NORMAL CERVICAL SPINE

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

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

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

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

Three major arteries supply the spinal cord and lie along its surface. One is anterior (the anterior spinal artery, which supplies 70% of the cord) and two are posterior (the posterior spinal arteries, which together supply 30% of the cord). In the cervical region, several radicular arteries supply the anterior spinal artery. In the thoracolumbar region, the artery of Adamkiewicz, which arises from a lower intercostal or upper lumbar artery, supplies the anterior spinal artery.

Magnetic resonance imaging (MRI) has the capability of providing flexion and extension views in the cervical spine. These scans can be of substantial clinical value. Flexion and extension views are typically acquired with some sort of rapid imaging technique, of which today there is a plethora. A very simple scheme, which can be used for rapid image acquisition on most scanners, is to acquire the T1-weighted scan with a reduced number of phase-encoding steps and the T2-weighted scan with FSE technique. Depending on the available coils, the range of possible motion may be limited. Flexion and extension views have substantial use in the demonstration of spinal cord compression not visualized in the neutral position (e.g., with rheumatoid arthritis) and in the evaluation of potential instability. The latter can be an important diagnostic question both after trauma and in chronic inflammatory disease (particularly at the occipitoatlantal and atlantoaxial levels).

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

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

SPINAL STENOSIS

Congenital

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

Degenerative (Acquired)

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

Neuroforaminal (Uncovertebral Joint) Spurring

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

Ossification of the Posterior Longitudinal Ligament

Ossification of the posterior longitudinal ligament is an uncommon cause of acquired spinal stenosis. More common causes include ligamentous and facet joint hypertrophy. Ossification of the posterior longitudinal ligament is more common in the oriental population. Patients are at risk for traumatic spinal cord injury. Multilevel involvement is typical. The ossified posterior longitudinal ligament will be very low signal intensity on both T1- and T2-weighted scans but may contain centrally intermediate- to high-signal-intensity soft tissue (fat and marrow).

CONGENITAL DISEASE

Klippel-Feil Syndrome

In the Klippel-Feil syndrome, there is congenital fusion of two or more cervical vertebrae, most commonly C2-3 and C5-6. At the affected levels, the intervertebral disk is absent. About half of all patients with Klippel-Feil syndrome demonstrate the classic triad, which consists of limited neck motion, a short neck, and a low posterior hairline. Common associated abnormalities include deafness, congenital heart disease, Sprengel’s deformity (elevation and rotation of the scapula), and uro-
FIGURE 6–1. Spinal stenosis, degenerative (acquired) in origin. A, On the sagittal fast spin echo T₂-weighted scan, there is encroachment anteriorly on the thecal sac by disk bulges and osteophytic spurs at the C4–5, C5–6, and C6–7 levels. In degenerative spinal stenosis of the cervical spine, these are the most commonly affected levels. Multilevel involvement, as in this case, is also common. The same findings are apparent on the sagittal gradient echo T₂-weighted scan (B). The latter scan is easily identified by the low signal intensity of vertebral body marrow, which is due to magnetic susceptibility effects. The osteophytic spurs are well visualized on the sagittal T₁-weighted scan (C), although the encroachment on the thecal sac is less evident. On axial imaging, the asymmetry of the canal compromise in this patient is clearly seen, together with the cord flattening and deformity. As with imaging in the sagittal plane, on axial imaging (D) the T₂-weighted scan depicts the interface between soft tissue and cerebrospinal fluid better than the T₁-weighted scan (E).
Figure 6–2. Degenerative stenosis of the cervical spinal canal, with both anterior and posterior compression. The patient is a 69-year-old woman with neck pain and intermittent numbness and weakness in both arms. A, The sagittal T2-weighted scan reveals canal compromise at the C2–3 through C6–7 disk space levels. Disk bulges and osteophytic spurs cause compression anteriorly on the thecal sac at the C3–4 level and below. At both C2–3 and C3–4, facet hypertrophy causes posterior compression. The subarachnoid space is obliterated at multiple levels, with accompanying cord deformity (flattening). There is mild reversal of the normal cervical lordosis in the lower cervical spine. B, The postcontrast T1-weighted image demonstrates thin curvilinear high signal intensity (enhancing epidural venous plexus) along the posterior margins of the vertebrae. With contrast enhancement, the true canal dimensions are better visualized. The failure to clearly visualize epidural soft tissue is one reason that precontrast T1-weighted scans are generally less useful than T2-weighted scans in imaging cervical degenerative disease (spondylosis). Disk space narrowing is identified at the C4–5 through the C6–7 levels on the T1-weighted scan. The disk bulges and spurs are also clearly seen, as on the T2-weighted scan.

