukomalacia (PVL) is the result of white matter hypoperfusion in watershed areas in the premature infant, which progresses to infarction. Clinical sequelae include spastic diplegia, quadriplegia, cerebral palsy, and mental retardation (in severe cases). MRI is often performed in the young child, visualizing chronic end-stage changes. These include decreased quantity of periventricular white matter and abnormal increased signal intensity (on T2-weighted images) in the adjacent white matter. The latter corresponds to gliosis. The areas most commonly affected include the white matter adjacent to the atrial trigone and frontal horn. Focal or generalized ventricular enlargement can be seen as a result of ex vacuo dilatation. There may also be thinning of the corpus callosum. Although neurosonography is used for evaluation of the neonate, the sensitivity of this modality is low in mild or moderate disease. Follow-up MRI in
symptomatic infants can confirm the diagnosis of PVL despite a negative neonatal ultrasound examination. The pattern of white matter involvement in PVL in the young child can resemble that of small vessel ischemic disease in the elderly. Age and clinical history clearly differentiate these two populations.

![Figure 10. Global hypoxia in the neonate. Hypoxemic injury (infarction) can be easily missed in the infant, particularly when it is symmetric in distribution, if one is not familiar with normal myelination and its appearance on magnetic resonance imaging. In the neonate, white matter on a T2-weighted scan has higher signal intensity than gray matter, a reversal of the normal adult pattern. However, this is not as high as the abnormal signal intensity seen in this neonate on the T2-weighted scan (A). Another striking finding is how thin the gray matter mantle is on both the T2- and T1-weighted scans. In the neonate, peripheral white matter is normally low signal intensity on a T1-weighted scan but not as low as seen in B. Also, the posterior limb of the internal capsule should be high signal intensity, as a result of myelination, but is not in this infant (because of edema).](image)

- **Toxic Demyelination**

Of the demyelinating diseases resulting from problems with nutrition or metabolites (with the exception of inborn errors of metabolism), central pontine myelinolysis (CPM) and Wernicke's encephalopathy are two that demonstrate characteristic findings on MRI. In CPM, there is symmetric destruction of myelin sheaths, which appears to start from the median raphe of the pons. The lesion can involve part of or the entire base of the pons. Contiguous spread into the dorsal pons (tegmentum) and superiorly into the mesencephalon (midbrain) has been reported. The cause is believed to be an osmotic injury secondary to rapid correction of severe chronic hyponatremia. In Wernicke's encephalopathy, there is involvement of the periventricular structures at the level of the third and fourth ventricles. Patients with classic Wernicke's encephalopathy exhibit
confusion, nystagmus (less commonly ophthalmoplegia), and truncal ataxia. These clinical findings reflect the localization of the lesions pathologically. MRI reveals lesions in these characteristic locations (Fig. 11). Untreated, Wernicke's encephalopathy is a progressive disease. The administration of thiamine reverses the disease over the course of days to weeks, although mortality even with treatment is 10% to 20%.

Figure 11. Wernicke's encephalopathy. T1-weighted scans pre(A) and postcontrast (B) are shown. Magnetic resonance findings include symmetric periventricular lesions that are hyperintense on T2-weighted scans and enhanced after contrast administration (in the acute phase) on T1-weighted scans. Bilateral involvement of the mammillary bodies, as seen in this case with enhancement postcontrast (arrows), is characteristic. This uncommon disorder is caused by thiamine deficiency. Clinical diagnosis is difficult; the disease is characterized by ophthalmoplegia, ataxia, and disturbances of consciousness. These clinical signs may or may not be present. Wernicke's encephalopathy is due to malnutrition or malabsorption (often after prolonged alcohol intake).

- **Radiation Injury**

Symmetric periventricular white matter hyperintensity on T2-weighted scans is a typical finding in radiation injury to the brain (Fig. 12). MRI evidence of injury is more likely to be seen in older patients, in cases involving higher radiation dose (and larger volume of radiated tissue), and when radiation is combined with chemotherapy. The injury to white matter by radiation consists of demyelination, edema, and fibrillary gliosis. The pattern may be focal, if radiation is restricted to a port, or diffuse. In diffuse disease, involvement of the white matter may extend to the interface with cortical gray matter. The scalloped appearance of radiation injury at the gray-white matter junction represents extensive white matter damage involving the more peripheral arcuate fibers. This pattern can be differentiated from transependymal absorption, which does not extend to the gray-white matter junction and demonstrates a sharp, rounded margin. The corpus callosum is usually spared in radiation injury. Diffuse white matter disease can also be caused by inhalation of organic solvents. However, uniform involvement of both central and peripheral white matter is more characteristic of radiation injury. Radiation-induced changes can mask recurrent tumors and other pathologic findings. MRI demonstrates high sensitivity to radiation-induced changes but low specificity. CT is relatively insensitive for detecting radiation damage; visualization of abnormalities is confined primarily to patients...
with severe disease. Both MRI and CT demonstrate the late sequelae of radiation therapy, which include sulci enlargement and ventriculomegaly. Abnormal contrast enhancement is seen in areas of radiation-induced necrosis (Fig. 13). MRI, like CT, lacks specificity in discriminating recurrent tumor from radiation necrosis (using conventional imaging sequences). Both are seen as focal enhancing lesions with surrounding edema. First-pass studies, acquired during bolus intravenous contrast injection, do, however, permit differentiation of these two entities. Classically, radiation necrosis demonstrates very low cerebral blood volume (CBV), whereas recurrent tumor manifests high CBV.

Figure 12. White matter changes as a result of therapeutic radiation. A and B, There is diffuse symmetric white matter hyperintensity on the T2-weighted scans. The involvement extends to the cortical gray matter and is scalloped laterally. The corpus callosum is spared. The white matter changes are typically accompanied by cortical atrophy, also present in this case. C and D, The atrophy is clearly seen on T1-weighted scans; the diffuse abnormality of white matter is less evident. Another typical finding is loss of gray-white matter differentiation, which is also present in this case.
Figure 13. Radiation necrosis. This 65-year-old patient underwent resection of a right temporal lobe glioblastoma followed by stereotactic radiation therapy (7 months before the current scans). A, On T2-weighted scan, there is abnormal high signal intensity in the right temporal lobe, confined mainly to white matter, consistent with edema. B, The T1-weighted scan demonstrates mass effect, with sulcal effacement and compression of the frontal horn and atria of the right lateral ventricle. C, Postcontrast, a large enhancing mass is noted within the area of edema defined on the T2-weighted scan. In the absence of a cerebral blood volume study (which can be acquired on magnetic resonance imaging during bolus contrast administration), an enhancing mass such as this could represent either recurrent tumor or radiation necrosis. On conventional scans such as that shown, there are no differentiating factors. The actual histologic diagnosis in this case, established by biopsy, was a mixture of recurrent tumor and radiation necrosis.

- Dilated Perivascular Spaces

Dilated perivascular spaces are a normal finding on MRI. They occur in three common locations. The perivascular space is an invagination of the subarachnoid space. Also known as the Virchow- Robin space, it surrounds perforating arteries entering the brain and contains CSF. The most common location for a dilated perivascular space is within the inferior one third of the basal ganglia adjacent to the anterior commissure and following the course of the lenticulostriate arteries. In this location, they are usually smaller than 5 mm in diameter but can be larger. Another common location is within the high convexity white matter of the centrum semiovale following the course of nutrient arteries (Fig. 14). Lesions in this location are usually less than 2 mm in diameter. A third common location is the midbrain, at the junction of substantia nigra and cerebral peduncle following the branches of collicular arteries (Fig. 15). In this location, they are usually less than 1.5 mm in diameter. Dilated perivascular spaces are commonly noted on MRI but rarely visualized on CT.
Figure 14. Supraventricular dilated perivascular spaces. T2-weighted fast spin echo (A) and (B) fluid-attenuated inversion recovery scans, together with T1-weighted pre- (C) and postcontrast (D) scans reveal multiple small punctate cerebrospinal fluid signal intensity lesions in the supraventricular white matter.
Figure 15. Dilated perivascular spaces (DPVSs) in the midbrain. Although described later in the literature than DPVSs in the basal ganglia and high convexity white matter, this normal variant is also not uncommon in the midbrain. Here, the location is very specific: at the junction of the substantia nigra and the cerebral peduncle. DPVS may be unilateral or bilateral, as in this case (arrows). The signal intensity is that of cerebrospinal fluid, as shown on fast spin echo T2-weighted (A), fluid-attenuated inversion recovery (B), and precontrast (C) T1-weighted scans.

It is important to distinguish this common variant from other pathologic entities, such as lacunar infarction, that carry more serious clinical implications. Dilated perivascular spaces are isointense compared with CSF on all pulse sequences. Except for cavitated old lesions, lacunar infarcts do not have CSF signal intensity on all scans and are hyperintense to CSF on intermediate T2-weighting. In general, dilated perivascular spaces are smaller than lacunar infarcts. The latter are often more slitlike and in the basal ganglia occur in the superior two thirds (as opposed to the inferior one third).

- Central pontine myelinolysis

Central pontine myelinolysis is an osmotic injury that occurs as a result of rapid correction of severe chronic hyponatremia (in alcoholism and severe malnutrition).
Figure 16. Central pontine myelinolysis. The pons and middle cerebellar peduncles have abnormal high signal intensity on the axial T2-weighted scan (A). The pons also demonstrates abnormal low signal intensity on the sagittal T1-weighted scan (B). In this instance, the pons is involved in its entirety. In mild cases, the abnormality may be confined to a smaller central triangular region.

There is symmetric destruction of myelin sheaths, starting at the median raphe of pons. Central pontine myelinolysis presents clinically with flaccid quadriplegia and facial, pharyngeal, and glottic paralysis. CT is usually negative. On MRI, abnormal high signal intensity is seen on T2-weighted scans within the pons, extending to include the middle cerebellar peduncles in severe cases. Differential diagnostic considerations include infarction, small vessel ischemic disease, metastasis, glioma, and radiation changes.

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INTRODUCTION

The value of magnetic resonance imaging (MRI) for assessing intracranial disease was quickly recognized after its clinical introduction in the early 1980s. Advantages of MRI over computed tomography (CT) include superior soft tissue contrast, absence of bone artifact, and ability to acquire high-resolution images in any plane. These features, combined with the variety of available scan types, lead to a highly sensitive and versatile imaging technique. As a result, MRI has become the principal imaging modality for intracranial tumor detection and evaluation.
The sensitivity of unenhanced MRI to detect brain neoplasms is primarily due to its ability to visualize small differences in extracellular fluid. Both T1 and T2, the two time constants describing the relaxation process of protons, are prolonged in most tumors. This leads to a decrease in lesion signal intensity on T1-weighted images and an increase on T2-weighted images. In clinical practice, it is the change on T2-weighted images that is most useful. Detection is possible even when lesions are small or isodense on CT. However, some brain tumors, such as neurofibromas, have only a small increase in water content. These lesions have less pronounced prolongation of T1 and T2.

Despite the sensitivity of unenhanced MRI, the visualization and characterization of many tumors were not possible before the introduction of intravenous MRI contrast media. Clinical examples of diagnostic difficulties encountered before the advent of contrast media include separation of tumor from surrounding edema, visualization of vascular extra-axial tumors, and detection of small metastatic lesions. The availability of MRI contrast media has largely overcome these drawbacks by providing information about blood-brain barrier (BBB) integrity and tissue vascularity.

- **Contrast media**

The iodinated contrast agents that play an essential role in x-ray-based imaging are not effective at clinical doses in MRI. This would be anticipated because of the difference in physical principles between the two imaging modalities. Iodinated contrast agents attenuate the x-ray beam, whereas MRI contrast agents change (decrease) T1 and T2. It is the shortening of T1 that is most important for contrast enhancement in clinical MRI.

Three MRI contrast agents dominate the worldwide market: Magnevist (gadolinium [Gd] DTPA), ProHance (Gd HP-DO3A), and Omniscan (Gd DTPA-BMA). No difference exists between these agents in contrast effect when given at the same dose. The gadolinium ion is the active ingredient. The ligand (DTPA, HP-DO3A, or DTPA-BMA) serves only to tightly bind (chelate) the gadolinium ion. This ensures complete renal excretion. It is possible that small differences exist in safety between the agents. The stability of the chelate is very important because of the toxicity of the free gadolinium ion (Gd). In this regard, ProHance has the greatest safety margin followed by Magnevist. Minor adverse reactions occur in a small percentage of patients with all three agents. Nausea and hives are the most common. Anaphylactoid reactions are rare but necessitate close monitoring and adequate safety measures.

The gadolinium chelates are distributed in the extracellular space after intravenous injection. Excretion is rapid and occurs by glomerular filtration. There is no hepatobiliary excretion. Patients with poor renal function (creatinine 2.5 mg/dL) should not receive contrast unless arrangements are made for repeated dialysis. To date, the preparations sold commercially are formulated at a concentration of 0.5 mol/L. The solutions are clear and colorless. The agents are given by weight; the standard dose is 0.1 mmol/kg. This equates to a 15-mL injection in a 75-kg individual. Injections are typically given as a fast infusion (over 10 to 20 seconds). Rates up to 10 mL/second have been used for specific applications, in particular first-pass studies of the brain. High dose (0.3 mmol/kg) is indicated in specific
situations. High dose is particularly important for screening and follow-up of brain metastases.

The mechanisms of lesion enhancement with gadolinium chelates are similar to that with iodinated contrast agents in CT. Enhancement can occur on the basis of either disruption of the BBB or a difference in vascularity. MRI is much more sensitive to soft tissue changes than CT. Thus, it should come as no surprise that abnormal contrast enhancement is better seen on MRI than on CT. Lesion detectability, when based on contrast enhancement, is higher on MRI. Enhancement is also seen in some pathologies on MRI and not on CT, providing a further improvement in diagnostic efficacy.

Whether an intra-axial neoplasm displays contrast enhancement depends largely on the degree of BBB disruption. Histologic studies reveal a structural alteration in the capillary walls in most neoplastic disease that allows interstitial accumulation of contrast. Generally, the more aggressive the tumor, the greater is the degree of BBB breakdown and thus contrast enhancement. The degree of vascularity plays an important role in tumors that occur outside the normal BBB (extra-axial neoplasms), including meningiomas, schwannomas, pituitary-origin tumors, and some parasellar tumors such as chordomas. Highly vascular lesions show strong enhancement. Enhancement of intra-axial lesions with BBB disruption occurs more slowly than that for extraaxial lesions with high vascularity. Thus, scans obtained at 5 to 10 minutes postcontrast may best show enhancement in intra-axial lesions (e.g., metastatic disease) as opposed to scans 1 to 2 minutes postcontrast in extra-axial lesions (e.g., acoustic schwannomas).

- Imaging sequences

Scans in MRI can be T1, T2, or proton density weighted. The latest hardware is also capable of acquiring images with diffusion weighting. Note that all scans are "weighted" in character. This provides, on any one scan, a sense of the parameter in question. However, the appearance of tissues can still be substantially influenced by the other parameters. Construction of an image that is a calculated map of one parameter, for example T1, is possible but rarely done in clinical practice. T1 is defined as the spin-lattice relaxation time and reflects the time required for a proton (a "spin") to return (or relax), once excited, by the process of giving off energy to the surrounding structure (the "lattice"). T2 is defined as the spin-spin relaxation time and reflects the time required for a proton to relax by giving its energy to a neighboring proton (thus "spin-spin"). Proton or spin density is the quantity of mobile protons (hydrogen atoms), principally water. Diffusion relates to the thermal (random) motion of protons at the molecular level.

Most scans currently in use fall within one of two general categories: spin echo or gradient echo. A third category, inversion recovery, also exists. However, scans of this type are used much less frequently. In a spin echo scan, a radiofrequency pulse is used to generate (refocus) the magnetic resonance (MR) signal from the patient. In a gradient echo scan, a small magnetic field gradient is used to generate the MR signal. TE (time to echo) and TR (time to repetition) are operator-selected parameters that specify in spin echo scans the parameter weighting (T1, T2, or proton density). A short TE (25 milliseconds) and short
TR (800 milliseconds) produces T1-weighting. A long TE (60 milliseconds) and long TR (2000 milliseconds) produces T2-weighting. Combining a short TE with a long TR gives a proton density-weighted scan. For inversion recovery scans, an additional parameter TI (time to inversion), which highly influences tissue contrast, must be specified along with TE and TR. T1-weighted scans can be recognized by the high signal intensity (white) of fat and low signal intensity (black) of cerebrospinal fluid (CSF). T2-weighted scans can be recognized by the high signal intensity of CSF. Proton density-weighted scans appear in between, with low overall tissue contrast. Proton density-weighted scans are not commonly used today in brain imaging.

The terms fast spin echo and turbo-spin echo refer to a more recent imaging development, a variant of spin echo imaging. With this technique, scan times are generally shorter. Overall image quality is usually also better, as judged by signal-to-noise and spatial resolution. The use of fast spin echo scans does make the interpretation of tissue contrast more difficult. Fat is generally high signal intensity on fast spin echo scans. Thus, whereas on a conventional spin echo T2-weighted scan fat will appear as intermediate to low signal intensity, on a fast spin echo scan it will be high signal intensity. A better marker of T1- and T2-weighting is the gray-white matter ratio. In adults, white matter is of higher signal intensity than gray matter on a T1-weighted scan. The reverse is true on a T2-weighted scan, with gray matter of higher signal intensity.

Correct identification of T1- and T2-weighting, by visual image inspection, has become even more difficult with the advent of a technique known as fluid-attenuated inversion recovery (FLAIR) scanning. In the clinical use of FLAIR in the brain, with the pulse parameters specified to obtain T2-weighting, CSF signal is attenuated (black). This provides markedly improved sensitivity to T2 abnormalities (such as edema), which are seen with high signal intensity. Gray and white matter are relatively isointense, both with lower signal intensity but not as dark as CSF. FLAIR is a type of inversion recovery scan.

With gradient echo scans, in addition to TE and TR, the "tip" or flip angle must be specified. Gradient echo scans typically have much shorter TEs and TRs than spin echo scans, with the relationship among TE, TR, and tip angle complex. Both T1- and T2-weighted scans can be produced with gradient echo technique. Only two common applications of gradient echo scans exist in the brain. The first is for improved sensitivity to iron, such as that in deoxyhemoglobin and hemosiderin. The second is for high-resolution three-dimensional (3D) imaging. In the latter application, images can be acquired with a spatial resolution of 1 mm 1 mm 1 mm. This allows postacquisition high-resolution image reformatting in any desired plane.

Contrast enhancement is best visualized on T1-weighted scans. The presence of the gadolinium ion causes a reduction in the T1 of nearby water protons. This leads to an increase in signal intensity or equivalently positive lesion enhancement. It should be noted that the presence of the gadolinium ion actually causes a reduction in both T1 and T2. This is of relevance in first-pass brain imaging, in which the T2 effect of the agent is actually visualized. Although both T1 and T2 are shortened, the T1 shortening is of larger magnitude.
One can only visualize the T2 effect at high concentrations, which occur during the first pass of the bolus through the brain (immediately after intravenous injection).

To appropriately identify all signal intensity abnormalities, both T1- and T2-weighted scans should be obtained before contrast administration. Acquisition of a postcontrast T1-weighted scan then completes the imaging set, with high efficacy for the evaluation of all brain disease. It is strongly recommended that all three scans be acquired in the same plane because this facilitates correlation between images of different weighting. Precontrast T1-weighted scans are also important for the differentiation of lesion enhancement from hemorrhage (methemoglobin) or fat (e.g., a corpus callosum lipoma). Supplemental scans in the coronal and sagittal planes are often useful for further lesion evaluation.

**INTRA-AXIAL TUMORS (SUPRATENTORIAL)**

- **Astrocytoma**

Astrocytomas are the most common brain tumor, accounting for 50% of all intracranial neoplasms. As the name implies, astrocytomas arise from the astrocyte or its primitive precursor. These tumors occur in white matter, where astrocytes are abundant. In adults, astrocytomas are more frequent above the tentorium in the cerebral hemispheres. In children, these arise more commonly in the cerebellar hemispheres and brainstem.

Astrocytomas are classified histologically according to several scales: World Health Organization (WHO) grades I to IV, Kernohan grades I to IV, and Rubenstein grades I to III. Higher grade equates with greater malignancy. The Rubenstein classification is simpler to remember and is easier to correlate with imaging findings.

According to this scale, a grade I astrocytoma is low grade, grade II is an anaplastic astrocytoma, and grade III is a glioblastoma multiforme (GBM). In the 1993 WHO classification, a distinction is made between lesions that are histologically well circumscribed (grade I) as opposed to diffuse (grades II-IV). Low-grade (grade I) astrocytomas are further divided into specific tumor subtypes, recognizing the favorable prognosis of these lesions, which include juvenile pilocytic astrocytoma and subependymal giant cell astrocytoma. Other WHO grade I nonastrocytic tumors include gangliocytoma, meningioma, and choroid plexus papilloma. In the WHO classification, the best possible grade for a diffuse astrocytoma (the "ordinary" type of astrocytoma seen in adults) is grade II. In this classification scheme, a low-grade astrocytoma is grade II, an anaplastic astrocytoma grade III, and a glioblastoma grade IV.

Although a lower grade (for an astrocytoma) implies a lesser degree of malignancy, the outcome of even these tumors is generally poor because of the infiltrative pattern of growth. Complete tumor resection is often impossible. Grade I astrocytomas carry a uniquely favorable prognosis; the juvenile pilocytic astrocytoma (referred to in the older literature as the cystic cerebellar astrocytoma of childhood) is the most common such lesion. Surgical removal of these tumors usually produces a clinical cure. MRI is the most sensitive imaging modality for detection of astrocytomas. Increased extracellular fluid...
occurs as a result of abnormal capillary walls. This is easily identified on T2-weighted scans as an area of increased signal intensity. On precontrast scans, this change may be the only or most convincing indication of the presence of a tumor. The change on T1-weighted scans (a decrease in signal intensity) may be subtle. CT, particularly in low-grade astrocytomases, may be nearly normal or show only subtle mass effect.

GBM has the most profound as well as characteristic imaging findings (Fig. 1). Thus, it is the most readily diagnosed tumor and the least frequently confused with other lesions. GBM usually has substantial mass effect, margin irregularity, and signal intensity heterogeneity on both T1- and T2-weighted scans. Low signal intensity on T1-weighted scans corresponds to necrotic and cystic areas. Necrosis occurs as the tumor outgrows its blood supply. High signal intensity on T2-weighted scans corresponds to associated, vasogenic edema, typically marked in amount. Hemorrhage may occur in higher grade tumors, frequently petechial in nature. GBMs spread via white matter tracts and frequently cross the corpus callosum to the opposite hemisphere (Fig. 2). Bifrontal corpus callosum tumors are referred to as "butterfly" gliomas.

![Figure 1. Glioblastoma multiforme (World Health Organization grade IV). A large right frontal lobe mass is noted on precontrast T2- (A) and T1-weighted (B) scans. There is extensive surrounding edema, which is high signal intensity on the T2-weighted scan. Substantial mass effect is noted, with obliteration of sulci, compression of the right lateral ventricle, and displacement of the falk. Irregular rim enhancement is present on the postcontrast T1-weighted scan (C), with no enhancement of the central necrotic portion of the tumor.](image)

Irregular enhancement of the tumor periphery ("rim") is seen after contrast administration in many higher grade astrocytomases and reflects the greater degree of BBB disruption. Other patterns of enhancement include homogeneous, garland-shaped, mixed or patchy, linear, and central. These enhancement patterns may occur in any tumor and thus are not grade specific. Occasionally, high-grade tumors will show little or no contrast enhancement. Thus, a completely accurate prediction of tumor grade by imaging appearance is not possible.
Contrast administration should, however, be routinely used in the assessment of all tumors. Because contrast use improves visualization, localization, and tumor margin delineation, a higher level of diagnostic confidence results. This is in part due to the separation of tumor nidus from surrounding edema, the former enhancing and the latter not. Unfortunately, histologic studies show that abnormal contrast enhancement in astrocytomas does not outline the entire extent of the tumor but simply the maximal site of BBB disruption (the area of greatest neovascularity). Astrocytomas are infiltrating lesions, with tumor often present beyond the border indicated by either precontrast T2-weighted or postcontrast T1-weighted scans. The area of maximal enhancement does identify the best site for diagnostic stereotactic biopsy. The irregular, finger-like growth pattern of these tumors produces many areas that are relatively uninvolved. If these areas are selected by chance for biopsy, the histologic diagnosis may be normal, although the imaging changes are consistent with tumor. This produces a management dilemma for the surgeon or radiation oncologist.

Contrast enhancement can also be used to separate viable tumor from frank necrosis. On postcontrast T1-weighted scans, areas of necrosis remain low signal intensity without evidence of enhancement. Central tumor necrosis may be difficult to distinguish from cystic change. With necrosis, the interface between viable and nonviable tissue is often irregular or ragged. With cyst formation, a fairly well-circumscribed area is seen, with a smooth inner margin and enhancement at the periphery. T1 and T2 are typically prolonged in both cystic and necrotic areas, with low signal intensity on T1-weighted scans and high signal intensity on T2-weighted scans. Cysts may demonstrate a fluid-debris level or a contrast-fluid level, the latter resulting from diffusion of contrast from adjacent tumor with BBB disruption.

As with most brain disease, the primary plane for imaging should be axial. Coronal images can provide important additional information in temporal lobe abnormalities. Sagittal images assist in evaluation of brain-stem and craniovertebral junction abnormalities. For preoperative evaluation, sagittal images are important, providing the neurosurgery team with improved visual localization of a lesion and thus assisting in craniotomy placement.

WHO grade III or anaplastic astrocytomas usually present with less severe imaging changes compared with GBM (Fig. 3). The margins are not as irregular, there is less mass effect, and the signal intensity changes on T1- and T2-weighted images are not as profound or heterogeneous. Hemorrhage is less frequently found. The degree of enhancement is variable. If the tumor lies near a convexity, enhancement may be difficult to assess without imaging in a second plane. Contrast enhancement often assists in differential diagnosis. Infarction, abscess, and resolving hematoma should be considered in the differential diagnosis of an anaplastic astrocytoma. WHO grade II, or low-grade, astrocytomas have the least severe imaging changes (Fig. 4). The tumor margin, as identified on imaging, may be relatively smooth or slightly irregular. Mass effect is typically minimal. Cystic changes and necrosis are infrequent, and contrast enhancement usually does not occur. Calcifications are more frequent, as assessed by CT. However, these are not usually seen on MRI. Low-grade astrocytomas may go undiagnosed by CT, particularly if they are located in the temporal lobe. Thus, a patient with temporal lobe seizures and a normal CT should have an MRI for complete evaluation. On occasion, it may be difficult to distinguish these
low-grade tumors from infarcts on a single study. Serial studies may be necessary to establish the diagnosis. Infarctions show a decline in mass effect over time and an increase in encephalomalacic changes. Tumors may show little change or a progression in mass effect with time. The major arterial territories should be kept in mind because both anterior and posterior cerebral artery infarctions, being less common, can be mistaken for an astrocytoma.

Figure 2. Glioblastoma multiforme with corpus callosum involvement. A large bifrontal lesion involving the genu of the corpus callosum is well seen on precontrast T2- (A) and T1-weighted (B) scans. C, There is irregular rim enhancement postcontrast. The enhanced scan also demonstrates a nonenhancing central component, which corresponds to necrotic debris and fluid. Glioblastomas are highly malignant, widely infiltrative lesions that grow along white matter tracts. A thick, irregular enhancing rim with central necrosis is characteristic.

Figure 3. Anaplastic astrocytoma (World Health Organization grade III). A, On the precontrast T2-weighted scan, a midline lesion with intermediate signal intensity is noted. There is subtle low signal intensity on the precontrast T1-weighted scan (B). Neither scan
depicts the lesion itself or its margins well. There is enhancement of the mass on the postcontrast scan (C), which also demonstrates involvement of the splenium of the corpus callosum. On magnetic resonance imaging, anaplastic astrocytomas (WHO grade III), as compared with low-grade astrocytomas (WHO grade II), tend to be less well defined and heterogeneous, with moderate mass effect, and may demonstrate contrast enhancement.
Figure 4. Low-grade astrocytoma (World Health Organization grade II). On precontrast T2-weighted (A) fast spin echo and (B) fluid-attenuated inversion recovery scans, a high-signal-intensity abnormality is noted involving the left temporal lobe. The lesion is low signal intensity on the precontrast T1-weighted scan (C) and does not demonstrate abnormal enhancement (D) postcontrast. On magnetic resonance imaging, low-grade astrocytomas appear well defined without substantial mass effect. Unlike higher grade tumors, these lesions usually do not enhance after contrast administration.

- Oligodendroglioma

Oligodendrogliomas are relatively rare, accounting for about 5% of all intracranial neoplasms. These slow-growing tumors are often large at diagnosis. Oligodendrogliomas tend to involve the anterior cerebrum. They are typically round or oval with fairly well-defined margins (Fig. 5).

Calcifications are more common in oligodendrogliomas than in other glia-origin tumors, occurring in more than 50% of cases. Because of the presence of calcification, CT has a diagnostic advantage. If the tumor is not calcified, it may be difficult to distinguish from other glia-origin tumors.

Figure 5. Oligodendroglioma. A large, hyperintense frontal lobe lesion is noted on the precontrast T2-weighted scan (A). The mass demonstrates moderate low signal intensity on the postcontrast T1-weighted scan (B). There is no abnormal contrast enhancement. Calvarial erosion resulting from location and slow growth is clearly depicted on both scans. Contrast enhancement is seen in about half of all oligodendrogliomas, which are typically being mild in degree and inhomogeneous.
- **Ganglioneuromas (Gangliocytomas) and Gangliogliomas**

Ganglioneuromas (gangliocytomas) and gangliogliomas share common characteristics in respect to incidence, macroscopic features, and biological behavior. These tumors are composed of mature ganglion cells with varying glial components. At one end of the spectrum is a tumor with mature neurons and scanty stromal glial cells. At the other end is a tumor that at first glance microscopically appears to be a glioma. Ganglioneuromas and gangliogliomas occur most frequently in children and young adults. The temporal lobe is the most common site. These tumors are usually small and well circumscribed. They are often cystic. Occasionally the cystic element dominates, with the tumor itself confined to a mural nodule (Fig. 6). These tumors grow slowly. Malignant change is rare. Their small size and good demarcation permit surgical resection in most cases. Prognosis is relatively good. By the WHO classification, gangliocytomas are grade I (with no malignant potential) and gangliogliomas grade I-II.

![Figure 6. Ganglioglioma. A large cystic lesion is noted in the right temporal lobe on precontrast T2- (A) and T1-weighted (B) scans. There is mild mass effect on the brainstem. A small amount of edema is seen lateral to the lesion on the T2-weighted scan. The signal intensity of the cyst is slightly different from that of cerebrospinal fluid on both images. C, The postcontrast T1-weighted scan, obtained at a level several centimeters lower, reveals an enhancing nodule (arrow) along the inferior wall of the cyst.]

- **Primitive Neuroectodermal Tumor (PNET)**

The term primitive neuroectodermal tumor (PNET) is controversial and refers to a group of tumors thought to originate from undifferentiated neuroepithelial cells.

There is considerable histopathologic heterogeneity. These tumors are highly malignant and carry a poor prognosis. Local spread, dissemination via the subarachnoid space, and distant metastases are frequent. When cerebellar in location (the most common type), the term medulloblastoma has also been used (this tumor is discussed in detail later).
supratentorial in location, the terms cerebral neuroblastoma and cerebral medulloblastoma have also been used.

Supratentorial PNETs are typically large, well-circumscribed frontal or parietal lesions. The lesion is often dominated by a cystic component, with enhancing tumor located around the periphery (Fig. 7). Hemorrhage into the cyst is not uncommon and often leads to clinical presentation.

Figure 7. Supratentorial primitive neuroectodermal tumor (PNET). A large well-demarcated cystic frontoparietal mass is seen on precontrast T2- (A) and T1-weighted (B) scans. Within the lesion, there is a fluid-fluid level representing separation of different hemoglobin degradation products. The body of the left lateral ventricle is completely obliterated by mass effect. C, The postcontrast T1-weighted scan reveals enhancement of a soft tissue component (arrow) along the lateral aspect of the mass as well as enhancement of the entire lesion rim.

- **Lymphoma**

There has been a marked increase in the last decade in the incidence of primary central nervous system (CNS) lymphoma. This tumor, once rare, is now quite common. The increase in incidence has occurred in both immunosuppressed and immunocompetent patient populations. Also known as reticulum cell sarcomas or microgliomas, these tumors are derived from microglial cells that histologically resemble lymphocytes. The basal ganglia, thalamus, and corpus callosum are the most frequently affected sites. There is an increased incidence of primary CNS lymphoma in the immunocompromised patient population. Thus, lymphoma should be considered in the differential diagnosis of brain lesions in patients who underwent organ transplantation and in those with AIDS.

Lesions not associated with AIDS are typically homogeneous in signal intensity and periventricular in location and enhance (uniformly) after contrast administration. In AIDS, lymphoma may have ring enhancement (Fig. 8). Lymphomas may be difficult to distinguish from abscesses, metastases, or glial tumors. Periventricular location and minimal mass
effect (little edema) favor lymphoma. Some solid lymphomas have mild hypointensity on T2-weighted scans.

Figure 8. Primary central nervous system lymphoma. A mass with intermediate signal intensity and extensive surrounding high-signal-intensity edema is noted on the axial T2-weighted scan (A). B, The postcontrast coronal T1-weighted scan demonstrates prominent peripheral enhancement with central hypointensity. Before the advent of AIDS, the majority of cerebral lymphomas were primary in origin with prominent homogeneous contrast enhancement. In AIDS, primary and secondary lymphomas occur with equal frequency, and enhancement is typically ringlike in nature with central lesion necrosis.

- **Metastasis**

Metastases comprise almost 40% of all intracranial tumors. The most common tumors that metastasize intracranially are lung, breast, melanoma, colon, and kidney. Multiplicity is the hallmark that distinguishes metastases from gliomas or other primary tumors (Fig. 9). Other imaging findings that suggest metastasis are a gray-white matter junction location, a small tumor nidus with a large amount of associated vasogenic edema, and less margin irregularity. MRI is markedly superior to CT for detecting metastatic disease. Contrast administration is mandatory (Fig. 10). In one published study, enhanced MRI revealed three times the number of lesions seen by enhanced CT. High-dose contrast administration on MRI provides a further improvement in sensitivity (Fig. 11). The multi-institutional study that examined contrast dose found that high dose (0.3 mmol/kg) revealed 32% more metastases compared with standard dose (0.1 mmol/kg). If stereotactic radiation therapy is an option (depending on geographic location of the patient and hospital), high-dose thin-section (5 mm or less) imaging in both the axial and coronal planes should be performed. This approach maximizes lesion detection. Small single metastases can also be missed on a standard dose (0.1 mmol/kg) examination.
Figure 9. Brain metastases (varied appearance). A cystic or necrotic mass is noted in the right cerebellum on the precontrast T2-weighted scan (A). Vasogenic edema, with abnormal high signal intensity, is seen bilaterally. Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals three enhancing lesions: large necrotic right-sided metastasis, a 1-cm-diameter solid left-sided metastasis, and a smaller pinpoint metastasis (arrow) just anterior and lateral to this lesion. The case illustrates the value of contrast enhancement in identification of metastatic lesions. Although the left cerebellar hemisphere appears abnormal precontrast, focal lesions cannot be identified. Also illustrated are the multiple patterns of lesion enhancement that can be seen in metastatic disease, including rim, solid, and pinpoint.
Figure 10. Brain metastasis (seen only postcontrast). Precontrast T2-weighted fast spin echo (A) and fluid-attenuated inversion recovery (B) scans are normal, as is the precontrast T1-weighted scan (C). D, Postcontrast, a single small enhancing lesion is noted (arrow), which is confirmed on the coronal scan (E). Small brain metastases may not elicit sufficient surrounding vasogenic edema to be recognized on precontrast magnetic resonance scans. Identification of blood-brain barrier disruption, provided by intravenous contrast administration, permits diagnosis of such lesions.

Figure 11. Brain metastases (improved lesion detection with high contrast dose). Precontrast T2- (A) and T1-weighted (B) scans are compared with postcontrast T1-weighted scans using doses of 0.1 mmol/kg (standard dose) (C) and 0.2 mmol/kg (high dose) (D). The contrast agent used in this instance was gadolinium (Gd BOPTA) (MultiHance), which has improved relaxivity compared with Gd DTPA (Magnevist) because of weak protein binding. In this patient, higher contrast dose improves the
enhancement of all lesions and makes two small lesions (arrows, D) more evident, one near
the right occipital horn and the other in the right temporal lobe.

The mechanism of enhancement for intra-axial metastases is similar to gliomas in that BBB
disruption is marked and separates the tumor nidus from surrounding edema. Various
types of enhancement are seen, including focal dotlike, larger rounded, and variable sized
areas of ring enhancement. Perhaps the greatest importance of contrast use in evaluating
metastatic disease is the greater number of lesions depicted. The diagnostic and therapeutic
impact is immense. The demonstration of multiple lesions may dictate radiation or
chemotherapy, whereas a solitary lesion may be more effectively treated with surgical
resection. Stereotactic radiation is often used in patients with only a few brain metastases.
Contrast enhancement is particularly critical in elderly individuals with age-related white
matter ischemic changes. These areas of increased signal intensity on T2-weighted images
may be impossible to distinguish from the signal intensity change of a metastatic lesion
with surrounding edema.

Although T2-weighted scans are quite sensitive in demonstrating vasogenic edema (as an
area of increased signal intensity), not all metastatic lesions have sufficient edema to be
detected on this basis alone. The lesions not visualized on unenhanced MRI are typically
small (5 mm). Common locations for metastases missed on T2-weighted scans include the
temporal lobes and the cortical-subcortical regions. Small lesions may also be missed when
adjacent to the ventricles or a larger metastatic lesion. Thus, a complete evaluation for
metastatic disease requires precontrast T2-weighted, precontrast T1-weighted, and
postcontrast T1-weighted scans. As with most brain disease, acquisition of two different
T2-weighted scans is suggested: one using FLAIR and one with fast spin echo technique.
On precontrast T1-weighted scans, large metastases are seen as low-signal-intensity
lesions. Small metastases are often not visualized on these scans. The primary purpose of
precontrast T1-weighted imaging is to distinguish areas of enhancement from subacute
hemorrhage (which also has high signal intensity on T1-weighted scans).

MRI also surpasses CT in its demonstration of subacute hemorrhage. Metastases with a
propensity toward hemorrhage include melanoma, choriocarcinoma, lung carcinoma (oat
cell), and kidney, colon, and thyroid carcinoma (Fig. 12). Petechial hemorrhage may be
seen in metastases following radiation therapy. Patients receiving chemotherapy
occasionally develop coagulopathies. Ensuing intracranial hemorrhage may produce a
sudden decline in mental status similar to the effect of a significant hemorrhage into an
intracranial metastasis. These hemorrhages may remain undiagnosed by CT, as do many
subacute hemorrhages.
Figure 12. Hemorrhagic brain metastases. A, The precontrast T2-weighted scan reveals (posteriorly) two hyperintense lesions. On the precontrast T1-weighted scan (B), the larger of the abnormalities is also hyperintense, whereas the smaller is difficult to identify. This appearance is compatible with two intraparenchymal hematomas of slightly different composition. C, Postcontrast, there is enhancement of abnormal soft tissue along the medial border of the larger lesion, with a thick circumferential rim of enhancement surrounding the smaller lesion. Both abnormalities were confirmed to represent metastatic disease. In the presence of acute and subacute hemorrhage, careful inspection of postcontrast scans is mandated to rule out an underlying abnormality, such as metastatic disease in this instance.

- Pineal Region Tumors

Pineal region tumors are classified by cell of origin (pineal or germ cell). Germ cell tumors include germinoma, teratoma, and teratocarcinoma. The occurrence of mixed germ cell tumors, with various cellular elements, is common (Fig. 13). All occur more frequently in males. Germinoma is the most common of these abnormalities and occurs almost exclusively in males. These tumors may be large and engulf the normal pineal gland. Less heterogeneity in signal intensity is seen in germ cell tumors compared with pineal cell tumors. Intense, homogeneous enhancement occurs. MRI defines the tumor margins better than CT.
Figure 13. Mixed germ cell tumor. A soft-tissue mass is identified just posterior to the third ventricle on precontrast T2- (A) and T1- weighted (B) scans. There is mild internal signal inhomogeneity. A striking finding on the mildly T2-weighted scan (A) is the abnormal high signal intensity surrounding the ventricles. This is consistent with transepndymal flow secondary to acute obstructive hydrocephalus. C, Postcontrast, there is intense enhancement of the lesion. Although contrast enhancement improves lesion identification, imaging characteristics for pineal region tumors on magnetic resonance imaging are nonspecific in regard to lesion type.

Pineal cell tumors include pineocytoma and pineoblastoma (Fig. 14). These are less common than other pineal region tumors, particularly germinoma. There is no sex predilection. These tumors may calcify. Pineoblastoma is the more malignant of the two and arises from a more primitive cell type. MRI is particularly helpful in assessing the extent of these rather large, bulky tumors and the degree of involvement of adjacent structures.

Figure 14. Pineocytoma. On T2- (A) and T1-weighted (B) axial scans, a well-demarcated very-low-signal-intensity mass is noted near the quadrigeminal plate. There is no associated edema. C, The precontrast sagittal T1-weighted scan reveals the lesion to be pineal in location. The lateral ventricles are dilated. Noncontrast computed tomography (not shown) demonstrated a 1-cm-diameter extremely dense calcification in the region of the pineal gland.
Metastases and gliomas may also occur in the pineal region. Obstructive hydrocephalus may accompany large tumors. All but teratomas are notorious for seeding by CSF pathways. Pineal cysts, which are benign, can cause difficulty in differential diagnosis. Typical pineal cysts have signal intensity only slightly different from CSF and demonstrate mild rim enhancement (Fig. 15).

Figure 15. Pineal cyst. A, The sagittal fast spin echo T2-weighted scan reveals a cystic midline pineal lesion with mild mass effect on the colliculi. On the intermediate T2-weighted spin echo scan (B), the lesion is hyperintense. C, Postcontrast, a faint rim of enhancement can be identified. Pineal cysts are common normal variants. These cysts are round, smoothly marginated, and rarely larger than 15 mm in diameter and have a thin wall that may demonstrate contrast enhancement.

INTRA-AXIAL TUMORS (INFRATENTORIAL SPACE)

Since its introduction, MRI has been well known for its efficacy in the diagnosis of posterior fossa lesions. CT is a poor imaging modality for evaluating the posterior fossa. The absence of bone artifacts and the ability to acquire images in multiple planes are the two main reasons that MRI is so effective in the posterior fossa.

- Astrocytoma

Cerebellar astrocytomas are predominantly tumors of early life (the first two decades). They are one of the most common posterior fossa tumors. Cerebellar astrocytomas are often well circumscribed and tend to be grossly cystic (Fig. 16). Anaplasia is uncommon in these lesions. This subtype is usually amenable to surgery. However, some cerebellar astrocytomas are solid tumors; infiltration of surrounding tissues is noted microscopically. Anaplastic change is more common in older patients.

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Figure 16. Pilocytic astrocytoma (cystic cerebellar astrocytoma, World Health Organization grade I). A large cystic lesion is seen within the cerebellum on precontrast T2- (A) and T1-weighted (B) axial scans. A small soft tissue component along the right lateral wall is noted to enhance (C, D) postcontrast. Enhancement of the nodule (arrow) is better seen on the coronal scan (D). With this type of tumor, the enhancing mural nodule corresponds to neoplastic tissue. The cyst wall, which does not enhance, is nonneoplastic.

Astrocytomas can involve any part of the cerebellum. If a tumor is located in the cerebellar hemispheres, the incidence of different tumor types favors diagnosis of an astrocytoma. Medulloblastomas and ependymomas are more likely to be midline.

In large cystic lesions, tumor tissue may be confined to a mural nodule, which enhances. In other instances, the cyst is lined circumferentially with tumor. Cerebellar astrocytomas consistently display contrast enhancement. This aids in differentiation between lesions with just a small tumor nidus (mural nodule), lesions with central cystic change or necrosis, and
solid lesions. Caution is indicated when a cystic mass in the cerebellum is noted on MRI. The tumor nidus in a cerebellar astrocytoma may be quite small and go unrecognized without contrast enhancement. Imaging in two planes, in addition to careful examination of the postcontrast scans, is highly recommended.

- **Brainstem Glioma**

Brainstem gliomas generally occur in older children and young adults. Most gliomas of the brainstem are diffusely infiltrating astrocytomas. Symptoms include progressive cranial nerve palsies, extremity weakness, and respiratory difficulty. MRI is markedly superior to CT for visualizing these lesions. With CT, only large extensive lesions are usually recognized. In more aggressive tumors, necrotic or cystic changes can be seen, with low signal intensity on T1-weighted scans and very high signal intensity on T2-weighted scans. The tumor itself, specifically the soft tissue component, is best seen on T2-weighted scans with high signal intensity but not that of CSF or fluid (Fig. 17). Contrast enhancement is variable.

![Figure 17. Brainstem (pontine) glioma. A, The T2-weighted axial scan demonstrates a high-signal-intensity expansile mass. The lesion occupies almost the entire pons, leaving only a residual rim of normal tissue. The mass is low signal intensity on the precontrast T1-weighted scan (B). C, Postcontrast, the more posterior portion of the lesion enhances. On histologic exam, brainstem (pontine) gliomas are often low-grade astrocytomas but have a tendency to undergo anaplastic change. Exophytic extension and cerebrospinal fluid seeding are common.](image)

- **Medulloblastoma (Cerebellar PNET)**

Medulloblastomas are one of the most common posterior fossa tumors in childhood, with a predilection for males. These embryonal tumors arise in the roof of the fourth ventricle or less commonly in the cerebellar hemisphere of older patients. They are difficult to distinguish from ependymomas, unless the ependymoma extends into the cerebellopontine
angle. As with most brain tumors, medulloblastomas have slightly low signal intensity on T1-weighted scans and moderately high signal intensity on T2-weighted scans (Fig. 18). Intense contrast enhancement is characteristic. Medulloblastomas are highly malignant and CSF spread is common.

Figure 18. Medulloblastoma. A midline mass with heterogeneous, although predominantly high, signal intensity is noted on the T2-weighted axial scan (A). B, The midline T1-weighted sagittal scan demonstrates the mass to fill the fourth ventricle. The brainstem is displaced anteriorly and the inferior aspect of the cerebral aqueduct widened. Leptomeningeal metastases were noted on the thoracic and lumbar magnetic resonance examinations performed on the same date (images not shown).

