The value of magnetic resonance imaging (MRI) for assessing intracranial disease was quickly recognized after its clinical introduction in the early 1980s. Advantages of MRI over computed tomography (CT) include superior soft tissue contrast, absence of bone artifact, and ability to acquire high-resolution images in any plane. These features, combined with the variety of available scan types, lead to a highly sensitive and versatile imaging technique. As a result, MRI has become the principal imaging modality for intracranial tumor detection and evaluation.

The sensitivity of unenhanced MRI to detect brain neoplasms is primarily due to its ability to visualize small differences in extracellular fluid. Both $T_1$ and $T_2$, the two time constants describing the relaxation process of protons, are prolonged in most tumors. This leads to a decrease in lesion signal intensity on $T_1$-weighted images and an increase on $T_2$-weighted images. In clinical practice, it is the change on $T_1$-weighted images that is most useful. Detection is possible even when lesions are small or isodense on CT. However, some brain tumors, such as neurofibromas, have only a small increase in water content. These lesions have less pronounced prolongation of $T_1$ and $T_2$.

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

**CONTRAST MEDIA**

The iodinated contrast agents that play an essential role in x-ray-based imaging are not effective at clinical doses in MRI. This would be anticipated because of the difference in physical principles between the two imaging modalities. Iodinated contrast agents attenuate the x-ray beam, whereas MRI contrast agents change (decrease) $T_1$ and $T_2$. It is the shortening of $T_1$ that is most important for contrast enhancement in clinical MRI.

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

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

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

Whether an intra-axial neoplasm displays contrast enhancement depends largely on the degree of BBB disruption. Histologic studies reveal a structural alteration in the capillary walls in most neoplastic disease that allows interstitial accumulation of contrast. Generally, the more aggressive the tumor, the greater is the degree of BBB breakdown and thus contrast enhancement. The degree of vascularity plays an important role in tumors that occur outside the normal BBB (extra-axial neoplasms), including meningiomas, schwannomas, pituitary-origin tumors, and some parasellar tumors such as chordomas. Highly vascular lesions show strong enhancement. Enhancement of intra-axial lesions with BBB disruption occurs more slowly than that for extra-axial lesions with high vascularity. Thus, scans obtained at 3 to 10 minutes postcontrast may best show enhancement in intra-axial lesions (e.g., metastatic disease) as opposed to scans 1 to 2 minutes postcontrast in extra-axial lesions (e.g., acoustic schwannomas).

### IMAGING SEQUENCES

Scans in MRI can be $T_1$-, $T_2$-, or proton density weighted. The latest hardware is also capable of acquiring images with diffusion weighting. Note that all scans are “weighted” in character. This provides, on any one scan, a sense of the parameter in question. However, the appearance of tissues can still be substantially influenced by the other parameters. Construction of an image that is a calculated map of one parameter, for example $T_1$, is possible but rarely done in clinical practice. $T_1$ is defined as the spin-lattice relaxation time and reflects the time required for a proton (a “spin”) to return (or relax), once excited, by the process of giving off energy to the surrounding structure (the “lattice”). $T_2$ is defined as the spin-spin relaxation time and reflects the time required for a proton to relax by giving its energy to a neighboring proton (thus “spin-spin”). Proton or spin density is the quantity of mobile protons (hydrogen atoms), principally water. Diffusion relates to the thermal (random) motion of protons at the molecular level.

Most scans currently in use fall within one of two general categories: spin echo or gradient echo. A third category, inversion recovery, also exists. However, scans of this type are used much less frequently. In a spin echo scan, a radiofrequency pulse is used to generate (refocus) the magnetic resonance (MR) signal from the patient. In a gradient echo scan, a small magnetic field gradient is used to generate the MR signal. $T_1$ (time to echo) and $T_R$ (time to repetition) are operator-selected parameters that specify in spin echo scans the parameter weighting ($T_1$, $T_2$, or proton density). A short TE ($<25$ milliseconds) and short TR ($<800$ milliseconds) produces $T_1$-weighting. A long TE ($>60$ milliseconds) and long TR ($>2000$ milliseconds) produces $T_2$-weighting. Combining a short TE with a long TR gives a proton density-weighted scan. For inversion recovery scans, an additional parameter TI (time to inversion), which highly influences tissue contrast, must be specified along with TE and TR. $T_1$-weighted scans can be recognized by the high signal intensity (white) of fat and low signal intensity (black) of cerebrospinal fluid (CSF). $T_2$-weighted scans can be recognized by the high signal intensity of CSF. Proton density-weighted scans appear in between, with low overall tissue contrast. Proton density-weighted scans are not commonly used today in brain imaging.

The terms fast spin echo and turbo-spin echo refer to a more recent imaging development, a variant of spin echo imaging. With this technique, scan times are generally shorter. Overall image quality is usually also better, as judged by signal-to-noise and spatial resolution. The use of fast spin echo scans does make the interpretation of tissue contrast more difficult. Fat is generally high signal intensity on fast spin echo scans. Thus, whereas on a conventional spin echo $T_2$-weighted scan fat will appear as intermediate to low signal intensity, on a fast spin echo scan it will be high signal intensity. A better marker of $T_1$- and $T_2$-weighting is the gray-white matter ratio. In adults, white matter is of higher signal intensity than gray matter on a $T_1$-weighted scan. The reverse is true on a $T_2$-weighted scan, with gray matter of higher signal intensity.

Correct identification of $T_1$- and $T_2$-weighting, by visual image inspection, has become even more difficult with the advent of a technique known as fluid-attenuated inversion recovery (FLAIR) scanning. In the clinical use of FLAIR in the brain, with the pulse parameters specified to obtain $T_1$-weighting, CSF signal is attenuated (black). This provides markedly improved sensitivity to $T_2$ abnormalities (such as edema), which are seen with high signal intensity. Gray and white matter are relatively isointense, both with lower signal intensity but not as dark as CSF. FLAIR is a type of inversion recovery scan.

