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 re-establishing 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 occa-
sionally 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. 3–1). 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

**FIGURE 3–1.** 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. (Courtesy of Dr. Larry Tanenbaum, New Jersey Neuroscience Institute.)
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.

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. 3–2). The $T_2^*$, 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.

**Figure 3–2.** 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). (Courtesy of Dr. Larry Tanenbaum, New Jersey Neuroscience Institute.)

**Figure 3–3.** 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 $T_2^*$-weighted scan (A) is indicative of early vasogenic edema. B, The thickening and increased prominence (visibility) of cortical gray matter (black arrows) on the $T_1$-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.
Once fully established, vasogenic edema is clearly seen with conventional MRI techniques. The increased water content causes prolongation of both $T_1$ and $T_2$. Vasogenic edema thus has low signal intensity on $T_1$-weighted scans and high signal intensity on $T_2$-weighted scans. $T_2$-weighted scans, however, are relied on in clinical practice for the visualization of vasogenic edema. Commonly used “$T_1$-weighted” spin echo sequences (i.e., short time to repetition [TR] and short time to echo [TE]) do not have optimal $T_1$ contrast. Such scans are only mildly $T_1$-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 $T_2$-weighted scans. Inversion recovery sequences or three-dimensional gradient echo $T_1$-weighted sequences (such as turbo-FLASH) are more heavily $T_1$-weighted. With the latter types of scans, the abnormal low signal intensity resulting from vasogenic edema is much better visualized than with conventional $T_1$-weighted spin echo scans.

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 $T_1$ relaxation times, increasing the signal intensity on $T_1$-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. 3–4). 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. 3–5). 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. 3–6). 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...
FIGURE 3–5. 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 3–6. 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.
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.

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. Glialosis 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. 3–7). 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. 3–8) 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. 3–9). 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 T1-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 3–7.** 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.
BRAIN: ISCHEMIC (AND ATROPHIC) DISEASE

FIGURE 3–8. 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 gyrmiform 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 gyrmiform enhancement postcontrast (image not shown), indicative of blood-brain barrier disruption in this late subacute infarct.

FIGURE 3–9. Hemorrhagic (deoxyhemoglobin) right middle cerebral artery (MCA) infarction. Abnormal high signal intensity is seen on the T1-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. Gyrmiform 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.

ARterial vascular territories

The arterial vascular territories of the brain are shown in Figure 3–10. 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 lenticulostriate 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. 3–11).

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. 3–18).

The anterior choroidal artery arises from the supraclinioid 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
FIGURE 3–10. Arterial vascular territories, in the axial (A–E) and coronal (F–J) planes. ACA = anterior cerebral artery; ACh = anterior choroidal artery; AICA = antero inferior 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 = posterior inferior cerebellar artery; SCA = superior cerebellar artery; WSCA = watershed region supplied predominantly by the SCA.
FIGURE 3–11. 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 3–12. 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 3–13. 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 3–14. 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 3–15. 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 3–16. 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.
artery (PICA) supplies the retro-olivary medulla, the cerebellar tonsil, the inferior vermis, and the posterior lateral inferior cerebellum (Fig. 3–19). 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.

**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 supraclinoid 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.

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
FIGURE 3–19. 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 3–20. 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 3–21. 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.

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.

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 decompression, 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.

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. 3–22). 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 arterial vascular 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. 3–23).
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. 3–24). 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. 3–25).

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.

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 iso- to low signal intensity on T1-weighted images (Fig. 3–26). 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 other chronic ischemic lesions, which are incidental to the patient's current medical problems.

Figure 3–25. 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.

Figure 3–26. 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.
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. 3–27). 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.

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 margined 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. 3–28). Contrast enhancement is consistently seen in late subacute pontine infarction (Fig. 3–29).

In the elderly population with arteriosclerotic disease, lateral medullary infarction (Wallenberg’s syndrome) is not uncommonly encountered (Fig. 3–30). 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...
**FIGURE 3–28.** 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 3–29.** 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 3–30.** 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.
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.

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. 3–31) and in the periatrial and supraventricular white matter (Fig. 3–32). A third common location is the midbrain (Fig. 3–33), 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.

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. 3–34). 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 com-

---

**Figure 3–31.** 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.
FIGURE 3–32. 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 3–33. 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.
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.

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.

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, Behçet’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. 3–35). The other pattern consists of small focal punctate white matter lesions, presumably corresponding to small microinfections. 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 Behçet’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 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. 3–36). 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. 3–37), 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 3–35.** 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.
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. 3–38). 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.

**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.

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 flow in these cases.

**MOYAMOYA**

Moyamoya is an ischemic vascular disease of unknown cause. There is progressive stenosis or occlusion of the supracaloid 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 supracaloid internal carotid artery or middle cerebral artery, and visualization of the dilated collateral moyamoya vessels as multiple signal flow voids (Fig. 3–39).

**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 reflect 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.

**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. 3–40). Long-term, the vessel can recanalize, and flow voids are again visualized.
FIGURE 3–41. Huntington’s disease. Coronal $T_2$- (A) and $T_1$-weighted (B) scans reveal substantial volume loss in the caudate nucleus bilaterally.

FIGURE 3–42. Central pontine myelinolysis. The pons and middle cerebellar peduncles have abnormal high signal intensity on the axial $T_2$-weighted scan (A). The pons also demonstrates abnormal low signal intensity on the sagittal $T_1$-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.

FIGURE 3–43. Cerebellar degenerative disease (alcoholic). A. The midline sagittal $T_2$-weighted scan demonstrates marked atrophy of the cerebellar vermis. The folia are small and the sulci enlarged. B. The coronal postcontrast $T_1$-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.
BRAIN: ISCHEMIC (AND ATROPHIC) DISEASE

FIGURE 3–44. 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.

Slow flow in 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.

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). 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 (Fig. 3–42). Differential diagnostic considerations include infarction, small vessel ischemic disease, metastasis, glioma, and radiation changes.

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 (Fig. 3–43). The atrophy is irreversible. Although much less common, phenytoin (diphenylhydantoin or Dilantin) can also cause global cerebellar atrophy.

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 thepons, middle cerebellar peduncles, olives, and cerebellar hemispheres (Fig. 3–44). There may also be accompanying gliosis.

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. 3–41). Cortical atrophy is seen in long-standing disease.