- **Hemangioblastoma**

Hemangioblastomas are histologically benign neoplasms of vascular structures. They may occur at any age but are more frequent in young and middle-aged adults. These tumors are usually solitary and located in the cerebellum. They can occur sporadically or as part of von Hippel-Lindau disease. In the latter, the tumors are typically multiple and patients present in childhood. About half of all hemangioblastomas are cystic, and half are solid. A characteristic feature is an enhancing mural nodule (Fig. 19). Because these tumors involve the cerebellar hemisphere, the main differential diagnosis is a cystic astrocytoma. Cystic astrocytomas tend to be larger tumors and occur in a younger population.
Figure 19. Hemangioblastoma. On the axial scan with intermediate T2-weighting (A), a high-signal-intensity lesion is noted within the posterior fossa. There is mass effect with compression of the fourth ventricle. The lesion is slightly higher in signal intensity than cerebrospinal fluid on this scan and the precontrast T1-weighted scan (B), suggesting a neoplastic origin. C, Postcontrast, there is enhancement of a small mural nodule (arrow), with a large prominent vein also identified adjacent to the mass. The most common appearance for a hemangioblastoma is that of a cystic mass with a peripheral mural nodule. Tumor vessels may also be apparent. Less commonly, these lesions present as solid masses.

- **Colloid Cyst**

Colloid cysts are benign congenital lesions and occur in the anterior third ventricle. These cysts are well defined and vary in diameter from a few millimeters to several centimeters. Larger colloid cysts may produce hydrocephalus by obstruction of the foramen of Monro. Growth is slow, and the lesion may not become symptomatic until adult life.

Colloid cysts are easily diagnosed by MRI because of their location and appearance. Signal intensity characteristics cover the entire spectrum from low to high on both T1- and T2-weighted scans (Fig. 20). If the contents are predominantly lipid, the signal intensity will be high on T1-weighted scans and fade to low on T2-weighted scans. Colloid cysts do not enhance.
Figure 20. Colloid cyst. A large round mass with signal intensity slightly lower than cerebrospinal fluid is noted on the axial T2-weighted scan (A). The mass is very high signal intensity on the axial T1-weighted scan (B). The lateral ventricles are enlarged, suggesting obstructive hydrocephalus. Comparison with sagittal and coronal scans (not shown) confirmed the location of the cyst at the anterior third ventricle. No abnormal contrast enhancement was noted (not shown).

- **Choroid Plexus Papilloma**

Choroid plexus papillomas originate from the ependyma (the lining of the ventricles). These are more common during the first decade of life and show a slight male predominance. Choroid plexus papillomas most frequently arise in the lateral ventricle in children, particularly the left lateral ventricle, and in the fourth ventricle in adults. In the lateral ventricles, hydrocephalus is asymmetric but bilateral and results from outlet obstruction of the ventricle, overproduction of CSF, or a combination of these two factors. Intermittent hemorrhage into these tumors is not uncommon and may contribute to the obstructive hydrocephalus. In fourth ventricular lesions, hydrocephalus is symmetric.

Choroid plexus papillomas are frequently lobulated. Focal calcifications are common. Contrast enhancement is intense. There is little difference in appearance between choroid plexus papilloma and choroid plexus carcinoma, although the latter is much less common. Differential diagnosis includes ependymoma, meningioma, and metastases, all of which are more common in the adult population.

- **Ependymoma**

Ependymomas are derived from ependymal cells that line the ventricles or from cell rests in the adjacent periventricular white matter. In adults, these tumors arise in the trigone of the lateral ventricle or near the foramen of Monro. Ependymomas can be periventricular
or intraventricular in location. They can grow through the septum pellucidum and involve both lateral ventricles. In children, ependymomas occur more commonly in the posterior fossa, arising in the fourth ventricle. These frequently extend through the foramen of Luschka into the cerebellopontine angle. Recognition of this feature, if present, improves differentiation from other posterior fossa tumors, such as medulloblastoma and astrocytoma.

Because of their intraventricular origin, seeding via the CSF is common. The prognosis is poor with the occurrence of drop metastases. Hydrocephalus is very common, particularly with ependymomas in the posterior fossa. Whether supra- or infratentorial in location, ependymomas are usually calcified, and about half have areas of cystic change.

MRI is helpful in confirming the intraventricular location, particularly if these tumors occur in the lateral ventricles. Ependymomas usually present as large, bulky, soft tissue masses. Cystic changes or dense calcifications appear as focal areas of low signal intensity on T1-weighted images. Ependymomas have high signal intensity on T2-weighted images (in both cystic and noncystic regions). These lesions do show contrast enhancement, which is variable in pattern.

- Meningioma

Intraventricular meningiomas are rare, occurring in the atrium of the lateral ventricle more commonly than in the third or fourth ventricles. They can occur at any age but show a predilection for older adults. As with all meningiomas, there is an increased incidence in neurofibromatosis. Intraventricular meningiomas are usually large, lobulated masses. There may be slight ventricular dilatation, either unilateral or bilateral. The signal intensity precontrast may be heterogeneous as a result of vascularity or dense calcifications. Enhancement is intense after contrast administration. MRI is more accurate in the assessment of intraventricular location than CT because of the availability of multiplanar imaging. The differential diagnosis should include other enhancing intraventricular tumors.

EXTRA-AXIAL TUMORS

- Meningioma

Meningiomas are the most common extra-axial adult tumor, comprising about 15% of all intracranial neoplasms. These tumors are more frequent in women between the ages of 40 and 70 years. The most common location is high over the convexity adjoining the superior sagittal sinus in its middle or anterior third (Fig. 21). Other sites, in decreasing order of frequency, are the lateral convexity, sphenoid ridge, olfactory groove, suprasellar-parasellar region, and posterior fossa (petrous bone, clivus, and foramen magnum). When these tumors are multiple or occur in childhood, they are usually associated with neurofibromatosis. Meningiomas are typically benign, slow-growing tumors that compress rather than invade adjacent brain tissue. Occasionally, more aggressive changes are seen.
such as dural sinus or bone invasion. With such changes, complete resection may not be possible, and recurrences are more likely to occur.

Figure 21. Falx meningioma. A soft tissue mass that is isointense to the brain is noted adjacent to the falx on precontrast T2- (A) and T1-weighted (B) scans. There is mild mass effect. C, Postcontrast, the mass is easily identified as a result of intense enhancement.

Regardless of location, meningiomas usually have a broad base that lies along a bony or dural margin. Features characteristic of extra-axial lesions are seen, including arcuate bowing of the white matter resulting from compression of the brain, a low-signal-intensity interface with brain on T1-weighted scans (caused in part by displacement of pial vessels), and a CSF cleft between the lesion and brain, seen best on T2-weighted images (Fig. 22). Displacement of the dura at the lateral margin of the lesion can be seen on occasion, more commonly with cavernous sinus lesions. Meningiomas are typically highly vascular; calcifications and cystic changes produce intrinsic tumor mottling. These findings are more obvious at higher field strengths perhaps because of differences in magnetic susceptibility. Meningiomas have a variable amount of associated edema. Occasionally, this edema will be the only evidence for the presence of a lesion on precontrast scans.

Unlike most intracranial tumors, meningiomas tend to be isointense with adjacent brain on both T1- and T2-weighted scans. Thus, small lesions and en plaque meningiomas can be difficult to detect without contrast administration. Contrast enhancement is intense because of the lack of a BBB. On occasion, a more intensely enhancing thin rim is present, surrounding the bulk of the tumor, which shows less but still substantial enhancement. Contrast use aids in lesion visualization, accurate localization, and assessment of lesion vascularity.

Meningiomas often invade adjacent dural sinuses (Fig. 23). MRI venography and postcontrast T1-weighted imaging are two effective ways to demonstrate sinus invasion. MRI venography is usually performed before contrast administration. Two-dimensional (2D) time-of-flight technique is used, depicting venous flow as high signal intensity. Sinus invasion is diagnosed on the basis of the irregular contour of the sinus, presence of a signal
void within the sinus, or absence of flow (with occlusion). On postcontrast T1-weighted scans, the venous sinus also has high signal intensity. Signs of sinus invasion are similar to that on MRI venography, except that the tumor is depicted as an enhancing soft tissue mass (although with lower signal intensity than that of venous blood). MRI is more sensitive in detecting sinus invasion than either CT or x-ray angiography.

Figure 22. Convexity meningioma. A soft tissue frontal lesion of slightly higher signal intensity than adjacent brain is noted on the precontrast T2-weighted scan (A). The mass is adjacent to both the falx and the calvarium. Erosion of the calvarium is evident on comparison of the diploic space from side to side. A cerebrospinal fluid cleft is seen posterior to the lesion, demarcating its extra-axial location. Intense uniform enhancement is seen on the postcontrast T1-weighted scan (B).
Figure 23. Posterior fossa meningioma with dural sinus invasion. Sagittal (A) and axial (B) T2-weighted scans reveal a subtle mass adjacent to the tentorium. The lesion is difficult to detect because it is isointense with adjacent brain. The lesion remains isointense on the T1-weighted precontrast scan (C). D, Postcontrast, the lesion is easily seen as a result of homogeneous enhancement. The lesion is also noted to invade the adjacent transverse sinus. With extra-axial lesions in particular, precontrast scans alone may fail to diagnose an abnormality or grossly underestimate its extent.

When meningiomas arise in the cavernous sinus or secondarily extend into this structure, encasement and displacement of the carotid artery are common. MRI offers improved evaluation of this type of vascular involvement over CT and angiography. Angiographically, it may be difficult to determine whether the change in vessel caliber is atherosclerotic in nature or caused by vascular encasement. With MRI, the soft tissue mass encasing the vessel, with narrowing of its caliber, is directly visualized. Contrast enhancement more clearly shows the enlargement of the cavernous sinus when meningiomas arise within or extend into it. The displaced lateral hypointense dural line
also becomes more evident. For small tumors and greater detail of involvement, thin section (3 mm) imaging is necessary.

En plaque meningiomas represent a special and often clinically frustrating type of meningioma (Fig. 24). These may become extensive, with involvement of the tentorium, cavernous sinus, brainstem, and cranial nerves. Transdural and subperiosteal spread may also occur. Total resection is often not possible, leading to recurrence and relentless enlargement. These lesions are also often not well seen, or go undetected, by CT.

Meningiomas in the cerebellopontine angle can be difficult to differentiate from acoustic schwannomas. Widening of the orifice of the internal auditory canal (IAC) favors an acoustic schwannoma. A wide dural base favors a meningioma. Although meningiomas can involve the sheath of cranial nerve VIII (and thus extend into the IAC), they typically do not cause focal enlargement within the canal.

CT often depicts osseous changes (secondary to a meningioma) better than MRI. However, MRI may detect osseous change not noted by CT because of the acquisition of scans in multiple planes; CT is restricted to the axial plane. Calcifications within lesions are better shown by CT. However, MRI rarely has difficulty with differential diagnosis because of the enhancement and extra-axial location of the lesion.

Figure 24. En plaque meningioma. A, The precontrast T2-weighted scan reveals edema adjacent to the atria of the right lateral ventricle. Sulcal effacement is seen in the right hemisphere on the precontrast T1-weighted scan (B). C, Postcontrast, an extensive homogeneous enhancing mass is identified extending along the posterior falx and right cerebral convexity. The mass follows the planes of the leptomeninges.

- **Acoustic Schwannoma**

Acoustic schwannomas (commonly and incorrectly referred to as "neuromas") are benign tumors that arise from the neurilemmal sheath of the vestibular division of cranial nerve VIII. Patients are usually 40 to 60 years of age and have unilateral sensorineural hearing
loss and tinnitus. Larger tumors with brainstem involvement cause unsteadiness, ataxia, vertigo, and diminished corneal reflexes (Fig. 25). Acoustic schwannoma is the most common benign extra-axial tumor of the posterior fossa.

On its clinical introduction, MRI rapidly replaced other imaging techniques for the diagnosis and evaluation of these tumors. Polytomography, iophendylate cisternography, and air-contrast CT were former techniques that involved significant radiation to the patient.

The latter two were also invasive, adding to patient morbidity. With MRI, the lack of signal from the adjacent dense bone allows direct visualization of cranial nerves VII and VIII. Thin-section (3 mm), high-resolution images are, however, necessary for appropriate diagnosis and evaluation of IAC tumors.

On precontrast scans, the tumor (if visualized) is isointense with brain on T1-weighted scans and iso to slightly hyperintense on T2-weighted scans. The lesion may be extracanalicular in location, intracanalicular, or both. Necrosis and hemorrhage are not uncommon in large extracanalicular lesions, causing further variability in signal intensity. Of all sequences, postcontrast scans best demonstrate both the intracanalicular and extra-canonical extent. Accurate knowledge of tumor extent is important in operative planning.

Contrast enhancement is important not only for assessing tumor extent but also for detecting small intracanalicular acoustic schwannomas (Fig. 26). Precontrast scans alone may miss small lesions within the IAC. Postcontrast, these are seen as brightly enhancing small soft tissue masses. The normal cranial nerve VIII does not enhance. Thus, any contrast enhancement in this region is abnormal. The degree of enhancement seen with acoustic schwannomas is greater than that for any other intracranial tumor. Enhancement is due to intrinsic lesion vascularity.

For accurate assessment, thin-section T1-weighted scans pre and postcontrast in both the axial and coronal planes are highly recommended in addition to a precontrast thin-section T2-weighted scan. MRI without contrast enhancement can produce both false-negative and false-positive results. In one series, the combination of these errors affected 10% of patients studied. Small intracanalicular tumors that went undetected without contrast could be seen with contrast. More alarming is the prospect of suggesting a tumor on precontrast scans when none can be found postcontrast. This can occur when the nerve appears (erroneously) to be enlarged on T1-weighted scans. Also, ectasia of the IAC can produce signal intensity on T2-weighted scans indistinguishable from that of intracanalicular tumor.
Figure 25. Acoustic schwannoma. A large soft tissue mass is noted in the left cerebellopontine angle on precontrast T2- (A) and T1-weighted (B) scans. C, Postcontrast, there is intense lesion enhancement consistent with a vascular extra-axial mass. Enlargement of the internal auditory canal (IAC) by the mass, with extension into the canal, favors diagnosis of an acoustic schwannoma. A meningioma, the other major consideration in differential diagnosis, is unlikely to enlarge the IAC.

Figure 26. Intracanalicular acoustic schwannoma. On precontrast T2- (A) and T1-weighted (B) scans, the question of a right-sided intracanalicular lesion is raised. C, Postcontrast, there is intense lesion enhancement (arrow), permitting definitive diagnosis. The clinical presentation was that of right-sided sensorineural hearing loss. Other entities to be considered in differential diagnosis include facial (seventh) nerve tumor and inflammatory disease, although the latter should not result in a mass lesion.

T1-weighted 3D gradient echo scans are used in some institutions for evaluating the IAC (replacing 2D axial and coronal T1-weighted spin echo scans). A high-resolution 3D scan can be acquired in less than 5 minutes. This approach offers high-resolution imaging in any desired plane, with postacquisition image reconstruction. Advantages over conventional
spin echo technique include thinner slices (typically 1 mm) and the absence of a gap between slices (true contiguous sections).

T2-weighted scans are not essential for the imaging evaluation of acoustic schwannomas. However, they are important for the differential diagnosis. Many abnormalities can mimic cranial nerve VIII disease clinically, and these are often better visualized with T2-weighted scans. Examples include multiple sclerosis, mastoiditis, and vascular brainstem compression.

A special word of caution is offered for evaluating postsurgical recurrence. In the translabyrinthine approach, the resected portion of the mastoid bone is often packed with an autologous graft that contains fat. The graft may be superimposed over the course of the nerve on axial scans. Coronal scans are then necessary to separate recurrent enhancing tumor from high-signal-intensity graft. Dural enhancement may also occur after surgery. Careful evaluation in both the axial and coronal planes is important for differentiation. Dural enhancement should be linear in character, with recurrent tumor presenting as a globular soft tissue mass.

- **Epidermoid**

Epidermoids (cholesteatomas) result from incomplete cleavage of neural from cutaneous ectoderm, with inclusion of ectodermal elements at the time of neural groove closure. Both midline (suprasellar and intraventricular) and more eccentrically located (cerebellopontine angle) lesions occur; the latter result from an inclusion at a slightly later stage of embryogenesis (Fig. 27). Epidermoids grow by desquamation of epithelial cells, which break down into keratin and cholesterol within the tumor capsule. These fatty elements are soft and pliable, and in the slow accumulation process they conform to the shape of the subarachnoid space or ventricle. The lesions are fairly well demarcated. Compression of adjacent structures occurs late. These congenital tumors may not become symptomatic until patients are 25 to 30 years old. Rupture occasionally produces chemical meningitis. As with other lipid tumors, their appearance on MRI depends on the type of fat and its physical state. Many contain cholesterol and show a prolongation of both T1 and T2 relaxation times. Such lesions are low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. A difference in the fat content or physical state yields brighter signal intensity on T1-weighted images. These tumors do not enhance. Thus, contrast administration is of little diagnostic value, except to exclude other cerebellopontine angle lesions with similar precontrast signal intensity (e.g., some meningiomas).
Figure 27. Epidermoid. An extra-axial mass with heterogeneous, but slightly higher signal intensity than cerebrospinal fluid (CSF), is noted in the right cerebellopontine angle cistern on an intermediate T2-weighted scan (A). The difference in signal intensity between the lesion and CSF is not apparent on a heavily T2-weighted scan (B). The mass is best demarcated on the precontrast T1-weighted scan (C). On this scan, the mass can again be differentiated from adjacent CSF, the latter with slightly lower signal intensity. The mass compresses the right middle cerebellar peduncle and right cerebellar hemisphere. There was no enhancement postcontrast (not shown).

- **Dermoid**

Dermoids are congenital tumors, like epidermoids, that arise from inclusion of ectodermal elements at the time of neural groove closure. The presence of hairs and other skin appendages differentiates a dermoid from an epidermoid tumor. Dermoids arise near the midline and are less common than epidermoids. Most intracranial dermoids are located in the posterior fossa. Most spinal canal dermoids occur in the lumbosacral region. Dermoids may contain fat, hair follicles, and glandular elements (sebaceous and apocrine). Those containing a large amount of fatty elements have high signal intensity on T1-weighted scans and lower signal intensity on conventional T2-weighted scans (Fig. 28). Dermoids are not vascular tumors and do not cause BBB disruption. Thus, they do not enhance after contrast administration.
Figure 28. Ruptured dermoid. A, The intermediate T2-weighted scan reveals scattered areas of abnormal hyperintensity, with many exhibiting a low-signal-intensity border along the frequency encoding direction (chemical shift artifact). Axial (B) and sagittal (C) precontrast T1-weighted scans confirm the presence of scattered abnormalities, which remain hyperintense. The more peripheral globules are noted to lie within cortical sulci. By recognition of chemical shift, high signal intensity resulting from fat (as in this case) can be differentiated from methemoglobin. This case also illustrates the importance of obtaining precontrast T1-weighted scans to identify fat or blood that might otherwise be mistaken for abnormal contrast enhancement.

- **Arachnoid Cyst**

Arachnoid cysts are benign lesions that contain CSF. Most are congenital in origin. Less common causes include inflammation, trauma, and subarachnoid hemorrhage. Their importance lies in differentiation from other masses, including epidermoids, dermoids, subdural hygromas or hematomas, and cystic tumors. Arachnoid cysts most frequently occur in the middle cranial fossa. Other common locations include the posterior fossa (retrocerebellar) (Fig. 29), the suprasellar region, the quadrigeminal plate, and the cerebral convexities. The cyst is lined by arachnoid membrane and filled with fluid that is usually clear but on occasion slightly xanthochromic. The margins of an arachnoid cyst are sharply defined. The signal intensity is usually identical to that of CSF. No contrast enhancement occurs.
Figure 29. Posterior fossa arachnoid cyst. Sagittal (A) and axial (B) T2-weighted scans reveal a lesion, with cerebrospinal fluid signal intensity, posterior to the vermis and right cerebellar hemisphere. Mass effect is evident both by the anterior displacement of the vermis and the upward bowing of the posterior portion of the tentorium.

- **Leptomeningeal Metastases**

Tumors that have access to the subarachnoid space may spread via the CSF or along the meninges. Tumors that, because of their origin in or near the ventricular system, spread via the CSF include ependymomas, medulloblastomas, pineal region tumors, and occasionally glioblastomas. Metastases from these primaries, often called "drop metastases," seed more commonly to the spine. Tumors that spread via cortical or meningeal involvement include metastatic breast carcinoma, melanoma, lymphoma, leukemia, and calvarial metastases with secondary meningeal involvement. Diffuse meningeal changes may be monitored by parenchymal deposits. These occur after the malignant meningeal lesions dip into the perivascular spaces of Virchow-Robin and spread to the parenchyma, forming nodular metastases.

Leptomeningeal metastases are not well seen by CT. Before approval of the gadolinium chelates, the same was true for MRI. Currently, contrast-enhanced MRI is the technique of choice for the diagnosis of leptomeningeal disease. In the brain, leptomeningeal metastases are visualized as abnormal contrast enhancement, linear or nodular in character, lining the meningeal surface and extending into sulci and cisterns (Fig. 30).
Figure 30. Meningeal carcinomatosis. Scans were taken 1 year after surgical resection and whole brain radiation for a right occipital metastasis from breast carcinoma. Abnormal high signal intensity, without a specific focal lesion, is noted in the right parietal and occipital lobes on the precontrast T2-weighted scan (A). No additional information is provided by the precontrast T1-weighted scan (B). C, After contrast administration, recurrent tumor is identified, marked by intense enhancement, along the surface of the brain in the area of prior resection.

PITUITARY AND PARASELLAR REGION TUMORS

After its clinical introduction, MRI rapidly replaced CT for evaluating the pituitary and parasellar region. The inherent advantages of MRI are of even greater importance in this small region. High-resolution imaging is possible in all planes without the need for image reformatting. Dental amalgam causes no artifacts. On CT, this often restricts the use of direct coronal scans. CT also poses the problem of radiation dose. Serial exams are often required in younger patients with hormonally active, but predominantly benign, lesions. Perhaps the greatest advantage of MRI is the superior depiction of soft tissue (without the presence of bone artifacts). This is particularly important in the imaging of such a small anatomic region situated in the dense skull base. Normal anatomic structures, including the cavernous sinus, internal carotid artery, and cranial nerves, are well visualized. Intrinsic abnormalities within the pituitary are easily recognized. Furthermore, the distinction between parasellar aneurysms and intrasellar tumor, a major pitfall with CT, is not a problem with MRI.

The identification and characterization of lesions in the sella and parasellar region require thin-section imaging (3 mm). Spin echo technique typically can provide no thinner than 2-mm sections, whereas 3D gradient echo technique can provide 0.5 to 1-mm sections. The latter technique is also advantageous in that the slices are truly contiguous without an intervening gap. Images from a high-resolution 3D data set can be reformatted in multiple planes, further improving the diagnostic value of the exam. For gradient echo scans, TEs should be short (1.5 milliseconds) to avoid susceptibility ("blow-out") artifacts at the air-soft tissue interface between the sella and the sphenoid sinus.

Regarding the relative utility of T1- and T2-weighted scans, the first provide excellent delineation of anatomy. T2-weighted scans are useful for recognizing necrosis and cystic changes and for characterizing areas of high signal intensity on T1-weighted scans.
Necrosis and cystic changes within the pituitary, as in the brain, are low signal intensity on T1-weighted scans and high signal intensity on T2-weighted scans. High signal intensity on precontrast T1-weighted scans corresponds to subacute hemorrhage or high lipid content; the latter is seen in some craniopharyngiomas. Extracellular methemoglobin is high signal intensity on both T2- and T1-weighted scans. Lipid has high signal intensity on T1-weighted scans yet low signal intensity on conventional T2-weighted scans. With the exception of these changes, pituitary abnormalities are characterized using T1-weighted scans before and after contrast administration. Most protocols call for precontrast T2-weighted coronal or sagittal scans (one plane only) and T1-weighted coronal and sagittal images (both planes) before and after contrast administration. Abnormalities are identified as a result of greater enhancement of normal adjacent structures, as in the case of many microadenomas, or of enhancement of the lesion itself (on the basis of intrinsic vascularity), as in the case of macroadenomas.

- Normal Pituitary Gland

The size of the normal pituitary gland varies widely. A height of 10 mm is considered the upper limit of normal, with two exceptions. During puberty and the early child-bearing years, the gland may be up to 12 mm in height. The upper surface of the gland may be flat, concave, or convex in the midline.

T1-weighted images provide excellent anatomic definition. In the coronal plane, the pituitary is localized as a soft tissue structure lying between the rounded areas of signal void from the internal carotid arteries. The signal intensity of the gland is similar to the white matter of brain. In the sagittal plane, the anterior and posterior lobes of the pituitary can be distinguished by the high signal intensity of the posterior lobe. Immediately posterior to the pituitary itself is the high-signal-intensity marrow of the dorsum sellae. Frequently, there is a normal area of increased signal intensity on T1-weighted images at the base of the pituitary. This may be mistaken for an abnormality but actually represents fatty marrow in the upper extreme of a sphenoid sinus septum.

The optic chiasm and pituitary stalk are outlined by low-signal-intensity CSF in the suprasellar cistern on T1-weighted images. These structures are easy to identify in both the sagittal and coronal planes. The coronal plane is more useful for assessing gland symmetry. The cavernous sinus, with the internal carotid artery, cranial nerves III through VI, and the lateral dural margin, is also best evaluated in the coronal plane. The anteroposterior dimension of the sella turcica is obtained from sagittal images, which also provide an important second view for lesion visualization. Postcontrast, the pituitary gland, stalk, and cavernous sinus show intense enhancement, greatly facilitating the diagnosis of sellar and parasellar disease.

On T2-weighted images, the gland is isointense with white matter (as on T1-weighted images). CSF in the suprasellar cistern is high signal intensity. The low signal intensity of the lateral dural margin of the cavernous sinus is better defined than on T1-weighted images.
• **Microadenoma**

Microadenomas are defined as lesions smaller than 10 mm in diameter. Production of hormones brings these lesions to clinical attention early and thus when small. The most common microadenoma is the prolactinoma. These tumors secrete prolactin and present with infertility, amenorrhea, and galactorrhea in women and galactorrhea and impotence in men. Imaging findings include focal asymmetry of the gland surface, displacement of the pituitary stalk to the contralateral side, and a low-signal-intensity focal mass on T1-weighted scans. Hemorrhage within the lesion may cause high signal intensity on precontrast T1-weighted scans. On T2-weighted scans, prolactinomas can be hypo-, iso-, or hyperintense. On early postcontrast scans, most prolactinomas are hypointense compared with the normal pituitary and infundibulum (which both enhance intensely). Contrast injection thus facilitates lesion detection (Fig. 31). A small number of tumors are isointense to the normal pituitary precontrast and hypointense postcontrast. With thin-section, high-resolution (small field of view) scans, evaluation of these tumors by MRI is superior to that by CT.

![Figure 31. Pituitary microadenoma (prolactinoma). Asymmetry of the sellar floor is noted on the T2-weighted coronal scan (A). A definite mass cannot be identified on either this scan or the T1-weighted coronal scan (B). C, Postcontrast, the normal pituitary demonstrates intense enhancement, revealing a large, hypointense, left-sided pituitary microadenoma (arrow).](image)

Cushing's syndrome is caused by adrenocorticotropic hormone (ACTH)-producing adenomas of the pituitary in 60% of cases. If not in the pituitary, these tumors arise in the adrenal gland or in ectopic sites. Clinical symptoms include truncal obesity, abdominal striae, moon facies, acne, hypertension, psychiatric disturbances, and amenorrhea and hirsutism in women. These occur because of excess cortisol production. Clinical symptoms usually bring these tumors to attention while still small. Detection on CT is difficult; less than half of all lesions are diagnosed. Presurgical localization still relies in some cases on petrosal vein sampling, an invasive and technically difficult angiographic procedure. Limited experience with MRI indicates a very high detection rate (80-100%).

• **Macroadenoma**

Large pituitary adenomas are rarely a diagnostic dilemma for CT or MRI. These bulky tumors are usually hormonally inactive, with a few tumors secreting prolactin. Because of the improved depiction of soft tissue, MRI can better assess suprasellar and lateral...
temporal extension (Fig. 32). The cavernous sinus can be displaced by tumor or on occasion can be invaded with encasement of the internal carotid artery. Macroadenomas are isointense with white matter on T1-weighted images, unless there is associated hemorrhage. Subacute hemorrhage in most tumors, including macroadenomas (with high signal intensity on T1-weighted images), is better demonstrated by MRI than CT. Hemorrhage within macroadenomas is more common than was once thought based on CT and clinical criteria. Pituitary apoplexy is defined as spontaneous hemorrhage into or ischemic necrosis of a normal pituitary or an adenoma. Before MRI, pituitary apoplexy was equated with severe neurologic symptoms, including sudden alteration in mental status and occasionally blindness. It is now known from MRI that small hemorrhages may be accompanied by no more than a severe headache.

On T2-weighted images, macroadenomas have intermediate, homogeneous signal intensity. Necrosis causes foci of high signal intensity. If the necrotic portion is substantial in size, differentiation from a craniopharyngioma can be difficult. A distinguishing feature is the size of the sella, usually substantially enlarged with a macroadenoma.

Macroadenomas demonstrate substantial enhancement postcontrast. The presence of liquefaction or necrosis, which does not enhance, produces patchy enhancement postcontrast. Tumor margins are better seen after contrast administration. Tumor extent can be underestimated precontrast, with greater extent demonstrated postcontrast. Involvement of the cavernous sinus is easier to assess postcontrast, with the sinus enhancing to a greater degree than the macroadenoma.

Figure 32. Pituitary macroadenoma. A large pituitary mass, with suprasellar extension, is identified on coronal pre- (A) and postcontrast (B) T1-weighted scans. The lesion is isointense to gray matter precontrast and demonstrates homogeneous enhancement postcontrast. The optic chiasm is markedly thinned as a result of compression by the suprasellar portion of the mass.
- **Craniopharyngioma**

Craniopharyngiomas are benign, slow-growing tumors that arise from nests of epithelium derived from Rathke's pouch. In regard to age of presentation, there are two peaks: one in childhood and the other in adults older than 50 years. These tumors most often are suprasellar in location; thus, the sella will not be enlarged. Occasionally, a portion of the tumor may extend into the sella, causing slight enlargement. However, the sella typically does not attain the size seen with macroadenomas. Although rare, a craniopharyngioma can arise within the sella. Intrasellar lesions are smaller at presentation than the more common suprasellar tumor and are difficult to differentiate from prolactinomas or Rathke's cleft cysts (a benign congenital cyst that can be intrasellar or suprasellar in location). Intrasellar craniopharyngiomas are usually accompanied by amenorrhea and galactorrhea resulting from low levels of prolactin.

Craniopharyngiomas are usually predominantly cystic with a small soft tissue component (Fig. 33). Most craniopharyngiomas are very low signal intensity on T1-weighted scans because of a large cystic component containing relatively clear fluid. High signal intensity on precontrast T1-weighted scans can also be seen resulting from high cholesterol content or byproducts (methemoglobin) from previous hemorrhage. The cystic portion of the tumor is usually very high signal intensity on T2-weighted scans.

![Figure 33. Craniopharyngioma. A, A high-signal-intensity suprasellar lesion is noted on the precontrast T2-weighted scan. Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals a solid enhancing nidus anteriorly and a cystic rim-enhancing component posteriorly. Craniopharyngiomas are complex heterogeneous masses with both cystic and solid components. Contrast administration aids in the differential diagnosis and definition of lesion extent.](image)

Suprasellar craniopharyngiomas vary from small lobulated to large multicystic septated lesions. Tumor margins are usually smooth and rounded. Craniopharyngiomas in children tend to be larger and contain more calcification. Postcontrast, the cyst walls of a craniopharyngioma enhance. There may also be areas of nodular enhancement. Contrast-
enhanced scans aid in differential diagnosis. The normal pituitary, which enhances brightly, can be separated from suprasellar tumor or from tumor that partially extends into the sella. Contrast-enhanced scans also aid in visualization of craniopharyngiomas that are not large enough to obliterate the suprasellar cistern. In this case, the signal intensity of the tumor blends with the signal intensity of the suprasellar cistern on T2-weighted studies. Although calcifications are not well visualized on MRI, the location of the lesion and dominant cystic component, along with the presence of septations and lobulations, enables correct diagnosis in most instances.

- **Other Parasellar Tumors**

MRI, especially when used in conjunction with contrast media, is particularly effective in visualizing and clearly localizing other parasellar tumors. MRI also well defines the relationship of these lesions to important adjacent structures, such as the cavernous sinus, brainstem, and optic chiasm. These tumors include chordomas, hypothalamic gliomas, and meningiomas. With the exception of the hypothalamic glioma, which shows variable enhancement, these tumors show excellent enhancement.

Because of their propensity to invade adjacent sinuses and encase arterial structures, meningiomas produce special imaging problems, particularly in view of their variable delineation on T2-weighted images. In the parasellar region, these tumors generally require thin-section imaging for definition of venous and arterial involvement. Examination of other parasellar tumors is also benefited by thin-section imaging because of the compact regional anatomy and proximity of crucial structures.

**TUMORS OF BONE**

- **Chordoma**

Chordomas are rare, slow-growing primary bone tumors that originate from remnants of the primitive notochord. The primitive notochord extends from Rathke's pouch to the clivus, continuing along the vertebral column. Remnants of the notochord can occur at any location along this line.

Thirty-five percent of chordomas are intracranial, and most of these arise from the clivus. Fifty percent are sacrococcygeal, and 15% arise from within a vertebral body. Within the calvarium, chordomas may involve the posterior or middle fossa by extension through the dura. The majority of these tumors cause extensive destruction of bony structures. Chordomas rarely metastasize to distant sites but are locally aggressive. Total surgical resection is rarely possible. Although locally invasive, chordomas are histologically benign. Macroscopically, chordomas are soft gelatinous tumors that frequently result in destruction of the clivus and skull base. They occur most commonly in men in the third and fourth decades. Patients present with headaches, facial pain, progressive cranial nerve palsies, and nasal stuffiness.
Calcification is identified in 50% to 60% of cases on CT. On MRI imaging, chordomas are usually well-defined, extra-axial tumors that show isointensity or mild hypointensity on T1-weighted images and moderate to extreme high intensity on T2-weighted images. Approximately 70% of chordomas have septations of low signal intensity separating lobulated areas of higher signal intensity on T2-weighted images. Chordomas typically enhance after contrast administration.

- **Metastases**

The normal diploic space does not enhance, except for diploic veins and the meninges near pacchionian granulations. Diploic veins appear as linear or small round (if cut in cross-section) foci of low to moderate signal intensity on precontrast MRI images, with enhancement postcontrast. The diploic space can appear inhomogeneous with areas of increased (resulting from fatty marrow) and decreased (caused by bony sclerosis or suture lines) signal intensity on precontrast scans. However, the diploic space should be symmetric from side to side. Gross asymmetry is highly suggestive of calvarial disease, even in the absence of appreciable destruction of the inner or outer table. Calvarial metastases enhance after intravenous contrast administration (Fig. 34).

![Figure 34. Calvarial metastases. There is widening of the diploic space in the right parietal and left frontal regions on A, the precontrast T2-weighted scan. B, The marrow space in the left frontal region appears enlarged on the precontrast T1-weighted scan. The soft tissue here is also of lower signal intensity than normal marrow fat. C, Postcontrast, there is intense enhancement of soft tissue within the diploic space in the right parietal and left frontal regions consistent with bony metastatic disease. Contrast administration, as in this case, can improve recognition of metastatic involvement of the diploic space as a result of the enhancement of neoplastic tissue. Comparison with precontrast scans is mandatory.](#)  

- **Eosinophilic Granuloma**

Langerhans cell (eosinophilic) granulomatosis is the term currently preferred for eosinophilic granuloma syndromes. This replaces older nomenclature, including
histiocytosis X, which referred to a spectrum of diseases now known to include this benign entity and malignant lymphoma.

Unifocal Langerhans cell granulomatosis is a disease of children and young adults, predominantly males, who present with a solitary osteolytic lesion (most often in the femur, skull, vertebrae, ribs, or pelvis). Diagnosis requires biopsy; treatment is simple excision. The typical presentation in neuroradiology is that of a solitary skull lesion. MRI demonstrates a soft tissue mass, centered in the diploic space, with adjacent bone destruction (Fig. 35). The lesion may extend into the epidural or subgaleal space. Eosinophilic granulomas enhance prominently postcontrast.

Multifocal Langerhans cell granulomatosis also presents in childhood, with multiple bony lesions in virtually any site. Diabetes insipidus occurs in one third as a result of hypothalamic involvement. The term Hand-Schuller-Christian syndrome was previously used to refer to the disease triad of destructive bone lesions, diabetes insipidus, and exophthalmos. However, only 25% of patients with multifocal Langerhans cell (eosinophilic) granulomatosis have this triad, which can also be caused by malignant lymphoma and carcinoma. Although benign, multifocal disease is treated with methotrexate, vinblastine, or prednisone.
Figure 35. Eosinophilic granulomatosis. An expansile diploic space mass is identified on axial T2- (A) and sagittal T1- weighted (B) images. On axial (C) and coronal (D) postcontrast scans, there is a thick peripheral rim of abnormal contrast enhancement. The sagittal and coronal scans demonstrate focal expansion of the diploic space. Differential diagnosis plays an important role in scan interpretation in this instance, with imaging findings (a solitary lesion) and clinical information (a young man with headaches and a "bump" on his head) favoring a diagnosis of eosinophilic granuloma (proven by subsequent resection).
POSTOPERATIVE TUMOR EVALUATION

The evaluation of tumors that recur after surgery is difficult without contrast administration. Subtle mass effect and postsurgical encephalomalacia are difficult to assess in regard to the question of tumor recurrence. Differentiation of encephalomalacia from edema associated with tumor recurrence is also difficult. Both have increased signal intensity on T2-weighted scans. Extension of abnormal high signal intensity into the corpus callosum, without volume loss, is, however, specific for tumor. Edema does not track into corpus callosum because of the compact nature of the nerve fibers. Cystic, necrotic, and hemorrhagic changes are well seen on precontrast T1-weighted scans. However, underlying tumor may be difficult to detect. Contrast-enhanced MRI is markedly superior to enhanced CT for demonstrating tumor recurrence (Fig. 36). One study demonstrated that 50% of postoperative tumor recurrences were primarily or more conclusively shown by contrast-enhanced MRI. Identification of recurrent tumor and delineation of the margin of tumor extent were both improved. Caution should be used, however, in the interpretation of tumor recurrence after radiation therapy. Both recurrent tumor and radiation necrosis can present as an enhancing lesion with surrounding edema and mass effect. These two entities cannot be differentiated on the basis of conventional MRI techniques. Regional cerebral blood volume (CBV) studies do offer the capability of distinguishing recurrent tumor (with high CBV) from radiation necrosis (with low CBV). This advanced type of study is performed by acquiring rapid images (on the order of one per second) after bolus contrast injection using a power injector during the first pass of the contrast agent through the brain.

Figure 36. Recurrent astrocytoma. A large postsurgical defect, communicating with the atria of the right lateral ventricle, is noted on precontrast T2(A) and T1-weighted (B) scans. The exam was performed to rule out tumor recurrence in this elderly patient with resection of an astrocytoma 4 years earlier. Medial to the postsurgical defect is soft tissue with signal intensity similar to that of normal brain. The question of tumor recurrence is raised by the slight hyperintensity of this soft tissue on the precontrast T1-weighted scan. C, After
contrast administration, there is intense enhancement, making possible definitive diagnosis of recurrent tumor.

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INTRODUCTION

Infection may reach the intracranial contents by hematogenous spread, direct extension (e.g., from sinusitis), and spread along peripheral nerves (e.g., herpes encephalitis). MRI is extremely valuable for early detection of parenchymal disease. Dystrophic calcification, which represents the primary finding on CT in chronic and congenital infection, is poorly visualized.
Figure 1. Brain abscess. A mixed low- and high-signal-intensity abnormality, with a thin hypointense rim and surrounding high- signal-intensity edema, is noted on the T2-weighted scan (A). On the postcontrast T1-weighted scan (B), a thin uniform rim of abnormal enhancement is noted. Characteristic features of a brain abscess include location at the corticomedullary junction and the presence of a smooth, well-defined, enhancing capsule. Necrotic contents are typically heterogeneous in signal intensity. Cultures for the lesion were positive for gram-positive cocci

- Parenchymal Disease

Staphylococcus, Streptococcus, and more recently Toxoplasmosis (in AIDS) are the common organisms responsible for focal parenchymal brain infections. The temporal evolution of brain infection has been carefully studied on both CT and MRI. An abscess evolves from an early focus of cerebritis to a more mature stage with a discrete capsule. Abnormal contrast enhancement occurs as a result of blood-brain barrier disruption (Fig. 1). Contrast enhancement on MRI permits early lesion identification (with sensitivity superior to that of unenhanced MRI and enhanced CT) and differentiation of cerebritis and capsule stages. Cerebritis demonstrates focal enhancement, often ill defined, while the capsule stage demonstrates ring enhancement (Figs. 2,3). Enhanced MRI also provides more precise delineation of disease extension. The evolution of intracranial infection, whether treated by antibiotic therapy or neurosurgical drainage, is well evaluated by MRI.

Incidental sinus disease is commonly seen on MRI. The spectrum of disease includes retention cysts and mucosal inflammation. Much less common is active infection. Intracranial complications from sinus infection include meningitis, abscess, and sinus thrombosis. The presence of a true air-fluid level within the sinus, opacification of the sinus by soft tissue with intermediate signal intensity on T2-weighted scans, and prominent
abnormal contrast enhancement (Fig. 4), given the appropriate clinical presentation, point toward acute sinus infection.

Figure 2. Cryptococcosis. Two areas of abnormal high signal intensity are noted on the T2-weighted scan (A) consistent with cerebral edema. Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals three ring-enhancing lesions, two of which (on the patient’s left) are adjacent to one another. Cryptococcus is a ubiquitous fungus that grows in tissue as yeast cells and spreads hematogenously. This organism usually causes leptomenigitis, which may be either acute or chronic. Parenchymal lesions, as featured in this case, are less common.
Figure 3. Neurocysticercosis. On the precontrast T2-weighted scan (A), an ovoid area of abnormal high signal intensity is noted in the region of the sylvian fissure. On the T1-weighted scan after contrast administration (B), there is ring enhancement of the lesion, with a suggestion of septations. In neurocysticercosis (infection by the larval stage of the pork tapeworm), the patient may present with either seizures, because of parenchymal cysts, or obstructive hydrocephalus, because of intraventricular cysts. On magnetic resonance imaging, the cysts have fluid signal intensity, with ring enhancement postcontrast of the cyst wall.
Figure 4. Mastoiditis, with transverse and sigmoid sinus thrombosis. The patient is a 7-year-old boy with right earache, nausea, vomiting, and low-grade fever. Physical exam revealed a right sixth nerve palsy and a very erythematous right tympanic membrane. On the precontrast T2-weighted scan (A), there is abnormal mixed signal intensity in the right mastoid air cells and petrous bone. Note that this abnormal soft tissue does not have high signal intensity, which is a
common finding as a result of inflammation (but without active infection). The presence of abnormal soft tissue is confirmed on the precontrast T1-weighted scan (B); the postcontrast scan (C) reveals prominent enhancement (white arrow). The sigmoid sinus remains at low signal intensity on all scans, suggesting occlusion. On a follow-up precontrast T1-weighted scan obtained 10 days later (D), there is abnormal hyperintensity (black arrow) in the right transverse sinus consistent with evolution of thrombus (in the transverse sinus) from deoxyhemoglobin to methemoglobin. Repeat exam 1 month later demonstrated recanalization of the sinus (scans not shown).

![Image](image-url)

Figure 5. Herpes simplex type 1 encephalitis. A, The T2-weighted scan reveals abnormal high signal intensity in the insula bilaterally and in the right frontal lobe. Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals abnormal meningeal enhancement within the sylvian fissure on the right. Herpes encephalitis in the adult most often affects the temporal and inferior frontal lobes. Meningeal enhancement is seen in the acute phase of the disease.

The most common cause of diffuse parenchymal infection is viral. The brain responds to insult with an inflammatory infiltration of lymphocytes and mononuclear cells. Petechial hemorrhage can result from vascular necrosis. Herpes simplex type 1 encephalitis typically involves the temporal lobe, although involvement may extend to the frontal or parietal lobes (Fig. 5). The basal ganglia are usually spared. MRI allows early diagnosis and can document effective response to therapy. Coronal imaging is useful for improved visualization of temporal lobe disease in this and other diseases. Herpes simplex type 2 encephalitis can occur in the infant exposed at birth during vaginal delivery. Infection in the infant causes a widespread necrotizing meningoencephalitis. Early in the disease course, brain edema may be patchy or widespread. Areas of involvement increase rapidly in size. Late findings include cortical atrophy and multicystic encephalomalacia. On CT, punctate or gyral calcification can also be seen at this stage.
Figure 6. Acute disseminated encephalomyelitis (ADEM). There are multiple high-signal-intensity white matter lesions, both infra(A) and supratentorial (B) in location, on the T2-weighted scans. In the supratentorial white matter, the lesions appear to be more peripheral than periventricular (unlike characteristic plaques in multiple sclerosis). The lesions also appear to have an indistinct margin (they appear "fluffy"). Computed tomography was within normal limits. ADEM is thought of as a monophasic illness and is known to occur after vaccination and minor viral infections.

Acute disseminated encephalomyelitis is an inflammatory and demyelinating disorder of white matter, which can occur after a childhood viral infection. CT is usually nondiagnostic. MRI demonstrates multiple foci of demyelination in the brainstem, cerebellum, and cerebrum (Fig. 6). Lesions are relatively few and nonhemorrhagic, with asymmetric involvement of the left and right hemispheres. Follow-up MRI exams can demonstrate resolution of lesions in conjunction with clinical improvement. MRI is an important modality for diagnosing acute disseminated encephalomyelitis because of its ability to identify the sites and extent of involvement and response to therapy.

