Cerebrovascular disease includes both structural vascular anomalies (aneurysms and vascular malformations) and ischemia. Vascular anomalies are discussed in this chapter; ischemia is discussed in Chapter 3. 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.

**HEMORRHAGE**

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 water 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 process 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 protons to undergo a sufficient number of collisions with the paramagnetic center to establish equilibrium.
water molecules to encounter greater local magnetic inhomogeneity, which increases dephasing and further shortens $T_2$.

Using knowledge about $T_2$ 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 $T_1$ weighting (short TR and short TE) are called $T_1$-weighted images. Sequences with relative $T_2$ weighting (long TR and long TE) are called $T_2$-weighted images. Long TR, short TE sequences are called proton density images. Even though a sequence is called "$T_1$-weighted" or "$T_2$-weighted," the signal intensity derives from both $T_1$ and $T_2$ 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 $T_1$- and $T_2$-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 ($Fe^{2+}$) 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 $T_1$ and $T_2$ relaxation times and is hypointense or isointense on $T_1$-weighted images and high signal intensity on $T_2$-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 ($Fe^{2+}$) 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 $T_2$ proton relaxation enhancement shortens $T_2$ but not $T_1$. As a result, intracellular deoxyhemoglobin is slightly hypointense or isointense on $T_1$-weighted images and has low signal intensity on $T_2$-weighted images (Fig. 2-1). The low signal intensity on $T_2$-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.

In the formation of methemoglobin, the heme iron is oxidized to the ferric state ($\text{Fe}^{3+}$). 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. 2–2).

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 proton density 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 ($\text{Fe}^{3+}$) 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 $T_1$-weighted, proton density, or $T_2$-weighted images. However, calcium is sometimes mixed with hemosiderin, and the iron in the mixture produces a $T_2$ 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 hemo-

**FIGURE 2–2.** Subacute hematoma (intracellular methemoglobin rim). A left frontal lobe mass is noted on precontrast $T_2$-weighted (A) and $T_1$-weighted (B) scans. The mass has a prominent low-signal-intensity rim on the $T_2$-weighted scan, with a thick high-signal-intensity rim on the $T_1$-weighted scan (findings consistent with intracellular methemoglobin). There is substantial surrounding vasogenic edema, best seen on the $T_2$-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.
siderin 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 inconspicuous because the BBB is not intact. In a simple intraparenchymal hematoma, the hemosiderin rim is well defined and continuous.

**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 to 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 on T₂-weighted images. The degree of hypointensity on T₁-weighted images increases with the increasing field strength. Consequently, on low-field systems, an acute hematoma can be nearly isointense with brain on T₁-weighted images. 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 deoxyhemo-

globin 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. 2–2). On T₁-weighted images, a subacute hematoma may 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. 2–3).

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 T₁-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 isointense with cerebrospinal fluid (CSF) (Fig. 2–4). 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.

**Subdural Hematoma**

Subdural hematomas result from a venous injury, with blood lying outside the brain parenchyma between the dura and arachnoid. Like intraparenchymal hematomas,
BRAIN: HEMORRHAGE AND VASCULAR ANOMALIES

FIGURE 2–3. 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.

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.

In a subacute subdural hematoma (Fig. 2–5), 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 T1- and T2-weighted images. As methoglobin 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. 2–6).

In the subacute and chronic phases, the membrane delimiting a subdural hematoma may enhance with intravenous injection of a gadolinium chelate (see Fig. 2–6). Subdural hematomas may have a combination of

FIGURE 2–4. 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 2–5. 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.

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 blood stream. On rare occasions, hemosiderin can be identified in patients with recurrent bleeds into chronic subdural hematomas.

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. 2–7). 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 layer.
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.

**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 ($P_{O_2}$) 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 $T_1$ proton relaxation enhancement, to $T_2$ shortening during this phase is negligible. Because $T_2$ shortening is proportional to the square of the concentration of the paramagnetic compound, deoxyhemoglobin in a concentration of 28% (100% – 72%) contributes only 8% ($0.28^2$) to $T_2$ shortening. The $T_2$ 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 $T_1$ caused by the higher protein content of the bloody CSF. This mild $T_1$ shortening leads to hypointense 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 $T_1$- and $T_2$-weighted images corresponding to methemoglobin within thrombus (Fig. 2–8). 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 $T_2$-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.

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. 2–9), 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 super-
Paramagnetic substances, retain their magnetism after the external magnetic field is removed.

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 (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 has also replaced CT as the screening modality of choice. CT readily visualizes acute hemorrhage and atypical symptoms. In these 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. 2–13). 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
FIGURE 2–10. 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 2–11. 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 2–12. 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 2–13. 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.
chronic subarachnoid hemorrhage resulting from a ruptured aneurysm is also clearly identified by MRI.

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 flow in 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 flow in 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. 2–14). 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 $1 \times 1 \times 1$ 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.

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. 2–15). 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. 2–16). 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 $T_1$- and $T_2$-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 $T_1$-weighted images and either hypointense or hyperintensity on $T_2$-weighted images because of the presence of intracellular or extracellular methemoglobin. Edema within the adjacent brain is hypointense on $T_1$-weighted images and hyperintense on $T_2$-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

![Figure 2–14. Right middle cerebral artery bifurcation aneurysm (arrow) 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 lesions and should be performed in all patients being evaluated for a possible intracranial aneurysm.](image-url)
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, Postcontrast 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. 2–17) 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,
FIGURE 2–16. 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. 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.

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 tangle 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. 2–18). 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. 2–20). 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
Figure 2–17. 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 2–18. 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 2–19. Perimesencephalic cistern arteriovenous malformation depicted on conventional spin echo scans. On proton density (A), T₁-weighted (B), and T₂-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.
draining veins (Fig. 2–21). 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. 2–22). 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.

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
FIGURE 2–22. 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.

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 T1-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).

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. 2–23). 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 transparent-chymal 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. 2–24).

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 2–23.** Infratentorial venous angioma. Precontrast T_{2-} (A) and T_{1}-weighted (B) scans reveal a linear, tubular flow void within the right cerebellar hemisphere. This is better seen on the T_{1}-weighted scan, in which there is also a suggestion of feeding branches. There is no surrounding edema or associated parenchymal abnormality. C, The postcontrast T_{1}-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 2–24.** 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 T_{2}-weighted scan. These two lesions have intermediate signal intensity on the precontrast T_{1}-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 T_{1}-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.
Cavernous Angioma and Capillary Telangiectasia

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. 2–25). Gradient echo imaging, using sequences with high sensitivity to T1* (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.