With gradient echo scans, in addition to TE and TR, the “tip” or “flip” angle must be specified. Gradient echo scans typically have much shorter TEs and TRs than spin echo scans, with the relationship among TE, TR, and tip angle complex. Both $T_1$- and $T_2$-weighted scans can be produced with gradient echo technique. Only two common applications of gradient echo scans exist in the brain. The first is for improved sensitivity to iron, such as that in deoxyhemoglobin and hemosiderin. The second is for high-resolution three-dimensional (3D) imaging. In the latter application, images can be acquired with a spatial resolution of 1 mm $\times$ 1 mm $\times$ 1 mm. This allows postacquisition high-resolution image reformating in any desired plane.

Contrast enhancement is best visualized on $T_1$-weighted scans. The presence of the gadolinium ion causes a reduction in the $T_1$ of nearby water protons. This leads to an increase in signal intensity or equivalently positive lesion enhancement. It should be noted that the presence of the gadolinium ion actually causes a reduction in both $T_1$ and $T_2$. This is of relevance in first-pass brain imaging, in which the $T_1$ effect of the agent is actually visualized. Although both $T_1$ and $T_2$ are shortened, the $T_1$ shortening is of larger magnitude.
One can only visualize the $T_2$ effect at high concentrations, which occur during the first pass of the bolus through the brain (immediately after intravenous injection).

To appropriately identify all signal intensity abnormalities, both $T_1$- and $T_2$-weighted scans should be obtained before contrast administration. Acquisition of a postcontrast $T_1$-weighted scan then completes the imaging set, with high efficacy for the evaluation of all brain disease. It is strongly recommended that all three scans be acquired in the same plane because this facilitates correlation between images of different weighting. Precontrast $T_1$-weighted scans are also important for the differentiation of lesion enhancement from hemorrhage (methemoglobin) or fat (e.g., a corpus callosum lipoma). Supplemental scans in the coronal and sagittal planes are often useful for further lesion evaluation.

INTRA-AXIAL TUMORS
(SUPRATENTORIAL SPACE)

Astrocytoma

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

Astrocytomas are classified histologically according to several scales: World Health Organization (WHO) grades I to IV, Kernohan grades I to IV, and Rubenstein grades I to III. Higher grade equates with greater malignancy. The Rubenstein classification is simpler to remember and is easier to correlate with imaging findings. According to this scale, a grade I astrocytoma is low grade, grade II is an anaplastic astrocytoma, and grade III is a glioblastoma multiforme (GBM). In the 1993 WHO classification, a distinction is made between lesions that are histologically well circumscribed (grade I) as opposed to diffuse (grades II-IV). Low-grade (grade I) astrocytomas are further divided into specific tumor subtypes, recognizing the favorable prognosis of these lesions, which include juvenile pilocytic astrocytoma and subependymal giant cell astrocytoma. Other WHO grade I nonastrocytic tumors include gangliocytoma, meningioma, and choroid plexus papilloma. In the WHO classification, the best possible grade for a diffuse astrocytoma (the “ordinary” type of astrocytoma seen in adults) is grade II. In this classification scheme, a low-grade astrocytoma is grade II, an anaplastic astrocytoma grade III, and a glioblastoma grade IV.

Although a lower grade (for an astrocytoma) implies a lesser degree of malignancy, the outcome of even these tumors is generally poor because of the infiltrative pattern of growth. Complete tumor resection is often impossible. Grade I astrocytomas carry a uniquely favorable prognosis; the juvenile pilocytic astrocytoma (referred to in the older literature as the cystic cerebellar astrocytoma of childhood) is the most common such lesion. Surgical removal of these tumors usually produces a clinical cure. MRI is the most sensitive imaging modality for detection of astrocytomas. Increased extracellular fluid occurs as a result of abnormal capillary walls. This is easily identified on $T_2$-weighted scans as an area of increased signal intensity. On precontrast scans, this change may be the only or most convincing indication of the presence of a tumor. The change on $T_1$-weighted scans (a decrease in signal intensity) may be subtle. CT, particularly in low-grade astrocytomas, may be nearly normal or show only subtle mass effect.

GBM has the most profound as well as characteristic imaging findings (Fig. 1–1). Thus, it is the most readily

![Figure 1-1. Glioblastoma multiforme (World Health Organization grade IV). A large right frontal lobe mass is noted on precontrast $T_2$- (A) and $T_1$-weighted (B) scans. There is extensive surrounding edema, which is high signal intensity on the $T_2$-weighted scan. Substantial mass effect is noted, with obliteration of sulci, compression of the right lateral ventricle, and displacement of the falx. Irregular rim enhancement is present on the postcontrast $T_1$-weighted scan (C), with no enhancement of the central necrotic portion of the tumor.](image-url)
diagnosed tumor and the least frequently confused with other lesions. GBM usually has substantial mass effect, margin irregularity, and signal intensity heterogeneity on both T₁- and T₂-weighted scans. Low signal intensity on T₁-weighted scans corresponds to necrotic and cystic areas. Necrosis occurs as the tumor outgrows its blood supply. High signal intensity on T₁-weighted scans corresponds to associated, vasogenic edema, typically marked in amount. Hemorrhage may occur in higher grade tumors, frequently petechial in nature. GBMs spread via white matter tracts and frequently cross the corpus callosum to the opposite hemisphere (Fig. 1–2). Bifrontal corpus callosum tumors are referred to as “butterfly” gliomas.

Irregular enhancement of the tumor periphery (“rim”) is seen after contrast administration in many higher grade astrocytomas and reflects the greater degree of BBB disruption. Other patterns of enhancement include homogeneous, garland-shaped, mixed or patchy, linear, and central. These enhancement patterns may occur in any tumor and thus are not grade specific. Occasionally, high-grade tumors will show little or no contrast enhancement. Thus, a completely accurate prediction of tumor grade by imaging appearance is not possible.

Contrast administration should, however, be routinely used in the assessment of all tumors. Because contrast use improves visualization, localization, and tumor margin delineation, a higher level of diagnostic confidence results. This is in part due to the separation of tumor nidus from surrounding edema, the former enhancing and the latter not. Unfortunately, histologic studies show that abnormal contrast enhancement in astrocytomas does not outline the entire extent of the tumor but simply the maximal site of BBB disruption (the area of greatest neovascularity). Astrocytomas are infiltrating lesions, with tumor often present beyond the border indicated by either precontrast T₁-weighted or postcontrast T₁-weighted scans. The area of maximal enhancement does identify the best site for diagnostic stereotactic biopsy. The irregular, finger-like growth pattern of these tumors produces many areas that are relatively uninvolved. If these areas are selected by chance for biopsy, the histologic diagnosis may be normal, although the imaging changes are consistent with tumor. This produces a management dilemma for the surgeon or radiation oncologist.