Two main patterns of brain involvement occur with sarcoidosis. Parenchymal disease presents with symptoms of an intracranial mass lesion. Periventricular and more peripheral white matter lesions can be seen. This pattern in certain instances is indistinguishable from that of MS. The parenchymal lesion, granulomatous in nature, is the result of disease spread via the Virchow-Robin spaces. Parenchymal involvement is typically accompanied by leptomenigitis. Meningeal disease can present with cranial nerve palsies, meningeal signs, and hypothalamic dysfunction. The granulomatous leptomeningitis seen in sarcoidosis involves the skull base and can be either focal or diffuse (Fig. 7). As with other brain infections, MRI is more sensitive than CT and better demonstrates the extent of disease.
**Figure 7. Neurosarcoïdosis.** A, The T2-weighted scan appears to be normal. On the postcontrast T1-weighted scan (B), there is diffuse enhancement of the leptomeninges. On imaging, two major patterns of brain involvement are seen with neurosarcoïdosis: (1) granulomatous leptomeningitis and (2) parenchymal involvement because of spread along the Virchow-Robin spaces.

- **Meningeal Disease**

Contrast-enhanced MRI is markedly superior to CT for the detection of meningeal disease. Unfortunately, neoplastic, inflammatory, and traumatic changes often cannot be differentiated. Contrast-enhanced MRI is also more effective than CT in the identification of complications of meningitis, including ventriculitis and cerebritis. Abnormal areas of contrast enhancement correlate pathologically with inflammatory cell infiltration (Fig. 8). Pathology studies also reveal that inflammation can extend beyond the region identified by abnormal contrast enhancement.

Dural enhancement is common after intracranial surgery (Fig. 9). Head trauma is also recognized as a cause of dural enhancement. Once present, dural enhancement can persist indefinitely. Abnormal enhancement is likely the result of a chemical arachnoiditis caused by blood. Involvement of the pia-arachnoid (with or without dural involvement) (Fig. 10), indicative of acute meningitis, should be distinguished from involvement of the dura alone, the latter commonly chronic in nature. MRI is also superior to CT for detecting extracerebral fluid collections (Fig. 11). Epidural and subdural hematomas appear smaller on CT as a result of Hounsfield artifact. Contrast-enhanced MRI plays an important role in the diagnosis and follow-up of subdural and epidural empyemas (Fig. 11). Early diagnosis is critical with subdural empyemas because of the possible sequelae of cortical
venous thrombosis and infarction. If all pulse sequences are compared, purulent fluid can be distinguished from CSF because of the shortening of T1 and T2. Contrast enhancement is marked, consistent with infection. Epidural empyemas can be caused by the extension of sinus or ear disease or can occur as a complication after neurosurgical intervention.

Figure 8. Viral meningitis. A, The T2-weighted scan is grossly normal, with the exception of ventricular dilatation. On the precontrast T1-weighted scan (B), the gray matter immediately adjacent to cortical sulci appears to have too low signal intensity. That the cortical gray matter is diffusely edematous is indirectly confirmed by the postcontrast T1-weighted scan (C), which demonstrates diffuse abnormal leptomeningeal enhancement. The imaging appearance of viral meningitis, with diffuse enhancement of the pia arachnoid, is indistinguishable from that of bacterial meningitis.
**Figure 9.** Postsurgical dural enhancement. Comparison of precontrast T2- (A) and T1-weighted (B) scans with postcontrast axial (C) and coronal (D) T1-weighted scans reveals diffuse intense dural enhancement. Identification of a ventricular shunt (arrow) on the coronal scan (D) suggests the cause: recent surgery. Dural enhancement, once present, is likely to remain for life. Although typically representative of chronic disease, it can be seen in acute settings and with active infection.

**Figure 10.** Bacterial meningitis (postoperative). A, On the T2-weighted scan, edema in the pons, middle cerebellar peduncle, and cerebellar hemisphere is noted. Postoperative
changes are present, including fat packing. The latter is best seen on the precontrast T1-weighted scan (B). The patient is in the early postoperative period after resection of a large acoustic neuroma. There is mass effect on the brainstem and fourth ventricle. C, On the postcontrast T1-weighted scan, there is intense enhancement of the dura, in particular at the site of recent surgery. Mild cases of meningitis may show no abnormality on magnetic resonance scans. Severe disease will display marked enhancement of the coverings of the brain.

Figure 11. Bilateral subdural hematomas. On the T1-weighted scan, high-signal-intensity extra-axial fluid collections are noted bilaterally. On the T2-weighted scan (not shown), the collection on the right was also high signal intensity, but the collection on the left was low signal intensity. This indicated that the subdurs were of different ages: the one on the right was made up of extracellular methemoglobin and that on the left, intracellular methemoglobin.

- AIDS

Greater sensitivity to disease involvement makes MRI superior to CT for the examination of patients with AIDS and its central nervous system complications. White matter lesions are clearly visualized on T2-weighted scans. Contrast enhancement is important for biopsy localization, judging lesion activity, and detecting small cortical lesions with minimal surrounding edema. Diffuse periventricular hyperintensity on T2-weighted scans is common in HIV encephalitis (Fig. 12). These changes are a result of a direct neurotrophic effect of the virus. Cortical atrophy and ventricular enlargement are found in virtually all patients with HIV encephalitis (Fig. 13), reflecting chronic infection and prolonged debilitation.

Toxoplasmosis is a ubiquitous obligate intracellular protozoan. Approximately 50% of the U.S. population have been exposed and have antibodies. Transmission is through insufficiently cooked meat and handling of cat feces. Toxoplasmosis is an important pathogen in the fetus and the immunocompromised patient. Transmission to the fetus occurs during acute infection of the mother.
Figure 12. Epidural abscess (empyema), with accompanying osteomyelitis. A 24-year-old patient presented with progressive right-sided headache, fever, and vomiting for 3 weeks. Blood cultures were positive for Salmonella. Abnormal high signal intensity, representing subcutaneous soft tissue edema, is seen on the T2-weighted scan (A) in the right frontal region. There is also a subtle abnormal increase in signal intensity of the adjacent diploic space (between the inner and outer tables of the skull). The presence of an extra-axial mass, together with involvement of the adjacent marrow, is confirmed by enhancement (white arrow) on the postcontrast T1-weighted scan (B). The disease involvement is better demonstrated by comparison of the pre- (C) and postcontrast (D) coronal scans. C, Note the hypointense rim, corresponding to the dura (black arrow), separating the lesion from normal brain.
Figure 13. HIV encephalitis. A man in his 40s presented with dementia and was found to be HIV positive. On the T2-weighted scans, there is diffuse increased abnormal high signal intensity in the white matter bilaterally. Diffuse periventricular white matter hyperintensity, on T2-weighted scans, is a hallmark of HIV encephalitis.
Figure 14. HIV encephalitis. A 33-year-old HIV-positive man with a CD4 count of 36 presented with progressive mental confusion over the past several years. A, The T2-weighted scan demonstrates diffuse abnormal high signal intensity in the periventricular white matter. B, The T1-weighted scan demonstrates central and peripheral (cortical) atrophy. There is also loss of the normal differentiation between gray and white matter (on the basis of signal intensity). There was no abnormal contrast enhancement (not shown). Atrophy is the most common magnetic resonance finding in the AIDS dementia complex. More severe grades of deep white matter abnormality are associated with the AIDS dementia complex. Distinguishing progressive multifocal leukoencephalopathy (PML), when extensive, from HIV infection can be problematic.

Figure 15. Toxoplasmosis. Bilateral high-signal-intensity abnormalities are noted in the basal ganglia on the T2-weighted scan (A). Comparison of pre- (B) and postcontrast (C) T1-weighted scans reveals faint rim enhancement indicative of active disease. Cerebral edema, depicted as high signal intensity on the T2-weighted scan and low signal intensity on the T1-weighted scan, is noted surrounding the larger lesion, specifically extending beyond the thin rim of enhancement defined on the postcontrast scan. The presence of multiple nodular or ring-enhancing basal ganglia (or gray-white matter junction) lesions in the immunocompromised patient suggests the diagnosis of toxoplasmosis, which is the most common intracranial opportunistic infection in AIDS. Other considerations include metastatic disease and lymphoma.
Figure 16. Progressive multifocal leukoencephalopathy (PML). A small focal area of abnormal high-signal-intensity white matter (arrow) is noted in the left frontal lobe on the T2-weighted scan (A). B, The precontrast T1-weighted scan reveals subtle abnormal low signal intensity in the corresponding region. There is no abnormal enhancement on the postcontrast T1-weighted scan (C). Focal areas of abnormal white matter with high signal intensity on T2-weighted scans, often in an asymmetric distribution, are characteristic of PML. Lesions most often involve the periventricular and subcortical white matter in the parieto-occipital or frontal lobes.

The result is a focal or diffuse encephalitis. Scattered intracranial calcifications and atrophy are seen in chronic disease. Toxoplasmosis is also the most common intracranial opportunistic infection in AIDS. The disease can be due to reactivation of latent infection or fulminant acquired infection. Toxoplasmosis lesions in the brain demonstrate nodular or ring enhancement postcontrast on T1-weighted scans, with surrounding cerebral edema clearly depicted on T2-weighted scans (Fig. 14). A common presentation is that of multiple small lesions smaller than 2 cm in diameter. Common locations include the basal ganglia and gray-white matter junction in the cerebral hemispheres. Lymphoma in AIDS, in distinction, is often a single lesion larger than 3 cm in diameter. Central necrosis, with irregular rim enhancement, is also not uncommon in lymphoma in the immunocompromised patient.

Progressive multifocal leukoencephalopathy is a viral demyelinating disease seen in immunocompromised patients, in particular AIDS. Disease progression is rapid; death occurs by 6 months in many cases. On MRI, focal areas of abnormal white matter are seen with high signal intensity on T2-weighted scans. Involvement is often asymmetric (comparing the two hemispheres) and distant from the ventricular system (Fig. 15). Lesions may be at first round or oval. These subsequently enlarge, becoming confluent. Mass effect is minimal or absent. There can be both cerebral and cerebellar (Fig. 17) involvement.
Figure 17. Progressive multifocal leukoencephalopathy (PML). Although less common, infratentorial white matter lesions also occur in PML. In the case illustrated, a large lesion confined to the white matter of the right cerebellar hemisphere and right middle cerebellar peduncle is seen with abnormal high signal intensity on the T2-weighted scan (A) and abnormal low signal intensity (without enhancement) on pre- (B) and postcontrast (C) T1-weighted scans. In a published study of 47 patients, 15 had posterior fossa lesions, and disease was limited to the posterior fossa in 2. In PML, as illustrated with this case, lesions typically lack mass effect and do not demonstrate contrast enhancement.

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INTRODUCTION

Magnetic resonance imaging (MRI) is the imaging modality of choice for the study of congenital brain disease. Here, T1-weighted scans play a dominant role. T1-weighted scans provide excellent information regarding structural lesions and disorders of white matter. For structural lesions, acquisition of images in multiple planes is important. T1-weighted and T2-weighted scans both chronicle the normal myelination process, making possible early diagnosis of the leukodystrophies. The role of T2-weighted scans is limited, as is the role of contrast enhancement. These only occasionally contribute information about structural defects and significant associated sequelae. Contrast enhancement is, however,
important in the evaluation of associated tumors, as may occur with the neurocutaneous syndromes.

NORMAL VARIANTS (VENTRICULAR SYSTEM)

- **Cavum Septum Pellucidum**

The septum pellucidum is a thin, translucent plate consisting of two laminae (leaves) lying in the midline between the frontal horns of the lateral ventricles. The septum pellucidum links the hippocampus to the hypothalamus. Abnormalities of the septum pellucidum may have subtle associated neuropsychiatric symptoms. The cavum septum pellucidum is a normal embryologic space. It is present in all fetuses and premature infants. By 3 months of age, it is seen in only 15%. Persistence into adulthood can occur and is considered a normal variant (Fig. 1). When the distance between the leaves is large (greater than 1 cm), obstruction can occur to cerebrospinal fluid (CSF) flow at the foramen of Monro.

![Figure 1. Cavum septum pellucidum and vergae. T2- (A) and T1-weighted (B) axial images demonstrate separation of the leaves of the septum pellucidum. Fluid, with cerebrospinal fluid (CSF) signal intensity, fills the intervening space. The central CSF space extends posteriorly to the splenium of the corpus callosum (with the posterior portion being the cavum septum vergae). The coronal T1-weighted image (C) is anterior to the columns of the fornix, thus depicting the cavum septum pellucidum component.](image)

- **Cavum Septum Vergae**

The cavum septum vergae is a normal embryologic cavity, like the cavum septum pellucidum. It is essentially a posterior extension of the cavum septum pellucidum. The cavum septum vergae is that part of the midline cavity posterior to the columns of the fornix. It ends at the splenium of the corpus callosum. The cavum septum vergae begins to disappear at 6 months gestational age. In the adult, it is considered a normal variant.
• **Cavum Velum Interpositum**

Cavum velum interpositi are much less common than cavum septum pellucidum or vergae. In this variant, there is separation of the crura of the fornix between the thalami and above the third ventricle. A cavum septum vergae lies superior to the internal cerebral veins. The latter lie within a cavum velum interpositum.

**CRANIOVERTEBRAL ANOMALIES**

• **Basilar Invagination (Impression)**

In this bony craniovertebral junction anomaly, the odontoid is high in position relative to the foramen magnum. A more specific definition is that the tip of the odontoid lies more than 5 mm above Chamberlain's line. The latter extends from the posterior edge of hard palate to the posterior lip of the foramen magnum. The dens can compress or displace the medulla (Fig. 2).

![Figure 2. Basilar invagination. T2- (A) and T1-weighted (B) sagittal images reveal the odontoid process to be abnormally high in position, lying within the foramen magnum. The odontoid tip, which lies 1 cm above Chamberlain's line, compresses and flattens the medulla.](image)

Basilar invagination can be the result of a primary bone anomaly. In this circumstance, it is often associated with assimilation of the posterior arch of C1 to the occiput. Basilar invagination can also be secondary to other diseases. These include osteoporosis, osteomalacia, Paget's disease, fibrous dysplasia, achondroplasia, and osteogenesis imperfecta. Platybasia may also be present. In platybasia, there is a flattened relationship between the anterior and middle cranial fossae, with the angle formed greater than 140 degrees. Patients with basilar invagination can present clinically with headache, neurologic deficits, and symptoms from vertebrobasilar artery compression.
CHIARI MALFORMATIONS

Chiari type I and II malformations are commonly encountered in clinical practice. The anatomic features of these conditions are well delineated by MRI. Because Chiari type I malformations may present with clinical symptoms suggesting demyelination or neoplastic disease, MRI is the usual mode of examination. Chiari type I malformations are also one of the more common congenital abnormalities encountered in asymptomatic patients. Requests for imaging are frequent with Chiari type II malformations because of the associated spinal anomalies and their sequelae. For the Chiari malformations, MRI is the easiest and most accurate method of diagnosis because of the anatomic detail visualized within the posterior fossa and upper cervical canal.

Table 1. Types of Chiari malformation

<table>
<thead>
<tr>
<th>Type</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chiari type I</td>
<td>Elongated peglike cerebellar tonsils that are displaced into the upper cervical canal through the foramen magnum</td>
</tr>
<tr>
<td>Chiari type II</td>
<td>Downward displacement of the medulla, fourth ventricle, and cerebellum into the cervical spinal canal, as well as elongation of the pons and fourth ventricle, probably due to a relatively small posterior fossa.</td>
</tr>
<tr>
<td>Chiari type III</td>
<td>Cervical spina bifida associated with herniation of the cerebellum through the foramen magnum (Encephalocele)</td>
</tr>
<tr>
<td>Chiari type IV</td>
<td>Severe cerebellar hypoplasia without displacement of brain through the foramen magnum,</td>
</tr>
</tbody>
</table>

- **Chiari Type I Malformation**

The primary feature of Chiari type I malformation is the abnormal cerebellar tonsil position and morphology. There is downward and posterior herniation of the cervicomedullary junction with a variable amount of tissue below the foramen magnum (Fig. 3). The tonsils are wedge shaped. The cisterna magna is small or absent. Two thirds of cases with Chiari type I have downward displacement of the tonsils inferior to C1. In one fourth, the herniation reaches the C3 level. Associated anomalies include hydrosyringomyelia (typically cervical), with variable extent, and hydrocephalus. The primary features of Chiari type I that separate it from Chiari type II are the normal position of the fourth ventricle, absence of supratentorial structural anomalies, and lack of an associated myelomeningocele.
Color plate 1. *Arnold Chiari malformation associated with hydrocephalus*

Chiari type I may also be associated with bony anomalies involving the skull base and cervical spine. These anomalies include basilar impression, fusion of C1 to the occiput, fusion of C2 and C3, Klippel-Feil deformity, and spine bifida occulta.
Figure 3. Chiari type I malformation with associated cervical syrinx. T2- (A) and T1-weighted (B) sagittal images reveal the tonsils to be wedge shaped in contour and displaced 5 mm below the foramen magnum. Within the spinal cord, beginning at the level of C2 and extending inferiorly, there is a cavity with the signal intensity characteristics of cerebrospinal fluid. C, The axial T1-weighted image demonstrates the syrinx to be central in position within the cord.
Color plate 2. **Two cases with cerebellar tonsillar herniation**

- **Chiari Type II Malformation**

Chiari type II refers to a cerebral dysgenesis associated with a neural tube closure abnormality, specifically a myelomeningocele. There is a myelomeningocele in nearly 100% of cases. Myelomeningoceles are the extreme form of spinal dysraphism. There is a midline defect of the posterior bony elements of the vertebral body, usually in the lumbosacral region. Although the muscle, fascia, and skin are split along the midline, the meninges typically remain intact. The incidence of this sporadically appearing syndrome is 0.3%. Chiari type III refers to a very rare dysgenesis, with Chiari type II features and a low occipital or high cervical encephalocele.

Common infratentorial structural changes in Chiari type II include the following (Fig. 4). There may be downward displacement of the cervical spinal cord. The brainstem may be elongated, with the medulla inferiorly displaced into the cervical canal. In extreme cases, the displaced medulla may fold over on itself behind the cervical spinal cord, forming a kink. The fourth ventricle is typically inferiorly displaced and may be at or below the foramen magnum. The cerebellum may protrude through the foramen magnum to create a cerebellar peg with compression of the inferior vermis. There may be forward displacement of the cerebellar hemispheres, enveloping the brainstem. These may touch
anteriorly, in front of the pons, in rare cases. In many cases, the folia of the superior cerebellum have an abnormal configuration, with an exaggerated craniocaudal orientation. The tentorial incisura is wide, creating the visual effect of a towering cerebellum with enlarged supracerebellar CSF spaces. The clivus and petrous ridges may have an altered contour.

Color plate 3. Intraoperative Chiari malformation
Figure 4. Chiari type II malformation. A, The midline sagittal T1-weighted image demonstrates elongation and inferior displacement of the fourth ventricle, a beak-shaped tectum, and a large massa intermedia. The corpus callosum is thin anteriorly and absent posteriorly. At the level of the fourth ventricle on the axial T1-weighted scan (B), the cerebellar hemispheres are displaced anteriorly, partially surrounding the pons. C, The coronal T1-weighted scan demonstrates abnormally wide margins of the tentorial incisura, a towering cerebellum, abnormal orientation of the cerebellar folia, an enlarged interhemispheric fissure, and interdigititation of gyri (with absence of the falx).

There are multiple common midbrain and supratentorial structural changes in Chiari type II. The colliculi are typically fused, creating a beaked or bulbous tectum. The massa intermedia may be large. Hydrocephalus is common, with inferiorly pointing frontal horns, large atria, and a prominent suprapineal recess of the third ventricle. Interdigitating gyri are common, accompanying fenestration or partial absence of the falx. The gyri are often thin and numerous, an appearance termed stenogyria. This is not to be confused with polymicrogyria, a finding not seen in Chiari type II and one in which the gross appearance is that of a smooth brain. There is agenesis of the corpus callosum in about one third of all cases. Also common is an abnormal interhemispheric CSF space, of variable size and configuration. Hydrosyringomyelia, which may be cervical or lumbar in location, is seen in about half of all cases.

OTHER ANOMALIES OF THE POSTERIOR FOSSA

- Dandy-Walker Malformation

The Dandy-Walker malformation is characterized by absence of the inferior vermis (Fig. 5). The fourth ventricle is large and communicates freely with a large cyst-like structure, posterior in location. The posterior fossa is usually expanded, with elevation of the torcular. CSF flow dynamics may be abnormal because of obstruction at the foramen of Magendie or Luschka. However, not all cases have obstruction at autopsy, and in vivo demonstration of foraminal patency is difficult.

These structural anomalies are thought to be due to an embryologic dysgenesis and not to a permanent obstructive process. Hydrocephalus is usually present and is highly variable in severity. The severity of hydrocephalus is the most important prognostic factor. Other cerebral anomalies associated with Dandy-Walker malformation are agenesis of the corpus callosum, cortical heterotopias, polymicrogyria, and brainstem lipomas.
Figure 5. Dandy-Walker malformation. On the midline sagittal T1-weighted scan (A), the posterior fossa is noted to be enlarged, containing principally cerebrospinal fluid. The inferior cerebellar vermis is absent and the torcular herophili elevated. There is scalloping of the inner table of the occipital bone. Communication between the fluid posteriorly and the fourth ventricle is confirmed on the axial T2-weighted scan (B).

In a few patients, the CSF collection is smaller, without posterior fossa expansion. In this Dandy-Walker variant, the torcular is in normal position. The foramen of Magendie is patent, and there are normal CSF dynamics.

- **Arachnoid Cyst**

With a retrocerebellar arachnoid cyst, the inferior vermis is intact and the CSF space anterior to the brainstem small because of mass effect. These features differentiate a retrocerebellar cyst from the Dandy-Walker malformation. However, as with the latter, the torcular may be elevated. The presence of mass effect is used to differentiate a retrocerebellar arachnoid cyst from a prominent cisterna magna.

Other characteristic locations for arachnoid cysts include the middle cranial fossa (the most common), brain convexity (Fig. 6), and perimesencephalic cistern. Hypogenesis of the temporal lobe is a common finding in middle cranial fossa arachnoid cysts. Arachnoid cysts are benign CSF-filled lesions. They should be CSF signal intensity on all pulse sequences and lack contrast enhancement. Most arachnoid cysts are congenital in origin. An arachnoid cyst may also form after head trauma, leptomeningitis, and subarachnoid hemorrhage. Although frequently asymptomatic, arachnoid cysts can be symptomatic as a result of mass effect.
Figure 6. Arachnoid cyst. A large mass with cerebrospinal fluid (CSF) signal intensity is noted over the right brain convexity on T2- (A) and T1-weighted (B) images. There was no abnormal contrast enhancement (not shown). Long-standing mass effect is evident, with scalloping of the adjacent calvarium.

- **Cerebellar Hypoplasia**

Cerebellar hypoplasia is characterized by absence of cerebellar or vermian tissue. In its place is a passive CSF space. Usually only the most anterior portions of the cerebellar hemispheres are present. The cerebellar hemispheric remnants may be asymmetric. The cerebellar peduncles and brainstem are hypoplastic. The posterior fossa is small, with a low torcular.

Other associated findings. The most common anomalies associated with agenesis are Chiari type II malformation, Dandy-Walker malformation, holoprosencephaly, neuronal migration abnormalities, encephaloceles, and interhemispheric cysts.

**DISORDERS OF CEREBRAL HEMISPHERIC ORGANIZATION**

- **Agenesis of the Corpus Callosum**

Agenesis of the corpus callosum is a relatively common congenital anomaly. Agenesis may be partial or complete. The hippocampal and anterior commissures may also be absent. The posterior commissure is typically intact. The corpus callosum is composed of four parts. Progressing anteriorly to posteriorly, the rostrum, genu, body, and splenium can be identified. These structures form embryologically from anterior to posterior. This temporal sequence of formation explains the consistent absence of the more posterior elements in partial agenesis.
Multiple features are associated with agenesis of the corpus callosum (Fig. 7). The third ventricle is large and high in location. In the coronal plane, the frontal horns are concave medially. White matter bundles (of Probst) run along the medial wall of the lateral ventricle. The lateral ventricles are widely separated and parallel. The gyri radiate in a medial direction because of the absent cingulate gyrus. The anterior commissure, if present, may be enlarged and dysplastic. The ventricular atria may be rounded because of the absence of the splenium and portions of the forceps major. There may be a wandering anterior cerebral artery. Heterotopic gray matter may be present.

Agenesis of the corpus callosum is easy to identify on MRI scans after the age of 2 years. In neonates, however, myelination is not complete, and agenesis is difficult to diagnose. At this age, the normal corpus callosum is very thin and difficult to visualize. However, if the sulci on the medial surface of the brain radiate directly from the lateral ventricle, the diagnosis may be suggested even in the neonate. Eighty percent of cases of agenesis have
Figure 7. Agenesis of the corpus callosum. A, The midline sagittal T1-weighted image shows a small, dysplastic genu. The remainder of the corpus callosum is absent. Adjacent to the genu, the cingulate gyrus has a normal orientation. Posteriorly, where the corpus callosum is absent, the cerebral gyri have an abnormal radiating appearance. B, The axial T1-weighted image reveals the lateral ventricles to be widely separated and oriented parallel to each other. On the T1-weighted coronal image (C), the lateral ventricles have an abnormal crescent-like appearance (with indentation medially).

- Lipoma

The incidence of intracranial lipomas is 0.1%. The most common location is midline within the interhemispheric fissure near the corpus callosum (Fig. 8) Other common sites include the quadrigeminal plate cistern, tuber cinereum, and cerebellopontine angle. Less common sites include the base of the cerebrum, the cerebellum, the brainstem, cranial nerve roots, ventral aspect of the midbrain, and choroid plexus of the lateral ventricles. Intracranial lipomas arise from the pia and envelop adjacent neural structures. MRI will demonstrate a fat intensity mass. If associated with agenesis of the corpus callosum, the lesion is most often situated in the midline where the genu of the absent callosum would lie. Approximately 50% of interhemispheric lipomas have associated hypoplasia of the corpus callosum. Calcifications may be present but are not readily identified on MRI.

Figure 8. Interhemispheric lipoma. Axial (A) and coronal (B) T1-weighted images reveal a midline high-signal-intensity mass immediately superior to the corpus callosum. The lesion was isointense with fat on all pulse sequences, and there was no abnormal contrast enhancement.
• Holoprosencephaly

In utero failure of hemispheric or thalamic separation leads to holoprosencephaly. Half of all cases have chromosomal abnormalities. The incidence is 0.01%. Holoprosencephaly is classically divided into three types by grade of severity. From most to least severe, these are alobar, semilobar, and lobar holoprosencephaly.

Absence of the falx and interhemispheric fissure, fused thalami (with absence of the third ventricle), and a horseshoe-shaped monoventricle characterize the alobar form. The superior sagittal, inferior sagittal, and straight sinuses are absent along with the internal cerebral veins. The roof of the third ventricle may balloon out posteriorly, giving the appearance of a large dorsal "cyst."

Semilobar holoprosencephaly is characterized by partial formation of the interhemispheric fissure and falx (Fig. 9). There is rudimentary differentiation of the occipital and temporal lobes and respective ventricular horns. A rudimentary corpus callosum is present. There is cleavage of the thalami to form a third ventricle.
Figure 9. Semilobar holoprosencephaly. A 9-month-old infant presented with microcephaly and developmental delay. Axial T2-weighted images demonstrate hypotelorism (A), rudimentary formation of the temporal horns (B), a small third ventricle with fusion of more anterior structures (C), and preservation of the corpus callosum posteriorly (D) (arrow). The falx is absent anteriorly. There are no identifiable frontal horns. Incidentally noted is a large retrocerebellar cyst.

A nearly complete interhemispheric fissure and falx characterize lobar holoprosencephaly. Only a small area of the frontal lobe is fused. There are well-formed occipital and temporal lobes. The thalami, third ventricle, and corpus callosum appear normal or nearly normal.
Septo-Optic Dysplasia

In septo-optic dysplasia, the septum pellucidum is abnormal and there is hypoplasia of the optic nerves (Fig. 10). The abnormality of the septum varies from mild dysplasia to complete absence. Half of all patients with septo-optic dysplasia also have schizencephaly. Septooptic dysplasia is not a single homogeneous entity. Clinical symptoms include blindness, seizures, hypothalamic-pituitary dysfunction, developmental delay, and growth retardation.

Figure 10. Septo-optic dysplasia. On sagittal (A) and axial (B) T1-weighted images, the optic chiasm and optic nerves are noted to be hypoplastic. Axial (C) and coronal (D) T1-weighted images reveal the leaves of the septum pellucidum to be incompletely formed and widely separated. Only the anterior portion of each leaf is present.
NEURAXONAL MIGRATION ABNORMALITIES

The neuronal migration anomalies are a varied group of entities caused by abnormal migration of the embryologic neuroblasts, which are arrested along their normal course from the ventricular germinal matrix to the cortical periphery. The primitive neuroblasts normally ascend along radial glial cells during the third and fifth gestational months. If this cellular migration is prevented, abnormal cortical structures result. The entities included in this group are agyria and pachygyria, polymicrogyria, schizencephaly, gray matter heterotopia, and unilateral megalencephaly.

- **Agyria and Pachygyria**

Agyria and pachygyria represent a spectrum of cortical malformations, with agyria being the most severe form.
Figure 11. Pachygyria. A-C, Axial T1-weighted images demonstrate a decreased number of cortical gyri that are also too broad. The gyri are abnormal bilaterally, and the gray matter is too thick. D, The coronal T1-weighted image demonstrates abnormal broad gyri in the parietal and superior temporal lobes. However, the inferior gyri of the temporal lobes are normal. Pachygyria may be focal and unilateral but is most commonly a diffuse, bilateral abnormality with relative sparing of the temporal lobes.

Lissencephalia is a term often used for agyria when gyral formation is very rudimentary, creating the appearance of a "smooth brain." Agyria and pachygyria describe regions of cortical brain that have diminished gyral formation, creating thick, broad gyri (Fig. 11). The ratio of the gray matter to white matter is reversed. Thus, the gray matter cortex is broad. The corpus callosum is thin. The brainstem is small because of failure of
corticospinal tract formation. On T2-weighted images, there is a band of increased signal intensity in the peripheral cortex. It is theorized that normal migration is impaired here at the cell-sparse layer.

- Polymicrogyria

On imaging in polymicrogyria, the cortex is thick, gyri are not detectable, and the underlying white matter is decreased in quantity (Fig. 12). The number of cerebral convolutions is much greater than normal; however, this feature can only be identified histologically. Polymicrogyria is also characterized by an abnormal cellular histology, with the cortex composed of four layers instead of the normal six. Polymicrogyria is to be differentiated from stenogyria. In the latter, the gyri are thin and too numerous but visible on imaging. The cortex in stenogyria has a normal number of cellular layers.

Color plate 5. A, Polymicrogyria. B, pachygyria with polymicrogyria, notice the subependymal nodular heterotopia
Polymicrogyria. On axial T1-weighted images (A-C), the gyri in the distribution of the left middle cerebral artery are noted to be abnormally broad and diminished in number. The cortical gray matter is also too thick. The left lateral ventricle is mildly enlarged and the quantity of white matter on the left diminished. Anomalous venous drainage of the abnormal cortex is also often seen in polymicrogyria. This can be noted in the present case by comparing A and D, the latter a scan with intermediate T2-weighting, which together show a network of vessels feeding a large abnormal vein within a deep sulcus.

- **Schizencephaly**

Schizencephaly is a disorder of cell migration within a segment of the brain, creating a cleft lined by gray matter traversing the hemisphere from the cortex to the ventricles (Fig. 13). The cleft may be unilateral or bilateral. Bilateral clefts are associated with severe clinical impairment. Covering the cleft is a "pial-ependymal" seam, representing fusion of the pial lining of the brain and the ventricular ependyma. The clefts are situated at the precentral
or postcentral gyri. Abnormal gyral patterns surround the clefts, including stenogryria and gray matter heterotopia. The clefts may be gaping or "open" or the clefts may be "closed," with only a gray matter seam extending from the peripheral cortex to the ventricular level. The identification of gray matter lining the cleft permits differentiation from porencephaly and other acquired destructive lesions. This differentiation is important because siblings of patients with schizencephaly have an increased incidence of brain anomalies.

Figure 13. Schizencephaly. Bilateral cerebrospinal fluid-filled clefts are noted on sagittal (A), axial (B), and coronal T1-weighted images (C). The clefts are lined by gray matter and traverse the brain from the cortex to the ventricular system. Schizencephaly is associated with absence of the septum pellucidum, also noted in this case. Clinical symptoms in this 9-month-old infant included decreased movement of the left arm and leg and a generalized increase in limb tone; the latter suggests bilateral brain involvement.
Color plate 6. Open-lip schizencephaly with cortical dysplasia

- **Heterotopic Gray Matter**

Heterotopic gray matter represents collections of neurons of varying size in aberrant locations (Fig. 14). These collections may be found anywhere between the ependyma and the cortex. Heterotopic gray matter may be an isolated asymptomatic lesion or may be associated with other anomalies, such as Chiari type II or neuronal migration anomalies. Sequences that provide high gray-white matter contrast, such as heavily T1-weighted inversion recovery scans, best delineate these lesions.
Figure 14. Heterotopic gray matter. Abnormal soft tissue, isointense with gray matter on all pulse sequences, is seen adjacent to the posterior right lateral ventricle on proton density (A), T2-weighted (B), and T1-weighted (C) images. The lesions project into the ventricle as small nodules. There is no abnormal contrast enhancement (C). Most patients

Color plate 7. Subependymal nodular heterotopia
present clinically with seizures, as this one did. Late-onset, mild symptoms are characteristic for isolated anomalies.

- **Unilateral Megalencephaly**

Unilateral megalencephaly is a rare anomaly of the brain characterized by overgrowth of part or all of a cerebral hemisphere, with distorted, thickened cortex and ipsilateral ventricular dilatation. Abnormal signal intensity within the centrum semiovale represents areas of decreased myelination. Because of the intractable seizures associated with this disorder, all areas of abnormal tissue should be delineated to permit surgical resection if possible.

**NEUROCUTANEOUS SYNDROMES (PHAKOMATOSES)**

The neurocutaneous syndromes refer to a group of disorders that are dysplasias of tissues primarily derived from the embryonic ectoderm. These congenital disorders may also affect the embryonic mesoderm and endoderm. The more common syndromes in this group are neurofibromatosis (von Recklinghausen's disease), tuberous sclerosis, von Hippel-Lindau disease, and Sturge-Weber syndrome.

- **Neurofibromatosis**

Neurofibromatosis (NF) is an autosomal-dominant disorder of neuroectodermal and mesodermal tissues in which the Schwann cell is the primary abnormal element. The incidence of NF is approximately 1 in 3000 births. Two main subtypes exist. NF1 is the classic von Recklinghausen's neurofibromatosis with multiple central nervous system (CNS), cutaneous, and osseous lesions. Most patients with NF1 have high signal intensity lesions in the brain on T2-weighted imaging. These
Figure 15. Neurofibromatosis 1. There is symmetric abnormal hyperintensity in the globus pallidus and posterior limb of the internal capsule on both T2- (A) and T1-weighted (B) axial images. There is no mass effect. This patient also had abnormal hyperintensity in the cerebellar white matter bilaterally (not shown).

Abnormalities are most often seen in the basal ganglia (specifically the globus pallidus), brainstem, and cerebellar white matter. The pathologic basis and clinical consequence of such abnormalities are unknown, although these lesions most likely represent hamartomas or heterotopias. Abnormal hyperintense foci on T1-weighted scans involving the globus pallidus and internal capsule bilaterally (usually symmetrically) with extension across the anterior commissure have also been described. The same lesions appear smaller and less prominent on T2-weighted scans. There is typically no associated mass effect or abnormal contrast enhancement (Fig. 15). Optic nerve gliomas are the most frequent intracranial tumor associated with NF1. Less common, but seen in 10% to 15% of patients, is a primary glioma. NF2, which is much less common than NF1, is characterized by bilateral acoustic neuromas (Fig. 16). Cranial nerve tumors, cranial and spinal meningiomas, paraspinal neurofibromas, and spinal cord ependymomas are often seen in NF2.
Figure 16. Neurofibromatosis 2. Bilateral acoustic neuromas, with prominent contrast enhancement, are identified on T2- (A), precontrast T1-, (B), and postcontrast T1-weighted (C) images. Two additional enhancing lesions are seen postcontrast, both adjacent to the dura, compatible with meningiomas. Dense calcification is the cause of the central hypointensity of several lesions.

- **Tuberous Sclerosis**

Tuberous sclerosis is an autosomal-dominant disorder with hamartomatous lesions of multiple organs. Seizures, mental retardation, and facial adenoma sebaceum define the classic clinical triad. On MRI, the combination of parenchymal lesions and subependymal nodules is pathognomonic. Rarely, a subependymal nodule may form a giant cell astrocytoma. These usually arise at the foramen of Monro and can be identified as an enlarging, enhancing mass. The subependymal nodules lie along the ventricular wall and have decreased signal intensity on T2-weighted scans. The parenchymal lesions involve both gray and white matter and have increased signal intensity on T2-weighted scans (Fig. 17). In some instances, involvement is limited to the subcortical white matter of an expanded gyrus, a "gyral core." Involvement of two adjacent gyri in this fashion may spare the intervening cortex lining the sulcus, forming a "sulcal island." The classic renal lesion is an angiomyolipoma, diagnosed by identification of fat within a renal mass.
Tuberous sclerosis. A 19-month-old infant presented with seizures and mental retardation. Multiple high-signal-intensity lesions are seen on the T2-weighted images (A and B). These involve both gray and white matter. In several instances, the abnormality appears confined to the subcortical white matter core of an expanded gyrus (a "gyral core" lesion). Involvement of two adjacent gyri in this fashion, with sparing of normal intervening cortex lining a sulcus (a "sulcal island"), can also be seen. The parenchymal lesions are of low signal intensity, but in general less well-seen, on T1-weighted images (C and D). There are multiple subependymal nodules, best seen on the T1-weighted scan (C).
• **Von Hippel-Lindau Disease**

Von Hippel-Lindau disease or retinocerebral angiomatosis is an autosomal-dominant disorder of the vascular elements within multiple organ systems. Hemangiomas and hemangioblastomas are found in the CNS, primarily in the cerebellum. The most common presentation is that of a cystic lesion with a highly vascularized mural tumor nodule. Noncystic, solid lesions do occur but are rare.

On angiography, characteristic findings include a densely staining nodule or an abnormal tangle of vessels. Both may have enlarged feeding arteries and draining veins. Malignant tumors may also involve the retina, kidney, adrenal gland, and pancreas. The retinal lesions are hemangioblastomas, but the malignancies involving the kidney and pancreas are carcinomas. The rare associated adrenal tumors are pheochromocytomas.

• **Sturge-Weber Syndrome**

Sturge-Weber syndrome or encephalotrigeminal angiomatosis is a sporadic disorder characterized by a facial cutaneous vascular nevus within the first and second divisions of the trigeminal nerve and an ipsilateral leptomeningeal angiomatosis involving the parietal and occipital lobes. The cutaneous lesion is a capillary angioma. The leptomeningeal lesion contains thin-walled venous structures confined to the pia mater. Diagnosis is readily made on the basis of focal atrophy and prominent leptomeningeal contrast enhancement (Fig. 18). Patchy, parenchymal increased signal intensity is also seen on T2-weighted scans. The T2 changes correspond to gliosis and demyelination, presumably caused by ischemic damage from the overlying angiomatous lesion. The gyriform calcifications seen on computed tomography and plain x-ray film are often not well identified on MRI.
Figure 18. Sturge-Weber syndrome. There is abnormal low signal intensity in a gyriform pattern in the posterior parietal lobe on precontrast T2- (A) and T1- weighted (B) scans. This is most compatible with dense calcification. Axial (C) and coronal (D) postcontrast T1-weighted scans reveal prominent leptomeningeal enhancement. Mild atrophy is also present in the involved region.

NORMAL MYELINATION

Of all radiologic modalities, MRI is the best for the assessment of myelination. MRI provides an excellent evaluation of the progression of normal myelination, delays in myelination, and changes caused by the dysmyelinating diseases. Changes of normal myelination follow a well-documented course on both T1-weighted and T2-weighted scans. In the newborn, the signal intensity relationship between gray and white matter is in general reversed compared with the adult because of the lack of myelination. This pattern is seen on both T1- and T2- weighted scans. On T1-weighted scans, peripheral gray matter is higher signal intensity than underlying white matter, the opposite of the adult pattern. These differences, and the changes that occur with age, are important to consider in clinical scan interpretation.
Myelination begins in the brainstem and progresses to the cerebellum and cerebral hemispheres. The order of myelination is central to peripheral, inferior to superior, and posterior to anterior. T1-weighted scans are particularly useful to assess myelination in the first 9 months of life. With normal myelination, white matter becomes higher signal intensity on T1-weighted scans as a result of increasing cholesterol and protein content. T2-weighted scans are more useful to assess myelination after 6 months of age. The time to repetition (TR) for a T2-weighted scan in the infant, however, needs to be longer than that typically used in adults. A TR of 4000 ms is sufficient. On T2-weighted scans, white matter becomes lower signal intensity as it myelinates. This change is due to the myelin becoming progressively hydrophobic, with lower water content, as it matures. Myelination on T1-weighted scans precedes that on T2-weighted scans as a result of the different components evaluated.

Figure 19. Normal myelination in a neonate. A portion of the posterior limb of the internal capsule is low signal intensity on the T2-weighted scan (A) and high signal intensity on the T1-weighted scan (B) consistent with normal myelination. Peripheral white matter (nonmyelinated) is high signal intensity on the T2-weighted scan and low signal intensity on the T1-weighted scan, the reverse of the normal adult pattern.

In the newborn, the dorsal pons, superior and inferior cerebellar peduncles, posterior limb of the internal capsule, and ventral lateral thalamus demonstrate partial myelination (Fig. 19). These structures will have increased signal intensity on T1-weighted scans and decreased signal intensity on T2-weighted scans. The corpus callosum is not yet myelinated and will be very thin.
Figure 20. Normal myelination at 6 months of age. Although the corpus callosum is thin, it is of increased signal intensity on the sagittal T1-weighted scan (A), indicating that it is myelinated. The posterior limb of the internal capsule is low signal intensity, consistent with normal myelination, on the axial T2-weighted scan (B). The genu and posterior limb of the internal capsule are high signal intensity on the axial T1-weighted scan (C). The perialtral and occipital white matter are also high signal intensity (consistent with normal myelination) on C, whereas the frontal white matter is close in signal intensity to that of gray matter. The perialtral and occipital white matter still have immature signal intensity on the T2-weighted scan. At a higher level with T1-weighting (D), it is evident that myelination has progressed further in the posterior portion of the centrum semiovale than the anterior.
Figure 21. Normal myelination at 1 year of age. On the T2-weighted scan (A), the deep white matter (internal capsule, corpus callosum) appears normally myelinated, with low signal intensity. Peripheral subcortical white matter is not yet mature (as judged by the T2-weighted scan), having signal intensity isointense to gray matter. The T1-weighted scan (B) looks much like that of an adult, with high signal intensity in both deep and peripheral white matter.

Figure 22. Normal myelination at 2 years of age. Deep and peripheral white matter have low signal intensity on T2-weighted scans (A-C) consistent with normal myelination. The signal intensity of white matter surrounding the ventricular trigones is not as low, reflecting terminal myelinization.

At 6 months of age, the cerebellum, posterior limb and genu of the internal capsule, occipital lobe, and posterior centrum semiovale are normally myelinated on T1-weighted
scans (Fig. 20). The corpus callosum is still thin but now partially myelinated (high signal intensity on T1-weighted scans). The genu myelinates slightly later than the splenium at 8 months of age as opposed to 6 months. On T2-weighted scans, only the posterior limb of the internal capsule demonstrates low signal intensity, indicative of myelination.

At 1 year of age, the adult pattern of myelination (for deep and peripheral white matter) is present on T1-weighted scans. Peripheral arborization continues up to 2 years of age, with visual thinning of the gray matter mantle. At 1 year of age on T2-weighted scans, the deep white matter (internal capsule, corpus callosum, and corona radiata) will appear mature, with low signal intensity (Fig. 21). However, the white matter of the frontal, temporal, parietal, and occipital lobes as well as the peripheral (subcortical) white matter will not appear mature. These structures will be isointense to gray matter on T2-weighted scans.

At 2 years of age, deep and superficial white matter of the frontal, temporal, parietal, and occipital lobes are low signal intensity, like the adult, on T2-weighted scans (Fig. 22). The signal intensity of these white matter structures may not, however, be as low as the internal capsule. This is achieved by 3 years of age. The deep white matter of the parietal lobes, surrounding the ventricular trigones, is the last region to completely myelinate. This process is referred to as terminal myelinization. Mild hyperintensity in this region on T2-weighted scans may persist up to 10 years of age. Histologically, however, myelination proceeds into late adolescence.

In addition to myelination patterns, other features of immaturity can be observed. The relative ventricular size and width of the extra-axial cerebral spaces may appear differently in the neonate and in the older child. The normal ventricle-to-brain ratio in the neonatal period, as measured at the frontal horns, should be approximately one third of the width of the brain. The extraaxial spaces are less variable. The normal width over the convexities should be 4 mm.

**DYSMYELINATING DISEASE**

Dysmyelination is defined as the improper laying down or subsequent breakdown of myelin. The dysmyelinating diseases are genetically determined and appear early in life. MRI is extremely sensitive to white matter disease of all types. Thus, it is the imaging modality of choice for the diagnosis and evaluation of the dysmyelinating diseases. Although not indicated in every patient, contrast enhancement provides definition of areas of active demyelination on the basis of focal blood-brain barrier disruption.

- **Adrenoleukodystrophy**

Patients with adrenoleukodystrophy present with adrenal insufficiency and progressive multifocal demyelination. Although there are many subtypes, childhood adrenoleukodystrophy is the most common. For this specific disease, the defective gene is located in the Xq28 region of X chromosome. There is impaired degradation of saturated very long chain fatty acids. The disease onset is from 5 to 14 years of age, with rapid neurologic deterioration. In most instances, at presentation, there will be involvement of
the splenium of the corpus callosum, the fornix, and the parieto-occipital white matter. These structures will have abnormal low signal intensity on T1-weighted scans and abnormal high signal intensity on T2-weighted scans (Fig. 23). On postcontrast scans, mild enhancement may be noted along the anterior margin (leading edge) of the involved white matter. If followed temporally, the disease can be seen to progress in anatomic involvement from posterior to anterior. Atypical patterns of white matter disease also occur, with frontal, cerebellar, and asymmetric involvement described.

Figure 23. Adrenoleukodystrophy. The patient is a 15-year-old with impaired vision, hearing loss, and intellectual decline. A and B, Sagittal T1-weighted scans reveal abnormal hypointensity in the splenium of the corpus callosum and parieto-occipital white matter. C and D, Axial T2-weighted scans demonstrate abnormal hyperintensity in the corresponding areas.

- **Canavan's Disease**

Canavan's disease presents clinically during the first 6 months of life. There is macrocephaly, which is helpful in differential diagnosis. The only other leukodystrophy with macrocephaly is Alexander's disease. Other clinical findings include hypotonia, developmental regression, and cortical blindness. Canavan's disease is autosomal recessive;
enzyme tests reveal a deficiency of aspartoacylase. Imaging findings include cortical atrophy, ventriculomegaly, and symmetric abnormal white matter (Fig. 24). These findings are not specific for Canavan's disease. Diffuse abnormal white matter, with high signal intensity on T2-weighted scans, is seen in most of the leukodystrophies.

Figure 24. Canavan's disease. The patient is a 4-year-old child who is blind and has macrocephaly, progressive weakness, and severe learning disabilities. Sagittal T1- (A), axial T2- (B), and axial T1-weighted (C) images reveal cortical atrophy and ventriculomegaly. Anteriorly, the central white matter may be of normal signal intensity. However, both posteriorly and peripherally, the signal intensity of white matter is abnormal for age (hyperintense on T2- and hypointense on T1-weighted scans relative to gray matter).

- **Leigh's Disease**

Leigh's disease presents in the first few years of life. Clinical findings include feeding difficulties, psychomotor retardation, and visual disturbances. Leigh's disease is autosomal recessive; tests reveal cerebral inhibition of adenosine triphosphate-thiamine pyrophosphate phosphoryl transferase. Imaging findings include abnormal high signal intensity on T2-weighted scans in the spinal cord, brainstem, basal ganglia (putamen), and optic pathways (Fig. 25).
Figure 25. Leigh's disease. On T2-weighted scans, there is abnormal hyperintensity in both the brainstem (A) and the optic radiations (B) (adjacent to the ventricular trigones). The corresponding T1-weighted scans (not shown) were normal.

- **Hurler's Disease**

Hurler's disease is the most common of the mucopolysaccharidoses. Hunter's syndrome, or type II, is the second most common. Both are lysosomal storage diseases; this group of congenital enzyme deficiencies includes two main types: the sphingolipidoses and the mucopolysaccharidoses. The sphingolipidoses include the gangliosidoses, including Tay-Sachs disease, as well as Krabbe's disease, Fabry's disease, Gaucher's disease, Niemann-Pick disease, and Farber's disease. The mucopolysaccharidoses include Hurler's disease, Hunter's syndrome, Sanfilippo's syndrome, Morquio's disease, Scheie's syndrome, and Maroteaux-Lamy syndrome. The mucopolysaccharidoses all display coarse facial features ("gargoylism") and have both skeletal and multiple organ involvement.

Patients with Hurler's disease present clinically with mental retardation, deafness, short stature, corneal clouding, and coarse facial features. Death is usually by the teenage years. Enzyme tests in Hurler's disease reveal a deficiency of alpha-L-iduronidase. Imaging findings include ventriculomegaly, cerebral atrophy, a J-shaped sella, cavitated white matter lesions, and diffuse white matter high signal intensity on T2-weighted scans (Fig. 26).
Figure 26. Hurler's disease. This 2-year-old child demonstrates diffuse abnormal white matter hyperintensity on the T2-weighted scan (A). Best demonstrated on the T1-weighted scan (B) are numerous small holes, principally in white matter, containing cerebrospinal fluid. Moderate ventriculomegaly is also noted.

Figure 27. GM1 gangliosidosis. This 11-month-old infant has diffuse abnormal white matter hyperintensity on the T2-weighted scan (A). The T1-weighted scan (B) is also grossly abnormal, with diffuse white matter hypointensity. The pattern of involvement is
nonspecific, other than suggesting an inherited metabolic storage disease. There is normal myelination, by signal intensity, of only the posterior limb of the internal capsule.

- **Other Inherited Metabolic Storage Diseases**

The inherited metabolic storage diseases share a common imaging appearance, particularly in end-stage disease. In most instances, there is cerebral atrophy and diffuse white matter abnormality (Fig. 27). The appearance of adrenoleukodystrophy and Leigh's disease can be distinct. Otherwise, however, MRI is not able to differentiate between the many types of dysmyelinating disease.
HUNTINGTON'S DISEASE

In Huntington's disease, there is premature death of certain neurons. Inheritance is autosomal dominant. Patients present clinically in the fourth to sixth decades with choreoathetosis and progressive dementia. MRI is substantially better than CT for demonstration of morphologic changes. Thin-section, coronal, heavily T1- or T2-weighted techniques are recommended. Findings include volume loss in the corpus striatum: the caudate nucleus, putamen, and globus pallidus (Fig. 1). Cortical atrophy is seen in long-standing disease.
Figure 1. Huntington’s disease. Coronal T2- (A) and T1-weighted (B) scans reveal substantial volume loss in the caudate nucleus bilaterally.

Color plate 1. A case of Huntington dementia showing atrophy of the caudate nucleus, with dilatation of the frontal horns.
Color plate 2. Huntington's Disease: A coronal section through a brain in a case of Huntington's disease reveals dilatation of the lateral ventricles due to degeneration and shrinkage of the caudate nucleus. Histologically, there is marked loss of neurons in both the caudate and the putamen.

CEREBELLAR DEGENERATIVE DISEASE

Cerebellar atrophy can be either primary or secondary in type. The most common cause is alcoholism. The pathogenesis is twofold, with alcohol having a direct toxic effect and thiamine deficiency also contributing. The clinical presentation includes ataxia, impaired heel- to-toe walking, truncal instability, and a broad-based staggering gait. Atrophy of cerebellar vermis and hemispheres is seen in up to 40% of chronic alcoholics. The atrophy is irreversible. Although much less common, phenytoin (diphenylhydantoin or Dilantin) can also cause global cerebellar atrophy.
Figure 2. Cerebellar degenerative disease (alcoholic). A, The midline sagittal T2-weighted scan demonstrates marked atrophy of the cerebellar vermis. The folia are small and the sulci enlarged. B, The coronal postcontrast T1-weighted scan demonstrates atrophy of the cerebellar hemispheres as well. The cerebellar atrophy is disproportionate relative to the cerebral atrophy, which is mild at most.

Primary forms of cerebellar degenerative disease are much less common. Olivopontocerebellar degeneration is one primary form. This disease is differentiated by olivary atrophy, which is not present in alcoholism. The clinical presentation is that of ataxia, first in the lower and then the upper extremities. MRI findings include atrophy of the pons, middle cerebellar peduncles, olives, and cerebellar hemispheres. There may also be accompanying gliosis.

Figure 3. Olivopontocerebellar degeneration. A, The axial T1-weighted scan at the level of the fourth ventricle demonstrates loss of the normal olivary bulge bilaterally (arrows) and atrophy of the middle cerebellar peduncles. Pontine and cerebellar atrophy is noted on additional axial (B) and sagittal (C) T1-weighted scans.
MOTOR NEURON DISEASE

This mixed group encompasses conditions that affect the first or second motor neuron, or both, such as amyotrophic lateral sclerosis (ALS), spastic paraparesis, spinal muscular atrophies, and bulbospinal muscular atrophy. When the second motor neuron is affected, as in spinal muscular atrophy, bulbospinal muscular atrophy, or bulbospinal neuronopathy, the atrophy is confined to the anterior horns and anterior spinal roots, owing to neuronal depletion and fibrillar astrocytosis. In ALS and spastic paraparesis affecting the first motor neuron, descending corticospinal long tracts undergo atrophy with subsequent reduction in volume of the spinal cord. When clinically severe and especially restricted to the first motor neuron, the precentral gyrus also may be involved. Dementia may be associated with MND and marked by additional atrophy of frontal and temporal lobes.
Color plate 4. Amyotrophic lateral sclerosis (ALS) is uncommon. It begins in middle age and proceeds to death in several years. There is loss of anterior horn cells, so that patients present with progressive weakness that proceeds to paralysis from neurogenic muscular atrophy. Because of the loss of anterior horn cells, the anterior (ventral) spinal motor nerve roots demonstrate atrophy, as seen here in comparison with a normal spinal cord.

- **Imaging in motor neuron disease and amyotrophic lateral sclerosis**
  - Brain or cervical spine MRI should be done to rule out dysmyelinating lesions (eg, in family history of Tay-Sachs disease) or to rule out cervical myelopathy.
  - The cervical spinal cord is often normal in appearance in ALS. Cord atrophy is generally a late manifestation of this disease. The most common finding noted in ALS is signal hyperintensity on T2-weighted images in the posterior limbs of the internal capsule and extending into the adjacent frontoparietal white matter. The phenomenon is caused by secondary Wallerian degenerative changes related to the neuronal abnormality in the anterior horn cells of the spinal cord. Low signal intensity in a gyral distribution in the posterior frontal and anterior parietal lobes-already described with AD-has also been observed with ALS.
Figure 4. The most common finding noted in ALS is signal hyperintensity on T2-weighted images in the posterior limbs of the internal capsule and extending into the adjacent frontoparietal white matter. The phenomenon is caused by secondary degenerative changes related to the neuronal abnormality in the anterior horn cells of the spinal cord. Notice moderate central atrophy.

Figure 5. A, Left corticospinal tract degeneration in a patient with ALS. Axial proton density–weighted FSE MR image demonstrates a single round hyperintense focus within the posterior limb of the internal capsule on the left. B, Bilateral corticospinal tract degeneration in a patient with ALS. Coronal T2-weighted FSE MR image demonstrates linear hyperintensity extending from the subcortical white matter of both cerebral hemispheres through the internal capsule to the cerebral peduncles.
PICK'S DISEASE

Pick disease (Friedel Pick) is a progressive dementia that is defined by clinical and pathologic criteria. Unlike Alzheimer's disease and other dementias that present with cognitive deficits localized to the posterior (parietal) cortex, Pick disease typically affects the frontal and/or temporal lobes. First described in 1892, Pick disease is now considered by some to be part of a "complex" of neurodegenerative disorders with similar or related histopathological and clinical features.

Color plate 5. The marked atrophy of Pick's disease, a senile dementia, produces "knife-like" thinning of the gyri in frontal lobes and temporal lobes.

Color plate 6. A, Pick's disease with the gross appearance of lobar atrophy is seen here involving the frontal lobe. Note the "knife like" gyri. B, Pick's disease is demonstrated grossly in this coronal section in which there is marked atrophy with ex vacuo ventricular dilation.

In Pick's dementia, MRI commonly shows central and cortical atrophy associated with fronto-temporal signal changes on the MRI T2 images
NORMAL PRESSURE HYDROCEPHALUS

- CT scan or MRI alone is not sufficient for diagnosis. Distinguishing features of NPH (which excludes hydrocephalus ex vacuo from the diagnosis) include the following:
  - Ventricular enlargement out of proportion to sulcal atrophy
  - Prominent periventricular hyperintensity consistent with transependymal flow of CSF
  - Prominent flow void in the aqueduct and third ventricle, the so-called “jet sign,” (presents as a dark aqueduct and third ventricle on a T2-weighted image where remainder of CSF is bright)
  - Thinning and elevation of corpus callosum on sagittal images
  - Rounding of frontal horns

Figure 6. A case with Picks dementia showing central and cortical atrophy associated with fronto-temporal signal changes on the MRI T2 images
FRIEDREICH ATAXIA

The major pathophysiological finding in FA is a "dying back phenomena" of axons, beginning in the periphery with ultimate loss of neurons and a secondary gliosis. The primary sites of these changes are the spinal cord and spinal roots. There is a loss of large myelinated axons in peripheral nerves, which increases with age and disease duration. Unmyelinated fibers in sensory roots and peripheral sensory nerves are spared.

The posterior columns, corticospinal, ventral, and lateral spinocerebellar tracts all show demyelination and depletion of large myelinated nerve fibers to differing extents. This is accompanied by a fibrous gliosis that does not replace the bulk of the lost fibers. Overall, the spinal cord becomes thin and the anteroposterior (AP) and transverse diameters of the thoracic cord are reduced. The dorsal spinal ganglia show shrinkage and eventual disappearance of neurons associated with proliferation of capsular cells. The posterior column degeneration accounts for the loss of position and vibration sense and the sensory ataxia. The loss of large neurons in the sensory ganglia causes extinction of tendon reflexes.

Large neurons of the dorsal root ganglia, especially lumbosacral, and nerve cells in Clarke's column are reduced in number. The posterior roots become thin. The dentate nuclei exhibit mild to moderate neuronal loss and the middle and superior cerebellar peduncles are reduced in size. There is patchy loss of Purkinje cells in the superior vermis of the cerebellum and of neurons in corresponding portions of the inferior olivary nuclei. There are mild degenerative changes in the pontine and medullary nuclei and optic tracts. The cerebellar ataxia is explained by loss of the lateral and ventral spinocerebellar tracts, involvement of Clarke's column, the dentate nucleus, superior vermis, and dentatorubral pathways.

The corticospinal tracts are relatively spared down to the level of the cervicomedullary junction. Beyond this point, the corticospinal tracts are severely degenerated, which becomes progressively more severe moving down the spinal cord. This explains the common finding of bilateral extensor plantar responses and weakness late in the disease. Loss of cells in the nuclei of cranial nerves VIII, X, and XII results in facial weakness, speech, and swallowing difficulty.
Myocardial muscle fibers also show degeneration and are replaced by macrophages and fibroblasts. Essentially, chronic interstitial myocarditis occurs with hypertrophy of cardiac muscle fibers; fibers become hypertrophied and lose their striations. This is followed by swelling and vacuolation and finally interstitial fibrosis. The nuclei appear hyperchromatic and occasionally vacuolated. The cytoplasm appears granular with frequent lipofuscin depositions. Kyphoscoliosis is likely, secondary to spinal muscular imbalance.

Figure 8. Friedreich Ataxia

- **Histologic Findings in Friedreich ataxia**

A cross-section through the lower cervical cord clearly shows loss of myelinated fibers of the dorsal columns and the corticospinal tracts (Weil stain). Milder involvement of spinocerebellar tracts is also present. The affected tracts show compact fibrillary gliosis (hematoxylin and eosin [H&E]) but no breakdown products or macrophages, reflecting the very slow rate of degeneration and death of fibers. The dorsal spinal ganglia show shrinkage and eventual disappearance of neurons associated with proliferation of capsular cells (H&E). The posterior roots are nearly devoid of large myelinated fibers. Within the thoracic spinal cord, degeneration and loss of cells of the Clarke column is apparent.

Figure 9. Friedreich Ataxia, Spinal cord
• Neuroimaging in Friedreich ataxia

In Friedreich ataxia MRI examination shows cervical cord atrophy, thinning with reduced anteroposterior diameter. A hyperintense line on the posterior portion of cord is commonly seen, which represents loss of myelinated fibers and gliosis. The thinned spinal cord is seen lying on the posterior wall of spinal canal with increased signal intensity in its posterior and lateral compartments.

Figure 10. MRI of the brain in a case with Friedreich ataxia showing normal findings

Figure 11. MRI T2 (A,B) and MRI T1 (C) in a case with Friedreich showing marked atrophy of the uppermost part of the cervical spinal cord
Figure 12. MRI T1 (A) and MRI T2 (B) in a case with Friedreich showing marked atrophy of the uppermost part of the cervical spinal cord.

Figure 13. MRI T2 images in a case with Friedreich showings cervical cord atrophy, thinning with reduced anteroposterior diameter. Notice the hyperintense line in posterior portion of cord. The thinned spinal cord is seen lying on the posterior wall of spinal canal with increased signal intensity in its posterior and lateral compartments. The anterior subarachnoid space is enlarged. The intramedullary signal changes reflect loss of myelinated fibers and gliosis.
The decreased anteroposterior diameter of the spinal cord at the upper cervical region confirms that atrophy of the upper cervical part of the spinal cord is a characteristic feature of Friedreich’s ataxia, as opposed to other forms of corticocerebellar and cerebellar-brainstem atrophy. This had been indicated on the basis of subjective evaluation in two previous studies.

No direct pathologic correlation of the intramedullary signal abnormalities is available. However, the sensitivity of MR imaging to degeneration of white matter tracts in the brain and spinal cord after stroke or in degenerative diseases of the CNS - that is manifested on the MRI T2 images as hyperintense lines- has been cited in several reports [1-5]. Because of the substantial similarities between the intramedullary signal abnormality pattern that is found in patients with Friedreich and the distribution of demyelination and gliosis of white matter tracts in the histopathologic pictures of the spinal cord in cases of Friedreich’s ataxia, we think it reasonable to assume that the MR appearance could reflect these pathologic findings. Obviously, the intramedullary signal abnormality pattern is not exclusive to Friedreich’s ataxia and can be observed in subacute combined degeneration, tabes dorsalis, wallerian degeneration, and AIDS myelopathy. In these conditions, however, associated clinical and laboratory findings usually allow the correct diagnosis. [2-5]

Detection of signal changes in the white matter tracts of the spinal cord of patients with Friedreich’s ataxia could be an index of severity or progression of the disease and in this respect it is more useful than cord atrophy. The association between the extent of intramedullary signal changes and the chronicity and severity of disease is well known by the author and was reported by others [2-5]. Although this analysis could be informative, it requires quantitation of the signal changes in the white matter tracts and evaluation of the thoracolumbar spine. Noteworthy is the fact that intramedullary signal changes are only in patients with Friedreich’s ataxia. No such findings were seen in any of the patients with corticocerebellar or cerebellar-brainstem atrophy in the author experience and by others [2-5]. Thus, it appears that evaluation of the cervical spinal cord for intramedullary signal changes might be useful for differential diagnosis in patients with progressive ataxia of uncertain clinical type.

In a broad sense, MR examination of the cervical spinal cord is more informative than examination of the brain in patients with Friedreich’s ataxia. Although spinal cord atrophy and intramedullary signal changes theoretically could be searched for in the thoracic spinal cord of patients with Friedreich’s’ ataxia, focusing on the cervical spinal cord is recommended because it usually allows concurrent evaluation of the brainstem and the cerebellum. This may help in the differential diagnosis with corticocerebellar and cerebellar-brainstem atrophies.

In conclusion, MR imaging of the cervical spinal cord can show thinning of the cord and intramedullary signal changes consistent with degeneration of white matter tracts in the lateral and posterior columns of patients with Friedreich’s ataxia. These MR findings might be helpful for differential diagnosis in patients with progressive ataxia of uncertain clinical type.
OLIVOPONTOCEREBELLAR ATROPHY

In 1900, Dejerine and Thomas first introduced the term olivopontocerebellar atrophy (OPCA). Since then, the classification of idiopathic acquired ataxias has evolved a great deal. The initial cases of Dejerine and Thomas involved 2 middle-aged patients with chronic progressive cerebellar degeneration and autopsy findings of gross atrophy of the pons, cerebellum, middle cerebellar peduncle, and inferior olives.

OPCA has not been proven to be a single entity. The nosology of these disorders has been extremely confusing, as the OPCAs overlap with spinocerebellar atrophies (SCAs) and multiple system atrophies (MSAs). Clinical distinction of these entities is based on the dominant feature, which may be cerebellar ataxia (observed in OPCA, SCA, and MSA), parkinsonism (observed in multiple system atrophy [MSA]), or autonomic failure (observed in MSA). The term OPCA has been retained to describe a form of progressive ataxia distinguished by pontine flattening and cerebellar atrophy on brain imaging studies and at autopsy. Thus defined, OPCA also may qualify as an SCA or as an MSA.

While MSAs are sporadic by definition, the genetic bases of the SCAs are increasingly well defined. Since OPCA may exist as a sporadic or inherited disease, categorizing sporadic OPCA as MSA and inherited OPCA as SCA may be appropriate. Differences between sporadic and inherited OPCA in microscopic pathology support this division.

When faced with an adult having progressive ataxia suggestive of OPCA, the role of the clinician includes (1) excluding readily treatable alternative diagnoses, (2) discussing the value of genetic testing with patients in whom such testing is informative, (3) managing symptoms, and (4) advising the patient and family regarding natural history and the need to plan for the future. No definitive therapy for OPCA exists.

- **Neuroimaging**

In patients with OPCA, MRI is the imaging study of choice, because CT scans do not provide adequate resolution of the pons and cerebellum. MRI scans typically show the following abnormalities:

- Pancerebellar and brainstem atrophy, with flattening of the pons
- Enlarged fourth ventricle and cerebellopontine angle
- Demyelination of transverse pontine fibers
- In the first year after the onset of cerebellar symptoms in patients with OPCA, MRI scan may be normal; therefore, serial MRI examinations are necessary for detecting infratentorial atrophy.
- Brain MRI also is useful in patients presenting with spinocerebellar syndromes in order to exclude diagnosis of multiple sclerosis, cerebrovascular disease, and malignancy.

MRI also permits the visualization of pontine atrophy, which distinguishes OPCA from other forms of SCA and MSA.
Figure 14. Olivopontocerebellar atrophy: Notice pancerebellar and brainstem atrophy, with flattening of the pons. Enlarged fourth ventricle and cerebellopontine angle. Olivopontocerebellar degeneration, (olivopontocerebellar atrophy, spinocerebellar degeneration type I) is an autosomal dominant inherited degenerative disorder of the central nervous system that predominantly involves neurons in the cerebellum, inferior olives in the brain stem, and tracts in the spinal cord. The condition results from CAG trinucleotide repeats within the ATX1 gene that encodes for the ataxin. Normal individuals contain 19-36 of the CAG repeats within the gene; affected persons have 40-81 CAG repeats. The disease is manifest by ataxia, an intention tremor, rigidity, loss of deep tendon reflexes, and a loss of vibration and pain sensation. Alpha synuclein is present in neuroglia and neurons of persons with olivopontocerebellar atrophy. The pons becomes markedly atrophic.

Figure 15. A patient with OPCA. The T2W spin-echo axial section shows atrophy of the pons and middle cerebellar peduncles with enlargement of the prepontine and pontocerebellar cisterns and moderate atrophy of the cerebellar hemispheres. The PDW image shows slight signal hyperintensity of the transverse pontine fibers (arrows), sparing the
pyramidal tracts. The midline sagittal T1W spin-echo image (far right) shows flattening of the pons and atrophy of the cerebellar vermis.

**MULTISYSTEM ATROPHY**

Multiple system atrophy is a rare neurological disorder characterized by a combination of parkinsonism, cerebellar and pyramidal signs, and autonomic dysfunction. The term "Multiple System Atrophy" is synonymous with striatonigral degeneration (SND) when Parkinsonism predominates, olivopontocerebellar atrophy (OPCA) when cerebellar signs predominate, and Shy-Drager syndrome when autonomic failure is dominant.

**Table 1. Clinical feature of MSA**

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<th>Name</th>
<th>Characteristics</th>
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<td>Striatonigral degeneration</td>
<td>Predominating Parkinson’s-like symptoms</td>
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<td>Shy-Drager syndrome</td>
<td>Characterized by Parkinsonism plus a more pronounced failure of the autonomic nervous system</td>
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<tr>
<td>Sporadic Olivopontocerebellar atrophy (OPCA)</td>
<td>Characterized by progressive ataxia (an inability to coordinate voluntary muscular movements) of the gait and arms and dysarthria (difficulty in articulating words)</td>
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The classical presentation of MSA are atypical parkinsonism with early autonomic dysfunction and cerebellar signs that usually manifests in middle age and progresses relentlessly with a mean survival of 6 to 9 years. Initial L-dopa response occurs in a third of patients, however 90% of them are unresponsive on long-term follow-up. Orofacial dystonia is a feature observed in more than half of all MSA patients and may occur spontaneously or more usually as a complication of L-dopa therapy. Disproportionate anterocollis is another characteristic feature seen in MSA. Early urinary incontinence and syncope are characteristic for MSA and contrast with the later autonomic involvement often seen in Parkinson disease (PD). Early erectile dysfunction is also common and urinary retention can rarely be an early symptom. There are two subtypes of MSA: parkinsonian (MSA-P) and cerebellar (MSA-C) subtypes. Neuropathologically, all subtypes of MSA are collectively characterized by the finding of a-synuclein glial cytoplasmic inclusions in the striatum and cerebellum (GCIs).

The clinical differential diagnoses for this patient would include the following: other atypical parkinsonian syndromes, adult-onset cerebellar ataxia that can be hereditary despite a negative family history (eg, Friedreich ataxia), spinocerebellar ataxia, Fragile X tremor ataxia syndrome (FXTA) syndrome, and autoimmune conditions in association with Anti-GAD in celiac disease, anti-Yo and anti-Hu in paraneoplastic syndromes. Toxic and metabolic conditions (eg, hypothyroidism, alcohol-related cerebellar degeneration) should also be considered, as some of these are potentially reversible.

The most common first sign of MSA is the appearance of an "akineti-rigid syndrome" (i.e. slowness of initiation of movement resembling Parkinson’s disease) found in 62% at first
Other common signs at onset include problems with balance (found in 22%), followed by genito-urinary problems (9%). For men, the first sign can be erectile dysfunction (unable to achieve or sustain an erection). Both men and women often experience problems with their bladders including urgency, frequency, incomplete bladder emptying or an inability to pass urine (retention). About 1 in 5 MSA patients will suffer a fall in their first year of disease.

As the disease progresses three groups of symptoms predominate. These are:

1-Parkinsonism (slow, stiff movement, writing becomes small and spidery). The parkinsonian subtype of MSA (MSA-P) or striatonigral degeneration

2-Cerebellar dysfunction (difficulty coordinating movement and balance). The Cerebellar subtype of MSA (MSA-C) or olivopontocerebellar atrophy.

3-Autonomic dysfunction. The autonomic subtype of MSA or Shy-Drager syndrome (impaired automatic body functions) including:

- Postural or orthostatic hypotension, resulting in dizziness or fainting upon standing up
- Urinary incontinence
- Impotence
- Constipation
- Dry mouth and skin
- Trouble regulating body temperature due to abnormal sweating abnormal breathing during sleep

**Neuroimaging**

Neuroimaging is not included in the consensus diagnostic criteria of MSA. Nevertheless, typical neurologic findings can assist in differentiating MSA from other causes of parkinsonism and cerebellar ataxia. The “hot-cross bun” sign observed in this case is characterized by cruciform signal hyperintensity on T2-weighted images in mid pons, which resembles a hot-cross bun, traditionally baked on the last Thursday before Easter. This finding is thought to correspond to the loss of pontine neurons and myelinated transverse cerebellar fibers with preservation of the corticospinal tracts. However, this sign is not specific to MSA and has been reported in other conditions such as spinocerebellar ataxia (SCA).

The more common typical radiological findings in MSA include atrophy of the cerebellum, most prominently in the vermis, middle cerebellar peduncles, pons, and lower brainstem. In addition to putaminal atrophy, a characteristic hypointense signal in T2 with hyperintense rim, corresponding to reactive gliosis and astrogliosis, can be observed in the external putamen, and is termed “slit-like void sign”. This combination of hypointense and hyperintense putaminal signal change is specific for MSA and its finding can be used to
differentiate MSA from PSP and PD. Hypointensity alone without hyperintense rim is a sensitive radiological feature but nonspecific for MSA.

Figure 16. A hot cross bun, or cross-bun, is a type of sweet spiced bun made with currants or raisins and leavened with yeast. It has a cross marked on the top which might be effected in one of a variety of ways including: pastry, flour and water mixture, rice paper, icing, or intersecting cuts.

Figure 17. (A) Axial T2-weighted MR imaging demonstrates cruciform hyperintense signal changes in mid pons, the so-called “hot-cross bun sign.” (B) Axial T2-weighted MR
imaging demonstrates hypointensity in association with hyperintense rim in the external putamen, which is termed “slit-like void sign.”

PROGRESSIVE SUPRANUCLEAR PALSY

Progressive supranuclear palsy is characterized by atypical parkinsonian features characterized by early postural instability and falls backward, a vertical supranuclear gaze palsy, axial rigidity, pseudobulbar palsy, frontal lobe signs, and a poor response to L-dopa. These are characteristic features of progressive supranuclear palsy (PSP), also known as Richardson disease. It has a prevalence of 5 per 100,000, but is commonly underdiagnosed. The clinical features are quite different from PD or other atypical parkinsonian syndromes such as MSA. However, there is a subgroup of progressive supranuclear palsy (PSP), patients, known as PSP-Parkinsonism (PSP-P), which presents with asymmetrical bradykinesia, jerky tremor, and an initial L-dopa response without vertical gaze palsy. Other unusual presenting features of progressive supranuclear palsy (PSP), include primary gait freezing, early frontotemporal dementia, and corticobasal syndrome.

- Neuroimaging

MR imaging of the brain can be normal in the early stages of disease. Nevertheless, certain MR imaging features can greatly assist in making the diagnosis especially in patients with PSP-P or an atypical presentation (Fig. 1). The first radiological clue for progressive supranuclear palsy (PSP), would be the presence of striking hyperextension of the neck on sagittal MR imaging. The characteristic MR imaging feature is selective atrophy of the midbrain in association with preservation of the pons. The resulting atrophy of the midbrain tegmentum gives a distinctive concavity with the appearance of the beak of a hummingbird or king penguin, and is termed the “hummingbird” or “penguin” sign. Quantitative measurements of midbrain atrophy have been shown to improve diagnostic accuracy of PSP. Midbrain diameter in PSP (13.4 mm) was shown to be significantly lower than that of PD (18.5 mm). Recent study indicated that the surface area of midbrain of progressive supranuclear palsy (PSP), (56 mm2) was significantly smaller than that of Parkinsonian subtype of MSA (MSA-P) (97.2 mm2), PD (103 mm2), and healthy controls (117 mm2). Some overlaps of the area measurements were observed in PSP and Parkinsonian subtype of MSA (MSA-P), but the ratio of the area of the midbrain to pons was significantly smaller in PSP when compared with Parkinsonian subtype of MSA (MSA-P).
Figure 18. (A) Sagittal T1-MR imaging demonstrates volume loss in the midbrain with relative preservation of the pons. The midbrain tegmentum has lost its normal convexity giving it the appearance of a hummingbird (or penguin), also known as the “hummingbird sign.” (B) T2-weighted axial MR imaging demonstrates “Mickey mouse” or “morning glory” sign with concavity of the lateral margin of midbrain tegmentum.

On axial views, the selective atrophy of the midbrain tegmentum with relative preservation of the tectum and cerebral peduncles produces the “Mickey mouse” sign (see Fig. 18). Sometimes, the concavity of the lateral margin of the midbrain tegmentum is referred to as the “morning glory” sign and has high specificity but rather low sensitivity for PSP.

Figure 19. Midsagittal T1-weighted MR images in a patient with PD (A), a patient with Parkinson variant of MSA (MSA-P) (B), and a patient with PSP (C). (A) There is no pontine or midbrain atrophy in the patient with PD. (B) Pontine atrophy (arrow) without
midbrain atrophy in the Parkinson variant of MSA (MSA-P) patient. (C) Midbrain atrophy without pontine atrophy (divided by the white line) in the PSP patient, forming the silhouette of the “penguin” or “hummingbird” sign, with the shapes of midbrain tegmentum (bird’s head; above the white line) and pons (bird’s body; below the white line) looking like the lateral view of a standing penguin (especially the king penguin) or hummingbird, with a small head and big body.

Other radiological findings of PSP include dilatation of the third ventricle, particularly the posterior portion, signal change in the periaqueductal gray matter indicative of gliosis, and atrophy of the superior cerebellar peduncle, which has a specificity of 94% and sensitivity of 74% and can aid the differentiation of PSP from MSA-P and PD. “Eye of the tiger” sign with hypointensity signal change in T2, a common finding in pantothenate kinase-associated neurodegeneration (PKAN), can occasionally be observed in PSP, indicating the presence of iron deposition in the putamen.

References

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NORMAL CERVICAL SPINE

There are seven cervical vertebral bodies and eight cervical nerves. C1 is called the atlas and is a bony ring. C2 is called the axis and features the dens anteriorly, which extends superiorly like a thumb. From C3 to C7, the size of the vertebral body progressively increases. There are bilateral superior projections (referred to as the uncinate processes) from C3 to C7, which indent the disk and vertebral body above (posterolaterally), forming the uncovertebral joints. The transverse foramen lies within the transverse processes of each cervical vertebral body and contains the vertebral artery. There is a slight increase in spinal cord size from C4 through C6. The neural foramina course anterolaterally at a 45-
degree angle with a slight inferior course, oblique to the sagittal and axial imaging planes. The epidural venous plexus is prominent in the cervical region; epidural fat is sparse (the opposite of the lumbar region). In regard to dermatomes, the hand is innervated by C6 (thumb), C7 (middle finger), and C8 (little finger).

On T1-weighted spin echo imaging, the vertebral body marrow, which is primarily fat, has high signal intensity. The cord and disks have intermediate signal intensity. On high signal-to-noise (SNR) and spatial resolution images, gray and white matter within the cord can be distinguished on the basis of signal intensity. In the cord, the gray matter is central and the white matter peripheral. Cerebrospinal fluid (CSF) is of low signal intensity. On sagittal images, the neural foramina are poorly visualized because of their oblique orientation. Advantages of T1-weighted spin echo imaging include the ability to acquire scans with high spatial resolution and SNR in a relatively short scan time. T1-weighted scans are used to visualize structural abnormalities, marrow infiltration, degenerative disease, and contrast enhancement (using a gadolinium chelate).

On T2-weighted spin echo imaging, CSF and hydrated disks are high signal intensity. The cord and soft tissues are intermediate signal intensity. Fat, including the vertebral body marrow, is intermediate to low signal intensity. Fast spin echo (FSE) techniques, using repeated 180-degree radiofrequency pulses, have for the most part replaced conventional T2-weighted spin echo techniques. The use of fast spin echo technique results in a much shorter scan time and less sensitivity to motion artifacts (especially CSF pulsation). T2-weighted scans are used to detect spinal cord abnormalities, including edema, gliosis, demyelination, and neoplasia, and to evaluate the thecal sac dimensions, looking for canal compromise.

Gradient echo imaging is still used in the cervical spine, particularly in the axial plane. Scans are acquired with a low flip angle, resulting in T2-weighting. On such scans, CSF and normal intervertebral disks are high signal intensity, the cord intermediate signal intensity, and the marrow low signal intensity (as a result of magnetic susceptibility effects). Gray and white matter within the cord are usually well differentiated on the basis of signal intensity. Myelographic-like sagittal and axial images can be acquired, with diagnostic utility for the detection of degenerative disease (disk herniation, canal compression, and foraminal stenosis) and the evaluation of intrinsic cord abnormalities in the axial plane (multiple sclerosis, tumors, edema, and hemorrhage). Canal and foraminal stenoses are typically exaggerated on gradient echo imaging as a result of magnetic susceptibility effects.

Three major arteries supply the spinal cord and lie along its surface. One is anterior (the anterior spinal artery, which supplies 70% of the cord) and two are posterior (the posterior spinal arteries, which together supply 30% of the cord). In the cervical region, several radicular arteries supply the anterior spinal artery. In the thoracolumbar region, the artery of Adamkiewicz, which arises from a lower intercostal or upper lumbar artery, supplies the anterior spinal artery.
Magnetic resonance imaging (MRI) has the capability of providing flexion and extension views in the cervical spine. These scans can be of substantial clinical value. Flexion and extension views are typically acquired with some sort of rapid imaging technique, of which today there is a plethora. A very simple scheme, which can be used for rapid image acquisition on most scanners, is to acquire the T1-weighted scan with a reduced number of phase-encoding steps and the T2-weighted scan with FSE technique. Depending on the available coils, the range of possible motion may be limited. Flexion and extension views have substantial use in the demonstration of spinal cord compression not visualized in the neutral position (e.g., with rheumatoid arthritis) and in the evaluation of potential instability. The latter can be an important diagnostic question both after trauma and in chronic inflammatory disease (particularly at the occipitoatlantal and atlantoaxial levels).

Intravenous administration of a gadolinium chelate (the most common type of contrast agent currently used in MRI) produces enhancement of the normal venous plexus on cervical spine exams. The external vertebral plexus consists of a network of veins along the anterior vertebral body, laminae, and spinous, transverse, and articular processes. The internal vertebral plexus consists of a network of veins lying within the epidural space both anteriorly and posteriorly. The internal plexus is more important in regard to the interpretation of MRI scans. The anterior part of this plexus is larger (than that posteriorly), with longitudinal veins lying on each side of the posterior longitudinal ligament. The anterior plexus tapers at the disk space level. Displacement and engorgement of the anterior plexus often accompany disk herniation. All of the plexus drain via intervertebral veins that accompany the spinal nerves within the foramina.

In regard to the reading of MRI scans of the cervical spine, there is a need for a consistent, thorough approach to scan interpretation. All structures, including the contents of the thecal sac, the bony vertebral column, and the surrounding soft tissues, should be consciously examined. The cerebellar tonsils, thyroid, facet joints (looking specifically for perched facets), and surrounding soft tissues (looking specifically for lymphadenopathy) deserve particular attention because disease is common and often overlooked in these areas.

SPINAL STENOSIS

- Congenital

Congenital stenosis in the cervical spine is caused by short pedicles. There may be an underlying primary disease, such as achondroplasia or Down syndrome. Cervical spinal stenosis causes myelopathic symptoms, which include extremity weakness, gait abnormalities, reflex changes, and muscular atrophy. Relative spinal stenosis is defined as a canal less than or equal to 13 mm in diameter (but greater than 10 mm). Patients with this degree of narrowing may be symptomatic. Absolute spinal stenosis is defined as a canal smaller than 10 mm in diameter. Patients with cervical spinal stenosis are predisposed to early, more severe degenerative changes and traumatic spinal cord injury.
Degenerative (Acquired)

Degenerative (also known as acquired) spinal stenosis is caused by advanced degenerative disk disease (Fig. 6-1). Advanced degenerative disk disease is also referred to by the term spondylosis. Factors contributing to narrowing of the spinal canal include decreased disk height with thickening and buckling of intraspinal ligaments, calcification of the posterior longitudinal ligament and ligamentum flavum, disk bulges and herniations, osteophytic spurs (anteriorly), and hypertrophy of facet joints (posteriorly) (Fig. 6-2). Symptom onset is usually in middle age or older patients. This is older than the population affected by disk herniation, although there is considerable overlap. Symptoms are typically myelopathic. These include progressive or intermittent numbness, weakness of the upper extremities, pain, abnormal reflexes, muscle wasting (specifically the interosseous muscles of hand), and a staggering gait. The dimensions of the canal are most accurately measured on axial images. The normal anteroposterior dimension in the cervical region is greater than 13 mm. Patients with a borderline size canal, 10 to 13 mm, may experience symptoms. An anteroposterior dimension of less than 10 mm is considered to be diagnostic of cervical stenosis. The most commonly affected levels are C4-5, C5-6, and C6-7. Multilevel involvement is also very common. On MRI, with mild disease, the ventral subarachnoid space is effaced. With severe disease, there may be cord flattening, impingement, and myelomalacia (edema, gliosis, and cystic changes within the cord).

- Neuroforaminal (Uncovertebral Joint) Spurring

The uncovertebral joints, also known as the joints of Luschka, lie along the posterolateral margins of the cervical vertebral bodies. These joints are formed by the uncinate process of the lower vertebral body extending superiorly to articulate with a depression in the inferior end plate of adjacent superior vertebral body. Uncovertebral joints are present from C3 to C7. Thus, degenerative disease of the uncovertebral joints can cause foraminal narrowing from C2-3 to C6-7. As part of the degenerative process, hypertrophic spurs may form around these joints, which then narrow the anteromedial part of the neural foramen. When combined with disk space narrowing, which causes decreased height of the neural foramen), uncovertebral joint spurs can cause nerve root compression (Fig. 6-3). This is a more common cause of radiculopathy in the cervical spine than disk herniation. Because of the anterolateral and slightly inferior course of the neural foramen, oblique images provide the best view of the foramina.

- Ossification of the Posterior Longitudinal Ligament

Ossification of the posterior longitudinal ligament is an uncommon cause of acquired spinal stenosis. More common causes include ligamentous and facet joint hypertrophy. Ossification of the posterior longitudinal ligament is more common in the oriental population. Patients are at risk for traumatic spinal cord injury. Multilevel involvement is typical. The ossified posterior longitudinal ligament will be very low signal intensity on both T1- and T2-weighted scans but may contain centrally intermediate to high-signal-intensity soft tissue (fat and marrow).
Figure 1. Spinal stenosis, degenerative (acquired) in origin. A, On the sagittal fast spin echo T2-weighted scan, there is encroachment anteriorly on the thecal sac by disk bulges and osteophytic spurs at the C4-5, C5-6, and C6-7 levels. In degenerative spinal stenosis of the cervical spine, these are the most commonly affected levels. Multilevel involvement, as in this case, is also common. The same findings are apparent on the sagittal gradient echo T2-weighted scan (B). The latter scan is easily identified by the low signal intensity of vertebral body marrow, which is due to magnetic susceptibility effects. The osteophytic spurs are well visualized on the sagittal T1-weighted scan (C), although the encroachment on the thecal sac is less evident. On axial imaging, the asymmetry of the canal compromise in this patient is clearly seen, together with the cord flattening and deformity. As with imaging in the sagittal plane, on axial imaging (D) the T2-weighted scan depicts the interface between soft tissue and cerebrospinal fluid better than the T1-weighted scan (E).
Figure 2. Degenerative stenosis of the cervical spinal canal, with both anterior and posterior compression. The patient is a 69-year-old woman with neck pain and intermittent numbness and weakness in both arms. A, The sagittal T2-weighted scan reveals canal compromise at the C2-3 through C6-7 disk space levels. Disk bulges and osteophytic spurs cause compression anteriorly on the thecal sac at the C3-4 level and below. At both C2-3 and C3-4, facet hypertrophy causes posterior compression. The subarachnoid space is obliterated at multiple levels, with accompanying cord deformity (flattening). There is mild reversal of the normal cervical lordosis in the lower cervical spine. B, The postcontrast T1-weighted image demonstrates thin curvilinear high signal intensity (enhancing epidural venous plexus) along the posterior margins of the vertebrae. With contrast enhancement, the true canal dimensions are better visualized. The failure to clearly visualize epidural soft tissue is one reason that precontrast T1-weighted scans are generally less useful than T2-weighted scans in imaging cervical degenerative disease (spondylosis). Disk space narrowing is identified at the C4-5 through the C6-7 levels on the T1-weighted scan. The disk bulges and spurs are also clearly seen, as on the T2-weighted scan.
Figure 3. Neuroforaminal narrowing caused by uncovertebral joint bony spurs. The patient is 54 years old and presents with neck and bilateral arm pain. A, The midline sagittal T2-weighted scan reveals disk bulges and osteophytic spurs at C3-4, C5-6, and C6-7 with effacement of the ventral subarachnoid space at each level. Because of their oblique orientation, the neural foramina are not well visualized in the cervical spine on sagittal images. Although the foramina would be best depicted on oblique scans, axial scans are used in most clinical practices for their assessment. B, The axial gradient echo T2-weighted scan in this case at the C6-7 level reveals narrowing of the right neural foramen as a result of hypertrophic changes and sclerosis (with accompanying bony spurring) of the right C6-7 uncovertebral joint.

CONGENITAL DISEASE

- Klippel-Feil Syndrome

In the Klippel-Feil syndrome, there is congenital fusion of two or more cervical vertebrae, most commonly C2-3 and C5-6. At the affected levels, the intervertebral disk is absent.
About half of all patients with Klippel-Feil syndrome demonstrate the classic triad, which consists of limited neck motion, a short neck, and a low posterior hairline. Common associated anomalies include deafness, congenital heart disease, Sprengel's deformity (elevation and rotation of the scapula), and urologic abnormalities. Other less frequently associated anomalies include syringomyelia and diastematomyelia. There are three types, defined on the basis of the extent and location of vertebral fusions. In type I, there is extensive cervical and thoracic fusion. In type II, the most common (Fig. 6-4), there are one or two cervical fusions; there may also be associated hemivertebrae and occipitoatlantal fusion. Type III is defined as type I or II with additional lower thoracic or lumbar fusions. Clinically, patients with Klippel-Feil syndrome are often asymptomatic from a neurologic point of view. They can, however, have cord or nerve root compression. Patients with Klippel-Feil syndrome are predisposed to spinal cord injury after minor trauma. Patients may have hypermobility (and thus instability) between the unfused segments.

Figure 4. Klippel-Feil syndrome. A 51-year-old presented with diffuse neck pain and otherwise no neurologic findings referable to the cervical spine. Midline sagittal T2- (A) and T1-weighted (B) scans reveal marked degenerative disease at the C4-5 level. More importantly, there is an abnormal shape to vertebral bodies C6, C7, and T1 (height greater than width) and decreased height to the C6-7 and C7-T1 disk spaces. The shape of these vertebral bodies, together with the absence of normal disk material, raises the question of
fusion. A syrinx is noted on the sagittal T2-weighted exam and confirmed on T1-weighted exam. Cervical spinal stenosis extends from C3 to C5 as a result of degenerative disease, best seen on the T2-weighted exam. The lateral cervical spine x-ray film (C) obtained in flexion confirms the fusion of C6-T1, forming a block vertebra. In Klippel-Feil syndrome, there is fusion of two or more cervical vertebral bodies, most commonly C2 and C3 or C5 and C6. With the advent of magnetic resonance imaging, associated cord abnormalities, including syringomyelia and diastematomyelia, have been reported. Patients with Klippel-Feil syndrome are predisposed to spinal cord injury.

- Abnormalities Involving the Cerebellar Tonsils
  - Ectopia

The position of the cerebellar tonsils is best evaluated on sagittal images. Mild inferior displacement (ectopia) can be seen in asymptomatic normal individuals. In the majority of normal individuals, the tonsils lie above the foramen magnum. The tonsils may, however, lie as far as 5 mm below the foramen magnum and still be normal. In individuals with tonsillar ectopia, the tonsils retain their normal globular configuration.

  - Chiari Type I

In the Chiari type I malformation, the cerebellar tonsils are low lying and pointed or wedge shaped (Fig. 6-5). Associated findings include syringomyelia (Fig. 6-6) and craniovertebral junction abnormalities (basilar impression, occipitalization of the atlas, and Klippel-Feil syndrome). The fourth ventricle will be in normal position, an important differentiating feature from the Chiari type II malformation. As with all congenital malformations of the brain, the Chiari type I malformation is best evaluated by MRI.

Figure 5. Chiari type I malformation. The patient is an 11-year-old with severe scoliosis. Fast spin echo T2-weighted (A) and spin echo T1-weighted (B) midline sagittal images reveal that the cerebellar tonsils are abnormally low in position (these extend 11 mm below the foramen magnum). The tonsils have also lost their usual globular configuration and are pointed (or wedge shaped) in appearance. Because of the patient's scoliosis, the lower cervical spine is seen in a parasagittal plane. The fourth ventricle is normal in shape and position, an important negative finding. C, The T1-weighted axial view at the level of the
The arch of C1 confirms the abnormally low position of the cerebellar tonsils, which are wedged posteriorly and laterally. These compress and deform the spinal cord (anteriorly) at the cervicomedullary junction.