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

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

WHO grade III or anaplastic astrocytomas usually present with less severe imaging changes compared with GBM (Fig. 1–3). The margins are not as irregular, there is less mass effect, and the signal intensity changes on

![Figure 1–2](image)
Anaplastic astrocytoma (World Health Organization grade III). A, On the precontrast T₂-weighted scan, a midline lesion with intermediate signal intensity is noted. There is subtle low signal intensity on the precontrast T₁-weighted scan (B). Neither scan depicts the lesion itself or its margins well. There is enhancement of the mass on the postcontrast scan (C), which also demonstrates involvement of the splenium of the corpus callosum. On magnetic resonance imaging, anaplastic astrocytomas (WHO grade III), as compared with low-grade astrocytomas (WHO grade II), tend to be less well defined and heterogeneous, with moderate mass effect, and may demonstrate contrast enhancement.

T₁- and T₂-weighted images are not as profound or heterogeneous. Hemorrhage is less frequently found. The degree of enhancement is variable. If the tumor lies near a convexity, enhancement may be difficult to assess without imaging in a second plane. Contrast enhancement often assists in differential diagnosis. Infarction, abscesses, and resolving hematoma should be considered in the differential diagnosis of an anaplastic astrocytoma.

WHO grade II, or low-grade, astrocytomas have the least severe imaging changes (Fig. 1–4). The tumor margin, as identified on imaging, may be relatively smooth or slightly irregular. Mass effect is typically minimal. Cystic changes and necrosis are infrequent, and contrast enhancement usually does not occur. Calcifications are more frequent, as assessed by CT. However, these are not usually seen on MRI. Low-grade astrocytomas may go undiagnosed by CT, particularly if they are located in the temporal lobe. Thus, a patient with temporal lobe seizures and a normal CT should have an MRI for complete evaluation. On occasion, it may be difficult to distinguish these low-grade tumors from infarcts on a single study. Serial studies may be necessary to establish the diagnosis. Infarctions show a decline in mass effect over time and an increase in encephalomalacic changes. Tumors may show little change or a progression in mass effect with time. The major arterial territories should be kept in mind because both anterior and posterior cerebral artery infarctions, being less common, can be mistaken for an astrocytoma.

Ganglieneuromas (Gangliocytomas) and Gangliogliomas
Ganglieneuromas (gangliocytomas) and gangliogliomas share common characteristics in respect to incidence, macroscopic features, and biological behavior. These tumors are composed of mature ganglion cells with varying glial components. At one end of the spectrum is a tumor with mature neurons and scanty stromal glial cells. At the other end is a tumor that at first glance microscopically appears to be a glioma. Ganglioneuromas and gangliogliomas occur most frequently in children and young adults. The temporal lobe is the most common site. These tumors are usually small and well circumscribed. They are often cystic. Occasionally the cystic element dominates, with the tumor itself confined to a mural nodule (Fig. 1–6). These tumors grow slowly. Malignant change is rare. Their small size and good demarcation permit surgical resection in most cases. Prognosis is relatively good. By the WHO classification, gangliocytomas are grade I (with no malignant potential) and gangliogliomas grade I-II.

Oligodendroglioma
Oligodendrogliomas are relatively rare, accounting for about 5% of all intracranial neoplasms. These slow-growing tumors are often large at diagnosis. Oligodendrogliomas tend to involve the anterior cerebrum. They are typically round or oval with fairly well-defined margins (Fig. 1–5).

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

Primitive Neuroectodermal Tumor (PNET)
The term primitive neuroectodermal tumor (PNET) is controversial and refers to a group of tumors thought to originate from undifferentiated neuroepithelial cells.
**Figure 1-4.** Low-grade astrocytoma (World Health Organization grade II). On precontrast T2-weighted (A) fast spin echo and (B) fluid-attenuated inversion recovery scans, a high-signal-intensity abnormality is noted involving the left temporal lobe. The lesion is low signal intensity on the precontrast T1-weighted scan (C) and does not demonstrate abnormal enhancement (D) postcontrast. On magnetic resonance imaging, low-grade astrocytomas appear well defined without substantial mass effect. Unlike higher grade tumors, these lesions usually do not enhance after contrast administration.

**Figure 1-5.** Oligodendroglioma. A large, hyperintense frontal lobe lesion is noted on the precontrast T2-weighted scan (A). The mass demonstrates moderate low signal intensity on the postcontrast T1-weighted scan (B). There is no abnormal contrast enhancement. Calvarial erosion resulting from location and slow growth is clearly depicted on both scans. Contrast enhancement is seen in about half of all oligodendroglialomas, which are typically being mild in degree and inhomogeneous.
There is considerable histopathologic heterogeneity. These tumors are highly malignant and carry a poor prognosis. Local spread, dissemination via the subarachnoid space, and distant metastases are frequent. When cerebellar in location (the most common type), the term medulloblastoma has also been used (this tumor is discussed in detail later). When supratentorial in location, the terms cerebral neuroblastoma and cerebral medulloblastoma have also been used.

Supratentorial PNETs are typically large, well-circumscribed frontal or parietal lesions. The lesion is often dominated by a cystic component, with enhancing tumor located around the periphery (Fig. 1–7). Hemorrhage into the cyst is not uncommon and often leads to clinical presentation.

Lymphoma

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

Lesions not associated with AIDS are typically homogeneous in signal intensity and periventricular in location and enhance (uniformly) after contrast administration. In AIDS, lymphoma may have ring enhancement (Fig. 1–8). Lymphomas may be difficult to distinguish from abscesses, metastases, or glial tumors. Periventricular location and minimal mass effect (little edema) favor lymphoma. Some solid lymphomas have mild hypointensity on T₂-weighted scans.