Clinical findings are variable. Most patients are asymptomatic. When symptomatic, clinical findings include those related to brainstem compression (headache, cranial nerve deficits, nystagmus, and ataxia) or a cervical syrinx (extremity weakness, hyperreflexia, and central cord syndrome). In rare cases, a syrinx can extend into the medulla (syringobulbia). Symptoms in these patients include hemifacial numbness, facial pain, vertigo, dysphagia, and loss of taste. Symptomatic patients may benefit from decompression of the foramen magnum or shunting of the syrinx.

Figure 6. Chiari type I malformation with hydrosyringomyelia. This 69-year-old woman presented with left arm weakness and atrophy of the left trapezius muscle. On both the fast spin echo T2-weighted (A) and the spin echo T1-weighted (B) midline sagittal images, the cerebellar tonsils are noted to be low in position, extending 8 mm below the foramen magnum. The cerebellar tonsils also have an abnormal wedge-shaped configuration. An extensive syrinx is present within the cervical and upper thoracic spinal cord. Small bony spurs are incidentally noted at C4-5 and C5-6. The syrinx (containing cerebrospinal fluid) is depicted with high signal intensity on the T2-weighted scan and low signal intensity on the T1-weighted scan.

Chiari Type II

The Chiari type II malformation is the most common major congenital malformation of the posterior fossa. It is nearly always associated with hydrocephalus and a myelomeningocele. Findings in the brain include low insertion of the tentorium cerebelli (small posterior fossa), hypoplastic tentorium cerebelli (large incisura), towering cerebellum, extension of the cerebellum around the brainstem (laterally and anteriorly), a flattened pons with
scalloping of the clivus and petrous bones, a prominent preponitne CSF space, an elongated midbrain, a small elongated slitlike fourth ventricle (10% of cases have a "ballooned" or trapped fourth ventricle), fusion of the colliculi (beaking of the quadrigeminal plate/tectum), fenestration of the falx (with interdigitation of cerebral gyri), agenesis of the corpus callosum, and a large massa intermedia. Findings in the spine (Fig. 6-7) include displacement of the brainstem and hypoplastic cerebellum into the upper cervical canal, cervicomedullary kinking (the medulla and cervical cord overlap), an enlarged foramen magnum and upper cervical canal, a small C1 ring with compression of the displaced brainstem and tonsils and vermis, a bifid C1 arch, posterior arch defects (C3-C7) and syringomyelia. The latter can occur in any location, more commonly in the low cervical and thoracic regions.

- Chiari Type III

The Chiari type III malformation is quite rare. Findings are similar to a Chiari type II but with the addition of a cervico-occipital encephalocele. There is an osseous defect of occiput and upper cervical spine, with cerebellar herniation into the encephalocele.

Figure 7. Chiari type II malformation. On the midline sagittal T2-weighted image, a complex congenital abnormality involving the brainstem and cerebellum is clearly depicted. The cerebellar tonsils are elongated and extend down to the C2-3 level. The fourth ventricle is slitlike. The insertion of the tentorium is low, making for a small posterior fossa. The colliculi are fused, forming a "beaked" tectum. All are common features of the Chiari type II malformation.

- Basilar Invagination
Patients in whom the tip of the odontoid process is 5 or more mm above Chamberlain's line (which is drawn from posterior margin of the hard palate to the posterior lip of the foramen magnum) are said to have basilar invagination. This anatomic variant can be primary or secondary (acquired) in type. The primary type is often associated with fusion of the atlas and occiput (occipitalization or assimilation). The secondary or acquired type is also called basilar impression. Acquired basilar invagination can be seen with osteomalacia, osteoporosis, fibrous dysplasia, Paget's disease, achondroplasia, and osteogenesis imperfecta. Platybasia can accompany basilar invagination. The normal angle formed by the clivus and floor of anterior cranial fossa measures 125 to 140 degrees. Platybasia is defined as an angle greater than 140 degrees.

- **Os Odontoideum**

Both congenital and acquired causes have been described for os odontoideum. In this structural anomaly, a corticate ovoid ossicle is present, distinct from the body of C2 (Fig. 6-8). Os odontoideum must be distinguished from a fracture of the dens, the latter being not uncommon after major trauma. Familial cases and associated congenital abnormalities support the existence of congenital lesions. Reports of development of this abnormality after trauma support the existence of acquired lesions. In a patient with os odontoideum, the anterior arch of C1 will also be enlarged and have a convex posterior margin.

- **Neurofibromatosis**

There are two major types of neurofibromatosis (NF). Both are autosomal dominant, but type 1 (NF1) is much more common. The abnormality has been localized to chromosome 17 in NF1 and to chromosome 22 in NF2.

Distinctive physical exam findings in NF1 include café-au-lait spots and iris hamartomas (Lisch nodules). Findings on MRI of the spine include scoliosis, a patulous dural sac, lateral meningoceles, and neurofibromas of the exiting nerve roots. Findings on MRI of the spine in NF2 include intradural extramedullary lesions (neurofibromas and meningiomas) and intramedullary lesions (ependymomas and low-grade astrocytomas) (Fig. 6-9). The presence of bilateral acoustic neuromas on imaging of the head is considered pathognomonic of NF2. These patients may also have schwannomas, meningiomas, gliomas, and hamartomas of the brain. Peripheral nerve lesions, either solitary or involving multiple nerves in plexiform manner, are considered hallmarks of NF, but these are less commonly seen on MRI because of the focus of the exam being the brain or spine.
Figure 8. Os odontoideum, with stable fibrous union. This 46-year-old is being seen for neck pain after a car accident. A, On the sagittal T1-weighted scan, the tip of the dens
appears separate from the base. There is intervening intermediate signal intensity soft tissue. The marrow signal intensities of both the tip and the base are normal. The anterior arch of C1 is large and has a convex, not concave, posterior margin. B, On the sagittal fast spin echo T2-weighted scan, no soft-tissue edema is noted. The cervical canal is normal in caliber. T1-weighted sagittal images obtained in flexion (C) and extension (D) reveal no change in the distance between the tip of the dens and the anterior arch of C1. No cord compression is noted. The normal marrow and soft-tissue signal intensity seen on magnetic resonance imaging makes acute trauma very unlikely, with substantial edema otherwise anticipated. E, The lateral radiograph confirms the nonunion of the superior dens with its base. The superior fragment (arrow) is well corticated.
Figure 9. Neurofibromatosis 2. A, The midline sagittal T2-weighted image reveals two intramedullary lesions (likely either ependymomas or low-grade astrocytomas) with abnormal high signal intensity. One lesion is at the level of C3 and the other at the cervicomedullary junction. A third lesion, at C2, was better seen on adjacent slices. B, The midline sagittal T1-weighted image reveals an extramedullary mass with soft tissue signal intensity along the posterior margin of the thecal sac anterior to the posterior arch of the
C1. The mass causes mild deformity of the upper cervical cord. C, On the postcontrast sagittal T1-weighted image, two foci of abnormal intramedullary enhancement are seen (corresponding to the abnormalities noted on the T2-weighted scan): one within the cervical spinal cord and one at the cervicomedullary junction. The cord shows mild enlargement at the lesion sites. An intradural extramedullary enhancing mass (a meningioma) with a broad base abutting the dura is also seen along the posterior thecal sac just below the margin of the foramen magnum. D, An axial contrast-enhanced T1-weighted image through the posterior fossa reveals bilateral (enhancing) acoustic schwannomas. Other images through the brain (not shown) demonstrated multiple meningiomas.

**INFECTION AND INFLAMMATORY DISEASE**

- **Epidural Abscess**

Causes for an epidural abscess include hematogenous spread, direct extension, and penetrating trauma. Staphylococcus aureus is the most common organism. On MRI, thickened inflamed soft tissue is seen initially, which progresses to a frank abscess with a liquid center (Fig. 6-10). Depending on the stage of disease, the enhancement on MRI after contrast administration can be homogeneous or rim-like with central low signal intensity (pus). An epidural abscess may cause cord compression as a result of the presence of inflammation, granulation tissue, or pus.
Figure 10. Epidural abscess. A cervical epidural catheter had previously been placed (now removed) for management of chronic left upper extremity pain. A, The T2-weighted axial scan reveals anterior displacement of the thecal sac. B, The T1-weighted scan raises the question of a posterior soft tissue mass. C, Postcontrast, an epidural fluid collection (arrow) is noted, with prominent enhancement of surrounding soft tissue. These findings are confirmed on the sagittal T2- (D), T1- (E), and postcontrast T1-weighted (F) scans. Contrast use permits identification of the fluid pocket, with surrounding inflammatory change (F, arrow), indicating the diagnosis of infection.

- **Sarcoidosis**

Sarcoidosis is a noncaseating granulomatous disease of unknown cause. The CNS is involved clinically in 5% of patients. The basal leptomeninges and floor of the third
ventricle are the most common sites of involvement. Spinal cord involvement is much less common.

MRI findings in sarcoidosis of the spine include fusiform cord enlargement, nodular parenchymal enhancement (broad based along the cord surface), and thin pial enhancement. Treatment is with steroids. Follow-up scans may demonstrate a return to normal appearance.

- **Rheumatoid Arthritis**

Rheumatoid arthritis is a synovitis. This disease can involve any synovium-lined joint. In the axial skeleton, the upper cervical spine is most commonly involved, usually at the articulation of the atlas and dens (Fig. 6-11). Imaging findings include increased distance between the atlas and dens (with instability), erosion of the dens (by surrounding inflammatory pannus), a retrodental soft tissue mass (resulting from involvement of the transverse ligament), and settling of the skull on the atlas. Rheumatoid arthritis, with involvement of the atlas and dens, can lead to cord compression.
Figure 11. Rheumatoid arthritis with atlantoaxial subluxation. A 72-year-old woman with advanced rheumatoid arthritis presented clinically with neck and left arm pain. A, The sagittal T2-weighted image reveals abnormal high signal intensity (resulting from fluid
and inflammation) between the anterior arch of C1 and the dens. Areas of low signal intensity consistent with fibrosis and chronic reactive changes are also present. B, The sagittal precontrast T1-weighted image demonstrates a large soft tissue mass predominantly anterior to the odontoid process. The cortex of the dens is mildly irregular. Enhancement of a portion of the abnormal soft tissue is seen on the postcontrast sagittal T1-weighted image (C). The distance between the dens and the anterior arch of C1 is normal on the axial gradient echo image obtained in neutral position (D). E, The axial gradient echo image obtained in flexion demonstrates increased space measuring 8 mm between the dens and C1 consistent with atlantoaxial subluxation. The upper cervical cord is compressed between the dens and the posterior arch of C1. Atlantoaxial subluxation was confirmed on a lateral plain radiograph of the cervical spine (not shown). The distance between the anterior arch of C1 and the dens measured 11 mm.

**BENIGN FOCAL LESIONS**

- **Osteochondroma**

An osteochondroma, also known as an osteocartilaginous exostosis, is a bony excrescence, with a cartilaginous covered cortex and a medullary cavity contiguous with the parent bone. Osteochondromas are rare in the spine. However, when present, the cervical spine is the most common location (half of all cases). The lesion is typically located in a spinous or transverse process.

- **Aneurysmal Bone Cyst**

Aneurysmal bone cysts are benign, nonneoplastic lesions. This lesion is typically osteolytic, multiloculated, expansile, and highly vascular. Aneurysmal bone cysts often contain blood degradation products. Eighty percent are seen in patients younger than 20 years. Twenty percent of all lesions are seen in the spine; the cervical and thoracic spine are the most common locations. Most spinal lesions occur in the posterior elements.

- **Eosinophilic Granuloma**

Eosinophilic granuloma is a benign, nonneoplastic disease. The preferred terminology for this disease is Langerhans' cell (eosinophilic) granulomatosis. Lesions may be solitary or multiple and are typically lytic without surrounding sclerosis. Eosinophilic granuloma is the classic cause of vertebra plana (a single collapsed vertebral body).

- **Cavernous Angioma**

Cavernous angioma is one of the four general types of vascular malformations; the other three are capillary telangiectasia, venous angioma, and arteriovenous malformation. Cavernous angiomas are angiographically occult. They are thus grouped together with capillary telangiectasias, which most commonly are solitary, occur in the pons, and are clinically silent, under the term occult cerebrovascular malformations. Cavernous angiomas occur throughout the CNS and are multiple in one third of all cases. Eighty
percent are familial. The majority of cavernous angiomas are clinically silent; the most common clinical presentation is seizure.

The typical cavernous angioma is small and smoothly marginated on imaging studies (Fig. 6-12). The border or rim of the lesion is markedly hypointense on T2-weighted scans as a result of hemosiderin and ferritin deposition within macrophages after hemorrhage. Centrally, a cavernous angioma contains a honeycomb of vascular spaces separated by fibrous stands, which appears as a mixture of high and low signal intensity on T2-weighted scans.
Figure 12. Cavernous angioma (cavernoma). The patient is a 34-year-old woman with right and left hand, arm, and neck pain and numbness. On T2-weighted scans using fast spin echo (A) and gradient echo (B) technique, a high-signal-intensity abnormality is noted within the cord at the T1 level, with a circumferential rim of hypointensity. The lesion is well marginated from surrounding tissue. Although the central high-signal-intensity portion of the lesion is clearly seen on the fast T2 scan, the rim of hypointensity is much less evident. Gradient echo scans, because of their sensitivity to susceptibility effects, clearly depict the presence of hemosiderin and ferritin. Fast T2-weighted scans are inferior in this
regard because of the acquisition of closely spaced spin echoes and thus relative insensitivity to susceptibility effects. The lesion is not well seen on the sagittal noncontrast T1-weighted image (C). D, The axial gradient echo scan demonstrates the central hyperintense fluid collection (methemoglobin), together with the smooth peripheral rim of hypointensity (hemosiderin/ferritin). A second lesion with similar characteristics was present within the medulla (not shown).

- **Hemangioblastoma/Von Hippel-Lindau Disease**

Hemangioblastomas of the spinal cord can be solid, with surrounding cord edema, or cystic, with an enhancing mural nodule. If the lesion is cystic, the fluid contents, although similar, will be differentiable from CSF on some pulse sequences. Hemangioblastomas are highly vascular lesions and thus enhance prominently on MRI. Their appearance on x-ray angiography is distinctive because of the tumor blush and enlarged feeding arteries and draining veins. Hemangioblastomas are most frequently found in the posterior fossa. They are much less common in the cord, but when in this location have an equal incidence in the cervical and thoracic spine. Spinal cord hemangioblastomas can be solitary or multiple, the latter pathognomonic of von Hippel-Lindau disease.

Von Hippel-Lindau disease is an autosomal-dominant syndrome. Features of this disease outside the CNS include renal cell carcinoma, pheochromocytoma, and cysts of the kidney and pancreas. In regard to neurologic disease, these patients present with hemangioblastomas of the cerebellum or spinal cord (Fig. 6-16).

- **Meningioma**

Of all intraspinal tumors, meningiomas represent 25% and are second in incidence to neurinomas. Meningiomas are usually solitary lesions. The peak age incidence is 45 years. Meningiomas are histologically benign and slow growing and cause symptoms because of cord and nerve root compression. On MRI, spinal meningiomas look much like meningiomas of the brain, often demonstrating a broad dural base and consistently displaying intense enhancement. One percent to 3% of all meningiomas occur at the foramen magnum (Fig. 6-17). Of extramedullary lesions in this location, three quarters are meningiomas and one quarter neurofibromas.

**NEOPLASTIC DISEASE**

- **Astrocytoma**

Astrocytomas are the most common intramedullary tumor in the cervical region. This tumor type has a lower incidence in the distal spinal cord, the opposite of ependymomas. The peak incidence for spinal cord astrocytomas is the third and fourth decades. The tumor grade tends to be lower than for brain astrocytomas.

On imaging, an astrocytoma causes fusiform enlargement of the spinal cord (Fig. 6-13). Typically, a long segment of cord is involved (several vertebral segments in length) along
with nearly the complete cross-section of the cord. Abnormal high signal intensity on T2-weighted scans reflects both tumor and edema. Enhancement postcontrast is common with cord astrocytomas but is not seen in all cases. This is one entity in which the addition of delayed scans (obtained 30 to 60 minutes after contrast administration) improves the detection of abnormal enhancement. Cord enlargement, limited to one or two levels, favors the diagnosis of an ependymoma over an astrocytoma. Contrast administration is mandatory in the MRI examination of postoperative cases for tumor recurrence (Fig. 6-14). Postsurgical changes can be difficult to distinguish from recurrent tumor without contrast administration, and recurrent tumor almost invariably enhances regardless of whether the primary lesion did so. Cord ischemia or infarction (in the subacute time frame) should be kept in mind in terms of differential diagnosis for an enhancing cord lesion of substantial craniocaudal extent (Fig. 6-15).

- **Metastases to Bone**

Vertebral metastases are a major source of morbidity in cancer patients. The spinal column is involved in up to 40% of patients dying of metastatic disease. Bone expansion, pathologic fractures, and cord compression are not uncommon. Plain x-ray films are insensitive for lesion detection; at least 50% of the bone needs to be destroyed in order for the lesion to be seen. Bone scans have high sensitivity but low specificity. Reasons for false-positive results on bone scan include infection, trauma, and degenerative disease. Computed tomography (CT) is typically limited in the extent of coverage and offers poor soft tissue contrast. With myelography, cord compression can be evaluated, but lesions are inferred (not directly visualized). MRI offers high sensitivity and specificity, excellent anatomic coverage, and excellent soft tissue lesion detection. MRI is universally accepted as the modality of choice for the detection and assessment of metastases involving the spinal column.
Figure 13. Cervical cord astrocytoma. A 10-year-old presented with arm weakness. A, On the precontrast T2-weighted sagittal scan, a hyperintense cord lesion is noted, which extends from C3 to C7. B, The precontrast T1-weighted scan reveals marked cord enlargement. C, Postcontrast, there is mottled abnormal enhancement within portions of the lesion. Although not all cord astrocytomas demonstrate enhancement postcontrast on magnetic resonance imaging, this finding, when present, improves differential diagnosis and provides guidance for biopsy. Administration of contrast is particularly important in the presence of a syrinx if a neoplastic origin is in question.
Figure 14. Recurrent astrocytoma. The magnetic resonance imaging (MRI) exam was performed several years after an extensive laminectomy for resection of a spinal cord astrocytoma. Examining the sagittal precontrast T2(A) and T1-weighted (B) scans, a syrinx cavity is noted, which expands the cord and extends from C2 to T2. The signal intensity characteristics of the syrinx differ from that of cerebrospinal fluid, suggesting a
neoplastic origin. On the sagittal (C) and axial (D) postcontrast T1-weighted scans, there is abnormal enhancement of a large soft tissue nidus within the syrinx at the C5-6 level. This finding was new from the prior MRI exam and represents recurrent tumor. Postcontrast scans in the spine are particularly valuable for detecting recurrent intramedullary neoplastic disease. Such lesions are often difficult to detect without contrast administration because of the distortion of normal structures and the isointensity of the lesion with surrounding soft tissue.
Figure 15. Cord ischemia (resulting from therapeutic radiation). This 7-year-old became quadriplegic after spinal axis radiation for acute lymphocytic leukemia. Biopsy revealed gliosis. A cervical spine magnetic resonance imaging (MRI) scan obtained before treatment was normal. A, On the sagittal T2-weighted scan, there is abnormal hyperintensity within the cervical cord and lower brainstem. Enlargement of the upper cervical cord is best visualized on the precontrast T1-weighted scan (B). Comparison of pre- (B) and postcontrast (C) sagittal T1-weighted scans reveals marked abnormal enhancement within the upper cervical cord. The MRI exam was repeated 5 months later with a sagittal T1-weighted scan (D). At that time, only atrophy of the upper cervical cord was noted. There was no abnormal contrast enhancement.

Figure 16. Spinal cord hemangioblastoma. A 42-year-old with known von Hippel-Lindau disease presented clinically with increasing gait disturbance. A cord syrinx is noted, with high signal intensity on the T2-weighted scan (A) and low signal intensity on the T1-weighted scan (B), which extends from the medulla to C2-3. There is secondary expansion of the spinal cord. No abnormal soft tissue mass is noted precontrast. The cerebellar tonsil is globular in shape and normal in position, ruling out a Chiari type I malformation. After contrast administration (C, sagittal; D, axial), an enhancing nodule is identified along the posterior wall of the upper portion of the syrinx. Hemangioblastomas are relatively rare benign epithelial tumors. In von Hippel-Lindau disease (with which there is an association), these tumors may be multiple.
Figure 17. Foramen magnum meningioma. On the precontrast T2- (A) and T1-weighted (B) axial images, a mass is seen at the level of the foramen magnum. There is substantial deformity of the medulla. Axial (C) and coronal (D) postcontrast images demonstrate intense lesion enhancement (D, arrow). The dural-based origin of the lesion, questioned on the basis of precontrast scans, is confirmed postcontrast.

High cervical vertebral metastases, in particular, can be a cause of great morbidity (Fig. 6-18). Sensory and motor deficits from such lesions can be extensive. Cranial neuropathies can occur as a result of spread to the skull base. Compression of the cervical cord above C3 can lead to death by respiratory embarrassment. In regard to tumor type, involvement of cervical spine and skull base by squamous cell carcinoma of neck is not uncommon. This tumor generally spreads by local invasion. Cervical vertebral metastases also commonly arise from a distant primary, with prostate, lung, and breast carcinoma common causes.
Figure 18. Cervical bony metastases with skull base involvement causing basilar impression. The 61-year-old patient had extensive laryngeal carcinoma and presented with increasing neck pain. Sagittal images just to the left of midline reveal abnormal signal intensity within the C1, C2, and C6 vertebral bodies, high on T2-weighted scans (A) and low on T1-weighted scans (B). The clivus is also involved. At both C1 and C6, there is anterior compromise of the thecal space resulting from the expansile nature of the lesions. Sagittal images more off to the side (not shown) revealed contiguity of the vertebral lesions with the patient's extensive laryngeal squamous cell carcinoma. The tip of the dens lies within the foramen magnum 1 cm superior to Chamberlain's line. C, Postcontrast, the affected vertebrae and skull base enhance to isointensity with normal marrow. Thus, it is difficult postcontrast to identify the marrow replacement by neoplastic disease on the basis of signal intensity alone. Epidural extension of the abnormality at C6, however, is more clearly depicted. D, A precontrast T1-weighted axial image at the C1 level confirms the abnormal low signal intensity within the dens and much of the anterior arch of C1. The abnormal soft tissue that infiltrates the arch of C1 on the left involves as well the adjacent occipital condyle. Abnormal soft tissue is also present within the anterior spinal canal, but
no cord compression is visualized at this level. Causes of basilar impression (acquired basilar invagination) include osteoporosis and osteomalacia, Paget's disease, and achondroplasia. However, any disease that produces abnormal bone softening, such as metastases, may lead to basilar impression.

Metastases to a vertebra, regardless of location, appear on MRI as low signal intensity lesions on T1-weighted scans because of the replacement of normal high signal intensity fatty marrow. Metastases are often high signal intensity on T2-weighted scans. The appearance on T2-weighted scans is, however, variable. Blastic metastases are often low signal intensity on T2-weighted scans. Thus, most MRI sites use precontrast T1-weighted scans for detection of vertebral metastases. After intravenous contrast administration, vertebral metastases often enhance to isointensity with normal surrounding marrow. Postcontrast scans, particularly as commonly used without fat saturation, are poor for detection of lesions within the bones of the spinal column. However, contrast enhancement generally improves the depiction of the epidural soft tissue extent of metastatic disease.
Figure 19. Leptomeningeal metastases from pineoblastoma. Two years before the current exam, this 9-year-old boy presented with persistent headaches and vomiting. Imaging revealed obstructive hydrocephalus, with a mass in the pineal region which proved (by subtotal resection) to be a pineoblastoma. The patient subsequently received brain and spinal axis radiation as well as chemotherapy. At this time, he presents with intractable vomiting, ataxia, and back pain. A bulky soft tissue mass is noted at the C1-2 level on sagittal T2- (A) and T1- weighted (B) scans, causing marked cord compression. The T2-weighted scan identifies an additional lesion at the C6 level, which is poorly seen on the T1-weighted exam. A portion of the larger lesion at C1-2 is of low signal intensity on the T2-weighted scan, suggesting tumoral hemorrhage. In the midthoracic region, multiple additional soft tissue masses were seen within the thecal sac (not shown). These were immediately adjacent to the cord and produced an irregular surface contour (C, D). Head magnetic resonance imaging obtained 2 weeks later reveals intracranial metastases. Two low signal intensity foci (arrows) can be identified precontrast on the T2-weighted exam (C). At least two enhancing lesions (arrows) are identified postcontrast on the T1-weighted exam (D). Pineoblastomas are primitive tumors of pinealocyte origin (as opposed to the more differentiated pineocytomas) that present in the first decade of life and are more common in males. Dissemination via the cerebrospinal fluid (CSF) is common. Another pediatric tumor with a propensity for early CSF spread is medulloblastoma.

- Leptomeningeal and Spinal Cord
  - Metastases

Five percent of all metastatic disease to the CNS will have intramedullary spinal metastases (metastasis to the spinal cord itself). The thoracic cord is most often involved. Bronchogenic carcinoma is the most common primary. On imaging, spinal cord metastases have a central enhancing focus with surrounding cord edema, an appearance expected from the imaging of brain metastases.

Leptomeningeal metastases in the cervical region can present on imaging as soft tissue nodules within the thecal sac (Fig. 6-19), irregularity of the cord surface contour (tumor adherent to or encasing the cord) (Fig. 6-20), or thin coating of the spinal cord (especially the dorsal aspect). Diffuse subarachnoid spread of tumor can cause coating and encasement (with deformity) of the spinal cord, leading to an appearance on gross exam resembling "icing." Most leptomeningeal metastatic disease enhances postcontrast on MRI; contrast administration is highly recommended for diagnosis. The entire spinal axis (cervical, thoracic, and lumbar) should be studied to rule out leptomeningeal metastases, with attention to the lumbar region (because of the effect of gravity). MRI, performed with and without contrast enhancement, has been consistently demonstrated in published studies to be superior to CT myelography for the detection of leptomeningeal metastases. This is particularly true for small tumor nodules and coating of the spinal cord by tumor. CT myelography is also not sensitive to intramedullary tumor involvement.
Figure 20. Leptomeningeal ("drop") metastases from medulloblastoma. The patient is a 4-year-old who had headaches for 9 months and now presents with diminished coordination. Head magnetic resonance imaging (not shown) revealed a midline enhancing posterior fossa mass with obstructive hydrocephalus. On the midline sagittal T1-weighted cervical image, multiple large soft tissue nodules are noted adjacent to the cervical cord. These demonstrated only very slight enhancement postcontrast (not shown). The posterior fossa mass was resected, followed by whole brain and spinal axis radiation. The follow-up scan 2 months later (not shown) revealed a normal thecal sac and spinal cord.

RADIATION THERAPY

The changes encountered with radiation therapy can at times be readily identified because of the confinement to the treatment area or port. After therapeutic radiation, there is uniform fatty replacement of bone marrow. This occurs as early as 2 weeks after initiation of therapy, with temporal progression. Imaging in the sagittal plane with T1-weighted scans is recommended. Vertebral bodies within the port will have substantially higher signal intensity on such scans (Fig. 6-21), assuming that the choice of time to echo and time to repetition has been made appropriately to obtain moderate to heavy T1-weighting.
Figure 21. Radiation therapy changes with fatty replacement of bone marrow. The patient is a 45-year-old woman with a clinical history of radical neck dissection and radiation therapy for squamous cell carcinoma of the mouth. The midline sagittal T1-weighted image demonstrates diffuse homogeneous high signal intensity throughout the marrow spaces of the C2-4 vertebral bodies. The high signal intensity in the marrow spaces shows an abrupt transition to the normal marrow signal intensity of the adjacent lower cervical vertebral bodies. Detection of this change was aided by comparison with the previous exam (not shown) and inspection of the relative signal intensity of the cord, disk spaces, and marrow. The increase in marrow signal intensity and uniformity of signal is due to radiation therapy with resultant replacement of normal red marrow by fat.

HYDROSYRINGOMYELIA

According to terminology developed for histopathology, syringomyelia is defined as an abnormal cavity within the spinal cord that is separate from but may communicate with the central canal. This is to be differentiated from hydromyelia, which is a dilatation of the central canal, lined by ependymal cells. These two entities are indistinguishable on imaging. Thus, the term hydrosyringomyelia should be used (Fig. 6-22). On imaging, hydrosyringomyelia is seen as a longitudinally oriented fluid cavity (with CSF signal intensity on all pulse sequences) within the spinal cord.

Of special note in the cervical spine is syringobulbia, which is simply extension of a syrinx into the brainstem (Fig. 6-23). This lesion is caused by obstruction of CSF flow at the foramen magnum, usually because of the presence of a Chiari type II malformation. Extension of the syrinx superiorly to involve the brainstem, with a tubular or saccular
configuration, is thought to be the result of episodes of increased intra-abdominal pressure (as a result of coughing or sneezing). Symptoms of syringobulbia include facial pain and numbness, dysphagia, vertigo, loss of taste, and respiratory problems (in severe cases).
Figure 22. Hydrosyringomyelia. Sagittal (A) and axial (B) T2-weighted images demonstrate apparent dilatation of the central canal of the spinal cord over a two-vertebral-body segment in the lower cervical spine. The abnormality is equally well seen on sagittal (C) and axial (D) T1-weighted images. The signal intensity of this centrally located fluid cavity is that of cerebrospinal fluid on all scans, being high signal intensity on T2-weighted images and low signal intensity on T1-weighted images. The patient's symptoms were unrelated to this incidental finding.

On MRI, the sagittal plane is typically used to define the extent of a syrinx. Imaging in the axial plane is often helpful to visualize small syrinxes and to confirm intermediate size lesions. Hydrosyringomyelia has many causes, including trauma (with development of the syrinx over years after the event), neoplasm, arachnoiditis, surgery, and developmental abnormalities such as the Chiari malformations.

Clinical symptoms of a cervical syrinx include progressive upper extremity weakness, muscle wasting, decreased upper extremity reflexes, and loss of pain and temperature sensation (with preservation of light touch and proprioception). A syrinx that enlarges in the post-traumatic patient can cause clinically significant neurologic deterioration. Large symptomatic syrinxes are treated surgically by shunting into the subarachnoid, pleural, or peritoneal spaces (Fig. 6-24).
Figure 23. Syringobulbia. This 31-year-old is status postcervical fusion 8 years ago for multiple fractures. A, The T2-weighted midline sagittal image reveals postoperative changes at C5-6. The C5 and C6 vertebral bodies have been surgically fused, with loss of the normal intervening disk space. A large amount of metallic artifact is present in the region of the posterior elements compatible with known stainless steel fixation wires. A fluid collection, which is noted to be septated on the T1-weighted image (B), is identified within the spinal cord above the site of fusion, extending superiorly to near the inferior extent of the fourth ventricle. The abnormality (a posttraumatic syrinx) is isointense with cerebrospinal fluid with on T1- and T2-weighted images.
Figure 24. Posttraumatic hydrosyringomyelia with interval shunting and collapse. This 44-year-old man suffered a fracture of T9 that was treated by laminectomy and fusion 12 years ago. The patient now presents with delayed, progressive neurologic deficits. A and B, The preoperative study demonstrates fluid signal extending down the central portion of the spinal cord from the cervicomedullary junction through the visualized lower cervical region. This is seen as high signal intensity within the cord on the sagittal T2-weighted image (A) and low signal intensity on the postcontrast T1-weighted image (B). No abnormal enhancement is noted. The lesion is a posttraumatic syrinx secondary to a severe wedge compression fracture of T9 (not shown). The spinal cord is expanded with effacement of the surrounding subarachnoid space. The patient underwent a thoracic laminectomy with placement of a syringoperitoneal shunt. C, The postoperative sagittal T1-weighted image of the cervical spine demonstrates collapse of the syrinx. The spinal cord is mildly atrophic, with cerebrospinal fluid now present surrounding the cord. Postoperatively, the patient's muscle strength and sensation improved.

TRAUMA

In flexion injuries, anterior wedging of the vertebral body and vertebral body fractures occur. In severe flexion injury, there can be disruption of the posterior longitudinal ligament and interspinous ligaments, facet distraction, and anteroposterior subluxation. In extension injuries, posterior element fractures occur. In severe extension injury, there can be rupture of the anterior longitudinal ligament and subluxation. Axial loading injuries (with vertical compression from diving or jumping accidents) produce vertebral body compression (burst) fractures and lateral element fractures. Rotation injuries, although rarely isolated and usually occurring with flexion-extension injury, produce lateral mass...
fractures and facet subluxations. High-resolution CT with multiplanar reconstruction is commonly used in acute trauma and best evaluates bony lesions. MRI is best in regard to the evaluation of the cord and soft tissues.

Cord hemorrhage after spinal cord injury carries in general a poor prognosis (Fig. 6-25). Cord edema, in the absence of hemorrhage, carries a much better prognosis, often with substantial neurologic recovery. Several patterns of acute spinal cord injury have been described on T2-weighted MRI scans. Type I injury has central hypointensity with a thin rim of hyperintensity (deoxyhemoglobin centrally with methemoglobin at the periphery) and carries a very poor prognosis, with little neurologic recovery anticipated. Type II injury has uniform hyperintensity as a result of spinal cord edema and carries an excellent prognosis, with substantial, often complete, neurologic recovery (Fig. 6-26). Type III injury has an isointense center with a thick rim of hyperintensity, representing a combination of hemorrhage and edema, and follows a variable course; some recovery of function is anticipated.

Myelomalacic changes in the spinal cord after trauma follow a well-known sequence. Early on there is cord edema, with compression and stasis within venules and blood-cord barrier disruption. In this early stage, the area of injury in the cord is high signal intensity on T2-weighted scans because of the presence of vasogenic edema. With progression of time, cystic necrosis occurs within the central gray matter. This has high signal intensity on T2-weighted scans and low signal intensity on T1-weighted scans. In the chronic stage, progressive cystic degeneration centrally may lead to a syrinx. The presence and extent of a syrinx is often best defined on axial T1 images; visualization on sagittal images suffers from partial volume imaging. Cord atrophy may also develop in the chronic period. Cord atrophy is defined by a cord diameter of less than 6 mm in the cervical region and less than 5 mm in the thoracic region.
Figure 25. Traumatic spinal cord injury, with cord hemorrhage (and edema) and canal compromise. The 34-year-old patient was an unrestrained passenger in a single-vehicle motor accident. A, On the midline sagittal T2-weighted image, there is abnormal high signal intensity within the cord extending from the tip of the dens to below the C7 level. There is compromise of the spinal canal posteriorly at the C5 and C6 levels. Abnormal high signal intensity is noted within the C4-5, C5-6, and C6-7 intervertebral disks, suggesting fluid accumulation or edema (secondary to trauma). B, On the corresponding T1-weighted sagittal image, there is abnormal hyperintensity within the cord from C4 to C6, corresponding to methemoglobin. Posterior compromise of the spinal canal is again noted. C, On the single axial T1-weighted image, the posterior soft tissues are asymmetric, suggesting additional injury, and the lamina on the left appears fractured. Lamina fractures were noted on computed tomography (not shown) at both the C5 and C6 levels on the left. The patient, who was quadriplegic after the accident, died 2 weeks later of multisystem failure.

Figure 26. Cord contusion with a small posterior epidural hematoma. The patient is a 25-year-old man who 12 hours earlier was involved in a motor vehicle accident and now complains of bilateral upper extremity and shoulder pain. A, On the T2-weighted exam, abnormal high signal intensity is identified within the cord from C5 to C6 consistent with edema (E) (cord contusion). There is obliteration from C4 to C6 of the cerebrospinal fluid space that normally surrounds the cord. Posteriorly in the epidural space, abnormal soft tissue (with mixed high and low signal intensity) is identified (A, white arrow), causing thecal sac compression. B, The sagittal T1-weighted exam at first glance appears
unremarkable, with perhaps only subtle loss of definition of the superior end plate of C7 (white arrow). Examining closely the epidural space at the C5 and C6 levels, abnormal high signal intensity corresponding to methemoglobin is identified (B, black arrows). Without comparison to the T2-weighted scan, this small extradural hematoma might have been mistaken for normal epidural fat. High signal intensity is identified on the T2-weighted image within the bodies of C6 and C7 as a result of microfractures and resultant marrow edema. This finding is consistent with the poor visualization of the superior end plate of C7 (B), which suggests gross bony damage. Extensive high signal intensity in the soft tissues posteriorly on the T2-weighted exam indicates substantial soft tissue and ligamentous injury.

In the imaging of spine trauma, as previously stated, CT is superior for the demonstration of osseous injury. CT is preferred (over MRI) for the evaluation of posterior element fractures and canal narrowing resulting from retropulsed fragments. MRI is preferred for evaluation of the spinal cord in trauma. MRI is superior for the demonstration of cord injury and cord compression by soft tissue, such as a traumatic disk herniation (Fig. 6-27). Traumatic disk herniations most commonly occur in the cervical spine as opposed to the thoracic or lumbar spine. The incidence of disease increases with the severity of trauma. A traumatic disk herniation is common with hyperextension injury, specifically at C5-6 (Fig. 6-28). In whiplash injuries (acceleration hyperextension), acute posterolateral disk herniations are primarily seen. Symptoms include immediate neck and arm pain. In patients with cervical fractures, a disk herniation is most common at the level immediately below the fracture. Cord compression can be due to a traumatic disk herniation, bone fractures or dislocations, or, not to be forgotten, an epidural hematoma (Fig. 6-29). T2-weighted images are important for the demonstration of marrow edema (vertebral body microfractures) and soft tissue injury.

A number of specific osseous injuries occur with some frequency after cervical trauma, and several carry colorful names. Atlanto-occipital dislocation is often fatal. Diagnosis is made on sagittal images (or a lateral x-ray film); the normal distance between the dens and the anterior margin of foramen magnum is no more than 12.5 mm. Jefferson's fracture is a burst fracture involving both the anterior and posterior arches of C1 (the atlas). Unless the transverse ligament is disrupted, the patient will be neurologically intact. This can be an unstable fracture. A fracture of the dens (Fig. 6-30) can occur with either hyperflexion or hyperextension. Dens fractures are classified by the anatomic location of the fracture line. Type I fracture involves the upper dens. Type II fracture involves the junction of the dens and the body. This is the most common type of injury and has the highest rate of nonunion. Type III extends into the C2 body. It is important to note that transverse fractures, such as those that occur in the dens, can be inapparent on axial images. Hangman's fracture, which is hyperextension injury, is a fracture or fracture dislocation at the level of C2 and C3 that extends through the pedicles of C2. The clay shoveler's fracture is a flexion injury, with avulsion of the spinous process, usually C6 or C7.

Injury to the cervical spine can be the result of abnormal flexion (or extension), rotation, or a combination of flexion and rotation. Bilateral facet fractures or dislocation are the result of flexion injury. Unilateral facet fractures are the result of flexion plus rotation (Fig. 6-31).
Vertebral body compression fractures result from flexion injury. Injury to the posterior musculature and ligaments occurs with flexion. Unilateral involvement suggests a rotational component.

Figure 27. Posttraumatic right foraminal disk herniation at C6-7. This 36-year-old presented with severe right arm and neck pain after a "whiplash" injury. A, The T2-weighted sagittal image, just to the right of midline, reveals a prominent extradural defect...
at the C6-7 level. B, On the corresponding T1-weighted sagittal image, the abnormal soft tissue is noted to be contiguous with the C6-7 disk (arrow) but also extends well above and below the level of the disk. After contrast administration (C), it is evident that the soft tissue above and below the disk space level corresponds to dilated epidural venous plexus (which enhances postcontrast, arrows). D, A postinfusion axial image at the C6-7 level confirms the disk herniation (arrow), which fills the right C6-7 neural foramen. Mild mass effect on the right side of the spinal cord is also noted.
Figure 28. Traumatic disk herniation (C5-6), with cord contusion and hemorrhage. This 30-year-old was an unrestrained driver in a motor vehicle accident. A, On the T2-weighted scan, abnormal soft tissue (isointense with disk material) is noted posterior to the C5-6 disk
space. Abnormal high signal intensity (consistent with edema) is also noted within the spinal cord, extending for at least two anatomic levels (C5-C6). B, On the T1-weighted scan, the abnormal soft tissue is again noted, abutting the spinal cord. The lesion is contiguous with the disk and has similar signal intensity. C and D, Two sagittal gradient echo images are also presented. The first scan (C) is along the midline, in the same anatomic position as the T1- and T2-weighted images. The traumatic disk herniation, contiguous with the C5-6 disk and of similar signal intensity, is again noted. This causes mild mass effect on the thecal sac. Cord edema is also confirmed. On the adjacent cut (D), slightly off the midline, the lesion is larger in size but remains contiguous with the disk space. Abnormal hypointensity is noted within the cord at the C5-6 level, consistent with hemorrhage (deoxyhemoglobin). A C5 pedicle fracture was noted on computed tomography (not shown). The patient was left with C5 quadriplegia on the right and C7 on the left. Drug screen was positive for cannabinoids and benzodiazepines.
Figure 29. Posttraumatic epidural hematoma. On the midline sagittal T2-weighted image, the cord is displaced anteriorly because of a large high signal intensity (methemoglobin) epidural fluid collection. This hematoma extends from C3 to C5 (and possibly below), obliterating the normal cerebrospinal fluid space. High signal intensity is seen within the cord, corresponding to edema, at the C3 and C4 levels.

Figure 30. Type II dens fracture. The patient is a 19-year-old woman who is being scanned 1 month after an unrestrained motor vehicle accident. Sagittal T2- (A) and postcontrast T1-weighted (B) images demonstrate a fracture through the base of the dens. Mild anterior slippage of the superior fracture fragment relative to the C3 vertebral body is also present. A small amount of enhancing granulation tissue or venous plexus is identified posterior to the dens on the T1-weighted scan. No evidence for cord compression or contusion is seen. The dens fracture and the offset of the C2 and C3 vertebral bodies were confirmed on a lateral x-ray film of the cervical spine (not shown).
Figure 31. Flexion-rotation injury of the cervical spine. The patient is a 28-year-old man with central cord syndrome who is being imaged 3 days after a motor vehicle accident. A, The midline sagittal T2-weighted image demonstrates mild increased signal intensity within the C3 and C4 vertebral bodies suggestive of microfractures. Increased signal is present within the spinal cord at the C3-4 level consistent with edema and cord contusion. There is also abnormal high signal intensity within the posterior musculature, as a result of edema. B, The corresponding precontrast T1-weighted image demonstrates a small central disk.
herniation at the C3-4 level with resultant canal compromise. No abnormal signal intensity is present in the cord to suggest hemorrhage. The vertebral bodies are grossly normal in height and alignment. C, The precontrast T1-weighted parasagittal image reveals discontinuity and deformity of the left C3 pedicle consistent with a fracture. The pre- (D) and postcontrast (E) T1-weighted axial images demonstrate asymmetric abnormal enhancement within the injured right paraspinous muscles. Plain cervical spine films (not shown) revealed a fracture-dislocation of C3-4 with approximately 3 mm anterior slippage of C3 on C4. No vertebral fracture was detected. Computed tomography (not shown) revealed a linear fracture through the left pedicle at C3. Mild anterior slippage of C3 on C4 was present with 20% compromise of the spinal canal but no direct impingement on the spinal cord. Traction was applied before the magnetic resonance imaging exam accounting for the normal alignment on this study.

- **Perched Facet**

Plain x-rays film may be suboptimal for evaluation of the lower cervical spine. On CT, misalignment of the facets may be inapparent unless sagittal reconstructions are performed. In distinction, MRI, with direct sagittal imaging, clearly delineates vertebral and facet alignment. It is incumbent on the radiologist to examine closely the alignment of the facets on all cervical spine MRI exams (Fig. 6-32). Perched or locked facets are not uncommonly missed in the setting of acute trauma; continued pain brings the patient back for further evaluation weeks to months later.

- **Brachial Plexus Injury**

Injury to the brachial plexus can lead to a posttraumatic neuroma, fibrosis, or meningocele (with or without nerve root avulsion). On MRI, meningoceles caused by brachial plexus injury are clearly seen. The lesion will follow the course of the nerve root in the foramen and manifest CSF signal intensity on all pulse sequences (Fig. 6-33). Nerve root avulsions per se are best evaluated by myelography.
Figure 32. C2 teardrop fracture and unilateral perched facet at C6-7. The patient presented 6 months after a motor vehicle accident with persistent left arm pain. Midline sagittal T2- (A) and T1-weighted (B) images demonstrate a teardrop fracture at the base (anteriorly) of C2, which had been noted on previous diagnostic exams. Mild anterior slippage of C6 on C7 is also apparent. The T1-weighted sagittal image (C) to the left of midline reveals a facet dislocation at C6-7 (arrow). The alignment of the facets on the right was normal (not shown).
Figure 33. Meningocele secondary to birth trauma. The patient is a 15-month-old infant with left upper extremity spasticity since birth. A, The T1-weighted left parasagittal image of the cervical spine reveals two low signal intensity extradural fluid collections within the C6-7 and C7-T1 neural foramina, respectively. These two abnormalities remained isointense with cerebrospinal fluid on T2-weighted scans (not shown). B, An axial T1-weighted spin echo scan confirms, at one level, the extradural location of the lesion and association with the exiting nerve root sleeve.

DISK HERNIATION

The cervical spine is most mobile at the C4-5, 5-6, and 6-7 levels. Thus, these are also the levels at which most disk herniations occur. Prior surgery with fusion at one level places the level above and below at increased risk for herniation. Cervical disk herniations are most commonly seen in the third and fourth decades of life. MRI and postmyelographic CT have equivalent sensitivity in the detection of acute cervical disk herniations; CT is better for demonstrating accompanying bony degenerative disease.

Clinical symptoms of a cervical disk herniation depend on its location. Large central herniations cause myelopathic symptoms (Fig. 6-34). Posterolateral or foraminal herniations can compress the exiting nerve root and cause radicular symptoms (Fig. 6-35).
Figure 34. Large central disk herniation at C4-5. The 42-year-old patient presented with recurrent neck pain. The clinical history is significant for a prior diskectomy and fusion at C5-6. A, The T1-weighted midline sagittal image reveals a prominent anterior extradural soft tissue mass contiguous with and posterior to the C4-5 intervertebral disk. The abnormality is of relatively low signal intensity, similar to the intervertebral disks, on this T1-weighted scan. On T2-weighted images (not shown), the abnormality remained isointense to disk material. The C5-6 disk space is narrowed and indistinct, compatible with the prior anterior diskectomy and fusion. B, A T1-weighted axial view through the C4-5 disk confirms the extradural soft tissue mass, with resultant central cord compression. The lesion is again noted to be contiguous with and isointense to the C4-5 disk. C, The corresponding axial gradient echo scan depicts this central disk herniation as high signal intensity.
Figure 35. Right foraminal disk herniation at C6-7. This 49-year-old patient presents with excruciating right arm pain. A, A midline T1-weighted sagittal image demonstrates small spurs and end plate degenerative changes at the C5-6 and C6-7 levels. B, A T1-weighted sagittal image to the right of midline demonstrates abnormal soft tissue (white arrow)
extending posterior to the vertebral bodies at the C6-7 level. This abnormality was isointense to disk material and contiguous with the C6-7 disk on both this image and the corresponding T2-weighted scan (not shown). C, An axial T1-weighted image at the C6-7 level does not clearly demonstrate the abnormality. However, the spinal cord does appear mildly shifted to the left. D, After contrast administration, the soft tissue abnormality (black arrow) is highlighted by the enhancement of the epidural venous plexus within the neural foramen. The disk herniation fills the right neural foramen at the C6-7 level.

Thin-section (less than 2 to 3 mm) images should be acquired in both the sagittal and axial planes on MRI when a disk herniation is suspected. T1-weighted spin echo and T2-weighted fast spin echo images are typically acquired in the sagittal plane. T2-weighted gradient echo images are of high value in the axial plane. On the latter, a thin rim of low signal intensity often outlines the high-signal-intensity disk herniation (along its posterior aspect). The low-signal-intensity rim corresponds to the dura and posterior longitudinal ligament. An acute disk herniation is seen on sagittal and axial images as an anterior epidural soft tissue mass. The abnormal soft tissue will be contiguous with the disk space unless a disk fragment is present. The signal intensity of the herniated material is similar to the native disk on both T1- and T2-weighted scans. A decade ago, postcontrast T1-weighted images were also commonly acquired. These can be very useful in diagnosis, but cost constraints led to their elimination in most clinical practices for the study of cervical disk disease. On postcontrast T1-weighted scans, the dilated epidural venous plexus surrounding a disk herniation will enhance, outlining the disk material and improving visualization of the neural foramina (Fig. 6-36). Without contrast administration, the epidural venous plexus is isointense with and cannot be distinguished from disk material on T1-weighted scans.

A "hard" disk is the result of a long-standing herniation (Fig. 6-37). A chronic disk herniation is covered above and below by bony spurs from the end plates. These form as a result of bone remodeling; elevation of the periosteum by the disk herniation leads to bone deposition at the site. Myelopathic symptoms are more common with chronic disk herniations as opposed to radicular symptoms, which are more common with acute disk herniations. Damage to the blood-spinal cord barrier, on the basis of chronic repetitive trauma at the level, can lead to enhancement within the cord at the level of a hard disk herniation (see Fig. 6-37). This is rarely visualized in current clinical practice because of the nonuse of contrast in the setting of chronic degenerative disease.
Figure 36. C3-4 right paracentral disk herniation. This 35-year-old patient presents with neck and right arm pain after a motor vehicle accident. Precontrast axial and sagittal T2- (A and D), and T1-weighted (B and E) scans reveal abnormal soft tissue at the C3-4 disk level anterior and to the right of the thecal sac, causing mild cord deformity. The lesion is difficult to separate from the contents of the right neural foramen. Postcontrast (C and F), the disk herniation itself (white arrow) can be differentiated from dilated epidural venous plexus (black arrows) because of prominent enhancement of the latter. There is no foraminal component.
Figure 37. Early compressive myelomalacia secondary to a large "hard" disk herniation at C3-4. This 44-year-old presented clinically with increasing pain and numbness in the upper extremities. A, On the midline fast spin echo T2-weighted sagittal image, abnormal increased signal intensity (likely a combination of edema and gliosis) is seen within the cord, extending from mid C3 to C4-5. At the C4-5 level, prominent osteophytes obliterate the cerebrospinal fluid space anterior to the cord. Mild retrolisthesis of C3 on C4 is identified on the midline sagittal T1-weighted image (B). The C3-4 intervertebral disk is narrowed, and abnormal soft tissue extends posterior to the disk, causing deformity of the cervical cord. C, The corresponding postcontrast T1-weighted image reveals prominent enhancement within the flattened cervical cord. Abnormal enhancement resulting from de novo scar and dilated epidural venous plexus is also apparent about the C3-4 disk.

HYPERTROPHIC END PLATE SPURS

Hypertrophic end plate spurs (osteophytes) are a common finding on MRI of the cervical spine (Fig. 6-38). Careful image inspection is necessary to distinguish these from a disk herniation. In most instances, spurs are asymptomatic. Imaging findings do not correlate well with clinical symptoms.

End plate spurs are the long-term result of a disk bulge or herniation. During healing, bone is laid down on elevated ligamentous attachments, resulting a bony spur. On MRI, these osteophytes can and should be distinguished from an acute disk herniation. T2-weighted
gradient echo images are very useful in this regard. Disk material is high signal intensity, and spurs are very low signal intensity. The high-signal-intensity CSF also tends to well outline these spurs. If large osteophytes are present along the anterior margin of the vertebral bodies at the levels in question, it is also likely that the compromise of the thecal sac posterior to the vertebral body is due to degenerative disease as opposed to an acute disk herniation. On postcontrast T1-weighted spin echo images, enhancement of the epidural venous plexus may outline the low signal intensity of the spur.

Figure 38. Hypertrophic osteophytic end plate spurs. A 57-year-old woman presented with right-sided neck and arm pain. Midline sagittal T2-weighted gradient echo (A) and T1-weighted spin echo images before (B) and after (C) contrast administration demonstrate a ventral extradural defect along the anterior margin of the thecal sac at the C5-6 level. A, The sagittal T2-weighted gradient echo image demonstrates, in addition to the low signal intensity spurs at C5-6, smaller spurs at C4-5 and C6-7 that partially efface the ventral subarachnoid space. B, The precontrast sagittal T1-weighted image shows pointed
extensions of bone marrow signal intensity along the posterior end plates adjacent to the C5-6 disk. C, Postcontrast, enhancement of dilated venous plexus is noted immediately above and below the C5-6 level. Mild irregularities of the posterior margin of the vertebral end plate are present on the axial T2-weighted gradient echo image (D). The signal intensity of these projections is very low, indicative of cortical bone. These findings are consistent with osteophytes extending from the posterior vertebral end plates.

SURGERY FOR CERVICAL SPONDYLOSIS

Damage to the spinal cord in cervical spondylosis is the result of ischemia from chronic compression. The aim of surgery is to prevent further deterioration. An anterior surgical approach is used for one to two-level stenosis (Figs. 6-39 and 6-40), and is the most common neurosurgical procedure in cervical disk disease. The disk is resected (using an anterior approach) and a bone graft placed between the two adjacent vertebral bodies to achieve a stable fusion. Portions of the adjacent vertebral bodies may or may not be removed. The signal intensity characteristics of the graft are variable. After more than 2 years, continuous marrow signal intensity is typically seen at the site of fusion, with no evidence of bone graft or native disk. There is a propensity over the long term for new disk herniations to develop above and below the site of fusion. The posterior approach, which is less common, involves a laminectomy and is used for congenital narrowing or extensive contiguous disease (multiple levels). MRI can be diagnostic in postoperative cases despite the presence of substantial metal hardware. Artifacts from metal will be greatest in general on gradient echo scans (because of the lack of a 180-degree refocusing pulse), moderate on spin echo scans, and least on fast spin echo scans (as a result of the short interecho interval). High signal intensity within the cord postoperatively on T2-weighted scans is seen occasionally and can be due to gliosis (present preoperatively) or postoperative complications (such as cord contusion and infarction).
Figure 39. Normal late appearance of anterior cervical diskectomy and fusion. The patient has continued left arm pain 4 months after anterior diskectomy and fusion for a C5-6 disk herniation. A, On the midline sagittal T2-weighted image, the C5-6 intervertebral disk is not seen. Small spurs with mild compromise of the thecal sac are noted at the levels above and below (C4-5 and C6-7). B, The sagittal T1-weighted image (obtained after contrast administration) demonstrates fusion of the C5 and C6 vertebral bodies. Mild decreased signal intensity is evident within the central portion of the fusion. The alignment of the cervical spine is normal.
Figure 40. Normal appearance after anterior diskectomy and titanium plate fusion at C6-7. This 48-year-old patient presented clinically with continued neck pain after surgery for a C6-7 disk herniation. A, The T1-weighted midline sagittal image reveals prominent metallic artifact anterior to and within the C6 and C7 vertebral bodies. The alignment of the cervical vertebral bodies is normal. No significant canal stenosis is present. The artifact is again present, but less apparent, on the fast spin echo T2-weighted sagittal image (B). A small osteophyte causes effacement of the ventral subarachnoid space at C4-5. C, On the axial gradient echo image at C6-7, the metal artifact is more extensive, with artifactual mild effacement of the anterior thecal sac. A lateral radiograph of the cervical spine (not shown) demonstrated a metallic plate and three screws that fused the C6 and C7 vertebral bodies.

MULTIPLE SCLEROSIS

Spinal cord multiple sclerosis (MS) plaques are best detected on T2-weighted scans. Short segments of the cord are typically involved and demonstrate abnormal high signal intensity. Focal cord enlargement is seen with acute lesions as a result of the presence of edema. Symptomatic (active) lesions may or may not demonstrate substantial surrounding edema but will consistently enhance postcontrast (Fig. 6-41). Edema, if present, can extend in a flamelike pattern above and below the lesion. Lesions are haphazard in distribution both in cross-section and longitudinally, disregarding anatomic boundaries. MS plaques tend to be elliptical in shape, with greatest dimension along the length of the cord. Cord
atrophy, which can be focal or generalized, is seen in long-standing disease (Fig. 6-42). Not all patients with spinal cord lesions will demonstrate characteristic brain lesions on MRI. The histologic appearance of spinal cord MS plaques is that of multifocal sharply marginated areas of demyelination.

Clinically, MS is characterized by recurrent focal neurologic attacks, progressive deterioration, and ultimately permanent neurologic dysfunction. Symptoms include decreased vibration and position sense, weakness of one or more extremities, and disorders of micturation (urination). Differential diagnosis, based on the results of MRI of the spinal cord, includes transverse myelitis. The presence of multiple cord lesions, combined with characteristic brain lesions, favors the diagnosis of MS.
Figure 41. Multiple sclerosis, with active spinal cord plaques. The patient is a 28-year old white woman with new onset 2 months ago of numbness below the waist, now involving the left arm. Several episodes of blurred vision in one eye have also occurred during the past 2 years. A, On the T2-weighted midline sagittal scan, two intramedullary lesions are noted, at C2-3 and C5-6, with the latter larger and exhibiting a flamelike pattern of edema extending superiorly and inferiorly. B, On the postcontrast T1-weighted midline sagittal scan, faint lesion enhancement (arrows) is identified at both levels. Axial T2-weighted gradient echo (C) and postcontrast T1-weighted spin echo (D) scans confirm the lower
lesion, which is eccentrically located, causes focal cord enlargement, and demonstrates prominent enhancement.

Figure 42. Multiple sclerosis (MS) (inactive or chronic disease). The patient is a 52-year-old white man with long-standing neurologic problems. He ambulates with a cane. Bowel function is intact; however, there is bladder incontinence. Heavily T2-weighted midline sagittal images of the cervical (A) and thoracic (B) spine are presented. A single hyperintense intrinsic cord abnormality is noted in the cervical spine at the C2 level, suggesting cord atrophy. Two thoracic cord lesions are also seen, both somewhat elongated in appearance. Incidental note is made of an osteophyte situated between the two thoracic cord lesions, causing anterior compression of the thecal sac. The lesions (all chronic MS
plaques) do not cause cord enlargement, and there was no abnormal contrast enhancement (not shown).

**ACUTE TRANSVERSE MYELITIS**

In acute transverse myelitis, a section of the cord demonstrates fusiform enlargement and abnormal high signal intensity on T2-weighted scans. The area involved usually extends over several vertebral segments. Clinical symptoms include a sudden loss of sensory and motor function in a segmental distribution. The pathogenesis is unknown. Possible causes include viral, vascular, and autoimmune disease.

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**References**

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INTRODUCTION

There are normally 12 thoracic vertebral bodies. The ribs articulate with the vertebrae both at the disk and at the transverse process. However, the latter articulation occurs only for T1 through T10. On sagittal magnetic resonance imaging (MRI) scans, the exit foramina for the basivertebral veins can be clearly identified posteriorly within the midvertebral body. Epidural fat is prominent posterior to the thecal sac.

The thoracic spine presents several unique problems in regard to MRI, necessitating attention to imaging technique to obtain a high-quality exam. A coronal saturation pulse (or presaturation slab) is routinely used to eliminate motion artifacts from the chest wall and heart. This saturation pulse is used in both sagittal and axial imaging of the thoracic
spine. A maximum slice thickness of 3 mm is recommended regardless of imaging plane. Thoracic disk herniations, in particular, are often small and not well visualized when thicker sections are acquired. With conventional T2-weighted spin echo techniques, focal signal loss within the cerebrospinal fluid (CSF) is common. This is due to the strong pulsatile nature of CSF in the thoracic region, which is also present in the cervical spine. Fast spin echo T2-weighted scans suffer substantially less from this problem and are routinely used for sagittal imaging. However, axial T2-weighted scans suffer from CSF flow artifacts regardless of specific technique (conventional or fast). Thus, gradient echo T2-weighted scans are routinely used for axial imaging in the thoracic spine.

Although not strictly "normal," one finding (indicative of a prior diagnostic exam) that can still be seen in older patients deserves comment: the presence of residual Pantopaque (iophendylate) from a myelogram performed before 1990. Pantopaque was an early contrast agent used for myelography. It is no longer used in part because of the high incidence of arachnoiditis after the exam. Pantopaque is an oily, non-water-soluble substance. It was not uncommon for a small amount to be left within the thecal sac after completion of a myelogram. This persists indefinitely. Currently, on MRI, Pantopaque is still occasionally seen in older patients either free within the thecal sac or trapped within a root sleeve or scar. It is easily recognized because of its appearance on MRI (typically a small globule) with high signal intensity on T1-weighted scans and low signal intensity on T2-weighted scans. Correlation with conventional x-ray films is recommended because Pantopaque is extremely x-ray dense.

**CONGENITAL OR DEVELOPMENTAL ABNORMALITY**

Butterfly vertebrae have concave superior and inferior end plates with a central osseous defect. In some instances, this is an incidental finding of no clinical significance. However, butterfly vertebrae can be associated with congenital abnormalities such as diastematomyelia, necessitating close review of images.

A lateral meningocele is produced by a protrusion (laterally) of the dura and arachnoid through an enlarged neural foramen. The adjacent pedicles and lamina may be thinned, and the dorsal surface of the vertebral body scalloped. The vast majority of lateral meningoceles (85%) are seen in neurofibromatosis. Thoracic paraspinal masses, when present in neurofibromatosis, are more likely to be meningoceles than neurofibromas. Most lateral meningoceles are right sided, occur in a single foramen, and are seen in the upper thoracic spine (T3-7). Lateral meningoceles are typically asymptomatic. They are easily characterized and diagnosed by MRI, with CSF signal intensity on all pulse sequences.

Neuroenteric cysts are an embryologic remnant. During early embryonic development, a temporary structure (the canal of Kovalevsky) connects the amnion and the primitive yolk sac. Persistence of this canal after embryologic development leads to a fistula from gut, through the vertebral bodies and spinal cord, to the dorsal skin. Persistence of only a portion of the canal is believed to be the origin of mesenteric cysts, enteric diverticula,
neuroenteric cysts, diastematomyelia, and spina bifida. Neuroenteric cysts are by definition enteric lined cysts that lie within the spinal canal. There can also be a component outside the canal. Neuroenteric cysts are usually ventral in location to the spinal cord and are most frequently seen at the cervicothoracic junction and conus medullaris. There are frequently associated vertebral body anomalies. The imaging appearance on MRI is varied depending on the blood and protein content, viscosity, and pulsatility. Included in the differential diagnosis is an arachnoid cyst. However, arachnoid cysts are isointense to CSF on all pulse sequences and not associated with vertebral body anomalies.

Epidural lipomatosis is the result of excessive fat deposition in the epidural space. It is seen in morbid obesity, chronic steroid use, and Cushing’s disease. Sixty percent of cases occur in the thoracic spine and 40% in the lumbar spine. In extreme cases, patients can be symptomatic; pain and weakness result from compression of the thecal sac by overabundant fat.

**INFECTION**

- **Osteomyelitis**

Vertebral osteomyelitis is often insidious; nonspecific symptoms make diagnosis difficult. Delay in treatment, however, dramatically increases morbidity. In children, osteomyelitis occurs after hematogenous spread of bacteria to the vascularized intervertebral disk. However, in adults, the infection is the result of hematogenous spread to the more vascular end plate, with the disk itself involved secondarily. Plain x-ray films are frequently unremarkable until late in the disease. MRI is the modality of choice for diagnosis; sensitivity is higher for MRI than for radionuclide scintigraphy. Findings on MRI include abnormal low signal intensity on T1 weighted scans and high signal intensity on T2-weighted scans within the vertebral body. These signal abnormalities are due to edema and inflammatory changes. There is typically a paraspinal and epidural soft tissue mass, which enhances after contrast administration. The longer the delay in diagnosis, the greater is the size of the associated abnormal soft tissue. Specific diagnosis is usually possible because of the presence of an irregular very high signal intensity area within the disk space on T2-weighted scans corresponding to fluid. Involvement of the intervening disk space distinguishes this disease (infection) from vertebral metastases.

- **Tuberculous Spondylitis**

Tuberculous spondylitis follows a more indolent clinical course than pyogenic infection. It is uncommon in the United States except among immigrants (specifically those from Southeast Asia and South America) and immunocompromised patients. From an imaging perspective, abnormal marrow signal intensity is seen within two or more adjacent vertebral bodies, with accompanying cortical bone destruction and abnormal extradural soft tissue. Distinguishing features from pyogenic infection include involvement of three or more levels (50% of cases), "skip" lesions, relative sparing of the disk, and a disproportionately large soft tissue mass. Tuberculous spondylitis often spreads along the anterior longitudinal ligament involving multiple contiguous vertebral bodies. The
extradural component is typically prevertebral in location, but can extend into the spinal canal. In longstanding disease, there can be extensive bone destruction, a gibbous deformity (vertebral collapse with anterior wedging), and cord compression (resulting from angulation or the soft tissue mass). Computed tomography (CT) clearly depicts the extensive bone destruction and soft tissue (paraspinous) mass. MRI, however, offers superior depiction of both the vertebral and paravertebral involvement. As medical treatment begins to take effect, there is a return to normal in signal intensity (on both T1- and T2-weighted scans) of the vertebral bodies and a decrease in the abnormal enhancement of paravertebral soft tissue.

- **AIDS-Related Infection**

AIDS-related infections involving the thecal space in the thoracic area may present as polyradiculopathy or myelopathy. The cause is generally viral but can be direct or indirect in nature. Cytomegalovirus, herpes simplex type 2, varicella-zoster, and toxoplasmosis have been implicated and represent "direct" disease as a result of viral infection. "Indirect" effects include postinfectious demyelination and parainfectious vasculitis. The differential diagnosis should include neoplasia and specifically lymphoma.

**NEOPLASTIC DISEASES IF DORSAL SPINE**

- **Metastases to Bone**

T1-weighted scans are generally the most useful for detection of vertebral body metastases because of their high sensitivity to disease and intrinsic high signal to noise ratio (and thus good image quality). Malignant lesions, with increased cellularity, are low signal intensity on T1-weighted scans and thus quite distinct from the normal high signal intensity marrow (Fig. 1). After contrast injection, metastatic lesions usually enhance and are thus less clearly seen unless fat suppression is used. Postcontrast scans can, however, display more effectively the soft tissue extent of disease and canal compromise (Fig. 2), although the latter is often also clearly seen on T2-weighted scans. Lytic and blastic lesions appear distinct from one another; the latter is very low signal intensity on T1-weighted scans (Fig. 3). With tumors that spread via the lymphatics (e.g., carcinoid), it is important to scrutinize the off-midline sagittal images (and axial scans) for retroperitoneal lymphadenopathy.

In regard to sensitivity, it is well established that MRI is overall more sensitive than radionuclide bone scanning for metastatic disease. MRI may detect lesions despite a normal bone scan. Furthermore, radionuclide bone scans suffer from lower specificity. Degenerative changes, infection, and fractures can all cause a false-positive bone scan. MRI better discriminates between benign and malignant processes.

MRI has replaced myelography in most institutions for the assessment of cord compression by epidural metastatic disease as a result of high sensitivity and low morbidity. Myeloma, prostate, and renal cell carcinoma all have a propensity to develop epidural metastatic disease. However, the highest incidence of epidural metastatic disease is with lung carcinoma; this is the most common cause of metastatic disease to the vertebral column.
terms of symptoms, there is a prodromal phase with central back pain at the level of
disease involvement. This is followed by a compressive phase with neurologic deficits,
which begin with motor impairment (resulting from anterior cord compression). In lesions
causing compression of the conus, autonomic dysfunction may occur without sensory or
motor deficits. Compression of the thecal sac and cord can occur from any direction,
 anterior or posterior (Figs. 7-4 and 7-5) or lateral (Figs. 7-6 and 7-7), necessitating close
image inspection and acquisition of two perpendicular planes (typically sagittal and axial).

1. Metastatic disease (from lung carcinoma), with mild anterior compromise of the thecal
sac. A, On the sagittal T2- weighted scan, the posterior margins of T7 and T8 bulge in a
convex manner posteriorly, encroaching on the spinal canal. The signal intensity of the
marrow (of T7 and T8) is misleading on this fast spin echo scan (obtained without fat
suppression), appearing isointense with adjacent normal vertebral bodies. Fast T2-
weighted spin echo scans should be acquired with fat saturation to improve their sensitivity
to bony metastatic disease. B, The T1-weighted scan clearly demonstrates the metastatic
involvement of T7 and T8. There is replacement of normal marrow in these vertebral
bodies by metastatic disease, which demonstrates substantially lower signal intensity. A
small focus of metastatic disease (B, arrow) is also present in the anterior superior quadrant of T11.

2. Vertebral metastatic disease (from lung carcinoma), with epidural extension and severe cord compression. A, The fast spin echo T2-weighted midline sagittal image demonstrates compromise of the thecal sac by abnormal soft tissue posteriorly and anteriorly. B, The corresponding T1-weighted sagittal scan, although not demonstrating as clearly the interface between cerebrospinal fluid (CSF), cord, and soft tissue, clearly depicts the involvement of the posterior portion of both T10 and T11 by metastatic disease. Both the vertebral and epidural lesions demonstrate inhomogeneous enhancement on the postcontrast T1-weighted sagittal image (C). Contrast enhancement decreases the conspicuity of the vertebral body involvement by metastatic disease but improves the visualization of canal compromise by soft tissue.
3. Metastatic disease (from prostate carcinoma) with both lytic and blastic lesions. Metastatic vertebral lesions most often demonstrate decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted scans. Blastic metastases, however, may remain low signal intensity on T2-weighted images. A, The fast spin echo T2-weighted scan, obtained with fat suppression, reveals at least two vertebral body lesions with slight high signal intensity (asterisks). Several low signal intensity lesions are also evident (white arrows). B, The T1-weighted scan better depicts the widespread extent of metastatic disease; the lytic lesions are seen as gray or intermediate low signal intensity (slightly lower in signal intensity than the intervertebral disks), and the blastic lesions as very low signal intensity (almost black). One of the lesions also contains methemoglobin, with abnormal high signal intensity on the T1-weighted scan. C, The axial section through this lesion reveals abnormal low and high signal intensity. D, A lower axial section
demonstrates a focal vertebral body lesion, with additional metastases seen in the ribs (black arrows). A common mistake in film reading is to examine only the midline sagittal scan for metastatic disease. The entire bony skeleton visualized on the scan should be inspected for the presence of metastatic disease, when clinically suspected.

- **Leptomeningeal Metastases**

Leptomeningeal metastases can be seen with central nervous system (CNS) tumors (including specifically glioblastoma, ependymoma, medulloblastoma, and pineal tumors) as well as with non-CNS tumors (most commonly lung carcinoma, breast carcinoma, melanoma, and lymphoma). The clinical presentation is varied and includes back pain, leg pain, headache, cranial and spinal nerve deficits, and gait disturbance. The gold standard for diagnosis is CSF cytology. However, this may require multiple samples and a large volume of CSF. The diagnosis of leptomeningeal metastases by CT is based on visualization of nodular filling defects within the CSF and clumping of nerve roots. The advent of high-quality contrast-enhanced spine MRI provided a major advance in the imaging diagnosis of leptomeningeal metastases. MRI is markedly more sensitive than CT for the detection of leptomeningeal metastases when intravenous contrast is used. Small and large enhancing nodules, direct invasion of the cord by metastases, and seeding along the cord surface or exiting nerve roots are all well visualized.

- **Hematologic Neoplasia**

Spinal involvement is seen in lymphoma (Fig. 8) in 15% of cases. Paravertebral, vertebral, and epidural lesions all occur. Spinal lymphoma is most commonly caused by local spread from retroperitoneal nodes and is thus paravertebral in location. Isolated epidural lesions do occur as a result of hematogenous spread or spread from epidural lymphatics. Epidural disease in lymphoma frequently results in clinically significant cord compression. The appearance of epidural disease is not specific for lymphoma but merely reflects the characteristics of an epidural soft tissue mass. On T1-weighted scans, a lymphomatous epidural mass is isointense to slightly hyperintense to the spinal cord and on T2-weighted scans hyperintense to cord. Contrast enhancement is typically homogeneous. Vertebral involvement is also nonspecific in appearance, sharing that of metastatic disease from many causes with inhomogeneous low signal intensity on T1-weighted scans and intermediate to high signal intensity on T2-weighted scans.
4. Cord compression (in the upper thoracic spine) by expansile bony metastatic disease (from lung carcinoma), revealed on an emergency magnetic resonance imaging scan. A, The fast spin echo T2-weighted scan with fat suppression reveals extensive abnormal high-signal-intensity bony metastatic disease involving the vertebral bodies and in the upper thoracic spine the spinous processes (posterior elements) as well. The cerebrospinal fluid space surrounding the cord is obliterated, with compression from abnormal soft tissue both anteriorly and posteriorly. B, The T1-weighted scan clearly depicts the extent of bony metastatic disease but provides a relatively poor view of the canal compromise. Unless intravenous contrast is administered, cord compression is seen best on fast spin echo T2-weighted scans.
5. Cord compression at multiple levels from metastatic colon carcinoma. Just because compression is demonstrated at one level, inspection of the film and the search for other areas of involvement (and possible canal compromise) should not be discontinued. Metastatic disease is typically widespread; therefore, presentation with more than one discrete level of canal compromise is not uncommon. Fast spin echo T2-weighted (A) and conventional T1-weighted (B) sagittal scans show severe canal compromise as a result of metastatic involvement of two adjacent vertebral bodies in the upper thoracic spine. However, not to be overlooked is significant anterior cord compression at a level two bodies higher, best seen on the T2-weighted scan.
6. Lateral cord compression resulting from pedicle involvement by metastatic disease. A, The midline sagittal T1-weighted scan reveals only mild anterior compression of the thecal sac by metastatic disease (which involves both the vertebral body and spinous process). Involvement of the superoposterior quadrant of the adjacent lower vertebral body, with a normal intervening disk space, favors the diagnosis of neoplastic disease as opposed to infection. However, it cannot be concluded from the midline sagittal scan alone that significant canal compromise is not present. Such may occur by involvement of the pedicles laterally, as shown in an adjacent slice (B). Lateral thecal sac compromise was confirmed on the axial scan (not shown).
7. Metastatic disease from prostate carcinoma, illustrating the importance of both sagittal and axial scans for routine evaluation of canal compromise. On the basis of the sagittal T2-weighted fast spin echo scans (A and B), there is significant cord compression (by abnormal posterior soft tissue) at T4 (only). The lack of fat suppression, however, makes assessment of the extent of bony metastatic involvement difficult. The multiplicity of lesions is readily appreciated from the sagittal T1-weighted scans (C and D). An enlarged lymph node (white arrow), involved by metastatic disease, is also noted anterior to T11. Although the T10 vertebral body is involved in its entirety by metastatic disease, there does not appear to be any substantial canal compromise at this level (on the basis of the sagittal scans alone). However, the axial gradient echo scan at T10 (E) demonstrates substantial anterior and lateral compromise of the canal.
8. Lymphoma. A, The T2-weighted midline sagittal image reveals a large epidural mass at the T8-9 level posterior to and compressing the cord. The lesion is slightly hyperintense to the cord but of lower signal intensity than cerebrospinal fluid (CSF). B, The axial postcontrast T1-weighted image reveals moderate homogeneous enhancement of the lesion (black arrow), with the cord displaced and compressed anteriorly (and to the right). On the precontrast axial T1-weighted scan (not shown), the cord and mass were isointense and could not be distinguished.

Leukemia is the most common malignancy of childhood and the ninth most common in adults. The disease arises in lymphoid tissue and bone marrow and from a simplistic point of view represents a malignant proliferation of hematopoietic cells. A common symptom is bone pain caused by pressure from rapidly proliferating cells. Bone involvement is most often diffuse but can be focal. The latter is most common in acute myelogenous forms. The CNS serves as a sanctuary for the disease during chemotherapy; thus, the CNS is a frequent site of relapse.

Multiple myeloma is caused by a neoplastic overgrowth of plasma cells. The peak incidence is from 50 to 70 years of age. Vertebral involvement is most common in the thoracic region. MRI is far more sensitive than either plain x-ray films or radionuclide bone scans for disease detection. The most common appearance on MRI is that of diffuse marrow infiltration (Fig. 9).
Another not uncommon pattern is that of nodular deposits surrounded by normal marrow. As with other hematologic neoplasias, paravertebral and epidural soft tissue masses can also be seen (Fig. 10).

9. Multiple myeloma with diffuse marrow involvement and an epidural mass at T6. An epidural soft tissue mass is noted on the midline sagittal T2-weighted scan (A). The mass is posterior to the cord, displacing it anteriorly. B, The precontrast T1-weighted scan depicts both the mass and the diffuse involvement of vertebral marrow. The latter has abnormal low signal intensity. Diffuse marrow involvement may elude detection if the marrow signal intensity is not compared with a standard, such as that of the normal intervertebral disks. On T1-weighted scans, normal marrow should be hyperintense to the intervertebral disk. The heterogeneity of the marrow signal intensity on both the T-1 and T2-weighted scans in this patient confirms the widespread metastatic involvement. C, After contrast administration, both the marrow and the epidural mass demonstrate substantial enhancement. The latter improves markedly the depiction of cord compression by the mass.
10. Multiple myeloma with prevertebral and epidural extent. A, On the axial gradient echo T2-weighted scan, a high-signal-intensity prevertebral soft tissue mass is noted. The mass partially encases the aorta. The cord is displaced posteriorly and compressed by abnormal epidural soft tissue. B, On the precontrast T1-weighted scan, the prevertebral mass is clearly seen, but the epidural mass and cord have similar signal intensity and are difficult to separate. C, Postcontrast, both the prevertebral and epidural (white arrow) portions of the mass (myeloma) enhance, improving differentiation from the cord (black arrow), which lies compressed posteriorly.

- **Astrocytoma/Ependymoma**

The majority of intramedullary spinal cord tumors are either astrocytomas or ependymomas. Astrocytomas are more common in children and ependymomas more common in adults. MRI cannot differentiate an astrocytoma from an ependymoma, although certain imaging features favor one or the other. Involvement of the entire width of the cord, with homogeneous high signal intensity on T2-weighted scans, favors an astrocytoma (Fig. 11). Extensive cord involvement, extending over three or more vertebral segments, also favors an astrocytoma. A small nodular lesion (especially with a cystic component) is more likely to be an ependymoma. Three fourths of all spinal astrocytomas occur in either a cervical or thoracic location.

- **Neurogenic Tumors (Nerve/Nerve Sheath Origin Tumors)**

The majority of paraspinal lesions in the thoracic region are neurogenic tumors. These tumors are also the most common cause of a posterior mediastinal mass. In adults, schwannomas and neurofibromas are most common. These two tumors have similar imaging characteristics. In young children, neuroblastoma is most common.

In the radiologic literature, the term schwannoma has been used interchangeably with neurinoma and neurofibroma. Schwannomas arise from the Schwann cells of the nerve root sheath. Thus, these lesions are seen, at dissection, to be extrinsic (eccentric) to the nerve root. On MRI, schwannomas are hypointense on T1-weighted images to the cord and hyperintense on T2-weighted images (Fig. 12). On the latter type of scan, schwannomas are also often heterogeneous in appearance; high-signal-intensity areas correspond to small cysts. Enhancement is typically heterogeneous and often more intense peripherally.

Neurofibromas are distinguished from schwannomas by the presence of abundant connective tissue and nerve cells. Neurofibromas enlarge the nerve itself. Neurofibromas are usually associated with neurofibromatosis, even when solitary. Homogeneous contrast enhancement makes the diagnosis of a neurofibroma more likely than that of a schwannoma.

Three related but different tumors-neuroblastoma, ganglioneuroblastoma, and ganglioneuroma—are thought to arise from primitive sympathetic neuroblasts (the embryonic neural crest). These are differentiated histologically by the degree of cellular maturation. On imaging, the three tumor types are indistinguishable. Neuroblastoma is a
malignant tumor composed of undifferentiated neuroblasts. Most neuroblastomas arise in the adrenals and the remainder along the sympathetic chain. The clinical prognosis is worse with increasing age of presentation. The prognosis, however, is better with spinal lesions as opposed to abdominal or pelvic lesions. Extradural extension is common with paravertebral lesions. Ganglioneuroblastoma is also a malignant tumor but contains mature ganglion cells in addition to undifferentiated neuroblasts. Ganglioneuroma (Fig. 13) is a benign tumor that contains mature ganglion cells. Ganglioneuromas are more common in adolescents and young adults.

- **Meningioma**

One third of all spinal meningiomas occur in the cervical region and two thirds in the thoracic region. There is a 3:1 female-male incidence. Spinal meningiomas are most often intradural in location but may be extradural. Complete removal can be achieved surgically in 95% of cases. Microsurgical technique is important to minimize neurologic deficits. Despite "complete" removal, 5% recur.
11. Astrocytoma. Sagittal T2- (A) and T1- weighted (B) scans reveal abnormal expansion of the lower cervical and upper thoracic spinal cord. The area involved spans more than three vertebral segments. The lesion is higher in signal intensity than normal cord on the T2-weighted scan and slightly lower in signal intensity than normal cord on the T1- weighted scan. C, Postcontrast there is no enhancement of the mass, which is again demonstrated to be intramedullary in location, expanding the cord to fill the spinal canal.

![Images of sagittal and parasagittal scans showing astrocytoma and paraspinal schwannoma]

12. Paraspinal schwannoma. T2- (A) and T1-weighted (B) parasagittal images reveal a 3.5-cm paraspinal soft tissue mass at T7. The lesion is high signal intensity, but somewhat heterogeneous, on the T2-weighted scan. Extension into the T7-8 neural foramen is also noted. C and D, Postcontrast there is intense enhancement of the lesion. Although by imaging appearance the lesion could be either a schwannoma or a neurofibroma, that enhancement is heterogeneous and more intense peripherally favors a schwannoma.
13. Ganglioneuroma. A, On the T2-weighted sagittal image to the right of midline, a large paraspinal soft tissue mass is noted. The patient is a 2-year-old child who presented with respiratory distress. The mass extends into and widens the T5-6 neural foramen. B, On the axial postcontrast T1-weighted image, the mass is noted to enhance. However, the epidural portion enhances more intensely than the remainder of the lesion (the large paravertebral portion). The thoracic spinal cord is severely compressed and displaced to the patient's left.

14. Meningioma. On precontrast T2- (A) and T1-weighted (B) sagittal scans, a mass is noted within the thecal sac, outlined by cerebrospinal fluid. The flattening and displacement of the cord favor an intradural extramedullary location. Intense enhancement postcontrast (C) improves demarcation of the lesion and places a meningioma first on the list of differential diagnoses. This 48-year-old woman presented with paraplegia and progressive back pain. The lesion was surgically removed.

Meningiomas are isointense to the spinal cord on both T1- and T2-weighted scans. This tumor displays marked contrast enhancement, which can improve lesion identification and
demarcation (Fig. 14). The capping of a meningioma inferiorly and superiorly by CSF is characteristic and demonstrates the lesion to be intradural and extramedullary in location (by far the most common location). On plain film and CT, dense calcification is common.

**VASCULAR AND HEMATOPOIETIC DISEASE (NON-NEOPLASTIC)**

- **Arteriovenous Malformation and Fistula**

An arteriovenous malformation (AVM) is defined as a nidus of pathologic vessels between enlarged feeding arteries and draining veins. This is to be differentiated from an arteriovenous fistula (AVF), in which the arteries drain directly into enlarged veins. Within this group of lesions, three types are described in the spine: dural AVF (the most common), intramedullary AVM, and intradural extramedullary AVF.

Dural AVFs occur along the dorsal aspect of the lower cord and conus (Fig. 15). These feature a single transdural arterial feeder. Dural AVFs are found in elderly men and present with progressive neurologic deficits resulting from venous stasis and infarction.

Intramedullary AVMs occur in young patients and are one cause of intramedullary hemorrhage. They are typically dorsal in location and occur most often in the cervico-medullary region. Multiple feeding vessels lead to a compact vascular plexus, which drains into a tortuous venous plexus surrounding the cord. Intramedullary AVMs present with acute hemorrhagic stroke. The imaging appearance on MRI is that of multiple flow voids within the cord together with enlarged extramedullary feeding vessels (typically anterior to the cord).

Intradural extramedullary AVFs occur in the third to sixth decades. The most common presentation is that of a lesion at the level of the conus but anterior to the cord with supply by the anterior spinal artery. Intradural extramedullary AVFs present with progressive neurologic deficits.

MRI is an important technique for the initial diagnosis of a spinal AVM or AVF. Abnormal large vessels are identified as filling defects on conventional two-dimensional scans. These are best appreciated within the cord on T1-weighted images and within the CSF space on T2-weighted images (see Fig. 15). Small lesions are clearly seen postcontrast because of the enhancement of the large draining veins. Associate cord findings include hemorrhage, edema, and myelomalacia. An important pitfall on image interpretation is that CSF flow-artifacts may mimic an AVM on T2-weighted scans. These artifacts can be prominent on conventional spin echo T2-weighted scans but may on occasion also be present on fast spin echo scans. On x-ray myelography, filling defects may be seen as a result of enlarged vessels and cord atrophy detected, if present. X-ray angiography is used for definitive diagnosis. Selective vessel catheterization, assessing feeding vessels and venous drainage, is performed after initial intra-aortic injection. Spinal AVMs and AVFs are also clearly visualized by contrast-enhanced magnetic resonance angiography, which may with future refinements replace x-ray angiography.
15. Dural spinal arteriovenous fistula. A, On the sagittal T2-weighted scan, the question of abnormal hyperintensity within the lower cord and conus is raised. Immediately posterior to the cord, multiple small serpiginous signal voids are identified, spanning at least two vertebral segments. B, The precontrast T1-weighted scan is normal. C, Postcontrast, abnormal enhancement (arrows) is noted along the dorsal aspect of the cord, confirming the presence of enlarged draining veins. The diagnosis was confirmed surgically. By enhancement of slow flow within dilated veins, contrast administration improves visualization of spinal arteriovenous malformations and fistulas.

- Spinal Cord Ischemia/Infarction

There are many causes for spinal cord ischemia and infarction, including atherosclerosis, vasculitis, embolism, infection, radiation, trauma, and surgery (specifically after abdominal aortic aneurysm resection). Infarction and ischemia typically involve the central gray matter of the cord. Anatomically, the lower thoracic cord and conus are most commonly involved. The artery of Adamkiewicz, typically arising from the 9th to 12th intercostal artery, supplies this region. Blood flow is highest to this section of the cord, given the abundance of gray matter and its higher metabolic need. Thus, it is this region of the cord that is most vulnerable to hypoperfusion.

MRI is, without question, the imaging modality of choice for the diagnosis of spinal cord ischemia and infarction. The extent of abnormality as visualized by MRI correlates well with clinical findings and prognosis. The area involved can be minimal (e.g., just the anterior horns). In severe cases, the entire cord is involved in cross-section. In intermediate cases, both the anterior and posterior horns are involved together with the adjacent central white matter. On T2-weighted scans, abnormal high signal intensity is noted in the involved
region (Fig. 16), corresponding to vasogenic edema in acute and subacute disease. On T1-weighted scans, cord enlargement may be the only finding. Abnormal contrast enhancement of the cord can be present as a result of disruption of blood-cord barrier (secondary to ischemia). There may be associated marrow changes also resulting from ischemia. Differential diagnostic considerations include multiple sclerosis (MS), transverse myelitis, and neoplasia. Recognition of the vascular distribution, both in craniocaudal extent and cross-section, aids in differentiation of spinal cord ischemia/infarction from other disease processes.

- **Hemorrhage**

Subarachnoid hemorrhage may be secondary to a spinal aneurysm or AVM or may originate from a cerebral source. With acute hemorrhage, a moderate increase in the signal intensity of CSF on T1-weighted scans may be observed, obscuring the cord and nerve roots. With subacute hemorrhage, high signal intensity is seen on T1-weighted scans because of the presence of methemoglobin.

Epidural and subdural hemorrhage has many causes, including lumbar puncture, trauma, bleeding diatheses, anticoagulant therapy, vascular malformations, vasculitis, and pregnancy. The signal intensity on MRI is dependent largely on the stage of hemorrhage and dilution by CSF. It can be difficult to identify whether a hemorrhage is epidural or subdural in location. When abnormal high signal intensity is seen in the epidural or subdural space, two other disease processes should be considered in the differential diagnosis. Angiolipomas are rare benign tumors composed of lipocytes and abnormal blood vessels. The latter cause hyperintensity on T2-weighted scans on the basis of slow flow. These tumors are usually epidural in location and occur in the midthoracic region. Angiolipomas can cause bone erosion, pathologic fractures, and cord compression. The other consideration should be extradural lipomatosis, although with this disease process the abnormal soft tissue should be readily identifiable as fat (by inspection of both T1 and T2-weighted scans).

**TRAUMA**

Burst fractures are the most common traumatic bone injury encountered in the thoracic spine. Burst fractures are caused by an axial loading injury. Vertical compression forces the nucleus pulposus into the vertebral body, with radial displacement of fragments. Burst fractures are most common from T9 to L5. The injury is typically limited to one vertebral body, but associated injuries are common. Neurologic deficits occur as a result of the retropulsed fragments. CT is often used for initial evaluation. MRI detects associated cord (edema and hemorrhage) and ligamentous injuries, which are not clearly seen by CT.
16. Spinal cord infarction. The mid- and lower thoracic cord is slightly expanded and has abnormal high signal intensity. These findings correspond to vasogenic edema. Infarcts can be limited to a few vertebral segments or can be very extensive as in this case. With sufficient time, cord atrophy will occur, although the abnormal high signal intensity on T2-weighted scans can persist (as a result of gliosis).

- Extramedullary Hematopoiesis

Extramedullary hematopoiesis is a compensatory response to insufficient red blood cell production by bone marrow. It is seen in thalassemia, hereditary spherocytosis, and myelosclerosis. Favored sites of involvement include the spleen, liver, and lymph nodes. Thoracic involvement is rare and usually asymptomatic. Thoracic involvement is seen on imaging as a paraspinal mass resulting from extrusion of proliferating marrow from vertebral bodies into a subperiosteal location. Intraspinal lesions can occur as a result of extrusion of bone marrow or development of marrow from embryonic hematopoietic rests. Intraspinal involvement can cause cord compression. The appearance on MRI of thoracic extramedullary hematopoiesis is that of multiple, smoothly margined, paraspinal masses.
without bone erosion (Fig. 17). The masses have the signal intensity of marrow on all pulse sequences. The differential diagnosis should include lymphoma and metastatic disease.

DEGENERATIVE DISK AND BONY DISEASE

In the thoracic spine, disk herniations are most common at the lower four interspaces, where the spine is more mobile. Thoracic disk herniations are less common than either cervical or lumbar herniations. The clinical presentation is often not clear-cut. Symptoms include back pain, paresthesias, and motor weakness. On high-quality MRI images, small thoracic disk herniations are clearly seen (Fig. 18). MRI also clearly demonstrates mass effect on the cord, when present, and contour deformities of the cord. As with cervical disk herniations, part of the abnormality may actually represent dilated epidural venous plexus. On contrast-enhanced images, the dilated, engorged epidural venous plexus above and below the herniated disk (Fig. 19) is readily identified.

In addition to dedicated thoracic spine images, a high-quality large field of view localizer should be acquired, on which the dens can be identified, to define the level of disk herniation correctly. The use of MRI markers can assist in correct level identification. Commonly used markers include vitamin E capsules (an oily vitamin that has high signal intensity on T1-weighted scans) or oil (such as Johnson's baby oil) in a strip of intravenous tubing.

ABNORMAL ALIGNMENT

Scoliosis is defined as a lateral curvature of the spine. Ninety percent of cases are idiopathic with no underlying cause. Idiopathic thoracic scoliosis is more common in females, and the thoracic curvature is typically convex to right (with an S-shaped curve). Progression beyond 50 degrees necessitates surgery.
17. Extramedullary hematopoiesis with severe cord compression. The patient is 32 years of age with thalassemia and presents clinically with progressive paraplegia. A, The T1-weighted coronal image demonstrates large bilateral lobulated paraspinal masses in the upper and midthoracic regions. B, On the T1-weighted midline sagittal view, large intraspinal masses with resultant severe cord compression are identified at the T6 to T8 levels. The soft tissue masses lie within the same space as the thoracic epidural fat. Also noted is a generalized decrease in signal intensity of the thoracic vertebral bodies. C, The corresponding fast spin echo T2-weighted sagittal image confirms the abnormal intraspinal soft tissue masses. The lesions remain relatively low in signal intensity on the T2-weighted scan. Abnormally increased signal intensity compatible with edema or gliosis is identified within the compressed thoracic spinal cord. On postcontrast scans (not shown), there was mild, homogeneous enhancement of the paraspinal and the intraspinal lesions.
18. Thoracic disk herniation. A, The midline sagittal fast T2-weighted scan reveals mild anterior indentation of the thecal sac (arrow) at a midthoracic level. B and C, Axial gradient echo T2-weighted scans reveal small left paracentral disk herniations at this level and two levels below. It cannot be determined, however, whether these lesions are acute or chronic in nature. Attention to detail and high-quality images are necessary to diagnose thoracic disk herniations because these are often very small in size (despite being clinically symptomatic).