Metastasis

Metastases comprise almost 40% of all intracranial tumors. The most common tumors that metastasize intracranially are lung, breast, melanoma, colon, and kidney. Multiplicity is the hallmark that distinguishes metastases from gliomas or other primary tumors (Fig. 1–9). Other imaging findings that suggest metastasis are a gray-white matter junction location, a small tumor nidus with a large amount of associated vasogenic edema, and less margin irregularity. MRI is markedly superior to CT for detecting metastatic disease. Contrast administration is mandatory (Fig. 1–10). In one published study, enhanced MRI revealed three times the number of lesions seen by enhanced CT. High-dose contrast administration on MRI provides a further improvement in sensitivity (Fig. 1–11). The multi-institutional study that examined contrast dose found that high dose (0.3 mmol/kg) revealed 32% more metastases compared with standard dose (0.1 mmol/kg). If stereotactic radiation therapy is an option (depending on geographic location of the patient and hospital), high-dose thin-section (5 mm or less) imaging in both the axial and coronal planes should be performed. This approach maximizes lesion detection. Small single metastases can also be missed on a standard dose (0.1 mmol/kg) exam.

The mechanism of enhancement for intra-axial metastases is similar to gliomas in that BBB disruption is
FIGURE 1–10. Brain metastasis (seen only postcontrast). Precontrast T2-weighted fast spin echo (A) and fluid-attenuated inversion recovery (B) scans are normal, as is the precontrast T1-weighted scan (C). D. Postcontrast, a single small enhancing lesion is noted (arrow), which is confirmed on the coronal scan (E). Small brain metastases may not elicit sufficient surrounding vasogenic edema to be recognized on precontrast magnetic resonance scans. Identification of blood-brain barrier disruption, provided by intravenous contrast administration, permits diagnosis of such lesions.

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

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

MRI also surpasses CT in its demonstration of subacute hemorrhage. Metastases with a propensity toward hemorrhage include melanoma, choriocarcinoma, lung carcinoma (oat cell), and kidney, colon, and thyroid
carcinoma (Fig. 1–12). Petechial hemorrhage may be seen in metastases following radiation therapy. Patients receiving chemotherapy occasionally develop coagulopathies. Ensuing intracranial hemorrhage may produce a sudden decline in mental status similar to the effect of a significant hemorrhage into an intracranial metastasis. These hemorrhages may remain undiagnosed by CT, as do many subacute hemorrhages.
Pineal Region Tumors

Pineal region tumors are classified by cell of origin (pineal or germ cell). Germ cell tumors include germinoma, teratoma, and teratocarcinoma. The occurrence of mixed germ cell tumors, with various cellular elements, is common (Fig. 1–13). All occur more frequently in males. Germinoma is the most common of these abnormalities and occurs almost exclusively in males. These tumors may be large and engulf the normal pineal gland. Less heterogeneity in signal intensity is seen in germ cell tumors compared with pineal cell tumors. Intense, homogeneous enhancement occurs. MRI defines the tumor margins better than CT.

Pineal cell tumors include pineocytoma and pineoblastoma (Fig. 1–14). These are less common than other pineal region tumors, particularly germinoma. There is no sex predilection. These tumors may calcify. Pineoblastoma is the more malignant of the two and arises from a more primitive cell type. MRI is particularly helpful in assessing the extent of these rather large, bulky tumors and the degree of involvement of adjacent structures.

Metastases and gliomas may also occur in the pineal region. Obstructive hydrocephalus may accompany large tumors. All but teratomas are notorious for seeding by CSF pathways. Pineal cysts, which are benign, can cause difficulty in differential diagnosis. Typical pineal cysts have signal intensity only slightly different from CSF and demonstrate mild rim enhancement (Fig. 1–15).

INTRA-AXIAL TUMORS
(INFRATENTORIAL SPACE)

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

Astrocytoma

Cerebellar astrocytomas are predominantly tumors of early life (the first two decades). They are one of the most common posterior fossa tumors. Cerebellar astrocytomas are often well circumscribed and tend to be grossly cystic (Fig. 1–16). Anaplasia is uncommon in these lesions. This subtype is usually amenable to surgery. However, some cerebellar astrocytomas are solid tumors; infiltration of surrounding tissues is noted microscopically. Anaplastic change is more common in older patients.

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

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

Brainstem Glioma

Brainstem gliomas generally occur in older children and young adults. Most gliomas of the brainstem are diffusely infiltrating astrocytomas. Symptoms include progressive cranial nerve palsies, extremity weakness, and respiratory difficulty. MRI is markedly superior to CT for visualizing these lesions. With CT, only large extensive lesions are usually recognized. In more aggressive tumors, necrotic or cystic changes can be seen, with low signal intensity on T1-weighted scans and very high signal intensity on T2-weighted scans. The tumor itself, specifically the soft tissue component, is best seen on T1-weighted scans with high signal intensity but not that of CSF or fluid (Fig. 1–17). Contrast enhancement is variable.

Medulloblastoma (Cerebellar PNET)

Medulloblastomas are one of the most common posterior fossa tumors in childhood, with a predilection for males. These embryonal tumors arise in the roof of the fourth ventricle or less commonly in the cerebellar hemisphere of older patients. They are difficult to distinguish from ependymomas, unless the ependymoma extends into the cerebellopontine angle. As with most brain tumors, medulloblastomas have slightly low signal intensity on T1-weighted scans and moderately high signal intensity on T2-weighted scans (Fig. 1–18). Intense contrast enhancement is characteristic. Medulloblastomas are highly malignant and CSF spread is common.

Hemangioblastoma

Hemangioblastomas are histologically benign neoplasms of vascular structures. They may occur at any age but are more frequent in young and middle-aged adults. These tumors are usually solitary and located in the cerebellum. They can occur sporadically or as part of von Hippel–Lindau disease. In the latter, the tumors are typically multiple and patients present in childhood. About half of all hemangioblastomas are cystic, and half...
FIGURE 1–16. Pilocytic astrocytoma (cystic cerebellar astrocytoma, World Health Organization grade I). A large cystic lesion is seen within the cerebellum on precontrast T₂- (A) and T₁-weighted (B) axial scans. A small soft tissue component along the right lateral wall is noted to enhance (C, D) postcontrast. Enhancement of the nodule (arrow) is better seen on the coronal scan (D). With this type of tumor, the enhancing mural nodule corresponds to neoplastic tissue. The cyst wall, which does not enhance, is nonneoplastic.