19. Thoracic disk herniation demonstrating utility of contrast administration. The patient is a 31-year-old woman with bandlike paresthesias in the midthorax after an automobile accident. A, The T2-weighted scan reveals anterior compression of the thecal sac at T7-8. Abnormal soft tissue can be noted on both the T2-weighted and the precontrast T1-
weighted (B) scans. C, Contrast use permits identification of dilated epidural venous plexus and granulation tissue surrounding the disk herniation (arrow). In comparing the pre- (D) and postcontrast (E) axial scans, enhancement aids, in particular, identification of the interface between the disk (arrow) and the thecal sac.

Ten percent of thoracic scoliosis can be attributed to congenital, neuromuscular, or posttraumatic causes. In the congenital category are both vertebral anomalies (butterfly vertebral body and hemivertebra) and abnormalities of the cord. The latter include Chiari malformations, hydrosyringomyelia, diastematomyelia, and spinal cord neoplasm. Cerebral palsy is the primary neuromuscular cause and leads to a C-shaped curve. Posttraumatic causes include fractures, old osteomyelitis, surgery, and radiation therapy. MRI is the imaging modality of choice for study of atypical or progressive scoliosis. In a patient with scoliosis, coronal images are particularly useful in conjunction with sagittal images. Plain x-ray film are used for quantitation of the curvature (degree) and monitoring of progression.

**MULTIPLE SCLEROSIS**

MS lesions of the thoracic cord are clearly seen on high-quality MRI images, with no difference in appearance than that described for the cervical spine. In acute disease, there can be focal cord swelling, edema (limited to a focal region in both cross-section and craniocaudal extent and best seen on T2-weighted images), and abnormal contrast enhancement (as a result of blood-spinal cord barrier disruption seen on contrast enhanced T1-weighted images). Chronic lesions can be identified on T2-weighted scans because of focal cord atrophy and gliosis (Fig. 20).
20. Multiple sclerosis (inactive disease). Three short-segment high-signal-intensity lesions (asterisks) are noted within the thoracic cord on a fast spin echo T2-weighted scan. Cord atrophy is noticeable at the level of the highest lesion, determining it to be chronic in nature (with the abnormal signal equating to gliosis). Neither of the lower lesions causes cord expansion, making it unlikely that either represents active disease. The lack of contrast enhancement (images not shown) confirmed the chronic nature of disease in this patient.
References

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INTRODUCTION

The lumbar spine consists of five lumbar segments (vertebral bodies), five (fused) sacral segments, and the coccyx. Each intervertebral disk is composed of a central gelatinous core (the nucleus pulposus, which is high signal intensity on T2-weighted images) surrounded by dense fibrous tissue (the annulus fibrosus, which is low signal intensity on T2-weighted images). The bony elements of the lumbar spine include the pedicles, transverse processes,
articular pillars (pars interarticularis, superior and inferior articular facets), laminae, spinous processes, and vertebral bodies. The facet joints are diarthrodial (synovial lined) and richly innervated. On axial imaging, the superior articular facet forms a "cap" anterolaterally with the inferior articular facet posteromedial and connecting to the lamina. The ligamentum flavum extends from the anterior aspect of the upper lamina to the posterior aspect of the lower lamina. The epidural venous plexus is prominent in the lumbar spine. In regard to important dermatomes (for clinical diagnosis with a disk herniation), L4 innervates the medial big toe, L5 the midfoot, and S1 the little toe.

In the sagittal plane, the conus can be seen to terminate between L1 and L2. The posterior longitudinal ligament lies immediately posterior to the vertebral bodies and anterior to the thecal sac. Normal dimensions for the posterior longitudinal ligament are 1-mm thickness (anteroposterior) and 5-mm width (left to right). The facet joints of the upper lumbar spine are oriented in the sagittal plane. Those of the lower lumbar spine are oriented more in the coronal plane. On off-midline sagittal (parasagittal) images, the dorsal root ganglion (and ventral root) can be seen within the superior portion of the neural foramen. Parasagittal images are used to evaluate foraminal stenosis. In regard to the margins of the foramen, the disk and vertebral body lie anteriorly, the pedicles superiorly and inferiorly, and the facet joints posteriorly. On axial imaging, the margins of the bony (spinal) canal consist of the vertebral body anteriorly, the pedicles laterally, and the lamina posteriorly.

On T1-weighted spin echo images, normally hydrated (nondegenerated) disks are slightly hypointense to vertebral marrow. The normal ligamentum flavum is clearly seen, with intermediate signal intensity. Slice thickness should be no greater than 4 mm in the sagittal plane and 3 mm in the axial plane. It is important that a coronal saturation slab be placed anteriorly to decrease artifacts (from the motion of structures anterior to the spine), which would otherwise degrade the images. Saturation of anterior structures is equally important on T2 weighted images in the lumbar spine (and on both types of scans in the cervical and thoracic regions as well). The disks are best visualized in the axial plane when the slices are angled to be parallel to each disk space. Fast spin echo has replaced conventional spin echo technique for T2-weighted imaging of the lumbar spine, and such scans are clinically valuable in both the sagittal and axial planes. Fat saturation is advocated (for fast spin echo T2-weighted scans), and when used normally, hydrated (nondegenerated) disks will be markedly hyperintense to vertebral marrow. In the sagittal plane in adults, a central horizontal band of low signal intensity is typically noted (the "intranuclear cleft") within the intervertebral disk as a result of fibrous transformation.

Surface coils are used to image the lumbar spine. Today these are often an integral part of the patient table. The signal received from the body falls with distance from the surface coil. This situation is quite different from that with cylindrical coils (such as those used for imaging the head), which are specifically designed to achieve homogeneous signal intensity across the entire field of view. Because of the use of a surface coil in lumbar imaging, superficially located structures (close to the coil) will have artifactual high signal intensity. In routine clinical practice, the window and center for the image are chosen to adequately display the spinal canal; thus, posterior structures (soft tissue) are obscured (because of marked hyperintensity). If it is important to view the posterior soft tissues (e.g., to rule out
an abscess after surgery), then the images should be rewinded specifically for these structures. On some magnetic resonance image (MRI) scanners, the images can be normalized with postprocessing software. The aim is to attenuate signal from tissues close to the coil and thus provide more homogeneous signal intensity across the field of view.

The injection of contrast media (specifically, a gadolinium chelate) plays an important role in lumbar imaging, primarily because of the large population of post-surgical disectomy patients presenting with recurrent pain. Normal enhancing structures include the epidural venous plexus (also known as Batson's plexus), the basivertebral vein, and the dorsal root ganglion. The capillaries of the epidural venous plexus have nonfenestrated endothelium, which confines the contrast to the intravascular space. The basivertebral vein is commonly visualized on midline postcontrast sagittal images, running from the center of the vertebral body posteriorly. The endothelium of the dorsal root ganglion is fenestrated, like that in muscle and marrow, permitting contrast to enter the interstitial space. Enhancement of the dorsal root ganglion is only moderate in degree. The most common indication for contrast use in the lumbar spine is for the differentiation of scar from disk in the postoperative patient. On scans obtained within 20 minutes after contrast injection, scar enhances whereas recurrent (or residual) disk herniation does not. On precontrast scans, scar and disk material have similar signal intensity; differentiation is not possible. Contrast injection can also be beneficial in the more general population with low back pain but without previous surgery. Contrast use improves definition of the disk-thecal sac interface, permits identification of the epidural venous plexus and (de novo) scar, and improves visualization of the neural foramina. Contrast injection is recommended in patients with a high clinical suspicion of intradural or soft tissue extradural involvement by neoplastic disease. Disease involving the spinal cord (in particular neoplasia, ischemia, and demyelinating disease) is often better evaluated with the addition of postcontrast scans. Contrast use is mandatory when infection is suspected because extensive, active disease can be missed on precontrast scans.

The lumbar spine undergoes a marked change in appearance on MRI during the first year of life. Changes occur more gradually thereafter, with distinct differences in appearance between the young adult and the elderly. There is absence of the normal adult lumbar lordosis in the infant. Before 1 month of age, the ossification center within the vertebral body has low signal intensity on both T1- and T2-weighted scans. A distinct band with slight high signal intensity on T1 -weighted images within the ossification center corresponds to the basivertebral venous plexus. The cartilaginous end plate has higher signal intensity on T1-weighted scans than paraspinous muscle and has high signal intensity on T 2 weighted scans. The disk itself is thin, isointense on T1- weighted images to paraspinous muscle, and very high signal intensity on T2-weighted images. The anteroposterior dimension of the ossification centers is less than that of the intervertebral disks.

From 1 to 6 months of age, the ossification center has low to intermediate signal intensity on T1-weighted images and is isointense with the end plates. On T2- weighted scans, the cartilaginous end plates have higher signal intensity than muscle or the ossification center.
The intervertebral disk is low signal intensity on T1-weighted scans and high signal intensity on T2-weighted scans.

By 7 months of age, the spine attains a more adult appearance. The ossification center is more rectangular and is now hyperintense to muscle on T1-weighted scans. On both T1- and T2-weighted scans, the signal intensity of the cartilaginous end plate is similar to that of the ossification center. The intervertebral disk is low signal intensity on T1-weighted scans (isointense to muscle) and high signal intensity on T2-weighted scans.

The vertebral body contains both red and yellow marrow; the relative proportion of the two determines the signal intensity on MRI. Red (hematologically active) marrow has lower signal intensity on T1-weighted scans than yellow (fatty) marrow. The change in signal intensity from the infant to the young adult to the elderly reflects the conversion from red to yellow marrow. With increasing age, both diffuse and focal replacement of red marrow by yellow marrow occurs. Focal changes (focal "fat") are more common near the end plates perhaps because of decreased vascularity and earlier marrow conversion in this location.

At birth, the conus should terminate above the L3-4 level. Termination below this level is abnormal regardless of age. By 2 months of age, the conus should lie in the adult location: L2-3 or above. The conus lies, on average, at the L1-2 level in children and adults.

**CONGENITAL DISEASE (INCLUDING STRUCTURAL ANOMALIES)**

- **Transitional Vertebrae**

Transitional vertebrae are common at the lumbosacral junction (occurring in 4% to 8% of the population). By definition, there is articulation or fusion of an enlarged transverse process of the lowest lumbar segment to the sacrum. The articulation or fusion can be unilateral or bilateral. On sagittal images, the body of a transitional segment may be square (normal configuration for lumbar), wedge shaped (like the sacral segments), or intermediate in shape. The presence of a transitional vertebra on MRI is readily apparent if one is aware of the following key. Because numbering of the lumbar vertebrae is critical in patients being examined for possible disk surgery, close attention should be paid to the curve formed by the anterior margin of the lumbar vertebral bodies and sacrum. There should be a smooth curve with the apex anteriorly encompassing the lumbar vertebrae. This should then reverse at L5-S1 to a smooth curve with the apex posteriorly encompassing the sacral segments. Any variation from these two smooth curves indicates the presence of a transitional vertebra; plain film correlation is necessary to determine whether the body in question is lumbarized or sacralized. Transitional vertebrae are a known cause of back pain. There is decreased mobility at the affected level and increased mobility and stress at the interspace immediately above.
• Spina Bifida Occulta
  
  (Occult Spinal Dysraphism)

In spina bifida occulta, skin covers a developmental anomaly involving incomplete midline closure. On physical exam, there is no visible neural tissue or mass. Spinal bifida occulta includes diastematomyelia, dermal sinus tracts, fibrous bands, dermoids, neurenteric cysts, and lipomas. This class of congenital malformations is distinct (separate) from meningoceles and myelomeningoceles. Spina bifida occulta is not associated with the Chiari type II malformation.

Figure 1. Sacral agenesis. A, On the midline sagittal T2-weighted scan, the cord is seen to terminate at the L1-2 level. The L5 vertebral body is dysplastic. Only a portion of S1 is present (the remainder of the sacrum is absent). B, On the midline sagittal T1-weighted scan, the contour of the cord terminus is noted to be unusual, with the cord ending in a wedge shape (the dorsal aspect extends further caudally). There is abnormal signal
intensity centrally within the cord, low signal intensity on the T1-weighted scan, and high signal intensity on the T2-weighted scan, suggesting a small syrinx. C, The axial T1-weighted scan just above the level of the cord terminus confirms the presence of a dilated central canal (hydromyelia). This 21-month-old infant presented with lower extremity sensory and motor deficits.

- **Caudal Regression (Sacral Agenesis)**

In caudal regression, there is absence of sacroccocygeal vertebrae with or without lumbar involvement. The level of regression is below L1 in most cases. Agenesis is limited to the sacrum in about half of all cases (Fig. 1). Associated anomalies include cord tethering, renal dysplasia, pulmonary hypoplasia, and neuromuscular weakness or paralysis. Caudal regression is associated with maternal diabetes. On MRI, a wedge-shaped cord terminus is seen in about half of patients, with the dorsal aspect extending further caudally than the ventral aspect. MRI clearly depicts the level of regression, presence of stenosis (in the area of vertebral absence), and associated structural anomalies.

- **Myelomeningocele**

Spina bifida is defined as incomplete closure of the posterior bony elements. The contents of the spinal canal can extend through this defect (with tethering of the cord). A meningocele contains dura and arachnoid. Neurologic deficits are uncommon with a simple meningocele. A myelomeningocele contains neural tissue within the expanded posterior subarachnoid space (Fig. 2). On intrauterine ultrasonography, the neural arch is open and the posterior elements are flared. There is an associated Chiari type II malformation in almost all cases. MRI is usually obtained postoperatively. A wide dysraphic defect is typically seen, together with a cerebrospinal fluid (CSF)-filled sac covered by skin. There is often retethering of the cord. MRI is the modality of choice for evaluation of the soft tissue elements in suspected spinal dysraphism.

- **Anterior Sacral Meningocele**

In an anterior sacral meningocele, there is protrusion of the dura and leptomeninges anteriorly through a defect in sacrum. On plain film, the lesion is recognized because of semicircular erosion of the sacrum (the "scimitar sign"). On MRI, the abnormal fluid collection will have CSF signal intensity on all pulse sequences. Myelography may not be diagnostic because the pedicle connecting the cyst and the thecal sac can be obstructed by adhesions and thus the cyst not filled with contrast.

- **Diastematomyelia**

In diastematomyelia, the spinal cord is split into two hemicords, each invested by pia (Fig. 3). Each hemicord contains a central canal and has both dorsal and ventral horns. In 60% of cases, the hemicords are contained within one subarachnoid space and dural sac. In 40% of cases, separate sacs with a fibrous band or an osteocartilaginous spur is nearly always present at the most inferior aspect of the cleft. Gradient echo scans are more sensitive than
T2-weighted scans, which are likewise more sensitive than T1-weighted scans, for detecting the spur. In 85% of cases, the cleft occurs between T9 and S1. In 50% of cases, the cleft is lumbar in location. Associated anomalies include vertebral segmentation anomalies, spina bifida (which is nearly always present), orthopedic foot problems such as clubfoot (half of patients), and hydromyelia. Associated vertebral segmentation anomalies, which are common, include fusion (block vertebrae), hemivertebrae, and butterfly vertebrae. Patients with diastematomyelia often present clinically with nonspecific symptoms. Symptoms may be related to cord tethering. Cutaneous stigmas (hairy patches, nevi, and lipomas) are seen in more than 50% of cases. On MRI it is critical to obtain axial scans; coronal scans are also useful. With sagittal scans alone, the split cord can be overlooked.
**Figure 2.** Meningomyelocele with tethered cord. The midline sagittal T1-weighted scan reveals a sac filled with cerebrospinal fluid located posteriorly in the lower lumbar region. The sac communicates with the normal thecal space. The spinal cord extends at least to the lumbosacral junction. The posterior bony elements are dysraphic from L4 to S1. Abundant fatty tissue is present immediately below the defect. Note also the distinct signal intensity and configuration of the vertebral bodies and intervertebral disks, normal for the patient’s age. This newborn presented with a normal neurologic exam and a low lumbosacral mass covered by skin. At the time of surgery for repair of this defect, a single nerve (not seen on magnetic resonance imaging or computed tomography) was identified within the fluid-filled sac.

- **Lipomyelomeningocele**

A lipomyelomeningocele is differentiated from a myelomeningocele (a protrusion of the membranes and cord through a defect in the vertebral column) by the presence of a lipoma and an intact overlying skin layer. The lipoma is firmly attached to the dorsal surface of the neural placode (cord terminus), which then herniates through the dysraphic spinal canal. The lipoma merges with and is indistinguishable from subcutaneous fat. The distal cord is tethered by the lipoma. Lipomyelomeningoceles occur in the lumbosacral region.
They make up 20% of skin-covered lumbosacral masses and 50% of occult spinal dysraphisms.

Associated anomalies include butterfly vertebrae (and other vertebral segmentation anomalies), sacral anomalies, scoliosis, and maldevelopment of the feet. Patients with lipomyelomeningoceles typically present clinically before 6 months of age with a fluctuant subcutaneous mass. Neurologic symptoms include lower extremity weakness, sensory loss, urinary incontinence, and gait disturbance. Symptoms are usually progressive if corrective surgery is not performed. Occasionally, lipomyelomeningoceles go undetected until adulthood because the lesion is covered with skin.

- **Dorsal Dermal Sinus**

A dorsal dermal sinus is a midline epithelium-lined tract that extends from the skin inward for a variable distance. More than 50% occur in the lumbosacral region (Fig. 8-4). The tract can terminate in the posterior soft tissue, at the dura, or within the thecal sac. Cord tethering is common. On the skin surface, there may be a hairy nevus, hyperpigmented patch, or capillary angioma. Half of all patients have an associated dermoid or epidermoid tumor at the tract termination. Patients present clinically in two different ways: either with infection or with symptoms of cord compression (by a tumor mass). On MRI, if an infection is present, intravenous contrast enhancement improves delineation of the sinus tract, particularly the intraspinal portion.

- **Tethered Cord**

A tethered cord is a congenital anomaly in which the conus is held at an abnormally low position. Causes include a short (tight) filum terminale, an intradural lumbosacral lipoma (Fig. 5), diastematomyelia, and a delayed consequence of myelomeningocele repair. With a tight filum, the age of presentation is variable. Adults present frequently with radiculopathy. The normal filum should be 2 mm or less in diameter. Caution should be exercised when interpreting postoperative cases (after myelomeningocele repair) because not all patients with evidence of tethering on imaging are symptomatic.

Clinical symptoms are due to cord ischemia caused by traction. The typical patient is a young child with progressive neurologic dysfunction. Symptoms include gait difficulty, motor and sensory loss in the lower extremities, and bladder dysfunction. On imaging, the cord is seen to extend without change in caliber to the lumbosacral region, where it is tethered posteriorly. Also commonly present is a lipoma and dysraphism of the posterior spinal elements. Hydromyelia may be present as well. When small, this is usually not symptomatic. T1-weighted scans in all three orthogonal planes are important for depiction of the abnormal anatomy. The axial plane is superior to the sagittal plane for determination of the level of the conus. On sagittal images, differentiation between the conus and cauda equina can be difficult. For the diagnosis of retethering, the presence of adhesions is a good criterion.
The aim of surgical therapy is to untether the cord and thus arrest symptom progression. Early diagnosis and surgery can prevent urinary incontinence. The associated lipoma is typically removed as completely as possible, with attention to release of the tether. The use of synthetic dural grafts decreases the incidence of retethering. After surgery, the level of the cord termination does not change.

A terminal myelocystocele is a rare congenital cystic dilatation of the caudal central spinal canal with an associated posterior bony defect. There is a trumpetlike flaring of the distal central canal, which is a pia-lined CSF space and may be larger than the accompanying surrounding meningocele. Associated anomalies of the gastrointestinal tract, genitourinary tract, and vertebral bodies are common.
**Figure 3.** Diastematomyelia. A, The midline sagittal T2-weighted scan demonstrates a segmentation anomaly (block vertebrae) at L23. A low-signal-intensity band spans the thecal sac at the L2-3 level. A central region of high signal intensity, consistent with a small syrinx (hydromyelia), is present within the lower thoracic spinal cord. B, The T1-weighted axial image at the L2 level reveals splitting of the spinal cord by the previously noted band or spur. Bony dysraphism is noted posteriorly. C, An additional T1-weighted axial image at a slightly higher level more clearly depicts the separation of the cord into two hemicords. This 18-month-old infant presented clinically with lower extremity spasticity.
Figure 4. Dorsal dermal sinus. Sagittal T2- (A) and T1-weighted (B) images of the lumbar spine reveal a sinus tract (A, arrows) coursing from the skin to the thecal sac. This abnormality is less apparent on the T1-weighted scan because of the high signal intensity.
of fat posteriorly, accentuated by the proximity to the surface coil. The conus lies at L2. A portion of tract (arrow) is also visualized on the axial T1-weighted image (C).
Figure 5. Tethered spinal cord with lipomyelomeningocele. A, The midline sagittal T2-weighted scan demonstrates spinal dysraphism at L4-5, a capacious lumbar thecal sac, and a large abnormal fat pad posteriorly. B, The corresponding T1-weighted scan reveals the cord to be low lying and tethered to a lipoma at the L4-5 level. C, On axial imaging, the low lying cord is seen in cross-section with a separate, but adjacent, intrathecal lipoma. D, On a lower axial section, the cord is tethered posteriorly and attached to a large lipoma that extends into both the thecal sac and the posterior soft tissues. This 2-month-old infant presented at birth with a posterior lumbar mass that subsequently increased in size. Motion and strength of the lower extremities were normal.

- **Spinal Meningeal Cysts**

Spinal meningeal cysts are diverticula of the meningeal sac, nerve root sheath, or arachnoid. Most cysts are congenital in origin. There are three types. Type I cysts are extradural in location and do not contain nerve roots. This group includes arachnoid cysts and sacral meningoceles. Type II cysts are extradural in location and contain nerve roots. This group includes spinal nerve root diverticula and Tarlov cysts. The latter are not infrequently seen in clinical practice. More correctly known as Tarlov perineural cysts, these lesions are simply nerve root sleeve cysts (focal dilatation of the nerve root sleeves). The nerves may be in the cyst or in the wall. The cyst communicates freely with the thecal sac (Fig. 6). Type III cysts are intradural in location and are simply intradural arachnoid cysts.

Spinal meningeal cysts are usually asymptomatic. These lesions are common in the sacral area. The cysts are frequently large, multiple, and bilateral. They can cause erosion and scalloping of the vertebral body, pedicle, and foramen. On MRI, the cysts are CSF signal intensity on all pulse sequences.

- **Lumbosacral Nerve Root Anomalies**

Lumbosacral nerve root anomalies occur in 1% to 3% of the population. These usually involve the L5 and S1 roots unilaterally. There are three types of lumbosacral nerve root anomalies. The first, type I, is a simple conjoined root (Fig. 7). This is the most common anomaly. Two roots arise from a single root sleeve but exit separately (in the appropriate foramina). In type II, two roots exit through a single foramen (and there may be one foramen without a root). In type III, an anastomotic root connects two adjacent roots. Lumbosacral nerve root anomalies are asymptomatic. However, it is important to recognize their presence and report this in the dictation. For lumbar disk surgery to be successful, in the presence of a nerve root anomaly, adequate decompression is required. On computed tomography (CT) without intrathecal contrast, a nerve root anomaly can be mistaken for a herniated disk.

- **Fatty Filum Terminale**

The normal filum terminale runs from the tip of the conus to the end of the thecal sac, inserting on the first coccygeal segment. As previously noted, the normal filum is 2 mm or
less in diameter at the L5-S1 level. One percent to 5% of the population have a small amount of fat within the filum. This is usually an incidental finding; however, it can be associated with cord tethering.

- **Achondroplasia**

Achondroplasia is an autosomal-dominant disorder of enchondral bone formation. In this disease, there is premature synostosis (bony ankylosis) of ossification centers of the vertebral bodies. In childhood, cervical changes may dominate the presentation, with canal narrowing and constriction at the foramen magnum. Classic findings in the lumbar spine include thick, short pedicles, an interpediculate distance that decreases from L1 to L5, canal stenosis (with a predisposition to disk herniation), and accentuated lumbar lordosis (horizontal sacrum).

**SPINAL STENOSIS**

- **Congenital**

In congenital spinal stenosis, both the anteroposterior and transverse dimensions of the canal are decreased. The pedicles are typically short and thick with a decreased interpediculate distance. The spinal canal tapers in the lumbar region (Fig. 8). This is the opposite of normal, in which the canal is usually equal in size to or greater (in anteroposterior dimension) than that in the thoracic region. The lateral recesses and neural foramina may also be narrowed. The lower limit of normal for the anteroposterior canal dimension is 11.5 mm, and the normal lateral recess should be 5 mm. The L4-5 level is the most common site for canal stenosis and tends to be the most severely affected level when the canal is diffusely narrowed. Congenital spinal stenosis predisposes the patient to early degenerative disk disease. Clinical presentation typically includes myelopathic symptoms. Radicular symptoms may be present as a result of nerve root impingement.
Figure 6. Tarlov cysts. A and B, Precontrast T1-weighted sagittal images reveal two oval areas of low signal intensity posterior to the S1 and S2 vertebral bodies, both to the right and left of midline. There is erosion and scalloping of the adjacent sacral segments. On the proton density (C and D) and heavily T2-weighted (E and F) sagittal images, the signal intensity of these cysts follows that of cerebrospinal fluid. Chemical shift artifact is noted at the interface between the lesions and the adjacent fatty marrow of the sacrum. There was no abnormal contrast enhancement (images not shown). G, The axial T1-weighted image through S2 reveals both cysts, which occupy (albeit markedly enlarged) the expected location of the nerve root sleeves.

Figure 7. Conjoined nerve root. A-E, Axial postcontrast T1-weighted scans are depicted from the middle of the L5 vertebral body to the middle of S1. On the first scan, the right L5 nerve root has already exited from the thecal sac (and is normal). A large abnormal nerve root sleeve is seen on the left, having not yet separated from the sac. On the next scan, two separate nerve roots are noted adjacent to one another on the left. On the third scan, at the level of the L5-S1 foramen, a nerve root (S1) is identified on the left medial to the enhancing dorsal root ganglion of L5. On the last two scans, the left S1 nerve root is seen to remain within the bony canal to descend to a position more symmetrical and normal relative to the right S1 nerve root.

- Degenerative (Acquired)

There are three types of degenerative spinal stenosis: central, lateral recess, and foraminal. The lateral recess is the space between the posterior margin of the vertebral body and the anterior margin of the superior facet. Its anatomic boundaries include the thecal sac medially and the pedicle laterally. The lateral recess is normally larger than 5 mm in
diameter. Patients with a lateral recess smaller than 3 mm in diameter are usually symptomatic.

Ligamentum flavum hypertrophy is one cause of degenerative spinal stenosis. The ligamentum flavum is a paired, thick, fibroelastic band. The normal thickness is 3 mm in the lumbar spine. The ligamentum flavum connects the lamina of adjacent vertebral bodies and is situated posterolaterally in the canal. It extends from the anteroinferior aspect of the superior lamina to the posterosuperior aspect of the inferior lamina. Anterolaterally, the ligamentum flavum is contiguous with the capsule of the facet joint. With degenerative spine disease, the ligamentum flavum becomes fibrotic, visibly thickened (Fig. 9), and buckled. It narrows the posterolateral canal and thus the lateral recess. It may also narrow the central canal and/or the neural foramina.
Figure 8. Congenital spinal stenosis. A, The midline sagittal T1-weighted scan demonstrates tapering of the spinal canal from the T12-L1 level through the lower lumbar spine. This is most prominent at the L3-4 and L4-5 levels. These findings are confirmed on the sagittal fast spin echo T2-weighted scan (B).

Diminished signal intensity consistent with disk degeneration is also seen at L3-4 and L4-5. Small posterior spurs are present, further compromising the anteroposterior dimension of
the canal. Axial T1-weighted images at the L3-4 (C) and L4-5 (D) levels show narrowing of the canal with deformity of the thecal sac and crowding of the nerve roots. The anteroposterior dimension of the sac measured 9 at both levels. The lateral recesses are also narrowed bilaterally at the L3-4 and L4-5 levels, resulting in minimal space for the passage of the nerve roots. This 35-year-old patient presented with low back pain and left leg pain and numbness.

Facet joint hypertrophy is another cause of degenerative spinal stenosis (Fig. 10). Hypertrophy of the superior articular facet is a primary cause of lateral recess stenosis. Failure to recognize lateral recess stenosis is a major cause of persistent symptoms after lumbar diskectomy.

A third cause of degenerative spinal stenosis is neural foraminal degenerative disease. The neural foramen is bounded by the pedicles superiorly and inferiorly, the vertebral body and disk anteriorly, and the facets posteriorly. In the lumbar spine, the nerve root exits from the lateral recess and enters the neural foramen. Stenosis of the neural foramen is most common at L4-5 and L5-S1. Degenerative disease of the disk, end plates, and posterior elements (facets) all contribute to foraminal stenosis (Fig. 11). The most common cause is hypertrophy of the superior facet. The stenosis is accentuated if the disk is narrowed. Foraminal stenosis causes radicular symptoms as a result of nerve root compression. Pain can also originate from the degenerated facet joints, which are richly innervated. The neural foramen is best imaged in the lumbar spine in the sagittal plane. Stenosis is easily visualized as a result of obliteration of the normal fat that surrounds the nerve root in the foramen. The clinical presentation for degenerative spinal stenosis is that of chronic pain in the lower back and buttocks. There may be paresthesias (abnormal sensation) or pain in the posterolateral leg. Standing and walking aggravate the pain, and resting (sitting or lying down) relieves it. This is the opposite of clinical symptoms for an acute disk herniation, in which the pain is aggravated by sitting. Neurologic deficits are minimal with degenerative spinal stenosis. The pathogenesis is nerve root ischemia.
Figure 9. Spinal stenosis with marked thickening of the ligamentum flavum. A, The sagittal T1-weighted scan just to the right of midline demonstrates narrowing of the thecal sac at L4-5, with indentation posteriorly by intermediate-signal-intensity soft tissue: the thickened ligamentum flavum (arrow). Less marked findings are present on the midline sagittal T1-weighted scan (B). These two sagittal images also reveal disk degeneration at L4-5 with disk space narrowing, a disk bulge with associated spurs, end plate irregularities, and adjacent degenerative end plate disease. C, The axial T1-weighted image at the L4-5
disk level demonstrates severe central stenosis of the spinal canal. The thecal sac is very small and triangular in shape, narrowed anteriorly by the disk bulge and spurs and posteriorly by the markedly thickened ligamentum flavum (extending along the posterolateral margins of the thecal sac). The thickened ligaments (measuring 6 mm in cross-section) and facet hypertrophy have obliterated the lateral recesses. A tiny amount of epidural fat is seen in the posterior canal.
Figure 10. Severe spinal stenosis and lateral recess stenosis at L4-5 resulting from facet joint hypertrophy. A, The midline sagittal postcontrast T1-weighted image reveals prominent narrowing of the lumbar canal at the L4-5 level. The canal stenosis is also well seen on the fast spin echo T2-weighted sagittal image (B). C, A T1-weighted sagittal image in the plane of the left lumbar facet joints reveals hypertrophy and sclerosis of the L4-5 facet (arrow). The neural foramen remains patent at this level. A similar appearance was present at the right facet joint of L4-5 (not shown). D, A T1-weighted axial image at the L4-5 level confirms the marked facet hypertrophic changes, left greater than right. The superior articulating facet of L5 is particularly affected. The facet hypertrophy results in bilateral lateral recess stenosis, more severe on the left, where the lateral recess is less than 3 mm in width. Sclerosis of the facet joints is also apparent. The spinal canal is narrowed, measuring 11 mm in anteroposterior dimension. Even more striking is the degree of narrowing of the thecal sac, which measures 4 mm in anteroposterior diameter.

Figure 11. Degenerative foraminal stenosis on the left at L5-S1. A, The T1-weighted sagittal image to the left of midline reveals a small neural foramen at L5-S1 (arrow), which is moderately narrowed secondary to degenerative spurs and hypertrophic facet disease. Only a minimal amount of fat is seen about the exiting L5 nerve root. The more normal keyhole appearance of the fat-filled neural foramina is present at L3-4 and L4-5. B, The
T1-weighted axial image at the inferior L5 level displays the L5 dorsal root ganglia bilaterally. The right dorsal root ganglion is surrounded by fat. The left ganglion is contacted posteriorly by the hypertrophied superior articulating facet of S1 (*) and anteriorly by an osteophyte arising from the L5 vertebral body.

INFECTION AND INFLAMMATORY DISEASE

- **Disk Space Infection**

Disk space infection can be either hematogenous (Fig. 12) or postoperative (Fig. 13) in origin. In children, with hematogenous seeding, the disk serves as the initial site of infection (because it is richly vascularized). In adults, the initial site of infection (with hematogenous seeding) is the vertebral body (subchondral portion) or soft tissue. Patients with postoperative disk space infection present clinically with severe back pain 1 to 4 weeks after surgery. Disk space infection is seen in 1% to 3% of all back surgery patients. Staphylococcus aureus is the most common organism. Delays in diagnosis are common. Fever, wound infection, and elevation of white blood cell count are seen in only a minority of patients. On lumbar spine x-ray films, disk space narrowing, poorly defined end plates, and sclerosis of the adjacent vertebrae may be seen. On CT, disk space narrowing, cortical bone loss (from the end plate), and abnormal paraspinal soft tissue may be seen. All are late changes. Radionuclide bone scans are sensitive but nonspecific in disk space infection.

On MRI, the disk itself will be narrow and irregular but with high signal intensity on T2-weighted scans. The adjacent vertebral end plates will also demonstrate high signal intensity on T2-weighted scans as a result of edema (with low signal intensity on T1-weighted scans). The edema within the adjacent vertebrae forms a horizontal band involving one third to one half of the vertebral body. This appearance can be confused with degenerative type I end plate changes. The signal intensity and irregularity of the disk permit differentiation. After intravenous contrast administration, the end plates and disk space enhance. Pockets of nonenhancing fluid, representing pus, are commonly seen within the disk space. The vertebral end plates will be indistinct. Also common is a paraspinal soft tissue mass, which enhances postcontrast. MRI is both sensitive and specific for the diagnosis of disk space infection. With adequate treatment, the edema within the adjacent vertebral bodies and the size of the paraspinal soft tissue mass will both gradually decrease.

- **Arachnoiditis**

In arachnoiditis, there is clumping and thickening of nerve roots on the imaging exam regardless of modality.
Figure 12. Hematogenous diskitis. A, On the T2-weighted scan, the L2-3 disk is high signal intensity (which by itself could be normal), yet irregular in contour. There is absence of the normal intranuclear cleft. The thecal sac is narrowed at the L2-3 level. B, On the precontrast T1-weighted scan, both the L2 and L3 vertebral bodies are of abnormal low signal intensity. There is loss of definition between the L2-3 disk and the adjacent vertebral end plates. C, On the postcontrast T1-weighted scan, there is enhancement of the L2 and L3 marrow space, with irregular enhancement along the disk margin and residual low-signal-intensity (nonenhancing) soft tissue within the disk space. The latter corresponds in
position to the high signal intensity noted on the T2-weighted scan and represents inflammatory exudates. The basis for thecal sac narrowing is now evident, with abnormal paraspinal enhancing the soft tissue. In the adult patient, noniatrogenic disk space infection is usually the result of hematogenous seeding to the soft tissue or to the subchondral portion of the vertebral body.

Figure 13. Postoperative disk space infection. A, Precontrast on the T2-weighted sagittal scan, diffuse abnormal high signal intensity (SI) is noted within the marrow of the L4 and L5 vertebral bodies. The disk is reduced in height, irregular, and of abnormal high SI. B, On the precontrast T1-weighted scan, the L4-5 disk is difficult to identify. Also noted is abnormal low SI within the lower half of L4 and the upper half of L5, paralleling the disk. C, Postcontrast, abnormal enhancement of the disk space is noted, together with a soft tissue mass that compresses the thecal sac. Comparison of pre(D) and postcontrast (E) T1-
weighted axial scans at the disk level reveals a paraspinous mass with enhancement. There is abnormal enhancement of the disk as well, permitting identification of fluid pockets that remain low SI (arrows).

Inflammation initially elicits only a minimal cellular response, which then progresses to collagenous adhesions. The pathogenesis includes infection, which is uncommon today, previous surgery, hemorrhage within the thecal sac, and prior myelography with Pantopaque.

CT findings in arachnoiditis, which are seen with moderate involvement, include nodular or cord-like intradural masses and nerve roots that are adherent to the dura. On myelography, with mild involvement, there can be blunting of the nerve root sleeves, fusion of nerve roots, and irregularity of the thecal sac margin. With moderate involvement, there can be obliteration of the nerve root sleeves, multisegmental fusion of nerve roots, adhesions, scarring of the thecal sac, and loculation of intrathecal contrast.

The nerve roots and abnormalities thereof are clearly seen on MRI. Several common patterns of nerve root involvement in arachnoiditis are subsequently described. In mild disease, nerve roots can be clumped and lie centrally within the sac (Fig. 14). Alternatively, individual nerve roots may be adherent to the periphery of the sac. With severe disease, abnormal soft tissue can fill the majority of the thecal sac, with no discernible individual nerve roots. With acute infection (viral or bacterial meningitis), the nerve roots themselves enhance (Fig. 15). Care should be exercised in the diagnosis of arachnoiditis when spinal stenosis is present. Spinal stenosis can lead to a false impression of nerve root clumping.
Figure 14. Arachnoiditis. Midline sagittal T2- (A) and T1-weighted (B) images suggest clumping of nerve roots along the posterior margin of the thecal sac. No individual nerve roots are visualized; rather a single thick strand is seen. The clumping of nerve roots is confirmed on the axial T2- (C) and T1-weighted (D) images.
Figure 15. Spinal meningitis with progression to arachnoiditis. Postcontrast sagittal (A) and axial (B) images reveal prominent enhancement (white arrows) of the lumbar nerve roots. These also appear mildly thickened but retain their usual position within the dependent portion of the thecal sac. The patient returned for follow up after 1 month of antibiotic therapy. Her back pain remained severe at this time. C, The postcontrast T1-weighted sagittal image reveals persistent enhancement of the lumbar nerve roots. The nerve roots now lie anteriorly within the thecal sac. D, A postcontrast axial image at L3-4 demonstrates the enhancing nerve roots to be clumped anteriorly. Cultures in this patient revealed Staphylococcus aureus as the causative organism.

NEOPLASTIC DISEASE

- Benign Neoplasms of Bone
  - Vertebral Body Hemangioma

Vertebral body hemangiomas are a common incidental finding on MRI. This benign neoplasm can be found, on autopsy, in more than 10% of the population. Solitary lesions are most common, although multiple lesions are not uncommon. The size is variable, ranging from small to large, involving the entire vertebral body. Posterior extension can cause canal compromise. A large lesion can weaken the vertebral body and lead to fracture. Histologically, vertebral hemangiomas are composed of a mixture of adipose and angiomatous tissue with prominent bony trabeculae. The coarse vertical trabeculation can be seen on plain film and CT, which also depict the lesion as generally lucent. On MRI, vertebral hemangiomas are classically high signal intensity on both T1- and T2-weighted scans (Fig. 16). Also commonly noted is a reticular pattern of low signal intensity (prominent vertically) corresponding to the thickened trabeculae. The major differential diagnosis on MRI is that of focal fat (within the vertebral bodies). The latter is a common finding, particularly with increasing age. Focal fat deposition will be seen to follow the signal intensity of fat on all pulse sequences.

  - Osteoid Osteoma

Osteoid osteoma is a common benign skeletal neoplasm found most often in young patients. The lesion consists of a central nidus of osteoid, woven bone, and fibrovascular tissue, with an overall diameter of less than 2 cm. Osteoid osteomas are sharply demarcated from surrounding bone with variable surrounding sclerosis. The classic clinical presentation is that of pain, which is relieved by aspirin. Ten percent of osteoid osteomas occur in the spine. Here the most common location is in the neural arch of a lumbar vertebra. Scoliosis is common. On CT, sclerosis will be seen surrounding a small lytic lesion. CT may also demonstrate the nidus to be calcified. Bone scintigraphy is useful for diagnosis; focal activity is seen on both immediate and delayed scans. On MRI, the nidus is low signal intensity on T1-weighted images. The nidus is commonly surrounded by extensive edema, which can involve the adjacent soft tissue in addition to the bone.

  - Giant Cell Tumor
In the lumbar spine, the most common location of a giant cell tumor is the sacrum. Patients with this tumor, predominantly female, typically present at between 20 and 40 years of age. Vertebral lesions carry a better prognosis than giant cell tumors elsewhere in the body, with a low rate recurrence after resection. Giant cell tumors are lytic and expansile lesions, but they rarely cross the periosteum. On MRI, a giant cell tumor is typically lobular, with intermediate signal intensity on T1-weighted scans and mixed signal intensity on T2-weighted scans. High signal intensity on T2-weighted scans corresponds to hemorrhagic and cystic foci. A low-signal-intensity rim is seen on both T1- and T2-weighted scans as a result of dense sclerosis at the tumor margin. Giant cell tumors are quite vascular and demonstrate contrast enhancement. The differential diagnosis includes osteoblastoma (more common in the posterior elements, less lobular), aneurysmal bone cyst (younger age group), and metastatic disease.

Figure 16. Vertebral hemangioma. A round, mottled area of increased signal intensity is seen in the central portion of the L3 vertebral body on the sagittal T1-weighted image (A). The postcontrast T1-weighted image (not shown) revealed mild enhancement. The lesion also exhibits high signal intensity on the sagittal T2-weighted image (B). Mottled signal intensity is demonstrated with interspersed areas of very low signal intensity on all imaging sequences corresponding to prominent trabeculae.
Malignant Neoplasms of Bone

- Lumbar Metastases

The vertebral column is the most common site of skeletal metastatic disease. Lung cancer is the most common cause. Other causes include breast cancer, prostatic carcinoma, renal cell carcinoma, and hematologic malignancies. Most cases of epidural compression of the cord or cauda equina are due to vertebral metastases, with either bony collapse or posterior extension. In most such patients, the compression is at only one level.

Most patients with lumbar metastatic disease present clinically with back pain. Motor impairment can occur and usually precedes sensory deficits. Radiculopathy is uncommon. However, compression of a single nerve root can occur (with epidural tumor extension), mimicking a disk herniation. Plain x-ray films are notoriously insensitive to metastatic disease. The classic finding was that of an absent pedicle. This led to the misimpression that vertebral metastatic disease most often originated in the pedicle. The advent of MRI showed this clearly not to be true but rather simply that pedicle lesions were better seen by plain film than lesions in other locations. In the past, myelography was, but is no longer, the modality of choice for examination of the patient with a suspected compressive lesion. Myelography carries a high risk in patients with a block. Neurologic deterioration is seen after the exam in up to 25% of patients. Lesions above a block are also missed by myelography.

MRI is the modality of choice for detecting and assessing vertebral metastatic disease. MRI is more sensitive (as well as more specific) than bone scintigraphy for detecting vertebral metastases. We now know, because of MRI, that the vertebral body is nearly always the initial site of involvement. Sagittal scans provide screening of the area of interest. These should be supplemented with axial scans in areas where canal compromise is questioned. Imaging of the entire spine in the body coil is not recommended for screening because smaller metastases and even compressive lesions in some instances will be missed. Bony metastatic lesions are low signal intensity on (precontrast) T1-weighted scans, which are used by most practices for lesion detection (Fig. 17). Epidural extension is also well seen on MRI; axial scans play an important role here as well (Fig. 18). It is important to compare the signal intensity of the disk and the vertebral body (on sagittal images) in order not to miss diffuse metastatic disease. On T1-weighted scans, normal marrow should always be higher in signal intensity than the intervertebral disk. If the marrow is isointense with the disk or lower signal intensity, then the marrow is diffusely abnormal and widespread metastatic disease is likely (although other causes should be considered, including hematologic abnormalities).

Fast short time inversion recovery (STIR) scans are used in some practices as the primary scan for detection of vertebral metastases. These scans, although more motion sensitive, can be slightly superior to T1-weighted spin echo scans for lesion detection. Although STIR images are predominantly T1-weighted, the gray scale is reversed compared with spin echo images, and metastases appear as hyperintense vertebral body lesions. On spin echo T1-weighted scans, contrast administration is not helpful for detecting bone metastases. Most
metastases enhance postcontrast to near isointensity with normal marrow, decreasing their conspicuity. Contrast enhancement is, however, useful for improved depiction of epidural and soft tissue extent of metastatic disease and for the detection of leptomeningeal metastases (Fig. 19). T2-weighted scans are not of great use in the evaluation of metastatic disease to the vertebral column, although they are routinely acquired (Fig. 20). Many bony metastases will have abnormal high signal intensity on T2-weighted scans, but many will also be isointense. Osteoblastic metastases, which are common with prostate carcinoma, deserve special comment. These are typically low signal intensity on both T1 and T2-weighted scans. When both osteoblastic and lytic lesions are present, it is commonly observed that the blastic lesions are substantially lower in signal intensity on T1-weighted scans than the lytic lesions. Metastatic lesions in lung and breast carcinoma are typically lytic but may be osteoblastic when treated. Bony sclerosis is seen, of course, on plain film with osteoblastic metastases.

- **Chordoma**

Chordomas are locally invasive, destructive, lytic, lobular, slow-growing lesions. Calcification is seen on x-ray exams in half. A mixture of solid and cystic components is common. In regard to location, 50% occur in the sacrum or coccyx, 35% at the skull base (clivus), and 15% in the vertebral body.

- **Plasma Cell Myeloma**

The term plasma cell myeloma is used to describe a malignant disease of plasma cells that includes both multiple myeloma and plasmacytoma. A plasmacytoma is a solitary lesion of bone. Laboratory blood studies may be positive or negative. Additional lesions can develop with time. The spine and pelvis are the most common locations for a plasmacytoma. This lesion is osteolytic and expansile.