FIGURE 1–17. Brainstem (pontine) glioma. A, The T₂-weighted axial scan demonstrates a high-signal-intensity expansile mass. The lesion occupies almost the entire pons, leaving only a residual rim of normal tissue. The mass is low signal intensity on the precontrast T₁-weighted scan (B). C, Postcontrast, the more posterior portion of the lesion enhances. On histologic exam, brainstem (pontine) gliomas are often low-grade astrocytomas but have a tendency to undergo anaplastic change. Exophytic extension and cerebrospinal fluid seeding are common.
are solid. A characteristic feature is an enhancing mural nodule (Fig. 1–19). Because these tumors involve the cerebellar hemisphere, the main differential diagnosis is a cystic astrocytoma. Cystic astrocytomas tend to be larger tumors and occur in a younger population.

**INTRAVENTRICULAR TUMORS**

**Colloid Cyst**

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

Colloid cysts are easily diagnosed by MRI because of their location and appearance. Signal intensity characteristics cover the entire spectrum from low to high on both T1- and T2-weighted scans (Fig. 1–20). If the contents are predominantly lipid, the signal intensity will be high on T1-weighted scans and fade to low on T2-weighted scans. Colloid cysts do not enhance.

**Choroid Plexus Papilloma**

Choroid plexus papillomas originate from the ependyma (the lining of the ventricles). These are more common during the first decade of life and show a slight male predominance. Choroid plexus papillomas most frequently arise in the lateral ventricle in children, particularly the left lateral ventricle, and in the fourth ventricle in adults. In the lateral ventricles, hydrocephalus is asymmetric but bilateral and results from outlet obstruc-

**FIGURE 1–18.** Medulloblastoma. A midline mass with heterogeneous, although predominantly high, signal intensity is noted on the T2-weighted axial scan (A). B, The midline T1-weighted sagittal scan demonstrates the mass to fill the fourth ventricle. The brainstem is displaced anteriorly and the inferior aspect of the cerebral aqueduct widened. Leptomingeal metastases were noted on the thoracic and lumbar magnetic resonance examinations performed on the same date (images not shown).

**FIGURE 1–19.** Hemangioblastoma. On the axial scan with intermediate T2-weighting (A), a high-signal-intensity lesion is noted within the posterior fossa. There is mass effect with compression of the fourth ventricle. The lesion is slightly higher in signal intensity than cerebrospinal fluid on this scan and the precontrast T1-weighted scan (B), suggesting a neoplastic origin. C, Postcontrast, there is enhancement of a small mural nodule (arrow), with a large prominent vein also identified adjacent to the mass. The most common appearance for a hemangioblastoma is that of a cystic mass with a peripheral mural nodule. Tumor vessels may also be apparent. Less commonly, these lesions present as solid masses.
tion of the ventricle, overproduction of CSF, or a combination of these two factors. Intermittent hemorrhage into these tumors is not uncommon and may contribute to the obstructive hydrocephalus. In fourth ventricular lesions, hydrocephalus is symmetric.

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

Ependymoma

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

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

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

Meningioma

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

EXTRA-AXIAL TUMORS

Meningioma

Meningiomas are the most common extra-axial adult tumor, comprising about 15% of all intracranial neoplasms. These tumors are more frequent in women between the ages of 40 and 70 years. The most common location is high over the convexity adjoining the superior sagittal sinus in its middle or anterior third (Fig. 1–21). Other sites, in decreasing order of frequency, are the lateral convexity, sphenoid ridge, olfactory groove, suprasellar-parasellar region, and posterior fossa (petrous bone, clivus, and foramen magnum). When these tumors are multiple or occur in childhood, they are usually associated with neurofibromatosis. Meningiomas are typically benign, slow-growing tumors that compress rather than invade adjacent brain tissue. Occasionally, more aggressive changes are seen such as dural sinus or bone invasion. With such changes, complete resection
BRAIN: NEOPLASTIC DISEASE

Figure 1–21. Falx meningioma. A soft tissue mass that is isointense to the brain is noted adjacent to the falx on precontrast T2- (A) and T1-weighted (B) scans. There is mild mass effect. C, Postcontrast, the mass is easily identified as a result of intense enhancement.

may not be possible, and recurrences are more likely to occur.

Regardless of location, meningiomas usually have a broad base that lies along a bony or dural margin. Features characteristic of extra-axial lesions are seen, including arcuate bowing of the white matter resulting from compression of the brain, a low-signal-intensity interface with brain on T1-weighted scans (caused in part by displacement of pial vessels), and a CSF cleft between the lesion and brain, seen best on T2-weighted images (Fig. 1–22). Displacement of the dura at the lateral margin of the lesion can be seen on occasion, more commonly with cavernous sinus lesions. Meningiomas are typically highly vascular; calcifications and cystic changes produce intrinsic tumor mottling. These findings are more obvious at higher field strengths perhaps because of differences in magnetic susceptibility. Meningiomas have a variable amount of associated edema. Occasionally, this edema will be the only evidence for the presence of a lesion on precontrast scans.

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

Meningiomas often invade adjacent dural sinuses (Fig. 1–23). MRI venography and postcontrast T1-weighted imaging are two effective ways to demonstrate sinus invasion. MRI venography is usually performed before contrast administration. Two-dimensional (2D) time-of-flight technique is used, depicting venous flow as high signal intensity. Sinus invasion is diagnosed on the basis of the irregular contour of the sinus, presence of a signal void within the sinus, or absence of flow (with occlusion). On postcontrast T1-weighted scans, the venous sinus also has high signal intensity. Signs of sinus invasion are similar to that on MRI venography, except that the tumor is depicted as an enhancing soft tissue.

Figure 1–22. Convexity meningioma. A soft tissue frontal lesion of slightly higher signal intensity than adjacent brain is noted on the precontrast T1-weighted scan (A). The mass is adjacent to both the falx and the calvarium. Erosion of the calvarium is evident on comparison of the diploic space from side to side. A cerebrospinal fluid cleft is seen posterior to the lesion, demarcating its extra-axial location. Intense uniform enhancement is seen on the postcontrast T1-weighted scan (B).
mass (although with lower signal intensity than that of venous blood). MRI is more sensitive in detecting sinus invasion than either CT or x-ray angiography.

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

En plaque meningiomas represent a special and often clinically frustrating type of meningioma (Fig. 1–24). These may become extensive, with involvement of the tentorium, cavernous sinus, brainstem, and cranial nerves. Transdural and subperiosteal spread may also occur. Total resection is often not possible, leading to recurrence and relentless enlargement. These lesions are also often not well seen, or go undetected, by CT.

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

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

**Acoustic Schwannoma**

Acoustic schwannomas (commonly and incorrectly referred to as “neuromas”) are benign tumors that arise from the neurilemmal sheath of the vestibular division of cranial nerve VIII. Patients are usually 40 to 60 years of age and have unilateral sensorineural hearing loss and tinnitus. Larger tumors with brainstem involvement cause unsteadiness, ataxia, vertigo, and diminished corneal reflexes (Fig. 1–25). Acoustic schwannoma is the most common benign extra-axial tumor of the posterior fossa.

On its clinical introduction, MRI rapidly replaced other imaging techniques for the diagnosis and evaluation of these tumors. Polytomography, iophendylate cisternography, and air-contrast CT were former techniques that involved significant radiation to the patient.
The latter two were also invasive, adding to patient morbidity. With MRI, the lack of signal from the adjacent dense bone allows direct visualization of cranial nerves VII and VIII. Thin-section (≤3 mm), high-resolution images are, however, necessary for appropriate diagnosis and evaluation of IAC tumors.

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

Contrast enhancement is important not only for assessing tumor extent but also for detecting small intracanalicular acoustic schwannomas (Fig. 1–26). Precontrast scans alone may miss small lesions within the IAC. Postcontrast, these are seen as brightly enhancing small soft tissue masses. The normal cranial nerve VIII does not enhance. Thus, any contrast enhancement in this region is abnormal. The degree of enhancement seen with acoustic schwannomas is greater than that for any other intracranial tumor. Enhancement is due to intrinsic lesion vascularity.

For accurate assessment, thin-section T₁-weighted scans pre- and postcontrast in both the axial and coronal planes are highly recommended in addition to a precontrast thin-section T₂-weighted scan. MRI without contrast enhancement can produce both false-negative and...
Intracanalicular acoustic schwannoma. On precontrast T2- (A) and T1-weighted (B) scans, the question of a right-sided intracanalicular lesion is raised. C, Postcontrast, there is intense lesion enhancement (arrow), permitting definitive diagnosis. The clinical presentation was that of right-sided sensorineural hearing loss. Other entities to be considered in differential diagnosis include facial (seventh) nerve tumor and inflammatory disease, although the latter should not result in a mass lesion.

false-positive results. In one series, the combination of these errors affected 10% of patients studied. Small intracanalicular tumors that went undetected without contrast could be seen with contrast. More alarming is the prospect of suggesting a tumor on precontrast scans when none can be found postcontrast. This can occur when the nerve appears (erroneously) to be enlarged on T1-weighted scans. Also, ectasia of the IAC can produce signal intensity on T2-weighted scans indistinguishable from that of intracanalicular tumor.

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

T2-weighted scans are not essential for the imaging evaluation of acoustic schwannomas. However, they are important for the differential diagnosis. Many abnormalities can mimic cranial nerve VIII disease clinically, and these are often better visualized with T1-weighted scans. Examples include multiple sclerosis, mastoiditis, and vascular brainstem compression.

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

Epidermoid

Epidermoids (cholesteatomas) result from incomplete cleavage of neural from cutaneous ectoderm, with inclusion of ectodermal elements at the time of neural groove closure. Both midline (suprasellar and intraventricular) and more eccentrically located (cerebellopontine angle) lesions occur; the latter result from an inclusion at a slightly later stage of embryogenesis (Fig. 1–27). Epidermoids grow by desquamation of epithelial cells, which break down into keratin and cholesterol within the tumor capsule. These fatty elements are soft and pliable, and in the slow accumulation process they conform to the shape of the subarachnoid space or ventricle. The lesions are fairly well demarcated. Compression of adjacent structures occurs late. These congenital tumors may not become symptomatic until patients are 25 to 30 years old. Rupture occasionally produces chemical meningitis. As with other lipid tumors, their appearance on MRI depends on the type of fat and its physical state. Many contain cholesterol and show a prolongation of both T1 and T2 relaxation times. Such lesions are low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. A difference in the fat content or physical state yields brighter signal intensity on T1-weighted images. These tumors do not enhance. Thus, contrast administration is of little diagnostic value, except to exclude other cerebellopontine angle lesions with similar precontrast signal intensity (e.g., some meningiomas).

Dermoid

Dermoids are congenital tumors, like epidermoids, that arise from inclusion of ectodermal elements at the time
FIGURE 1–27. Epidermoid. An extra-axial mass with heterogeneous, but slightly higher signal intensity than cerebrospinal fluid (CSF), is noted in the right cerebellopontine angle cistern on an intermediate T₂-weighted scan (A). The difference in signal intensity between the lesion and CSF is not apparent on a heavily T₂-weighted scan (B). The mass is best demarcated on the precontrast T₁-weighted scan (C). On this scan, the mass can again be differentiated from adjacent CSF, the latter with slightly lower signal intensity. The mass compresses the right middle cerebellar peduncle and right cerebellar hemisphere. There was no enhancement postcontrast (not shown).

of neural groove closure. The presence of hairs and other skin appendages differentiates a dermoid from an epidermoid tumor. Dermoids arise near the midline and are less common than epidermoids. Most intracranial dermoids are located in the posterior fossa. Most spinal canal dermoids occur in the lumbosacral region. Dermoids may contain fat, hair follicles, and glandular elements (sebaceous and apocrine). Those containing a large amount of fatty elements have high signal intensity on T₁-weighted scans and lower signal intensity on conventional T₂-weighted scans (Fig. 1–28). Dermoids are not vascular tumors and do not cause BBB disruption. Thus, they do not enhance after contrast administration.