- **Intraspinal Neoplasms**
  - **Intradural Lipoma**

Lipomas within the thecal sac lie on the benign end of the spectrum that includes lipomyelomeningocele. A dorsal spinal defect, if present, is minimal. Developmentally, there is premature separation of cutaneous ectoderm from neuroectoderm, with mesenchyma entering the neural tube and later differentiating into fat. Lipomas compose 1% of all intraspinal tumors. Most lie along the dorsal aspect of the spinal cord. On MRI, lipomas will have fat signal intensity on all pulse sequences. At high field (1.5 T and above), chemical shift artifact is commonly observed at the interface between fat and CSF along the frequency encoding direction. Nerve roots can in some cases be identified coursing through the lesion (Fig. 21).

Care should be exercised in the diagnosis of a lipoma. The lesion should be of the exact same signal intensity as that of fat on all pulse sequences. The presence of septations, a
slight difference in signal intensity from fat, or contrast enhancement make it very unlikely that a fatty lesion is a lipoma (Fig. 22).

Figure 17. Lumbar vertebral metastatic disease. Sagittal (A-C) and axial (D and E) precontrast T1-weighted images reveal multiple low-signal-intensity vertebral body lesions. These involve T12, L1, L4, and S1. The metastases are in general round and well demarcated, occasionally extending to the cortex of the vertebral body. Incidental note is made of a lumbarized S1 vertebral body.
Figure 18. Sacral metastases with epidural tumor causing right S1 nerve root compression. A, The T1-weighted midline sagittal image demonstrates abnormally decreased signal intensity throughout the sacrum, most prominent at the S1 level. Epidural soft tissue involvement is apparent posterior to both S1 and S2. These abnormalities are increased signal intensity on the corresponding T2-weighted sagittal image (B). Irregular enhancement of the sacrum is apparent on the postcontrast T1-weighted sagittal image (C). The epidural disease demonstrates homogeneous enhancement. D, A precontrast T1-weighted axial image at the S1 level confirms the abnormal low signal intensity within the sacrum. The epidural soft tissue mass distorts the thecal sac and severely compresses the right S1 nerve root. The normal left S1 nerve root (arrow) is unaffected. Expansion of the right sacral ala with paraspinal extension is also apparent. This 79-year-old patient with lung cancer presented clinically with a right S1 radiculopathy.
Figure 19. Vertebral body and leptomeningeal metastases. The patient is 35 years old, has breast cancer, and presents with increasing pain and numbness in the legs. A, On the precontrast T1-weighted midline sagittal scan, vertebral body metastases with low signal intensity relative to normal marrow are noted in L1, L3, and L4. The vertebral body lesions are less apparent on the corresponding T2-weighted scan (B). The lesions in L1 and L4 do demonstrate slight hyperintensity relative to normal marrow. Posterior within the thecal sac, a questionable area of abnormal hyperintensity is noted at the L2 level. C, Postcontrast on the T1-weighted scan, the vertebral body lesions demonstrate enhancement to near isointensity with normal marrow. Partial collapse of L4 is now evident. Critical for prognosis and treatment is, however, the identification of two enhancing nodules (small arrows) within the thecal sac, consistent with leptomeningeal tumor spread.
Figure 20. Expansile L3 vertebral body metastasis. A, The T2-weighted scan reveals abnormal high signal intensity within the L3 vertebral body. This vertebral body also has an abnormal configuration, consistent with a compression fracture. The posterior margin has a convex outward curvature, compressing the thecal sac. The L3 vertebral body is low signal intensity on the precontrast T1-weighted sagittal scan (B) and enhances postcontrast (C). Of the axial scans—T2-weighted (D), precontrast T1-weighted, (E), and postcontrast T1-weighted (F)—the postcontrast scan best delineates the thecal sac (arrow), which is severely compressed. The patient, who had nonsquamous cell lung carcinoma, presented clinically with pain radiating into the right lower extremity.

- Dermoid and Epidermoid

Dermoids and epidermoids are two of the "pearly" tumors, so named for their gross appearance. Both are ectodermal inclusion cysts, containing squamous epithelium, keratin, and cholesterol. Dermoids are differentiated by the presence of dermal appendages (hair and sebaceous glands). In the spine, most dermoids and epidermoids occur in the
lumbosacral region. Dermoids are more common. The lesion can be either intra or extramedullary in location. Dermoids and epidermoids are well-defined, rounded lesions. A portion of the tumor may be cystic, containing desquamated epithelium and, in the case of dermoids, sebaceous gland secretions. Frequently associated anomalies include dermal sinus and spinal dysraphism.

- **Teratoma**

Teratomas are rare in the spinal canal, except for the sacrococcygeal form. The latter is the most common presacral mass in a child. Sacrococcygeal teratomas can undergo malignant transformation. Teratomas by definition are composed of tissue from all three germinal layers.

- **Lymphangioma**

This is a congenital lesion resulting from obstruction of lymphatic drainage. Seventy-five percent occur in the neck (posterior triangle). In this location, lymphangiomas are more common in children younger than 2 years.

Lymphangiomas are typically asymptomatic and treated by surgical resection. These lesions have fluid signal intensity, low on T1- and high on T2-weighted scans. Septa and fat may be present between the fluid spaces.
Figure 21. Intradural lipoma. An intradural, high-signal-intensity soft tissue mass is noted at the L1-2 level on the sagittal T1-weighted scan (A). B, On the axial T1-weighted scan, nerve roots (with lower signal intensity) are noted to course through the lesion. The mass is isointense with fat on the intermediate T2-weighted scan (C). This was also the case on all other pulse sequences. An artifactual low-signal-intensity line is noted at the inferior margin of the mass, at the interface with cerebrospinal fluid. This dark band occurs in the direction of the readout gradient and is caused by chemical shift artifact, with the image being acquired at 1.5 T.

Figure 22. Angiolipoma. A, On the T2-weighted scan, the conus is displaced anteriorly, but a soft tissue mass is not clearly identified. B, The precontrast T1-weighted scan reveals a posterior epidural mass, extending from T1 to L1, with mixed high signal intensity. C, On the postcontrast T1-weighted scan, the abnormality is noted to enhance to isointensity with fat. Although the lesion is similar in signal intensity to fat, it is heterogeneous and displays abnormal contrast enhancement. These characteristics suggest a neoplastic origin. Angiolipomas are rare benign tumors composed of lipocytes and abnormal blood vessels. These tumors are epidural in location, occur most commonly in the midthoracic region, and can cause cord compression.

- Ependymoma

Ependymomas are slow-growing, well-circumscribed, benign tumors. Complete surgical resection is possible. Ependymomas make up 60% to 70% of all spinal cord tumors. They occur in the third to sixth decades of life. Most arise in the conus, cauda equina, or filum terminale. The cervical cord is the most common site for an intramedullary ependymoma. The clinical presentation is nonspecific and can include motor and sensory deficits and sphincter dysfunction. On MRI, focal cord enlargement limited to two or three levels
favors the diagnosis of an ependymoma over an astrocytoma. Virtually all ependymomas enhance strongly after contrast administration (Fig. 23).

- **Neurofibroma and Schwannoma**

Neurofibromas (Fig. 24) and schwannomas (Fig. 25) are the most common of the nerve root sheath tumors. Most are intradural extramedullary in location.

One third are extradural. A foraminal lesion may be mistaken for a herniated disk (Fig. 26). Enhancement postcontrast allows differentiation.

It is difficult to differentiate schwannomas and neurofibromas on MRI or, likewise, any imaging exam. Schwannomas are typically solitary and well circumscribed and lie eccentric to the nerve itself (whereas a neurofibroma causes fusiform enlargement of the nerve). Schwannomas tend to be heterogeneous in signal intensity on T2-weighted scans. Neurofibromas tend to be homogeneous in signal intensity on T2-weighted scans and may have a target appearance (high signal intensity peripherally, lower signal intensity centrally). Multiplicity of lesions favors the diagnosis of neurofibroma.

- **Leptomeningeal Metastases**

The presence of leptomeningeal metastases portends a poor prognosis. One third of all patients with metastases to the brain or spine will eventually acquire leptomeningeal metastatic disease. Breast and lung carcinomas are the most common visceral neoplasms to spread to the subarachnoid space. In the lumbar region on MRI, leptomeningeal metastases can take on several different appearances. There can be large or small nodules or a combination (Fig. 27). Alternatively (or concurrently), there can be (smooth) coating of nerve roots and the cord (Fig. 28). The nerve roots can also appear "beaded" as a result of nodular metastatic deposits (Fig. 29). Intramedullary extension of leptomeningeal metastatic disease, although rare, can occur. Contrast-enhanced MRI is markedly superior to CT myelography for detection. The differential diagnosis should include meningeal infection (in immunosuppressed patients), toxoplasmosis, and sarcoidosis. In the latter disease, cord involvement usually dominates.
Figure 23. Mixed papillary ependymoma of the conus medullaris. A, On the sagittal T2-weighted scan, an intradural extramedullary soft tissue mass is noted. The spinal cord is displaced anteriorly and flattened. B, On the axial T2-weighted scan, the mass is seen posteriorly and to the right, with severe compression of the cord. C, On the postcontrast T1-weighted scan, there is heterogeneous enhancement of the mass (arrows), greater peripherally and less centrally. The cord itself is thinned and lies anterior and slightly to the left. The patient presented with slowly increasing low back pain and left lower extremity weakness. Virtually all ependymomas demonstrate strong enhancement after intravenous contrast injection on magnetic resonance imaging.
Figure 24. Neurofibroma. Pre- (A) and postcontrast (B) sagittal T1-weighted scans reveal a large enhancing soft tissue mass in the left L3-4 neural foramen. The mass is of high signal intensity on the T2-weighted scan (C). Comparison of pre- (D) and postcontrast (E) axial T1-weighted scans reveals a smoothly marginated enhancing lesion, which has expanded the foramen. Contrast enhancement of the mass favors a neural origin and improves lesion demarcation from surrounding soft tissue. Schwannomas tend to enhance in a heterogeneous fashion, often more intense peripherally. Neurofibromas typically demonstrate homogeneous contrast enhancement. The patient is a 66-year-old veteran with neurofibromatosis.
Figure 25. Lumbar nerve root schwannoma. A, On the T2-weighted scan, a small round lesion with intermediate signal intensity is noted within the thecal sac at the L5 level. The lesion appears to be immediately adjacent to or part of the L5 nerve root. The lesion is nearly isointense with cerebrospinal fluid on the precontrast T1-weighted scan (B) and demonstrates prominent enhancement (arrow) postcontrast (C). The lesion, a schwannoma, was confirmed on subsequent surgery performed for lumbar disk disease. Incidental note is made of an L3 vertebral body hemangioma.
Figure 26. Neurofibroma, mimicking a free disk fragment. Parasagittal T2- (A) and T1-weighted (B) images reveal a soft tissue mass (B, arrow) in the left L4-5 neural foramen. The L4 nerve root is not identified. Comparison of pre- (C) and postcontrast (D) axial T1-weighted scans reveals homogeneous enhancement of the mass (D, arrow). Contrast enhancement in this instance provides important information for differential diagnosis, eliminating from consideration a free disk fragment.
Figure 27. Leptomeningeal ("drop") metastases from medulloblastoma. The midline sagittal T2-weighted scan reveals multiple large soft tissue nodules adjacent to the conus, adherent to the cauda equina, and near the termination of the thecal sac. The size and extent of these intrathecal metastases lead to their excellent visualization on the T2-weighted scan in this instance. The patient, a 4-year-old with metastatic medulloblastoma, presented clinically with diminished coordination.
EFFECT OF TRAUMA

- Flexion Injury

Flexion injuries are seen in motor vehicle accidents when the patient is confined by a lap belt without a shoulder strap. Flexion occurs with the fulcrum centered on the anterior abdominal wall. The principal bony injury is a lumbar spine fracture (Chance fracture). The Chance fracture is a transverse fracture through the body of the vertebra, extending posteriorly through the pedicles and the spinous process. However, fracture of the posterior elements need not be present. This flexion injury is principally a distraction injury with ligamentous disruption. There may be little or no anterior vertebral body compression, and the injury may be unstable.

When the occupant is unrestrained, flexion occurs with the fulcrum centered on the posterior portion of the vertebral body. This results in an anterior body compression fracture. There is accompanying distraction of the posterior elements. This injury is most common at the thoracolumbar junction.

- Osteoporotic Compression Fracture

Osteoporotic compression fractures occur in the elderly as a result of insufficiency of bone (senile osteoporosis). They are more common in postmenopausal women. With an acute osteoporotic compression fracture, areas of low signal intensity on T1-weighted and high signal intensity on T2-weighted scans, corresponding to edema, will be present within the vertebral body. However, there will also be areas of preserved, normal marrow. Unfortunately, there is little to differentiate an acute benign compression fracture from a pathologic compression fracture. Over the years, value has been placed on many different MRI signs, none of which have proved to be specific. However, with an osteoporotic compression fracture, the edema will eventually resolve (after many months). Chronic osteoporotic fractures can be recognized by their anatomic deformity but demonstrate signal intensity isointense to that of normal marrow.

- Pathologic Compression Fracture

Pathologic compression fractures demonstrate low signal intensity on T1-weighted scans and high signal intensity on T2-weighted scans. The abnormal signal intensity is principally due not to edema but rather to the presence of neoplastic disease. There may be complete replacement of normal marrow signal intensity within the body, and this may extend into the pedicle. Most patients have multiple lesions in other vertebral bodies (round to oval in appearance), an important differentiating feature from an acute osteoporotic compression fracture. Sagittal T1-weighted imaging is thus very valuable in screening patients. With the advent of fast spin echo technique, T2-weighted scans have improved substantially in image quality. Thus, today both T1 and T2-weighted scans are typically acquired; axial scans are important in addition to sagittal scans. Although epidural extension and canal compromise are usually well demonstrated on sagittal scans, it is actually the central component that is
well visualized. Depiction of abnormal lateral soft tissue and compromise of the canal from either the right or left side is best accomplished with axial scans.

- **Spondylolysis and Spondylolisthesis**

In spondylolysis, there is interruption of the pars interarticularis. This may be unilateral or bilateral. Bilateral involvement allows motion of the posterior elements relative to the adjacent vertebrae. The superior and inferior facets at the involved level can move independently. The superior facet remains attached to the vertebral body. The inferior facet articulates and moves with the more inferior vertebral body. On axial CT, the defects are seen as lucent clefts, oriented in the coronal plane. On axial MRI, the discontinuity of bone may be difficult to visualize. One key to diagnosis is the presence of a "continuous facet" sign from the disk space above to the disk space below. The bony defect is often clearly seen on sagittal MRI.

Spondylolisthesis is defined as forward slippage of one lumbar vertebral body relative to the adjacent lower vertebral body (or sacrum). There are many causes, including trauma, surgery, degenerative disease (of the facet joints), and congenital disease. Spondylolisthesis causes narrowing of the neural foramen, which may cause nerve root impingement. The foramen assumes a more horizontal orientation as seen on sagittal scans.

Spondylolisthesis is graded according to the degree of subluxation. Grade I is up to one fourth of the vertebral body, grade II between one fourth and one half, grade III between one half and three fourths, and grade IV greater than three fourths.

With degenerative spondylolisthesis, the midline sagittal image demonstrates narrowing of the spinal canal (Fig. 30). The posterior elements are contiguous with and, therefore, move anteriorly with the displaced vertebral body. When spondylolisthesis occurs in combination with spondylolysis, the canal is typically not narrowed because the posterior elements move independently from the vertebral body (Fig. 31). The adjacent posterior elements remain in alignment, and the spinal canal may widen in this situation.
**Figure 28.** Leptomeningeal metastases. The presence of an intradural soft tissue mass at T12-L1 is questioned on the basis of precontrast sagittal T2(A) and T1-weighted (B) scans. C, Postcontrast, the lesion is confirmed because of intense enhancement (white arrow). Also noted postcontrast is an enhancing nerve root within the filum terminale and a second smaller mass within the thecal sac at the L2 level (black arrows). Leptomeningeal metastases are best identified postcontrast; enhancement in this case permits diagnosis. This elderly individual with lung carcinoma presented 6 months before the current exam with brain metastases.

![Image](https://www.yassermetwally.com/images/figure28.jpg)

**Figure 29.** Leptomeningeal metastases. A, On the midline sagittal T2-weighted image, there is diffuse disk degeneration, with narrowing of the thecal sac at multiple levels on the basis of degenerative disease. The lumbar nerves within the thecal sac appear prominent (suggesting nerve root thickening) on both the T2- and precontrast T1-weighted (B) images. C, Postcontrast, there is striking abnormal enhancement of the cauda equina and lumbar nerves, which now also appear somewhat "beaded." Head computed tomography (not shown) revealed multiple brain metastases. This 83-year-old patient was diagnosed with and treated for small cell carcinoma of the lung 1 year before the current exam. The patient is now admitted with a 2-week history of low back pain, leg weakness, and mental status changes.

- **Retrolisthesis**

A retrolisthesis is a posterior subluxation of a vertebral body relative to the adjacent lower body. This is caused by disk degeneration with preservation of the facet joints. A retrolisthesis can occur after surgery or other intervention (Fig. 32), with resultant neural foraminal narrowing (and nerve root impingement). This is one cause of the failed back surgery syndrome. Retrolisthesis is most common in the lumbar and cervical spine. In the lumbar spine, L3-4 and L4-5 are the most frequently involved levels. Disk bulges and spurs
commonly accompany a retrolisthesis. Central canal stenosis is uncommon, but neural foraminal narrowing is common.

Figure 30. Spondylolisthesis secondary to degenerative facet changes. A, The midline sagittal T1-weighted image demonstrates grade I anteriorlisthesis of L4 on L5. The right (B) and left (C) parasagittal T1-weighted images show the pars interarticularis to be intact bilaterally at L4. This excludes spondylolysis as a cause of the spondylolisthesis. These images reveal facet degeneration with irregular, narrowed facet joints. D, The axial T1-weighted image through the L4-5 disk again demonstrates the anteriorlisthesis of L4 on L5. The curvilinear low signal intensity of the posterior L4 body (small white arrows) projects 7 mm anterior to the posterior L5 body (small black arrows). E, The axial T1-weighted image through the L4-5 facets reveals irregularity and narrowing of the facet joints. Compare these to the axial image (F) showing normal smooth facets at L3-4.
Figure 31. Bilateral spondylolysis with spondylolisthesis. A, A right parasagittal T1-weighted image shows a break (arrow) in the right pars interarticularis at the L5 level. The neural foramen is narrowed because of the anterior listhesis. B, A left parasagittal section also demonstrates a left L5 pars interarticularis defect. A disk herniation is also present at the L5-S1 level extending into the left neural foramen. C, The midline sagittal T1-weighted image shows grade I spondylolisthesis at the L5-S1 level. The axial pre- (D) and postcontrast (E) T1-weighted images show bilateral irregular pseudarthroses in the posterior ring of L5 corresponding with the pars defects. Mild enhancement of the pseudarthroses is present presumably because of volume averaging with the surrounding soft tissues. The disk herniation is again demonstrated (arrow). The axial image at the level of the articular facets at L4-5 (F) demonstrates the appearance of the normal facet joints above the pars defects.

- Pseudomeningocele

A pseudomeningocele is an accumulation of CSF (outside the normal confines of the thecal sac) caused by a tear in the dura with (most common) or without a tear in the arachnoid membrane. The connection to the subarachnoid space is variable in size. Pseudomeningoceles can occur after laminectomy. In this instance, they are most common in the cervical spine, particularly after surgery involving the occiput. Pseudomeningoceles
are rare after laminectomy in the lumbar spine, but here they can produce radicular symptoms. A pseudomeningocele will follow CSF signal intensity on all pulse sequences.

- **Postoperative Lumbar Spine**

The recurrence of symptoms after lumbar surgery, which occurs in 10% to 40% of patients, defines the failed back surgery syndrome. Causative factors include recurrent disk herniation, spinal stenosis, arachnoiditis, and epidural fibrosis (scar). MRI plays an extremely valuable role in the evaluation of the patient with recurrent pain after lumbar spine surgery. On postcontrast scans, postoperative scar can be differentiated from a recurrent or residual disk herniation; the distinction is critical for the therapeutic decision making process (Fig. 33). This use of intravenous contrast accounts for a substantial amount of the contrast used overall in spine MRI.
Figure 32. Retrolisthesis. A, The sagittal T1 weighted image near the midline shows disk space narrowing at L4-5 and mild posterior displacement of the L4 vertebra on L5. A small disk bulge is present at L4-5 with thin, high- signal-intensity type II end plate changes adjacent to the L4-5 and L3-4 disks. B, The parasagittal T1-weighted image through the right neural foramina demonstrates narrowing of the L4-5 bony foramen. The inferior aspect of the foramen is obliterated by the posteriorly displaced L4 vertebra and the associated disk bulge. The right L4 nerve root exits under the L4 pedicle with a small amount of surrounding high signal intensity fat. The superior articular facet of L5 (arrow) has moved in an anterior and cephalad direction, obliterating the inferior aspect and narrowing the superior aspect of the neural foramen. C, The sagittal T1-weighted image
after intravenous contrast administration confirms the retrolisthesis at L4-5. The malalignment is more easily detected because of enhancement of the epidural venous plexus along the posterior margin of the vertebrae. D, The comparable sagittal, intermediate T2-weighted image again demonstrates the malalignment at L4-5. Decreased T2 signal intensity in the L3-4 and L4-5 disks is due to disk degeneration at these levels. The patient, 41 years old, presents with recurrent low back and bilateral leg pain after L4-5 disk surgery.

Figure 33. Postdiskectomy scar tissue. Two months after a right laminectomy and diskectomy, a soft tissue mass is identified anterior and to the right of the thecal sac on the precontrast T1-weighted axial scan (A). B, Postcontrast, there is uniform enhancement of this abnormal soft tissue (arrow), consistent with scar. The right S1 nerve root can only be identified postcontrast surrounded by scar.

In the postoperative back, postcontrast scans should be obtained within 20 minutes after intravenous contrast administration. After this time, there may be diffusion of contrast from enhancing to nonenhancing tissue, making interpretation difficult. Postoperative scar demonstrates homogeneous enhancement as a result of intrinsic vascularity. However, this is not seen consistently until 3 months after surgery. Scar is one cause of persistent pain after lumbar disk surgery and is in general a contraindication to further surgery. Although the presence of a soft tissue mass favors the diagnosis of a recurrent disk, scar can also have this appearance (Fig. 34). Thus, noncontrast scans are not reliable for differentiation. In the patient with recurrent pain and postoperative scar, the fibrosis is often extensive and surrounds an exiting nerve root, presumably the basis for symptoms. A recurrent or residual disk herniation (Fig. 35) will not show enhancement on MRI scans obtained after contrast administration (assuming, of course, that these are obtained within 20 minutes of injection). Correct diagnosis on MRI mandates the use of thin sections, 3 mm or less, to avoid partial volume effects. A recurrent disk herniation will be seen as a focal, smooth posterior protrusion of nonenhancing soft tissue (contiguous with the native disk). The disk
is commonly circumscribed posteriorly by a thin rim of enhancing soft tissue (Fig. 36) corresponding to scar (but in minimal amounts with a normal expected finding).

On contrast-enhanced MRI in the postoperative patient, the decompressed nerve may also enhance. This should resolve by 6 months after surgery. The facet joints may enhance presumably because of surgical manipulation. This can persist long term.

**ARTHRITIS**

Ankylosing spondylitis is an inflammatory disease of unknown etiology. The sacroiliac joints are involved early in the disease course. Erosion of cortical margins with subchondral bony sclerosis is seen first. Joint space widening, due to bony erosion, follows. The end result is fusion (obliteration) of the sacroiliac joints. In the spine, syndesmophytes are the hallmark of ankylosing spondylitis (Fig. 37). These slender, vertical ligamentous calcifications extend from the osseous excrecence of one vertebral body to the next. In the spine, the inflammation associated with ankylosing spondylitis occurs at the junction of the annulus fibrosus and the vertebral body. The outer annular fibers become replaced by bone, or syndesmophytes, which eventually bridge adjacent vertebral bodies. In advanced disease, this leads to the appearance on plain film of a "bamboo spine." One significant complication of ankylosing spondylitis is bony fracture after minor trauma. In the cervical spine, this can lead to quadriplegia.

**DEGENERATIVE DISEASE**

Spondylosis is a term that refers nonspecifically to any lesion of the spine of a degenerative nature (but usually involving specifically bone). Common degenerative processes seen in the lumbar spine include Schmorl's nodes, osteophytes, and end plate sclerosis.

- **Focal Fat Deposition**

Focal fat deposition in the vertebral marrow can occur at any level and is frequently seen in multiple vertebral bodies. These deposits are round and up to 15 mm in diameter. Focal fat deposition is more common in elderly patients. It is seen on MRI in more than 90% of patients older than 50 years. The pathogenesis is focal marrow ischemia, with fatty replacement of hematopoietic marrow. On MRI, focal fat deposition follows the signal intensity of fat on all pulse sequences.

- **Schmorl's Node**

A Schmorl's node represents a prolapse of the nucleus pulposus through the end plate into the medullary space of a vertebral body. The prolapse occurs as a result of axial loading. Schmorl's nodes are typically asymptomatic. On plain film, a focal depression, contiguous with the vertebral end plate, is seen with a sclerotic rim. On MRI, Schmorl's nodes will be of lower signal intensity than marrow on T1-weighted scans and of higher signal intensity on T2-weighted scans. There is often surrounding focal end plate changes. Contrast enhancement occurs, often peripheral in location, because of the presence of granulation
tissue. Sagittal scans demonstrate the lesion to be immediately adjacent to the disk space and are thus most useful for diagnosis.

- **Synovial Cyst**

In the spine, synovial cysts are associated with degenerative facet disease. When symptomatic, a synovial cyst can present with radicular pain, often sciatic in nature. This can mimic a disk herniation. Large synovial cysts can compress the thecal sac. On CT, the lesion can be hypo or hyperdense. Synovial cysts may be calcified and are recognized by their location adjacent to a facet joint. On MRI, the signal intensity of the fluid within the cyst is variable; synovial cysts can have any combination of low or high signal intensity on T1- and T2-weighted scans. Postcontrast, the cyst capsule and any solid component will demonstrate enhancement (Fig. 38). Delayed enhancement of the cyst contents has been observed. Recognition of the relationship to the facet joint is critical for diagnosis.

- **Degenerative Disk and End Plate Changes**

There are many signs of disk degeneration on MRI. There can be loss of disk height. Annular tears, with high signal intensity on T2-weighted scans, may be seen. However, decreased signal intensity of the disk itself on T2-weighted scans is the most sensitive indicator of early disk degeneration. This finding is often referred to in clinical dictations as disk dehydration or desiccation and occurs with varying degrees and may early on involve only a part of the disk. The actual cause of the decrease in signal intensity is a decrease in proteoglycans and in the ratio of chondroitin sulfate to keratin sulfate. With the exception of trauma, disk herniation without changes of disk degeneration is extremely unusual. This can be very helpful in directing the film reader toward the disk space levels that should be more closely examined (those demonstrating disk desiccation).
Figure 34. Differentiation of scar from disk in the postoperative back. A, The T1-weighted sagittal image to the right of midline reveals abnormal soft tissue (arrow) projecting posterior to the L5-S1 intervertebral disk. B, After contrast administration, this tissue enhances intensely. Enhancement is also apparent both superior and inferior to the L5-S1 intervertebral disk, at the interface with the adjacent vertebral bodies. C, A T1-weighted axial view at the inferior L5 level confirms the abnormal soft tissue in the ventral epidural space. The right laminectomy defect is also apparent. D, Postcontrast, the abnormal extradural soft tissue (which is now seen to surround the right S1 nerve root) is noted to enhance. Enhancement within soft tissue posteriorly at the laminectomy site and within the right paraspinal musculature is also noted. The patient presented with continued right leg pain 2 months after diskectomy at L5-S1. The anterior epidural mass in this case, which
appears contiguous to the L5-S1 disk, would be suspicious for a recurrent disk herniation on the precontrast scans. The homogeneous enhancement of the abnormality, however, allows confident diagnosis of the lesion as epidural fibrosis (scar).

Figure 35. Postdiskectomy recurrent disk extrusion. T2- (A) and T1-weighted (B) midline sagittal scans reveal abnormal soft tissue anterior to the thecal sac at the L4-5 and L5-S1 levels. Two previous percutaneous diskectomies had been performed. C, Postcontrast, the majority of abnormal soft tissue at each level does not enhance. Enhancing soft tissue (C, arrows) above and below the L4-5 disk space level corresponds to a dilated epidural venous plexus. Comparison of pre- (D) and postcontrast (E) T1-weighted axial scans at the L4-5 level confirms the presence of a recurrent disk herniation (arrow), with a small amount of surrounding enhancing granulation tissue. Lumbar microdiskectomy was subsequently performed.
Figure 36. Pre- and postdiskectomy exams in a patient presenting with a disk extrusion and recurrence after surgery. Also important to the clinical case and surgical approach is the presence of a transitional vertebra. The preoperative exam includes sagittal T2- (A), sagittal precontrast T1- (B), sagittal postcontrast T1- (C), axial precontrast T1- (D) and axial postcontrast T1-weighted (E) images. The postoperative exam, performed 1 year later, includes the same sequences, specifically sagittal T2-
Figure 36. (F), sagittal precontrast T1- (G), sagittal postcontrast T1- (H), axial precontrast T1- (I), and axial postcontrast T1-weighted (J) images. It should be recognized first that the patient has a transitional vertebra. The level with significant disease is likely to be L4-5, with L5 being sacralized. This was confirmed by reference to plain radiographs. This patient actually had four subsequent magnetic resonance imaging (MRI) exams, with one reader dictating the level as L5-S1 twice and two other readers dictating the level correctly as L4-5 once each. On the preoperative exam, there is a moderate-size right paracentral disk extrusion. Contrast enhancement provides minimal improvement in demarcation of the abnormal disk. On the MRI scan obtained a year later, with intervening surgery, there is a larger recurrent right paracentral disk extrusion. Postcontrast, there is a thin circumferential rim of enhancing scar tissue, which improves differentiation of the disk from adjacent cerebrospinal fluid. The lack of enhancement of the majority of the soft tissue mass confirms that this represents recurrent disk disease. Postoperative changes caused by the right-sided laminectomy are also noted.
Figure 37. Ankylosing spondylitis. A and B, On parasagittal T1-weighted images of the lumbar spine, there are prominent anterior osteophytes (curved arrows), which appear to bridge the disk space at several levels. C, The anteroposterior plain film of the lumbar spine reveals the sacroiliac joints to be obliterated, with bony bridges (marginal syndesmophytes) connecting adjacent vertebral bodies.
Figure 38. Synovial cyst. Images from two patients with similar symptoms are presented. The first is 60 years old and has experienced increasing left leg pain and intermittent numbness over the last 6 months. A, On the sagittal T2-weighted scan, a low-signal-intensity abnormality is noted within the bony spinal canal, immediately posterior to the L4-5 intervertebral disk. There is displacement and compression of the thecal sac. B, Before contrast administration, the lesion is difficult to identify. C, After contrast injection, there is rim enhancement. On this scan, the lesion (a synovial cyst) appears (correctly) to be extradural. Pre- (D) and postcontrast (E) axial images at the L4-5 level are presented from the second patient’s exam. There is facet hypertrophy bilaterally. D, Precontrast, the question is raised of a left-sided lesion causing compression posteriorly of the thecal sac. E, Postcontrast, there is rim enhancement, which improves the differentiation of the lesion from cerebrospinal fluid within the thecal sac. The lesion appears cystic in nature by signal intensity and enhancement characteristics. The lesion (another synovial cyst) is contiguous with the left facet joint.

A vacuum disk is a degenerated disk with gas (nitrogen) in clefts within the annulus fibrosus and nucleus pulposus. Vacuum disks are more common in the lumbar spine and in elderly patients. On CT, very low density is seen within the disk. On MRI, linear low signal...
intensity (with the presence of gas resulting in a signal void) is seen on both T1- and T2-weighted scans.

Degenerative vertebral body end plate changes are a common finding on MRI of the lumbar spine. A change in signal intensity of the marrow space adjacent to the end plate is by far the most clear indicator of degenerative end plate disease (Fig. 39). These changes are parallel and directly adjacent to the disk space. Such changes typically involve the entirety of both end plates (surrounding a degenerated disk), although involvement of just one end plate (and even just a portion of one) can occur.

Type I end plate changes reflect increased water content and are low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Type I end plate changes enhance after contrast administration, often to isointensity with marrow fat. Type I end plate changes can be mimicked by two other disease entities; differential diagnosis is critical. Metastatic disease can at times resemble type I end plate changes. However, typically, there are multiple additional lesions. Isolated involvement of the end plate by metastatic disease is uncommon. Disk space infection and adjacent osteomyelitis can also resemble type I end plate changes. However, with infection, the disk should be grossly abnormal, the demarcation between disk and body lost, and a paraspinous mass often present.
Figure 39. Degenerative end plate changes. A-C, Type I end plate changes histologically show vascular infiltration, fibrosis, and granulation tissue between thickened bony trabeculae. Increased water content results in both T1 and T2 lengthening. On magnetic resonance imaging (MRI) the end plates show increased signal intensity on T2-weighted scans (A) and decreased signal intensity on T1-weighted scans (B). The signal is usually parallel to the end plates and directly adjacent to the intervertebral disk. C, The affected end plates commonly enhance (to isointensity with normal marrow) after intravenous contrast administration. D, Type II end plate changes show fatty infiltration interposed between thickened trabeculae histologically. MRI reveals increased signal intensity on both T1- (D) and T2-weighted scans (not shown) compared with normal marrow. E and F, Another end plate pattern, Type III, consists of sclerotic changes. On MRI, the end plates are low signal intensity on both T2- (E) and T1-weighted (F) scans. These areas correspond with sclerosis on plain x-ray films.

Type II end plate changes reflect fatty infiltration. There is increased signal intensity within the end plate on both T1- and T2-weighted scans paralleling fat. The progression of type I to type II has been observed on occasion, leading to the conclusion that type I is an early form of end plate disease, which eventually converts to type II. In clinical cases, type II is by far the most common type of end plate disease observed. Mixed type I and II
patterns are also seen. Type III is very rare and corresponds to bony sclerosis, with low signal intensity seen on both T1- and T2-weighted scans.

**DISK HERNIATION**

The strict definition of a disk herniation is the protrusion of degenerated or fragmented disk material into the foramen compressing a nerve root or into the spinal canal compressing the spinal cord or cauda equina. Medicolegal considerations have led many radiologic practices to discard the use of the term disk herniation and adopt a terminology more descriptive of the process and its extent. This terminology, advanced by Michael Modic and others, is described in detail later. It classifies disk disease into four categories: disk bulge, protrusion, extrusion, and free fragment. Tears of the annulus fibrosus are also described; these can be seen on MRI and are no doubt a precursor to more advanced, symptomatic disk disease.

Tears of the annulus fibrosus are classified into three types. Concentric, or type I, is parallel to the curvature of the outer disk. Radial, or type II, involves all the layers of the annulus from the nucleus pulposus to the surface. Transverse, or type III, involves the insertion of Sharpey's fibers into the ring apophysis. Tears of the annulus fibrosus are high signal intensity on T2 weighted scans. A tear will also enhance after intravenous gadolinium chelate administration (Fig. 40). Contrast enhancement is due to the presence of fibrovascular (granulation) tissue, a result of the body's normal reparative process.

A disk or annular bulge is an extension of the posterior disk beyond the margin of the adjacent vertebral end plates but without focal disk protrusion (Fig. 41). The posterior disk margin forms a smooth curvilinear contour. A disk bulge by definition is broad based and circumferential. A disk bulge occurs as a result of laxity of and tears within the annulus fibrosus. It is a sign of early disk degeneration. A disk bulge can, however, narrow the spinal canal and the inferior neural foramen.

A disk protrusion is a herniation of the nucleus through a (small) tear in the annulus but still contained by outer fibers of the annulus. A disk protrusion is differentiated from a bulge by axial imaging, with demonstration of focal extension of disk material beyond the margin of the vertebral end plates (Fig. 42). Although disk protrusion and extrusion are distinct entities, differentiated by the degree of rupture of the annulus, this can rarely be appreciated on MRI. In common usage, the term disk protrusion is reserved for a small herniation and disk extrusion for a large herniation of disk material through the ruptured annulus.

A disk extrusion is a herniation of the nucleus through the ruptured annulus with no intact remaining annular fibers. It is important to specify, when interpreting MRI exams, whether a disk extrusion (or protrusion) is central (Fig. 43), paracentral (Fig. 44), foraminal (Fig. 45), or lateral (Fig. 46) in location. A disk extrusion, when combined with lateral stenosis, can cause nerve root ischemia with eventual fibrosis, leading to irreversible axonal damage. In an extrusion, the disk material remains in contiguity with the parent disk. This distinction differentiates a disk extrusion from a free fragment.
Even without surgery, granulation tissue forms around the extruded disk, part of the body's normal reparative process. This tissue enhances postcontrast, forming a thin rim of high signal intensity "wrapping" the extruded disk material on enhanced T1-weighted exams. This appearance can be confusing to radiologists who have experience principally with nonenhanced MRI scans. Scar in the nonoperated back may potentially assist in recovery by limiting the herniation and with contraction decreasing the degree of compression of neural structures. The neurosurgeons of yesteryear were very familiar with the fact that a substantial reduction in size of a disk herniation could be observed with conservative therapy. Thus, follow-up MRI scans can demonstrate a reduction in size of a disk extrusion without intervening surgery (Fig. 47).

Figure 40. Annular tear. T1-weighted pre- (A) and postcontrast (B) axial images demonstrate a mild, focal, asymmetrical extension of the posterior disk margin. The disk abuts, but does not significantly displace, the right L5 nerve root sleeve. The postcontrast image reveals a curvilinear area of high signal intensity (arrow) paralleling the posterior disk margin because of enhancement of a concentric tear in the outer fibers of the annulus fibrosus. A T2-weighted image was not acquired in this patient in the axial plane. Such an image would have also clearly depicted the tear, with abnormal hyperintensity.
Figure 41. Disk bulge. A, The sagittal T2-weighted image demonstrates decreased signal intensity in the L3-4, L4-5, and L5-S1 disk spaces consistent with disk degeneration. The posterior disk margins extend beyond the adjacent vertebral end plates and indent the anterior thecal sac at these levels. The sagittal pre- (B) and postcontrast (C) T1-weighted images again show mild posterior extension of disk material from L3-4 through L5-S1. The disk margin is better delineated after the administration of intravenous contrast because of enhancement of the epidural venous plexus. D, The axial T1-weighted image through the L3-4 level reveals a generalized disk bulge with mild convexity of the posterior disk margin. The disk material narrows the lateral recesses bilaterally (arrows). The posterior disk margin has a smooth curvilinear contour with no focal disk protrusion.
Figure 42. Disk protrusion. Sagittal precontrast T2- (A) and T1-weighted (B) scans reveal extension of disk material beyond the vertebral end plates at L4-5. C, Enhancement of de novo scar and epidural venous plexus postcontrast improves delineation of the disk margin from cerebrospinal fluid on the T1-weighted scan. Comparison of pre- (D) and postcontrast (E) axial T1-weighted scans through the L4-5 disk level reveals the extension of disk material, which is relatively small, to be focal and central in location.
Figure 43. Central disk extrusion. Midline sagittal precontrast T2-weighted (A) and postcontrast T1-weighted (B) scans demonstrate moderate compression of the thecal sac by posterior extension of disk material at the L4-5 level. Loss of the normal high signal intensity (on the T2-weighted scan) of the intervertebral disks at L4-5 and L5-S1 is compatible with disk degeneration. Axial pre- (C) and postcontrast (D) scans at the L4-5 level demonstrate the disk extrusion to be central in location. At high field, with current software, an alternative imaging approach (driven by cost) is to add an axial fast spin echo T2-weighted scan and not acquire the postcontrast scans.
Figure 44. Right paracentral disk extrusion. Sagittal precontrast T2-weighted (A) and postcontrast T1-weighted (B) scans, just to the right of midline, demonstrate substantial compression of the thecal sac by disk material at the L5-S1 level. Axial pre- (C) and postcontrast (D) scans at the L5-S1 level demonstrate this large disk extrusion to be paracentral in location. This 32-year-old patient presented with right leg pain.
Foraminal disk extrusion. Parasagittal precontrast T2- (A), precontrast T1- (B), and postcontrast T1-weighted (C) scans reveal extension of disk material into the inferior portion of the foramen at the L5-S1 level. After contrast administration, there is enhancement of a thin line (presumably scar) separating the disk extrusion from the superior portion of the foramen, which contains the L5 nerve root (surrounded by fat). Axial pre- (D) and postcontrast (E) scans at the L5-S1 level depict very clearly the focal extrusion of disk material within the foramen. The dorsal root ganglion is seen just lateral to the extrusion, with normal enhancement postcontrast. Axial T2-weighted scans (not shown), although excellent for demonstrating central and paracentral disk disease, are poor for foraminal disease; differentiation of disk material and other foraminal contents is difficult.
Figure 46. Lateral disk extrusion. Precontrast sagittal T2- (A) and T1-weighted (B) scans to the left of midline demonstrate extension of the L4-5 disk posteriorly into the left L4-5 neural foramen. The exiting left L4 nerve root is identified just above the disk extrusion. C, The precontrast axial T1-weighted scan reveals a large focal lateral herniation of disk material (black arrow). The exiting L4 nerve root (small white arrow) is seen on the right but is obscured by the herniated disk material on the left. D, The postcontrast T1-weighted axial scan provides clearer delineation of the disk extrusion as a result of enhancement of the epidural venous plexus and foraminal veins. The displaced left L4 nerve root (D, arrow)
can now be distinguished from the nonenhancing extruded disk. Mass effect on the left side of the thecal sac is also more apparent.

Ninety percent of lumbar disk extrusions occur at L4-5 or L5-S1. Of the remainder, most occur at L3-4. Central lesions may cause no symptoms, with the exiting nerve roots unaffected. Paracentral lesions cause symptoms as a result of compression of the exiting nerve root. For example, the S1 nerve root will be compressed by a paracentral L5-S1 disk extrusion. Lateral disk extrusions are the least common because the annulus is thinnest posteriorly. Superior migration of lateral fragments is common. A lateral disk extrusion will compress the ganglion or nerve root within the neural foramen. This causes radiculopathy of the nerve root above the interspace. For example, a lateral disk extrusion at the L3-4 level will compress the L3 nerve. Lateral disk extrusions occur beyond the termination of the nerve root sleeve. Thus, myelography is relatively insensitive to lateral disk disease. Myelographic findings with a disk extrusion include displacement of the contrast-filled sac, elevation, displacement, or amputation of the nerve root sleeve, and nerve root enlargement (as a result of edema). When a nerve is acutely compressed by a disk extrusion, edema of the nerve root in question can occasionally be seen within the thecal sac on MRI (with nerve root enlargement and abnormal high signal intensity on the T2-weighted exam). Lumbar nerve root enhancement is not uncommon with acute disk extrusions, although many radiologists are unfamiliar with this appearance. Their unfamiliarity is due to the fact that most screening exams of the lumbar spine for disk disease are performed without contrast enhancement. Lumbar nerve root enhancement occurs as a result of disruption of the blood-nerve root barrier. Its presence supports the clinical significance of a compressive lesion (Fig. 48).
Figure 47. Resolution of L4-5 paracentral disk protrusion with conservative therapy. Precontrast sagittal (A) and postcontrast axial (B) T1 weighted scans from the patient's initial clinical presentation are compared with scans obtained 1 year later (C and D). At presentation, disk material protrudes posteriorly on the sagittal image at the L4-5 level (A). The protrusion (B, arrow) is well delineated by a thin rim of enhancement on the postcontrast axial scan and is noted to be paracentral in location. On the follow-up exam
obtained 1 year later, there is no abnormal posterior extension of disk material on the sagittal scan (C). Enhancing scar tissue is noted on the postcontrast axial T1-weighted scan (D) but without compression of the thecal sac.
Figure 48. Enhancing nerve root resulting from compression by a large free fragment. Comparison of pre- (A) and postcontrast (B) T1-weighted axial scans at the L5-S1 level reveals intense enhancement of the left S1 nerve root (arrow) within the thecal sac. This is confirmed on the postcontrast T1-weighted sagittal scan (C, arrow), which also identifies nerve root compression by a large disk fragment. The patient was referred for a magnetic resonance imaging scan because of recent onset of a left S1 radiculopathy.

Figure 49. Free disk fragment. A, On the T2-weighted sagittal scan, a soft tissue mass with abnormal high signal intensity is identified posterior to S1. This mass (white arrow) is isointense with the remaining disk material at the L5-S1 level on the T1-weighted sagittal scan (B). C, Postcontrast, the periphery of the mass enhances. Inspection of pre- (D) and postcontrast (E) axial scans through the S1 vertebral body confirms the presence of a free disk fragment. The fragment is "wrapped" by enhancing scar (arrows), deforms the thecal sac, and compresses the left S1 nerve root.
Figure 50. Free disk fragment. A, The sagittal T2-weighted scan reveals only mild disk degeneration at L4-5. B, On the precontrast T1-weighted sagittal scan, abnormal soft tissue (arrow) is noted partly contiguous with the L4-5 disk but posterior to the L5 vertebral body. C, Postcontrast, the lesion (a free fragment) is better delineated because of the surrounding rim of enhancing tissue. Examining the T2-weighted scan in retrospect, the lesion is noted to be of high signal intensity and thus difficult to differentiate from cerebrospinal fluid. The free fragment (D, white arrow), which has migrated inferiorly, is well demonstrated on pre- (D) and postcontrast (E) axial T1-weighted scans, which also reveal compression of the right L5 nerve root (E, black arrow). The patient presented 5 days after injury with low back and right leg pain.
Figure 51. Free disk fragment within the L4-5 foramen. A, Abnormal soft tissue is noted within the left neural foramen on the precontrast axial T1-weighted scan. B, After contrast administration, a thin rim of enhancement better delineates the lesion, which otherwise does not change in signal intensity. Contrast administration in this instance permits the differentiation of a neural origin tumor within the foramen (which would enhance; see Fig. 26) from a migrated free fragment (which, as illustrated by this case, does not enhance).

With a free fragment or sequestered disk, the herniated disk material is separate from the parent disk. A free fragment may be anterior (contained by) or posterior to the posterior longitudinal ligament. When anterior, a thin midline septum directs the fragment paracentrally away from the midline. Free fragments have characteristic signal intensity on MRI, intermediate to low signal intensity on T1-weighted scans, and high signal intensity (but less than that of CSF) on T2-weighted scans (Fig. 49). Free fragments can migrate superiorly or inferiorly (Fig. 50) within the epidural space or into the neural foramen (Fig. 51).