Arachnoid Cyst

Arachnoid cysts are benign lesions that contain CSF. Most are congenital in origin. Less common causes include inflammation, trauma, and subarachnoid hemorrhage. Their importance lies in differentiation from other masses, including epidermoids, dermoids, subdural hygromas or hematomas, and cystic tumors. Arachnoid cysts most frequently occur in the middle cranial fossa. Other common locations include the posterior fossa (retro cerebellar) (Fig. 1–29), the suprasellar region, the quadrigeminal plate, and the cerebral convexities. The cyst is lined by arachnoid membrane and filled with fluid.
FIGURE 1–29. Posterior fossa arachnoid cyst. Sagittal (A) and axial (B) T₂-weighted scans reveal a lesion, with cerebrospinal fluid signal intensity, posterior to the vermis and right cerebellar hemisphere. Mass effect is evident both by the anterior displacement of the vermis and the upward bowing of the posterior portion of the tentorium.

that is usually clear but on occasion slightly xanthochromic. The margins of an arachnoid cyst are sharply defined. The signal intensity is usually identical to that of CSF. No contrast enhancement occurs.

**Leptomeningeal Metastases**

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

Leptomeningeal metastases are not well seen by CT. Before approval of the gadolinium chelates, the same was true for MRI. Currently, contrast-enhanced MRI is the technique of choice for the diagnosis of leptomeningeal disease. In the brain, leptomeningeal metastases are visualized as abnormal contrast enhancement, linear or nodular in character, lining the meningeal surface and extending into sulci and cisterns (Fig. 1–30).

**PITUITARY AND PARASELLAR REGION TUMORS**

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

FIGURE 1–30. Meningeal carcinomatosis. Scans were taken 1 year after surgical resection and whole brain radiation for a right occipital metastasis from breast carcinoma. Abnormal high signal intensity, without a specific focal lesion, is noted in the right parietal and occipital lobes on the precontrast T₂-weighted scan (A). No additional information is provided by the precontrast T₁-weighted scan (B). C. After contrast administration, recurrent tumor is identified, marked by intense enhancement, along the surface of the brain in the area of prior resection.
The identification and characterization of lesions in the sella and parasellar region require thin-section imaging (≤3 mm). Spin echo technique typically can provide no thinner than 2-mm sections, whereas 3D gradient echo technique can provide 0.5- to 1-mm sections. The latter technique is also advantageous in that the slices are truly contiguous without an intervening gap. Images from a high-resolution 3D data set can be reformatted in multiple planes, further improving the diagnostic value of the exam. For gradient echo scans, TRs should be short (1 – 5 milliseconds) to avoid susceptibility (“blow-out”) artifacts at the air-soft tissue interface between the sella and the sphenoid sinus.

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

**Normal Pituitary Gland**

The size of the normal pituitary gland varies widely. A height of 10 mm is considered the upper limit of normal, with two exceptions. During puberty and the early child-bearing years, the gland may be up to 12 mm in height. The upper surface of the gland may be flat, concave, or convex in the midline. T₁-weighted images provide excellent anatomic definition. In the coronal plane, the pituitary is localized as a soft tissue structure lying between the rounded areas of signal void from the internal carotid arteries. The signal intensity of the gland is similar to the white matter of brain. In the sagittal plane, the anterior and posterior lobes of the pituitary can be distinguished by the high signal intensity of the posterior lobe. Immediately posterior to the pituitary itself is the high-signal-intensity marrow of the dorsum sellae. Frequently, there is a normal area of increased signal intensity on T₁-weighted images at the base of the pituitary. This may be mistaken for an abnormality but actually represents fatty marrow in the upper extreme of a sphenoid sinus septum.

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

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

**Microadenoma**

Microadenomas are defined as lesions smaller than 10 mm in diameter. Production of hormones brings these lesions to clinical attention early and thus when small. The most common microadenoma is the prolactinoma. These tumors secrete prolactin and present with infertility, amenorrhea, and galactorrhea in women and galactorrhea and impotence in men. Imaging findings include focal asymmetry of the gland surface, displacement of the pituitary stalk to the contralateral side, and a low-signal-intensity focal mass on T₁-weighted scans. Hemorrhage within the lesion may cause high signal intensity on precontrast T₁-weighted scans. On T₂-weighted scans, prolactinomas can be hypo-, iso-, or hyperintense. On early postcontrast scans, most prolactinomas are hypointense compared with the normal pituitary and infundibulum (which both enhance intensely). Contrast injection thus facilitates lesion detection (Fig. 1–31). A small number of tumors are isointense to the normal pituitary precontrast and hypointense postcontrast. With thin-section, high-resolution (small field of view) scans, evaluation of these tumors by MRI is superior to that by CT.

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

**Macroadenoma**

Large pituitary adenomas are rarely a diagnostic dilemma for CT or MRI. These bulky tumors are usually
hormonally inactive, with a few tumors secreting prolactin. Because of the improved depiction of soft tissue, MRI can better assess suprasellar and lateral temporal extension (Fig. 1–32). The cavernous sinus can be displaced by tumor or on occasion can be invaded with encasement of the internal carotid artery. Macroadenomas are isointense with white matter on T1-weighted images, unless there is associated hemorrhage. Subacute hemorrhage in most tumors, including macroadenomas (with high signal intensity on T1-weighted images), is better demonstrated by MRI than CT. Hemorrhage within macroadenomas is more common than was once thought based on CT and clinical criteria. Pituitary apoplexy is defined as spontaneous hemorrhage into or ischemic necrosis of a normal pituitary or an adenoma. Before MRI, pituitary apoplexy was equated with severe neurologic symptoms, including sudden alteration in mental status and occasionally blindness. It is now known from MRI that small hemorrhages may be accompanied by no more than a severe headache.

On T1-weighted images, macroadenomas have intermediate, homogeneous signal intensity. Necrosis causes foci of high signal intensity. If the necrotic portion is substantial in size, differentiation from a craniopharyngioma can be difficult. A distinguishing feature is the size of the sella, usually substantially enlarged with a macroadenoma.

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

**Craniopharyngioma**

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

Craniopharyngiomas are usually predominantly cystic with a small soft tissue component (Fig. 1–33). Most craniopharyngiomas are very low signal intensity on T1-weighted scans because of a large cystic component containing relatively clear fluid. High signal intensity on precontrast T1-weighted scans can also be seen resulting from high cholesterol content or byproducts (methemoglobin) from previous hemorrhage. The cystic portion...
of the tumor is usually very high signal intensity on T2-weighted scans.

Suprasellar craniopharyngiomas vary from small lobulated to large multicystic septated lesions. Tumor margins are usually smooth and rounded. Craniopharyngiomas in children tend to be larger and contain more calcification. Postcontrast, the cyst walls of a craniopharyngioma enhance. There may also be areas of nodular enhancement. Contrast-enhanced scans aid in differential diagnosis. The normal pituitary, which enhances brightly, can be separated from suprasellar tumor or from tumor that partially extends into the sella. Contrast-enhanced scans also aid in visualization of craniopharyngiomas that are not large enough to obliterate the suprasellar cistern. In this case, the signal intensity of the tumor blends with the signal intensity of the suprasellar cistern on T1-weighted studies. Although calcifications are not well visualized on MRI, the location of the lesion and dominant cystic component, along with the presence of septations and lobulations, enables correct diagnosis in most instances.

Other Parasellar Tumors

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

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

TUMORS OF BONE

Chordoma

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

Thirty-five percent of chordomas are intracranial, and most of these arise from the clivus. Fifty percent are sacrococcygeal, and 15% arise from within a vertebral body. Within the calvarium, chordomas may involve the posterior or middle fossa by extension through the dura. The majority of these tumors cause extensive destruction of bony structures. Chordomas rarely metastasize to distant sites but are locally aggressive. Total surgical resection is rarely possible. Although locally invasive, chordomas are histologically benign. Macroscopically, chordomas are soft gelatinous tumors that frequently result in destruction of the clivus and skull base. They occur most commonly in men in the third and fourth decades. Patients present with headaches, facial pain, progressive cranial nerve palsies, and nasal stuffiness.

Calcification is identified in 50% to 60% of cases on CT. On MRI imaging, chordomas are usually well-defined, extra-axial tumors that show isointensity or mild hypointensity on T1-weighted images and moderate to extreme high intensity on T2-weighted images. Approximately 70% of chordomas have septations of
low signal intensity separating lobulated areas of higher signal intensity on T2-weighted images. Chordomas typically enhance after contrast administration.

**Metastases**

The normal diploic space does not enhance, except for diploic veins and the meninges near pacchionian granulations. Diploic veins appear as linear or small round (if cut in cross-section) foci of low to moderate signal intensity on precontrast MRI images, with enhancement postcontrast. The diploic space can appear inhomogeneous with areas of increased (resulting from fatty marrow) and decreased (caused by bony sclerosis or suture lines) signal intensity on precontrast scans. However, the diploic space should be symmetric from side to side. Gross asymmetry is highly suggestive of calvarial disease, even in the absence of appreciable destruction of the inner or outer table. Calvarial metastases enhance after intravenous contrast administration (Fig. 1–34).

**Eosinophilic Granuloma**

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

Unifocal Langerhans cell granulomatosis is a disease of children and young adults, predominantly males, who present with a solitary osteolytic lesion (most often in the femur, skull, vertebrae, ribs, or pelvis). Diagnosis requires biopsy; treatment is simple excision. The typical presentation in neuroradiology is that of a solitary skull lesion. MRI demonstrates a soft tissue mass, centered in the diploic space, with adjacent bone destruction (Fig. 1–35). The lesion may extend into the epidural or subgaleal space. Eosinophilic granulomas enhance prominently postcontrast.

Multifocal Langerhans cell granulomatosis also presents in childhood, with multiple bony lesions in virtually any site. Diabetes insipidus occurs in one third as a result of hypothalamic involvement. The term Hand-Schüller-Christian syndrome was previously used to refer to the disease triad of destructive bone lesions, diabetes insipidus, and exophthalmos. However, only 25% of patients with multifocal Langerhans cell (eosinophilic) granulomatosis have this triad, which can also be caused by malignant lymphoma and carcinoma. Although benign, multifocal disease is treated with methotrexate, vinblastine, or prednisone.

**POSTOPERATIVE TUMOR EVALUATION**

The evaluation of tumors that recur after surgery is difficult without contrast administration. Subtle mass effect and postsurgical encephalomalacia are difficult to assess in regard to the question of tumor recurrence. Differentiation of encephalomalacia from edema associated with tumor recurrence is also difficult. Both have increased signal intensity on T2-weighted scans. Extension of abnormal high signal intensity into the corpus callosum, without volume loss, is, however, specific for tumor. Edema does not track into corpus callosum because of the compact nature of the nerve fibers. Cystic, necrotic, and hemorrhagic changes are well seen on precontrast T1-weighted scans. However, underlying tumor may be difficult to detect. Contrast-enhanced MRI is markedly superior to enhanced CT for demonstrating tumor recurrence (Fig. 1–36). One study demonstrated that 50% of postoperative tumor recurrences were primarily or more conclusively shown by contrast-
FIGURE 1–35. Eosinophilic granulomatosis. An expansile diploic space mass is identified on axial T2- (A) and sagittal T1-weighted (B) images. On axial (C) and coronal (D) postcontrast scans, there is a thick peripheral rim of abnormal contrast enhancement. The sagittal and coronal scans demonstrate focal expansion of the diploic space. Differential diagnosis plays an important role in scan interpretation in this instance, with imaging findings (a solitary lesion) and clinical information (a young man with headaches and a “bump” on his head) favoring a diagnosis of eosinophilic granuloma (proven by subsequent resection).

FIGURE 1–36. Recurrent astrocytoma. A large postsurgical defect, communicating with the atria of the right lateral ventricle, is noted on precontrast T2- (A) and T1-weighted (B) scans. The exam was performed to rule out tumor recurrence in this elderly patient with resection of an astrocytoma 4 years earlier. Medial to the postsurgical defect is soft tissue with signal intensity similar to that of normal brain. The question of tumor recurrence is raised by the slight hyperintensity of this soft tissue on the precontrast T1-weighted scan. C, After contrast administration, there is intense enhancement, making possible definitive diagnosis of recurrent tumor.
enhanced MRI. Identification of recurrent tumor and delineation of the margin of tumor extent were both improved. Caution should be used, however, in the interpretation of tumor recurrence after radiation therapy. Both recurrent tumor and radiation necrosis can present as an enhancing lesion with surrounding edema and mass effect. These two entities cannot be differentiated on the basis of conventional MRI techniques. Regional cerebral blood volume (CBV) studies do offer the capability of distinguishing recurrent tumor (with high CBV) from radiation necrosis (with low CBV). This advanced type of study is performed by acquiring rapid images (on the order of one per second) after bolus contrast injection using a power injector during the first pass of the contrast agent through the brain.