Figure 6–3. Neuroforaminal narrowing caused by uncovertebral joint bony spurs. The patient is 54 years old and presents with neck and bilateral arm pain. A, The midline sagittal T2-weighted scan reveals disk bulges and osteophytic spurs at C3–4, C5–6, and C6–7 with effacement of the ventral subarachnoid space at each level. Because of their oblique orientation, the neural foramina are not well visualized in the cervical spine on sagittal images. Although the foramina would be best depicted on oblique scans, axial scans are used in most clinical practices for their assessment. B, The axial gradient echo T2-weighted scan in this case at the C6–7 level reveals narrowing of the right neural foramen as a result of hypertrophic changes and sclerosis (with accompanying bony spurring) of the right C6–7 uncovertebral joint.
logic abnormalities. Other less frequently associated anomalies include syringomyelia and diastematomyelia. There are three types, defined on the basis of the extent and location of vertebral fusions. In type I, there is extensive cervical and thoracic fusion. In type II, the most common (Fig. 6–4), there are one or two cervical fusions; there may also be associated hemivertebrae and occipitooatlantal fusion. Type III is defined as type I or II with additional lower thoracic or lumbar fusions.

Clinically, patients with Klippel-Feil syndrome are often asymptomatic from a neurologic point of view. They can, however, have cord or nerve root compression. Patients with Klippel-Feil syndrome are predisposed to spinal cord injury after minor trauma. Patients may have hypermobility (and thus instability) between the unfused segments.

Abnormalities Involving the Cerebellar Tonsils

Ectopia

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

Chiari Type I

In the Chiari type I malformation, the cerebellar tonsils are low lying and pointed or wedge shaped (Fig. 6–5).
Figure 6–5. Chiari type I malformation. The patient is an 11-year-old with severe scoliosis. Fast spin echo T2-weighted (A) and spin echo T1-weighted (B) midline sagittal images reveal that the cerebellar tonsils are abnormally low in position (these extend 11 mm below the foramen magnum). The tonsils have also lost their usual globular configuration and are pointed (or wedge shaped) in appearance. Because of the patient’s scoliosis, the lower cervical spine is seen in a parasagittal plane. The fourth ventricle is normal in shape and position, an important negative finding. C, The T1-weighted axial view at the level of the arch of C1 confirms the abnormally low position of the cerebellar tonsils, which are wedged posteriorly and laterally. These compress and deform the spinal cord (anteriorly) at the cervicomедullary junction.

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

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

Chiari Type II

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

Chiari Type III

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

Basilar Invagination

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

Os Odontoideum

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

Neurofibromatosis

There are two major types of neurofibromatosis (NF). Both are autosomal dominant, but type 1 (NF1) is much more common. The abnormality has been localized to chromosome 17 in NF1 and to chromosome 22 in NF2. Distinctive physical exam findings in NF1 include café au lait spots and iris hamartomas (Lisch nodules). Findings on MRI of the spine include scoliosis, a patulous dural sac, lateral meningoceles, and neurofibromas of the exiting nerve roots. Findings on MRI of the spine in NF2 include intradural extramedullary lesions.
Os odontoideum, with stable fibrous union. This 46-year-old is being seen for neck pain after a car accident. A, On the sagittal T1-weighted scan, the tip of the dens appears separate from the base. There is intervening intermediate signal intensity soft tissue. The marrow signal intensities of both the tip and the base are normal. The anterior arch of C1 is large and has a convex, not concave, posterior margin. B, On the sagittal fast spin echo T2-weighted scan, no soft-tissue edema is noted. The cervical canal is normal in caliber. T1-weighted sagittal images obtained in flexion (C) and extension (D) reveal no change in the distance between the tip of the dens and the anterior arch of C1. No cord compression is noted. The normal marrow and soft-tissue signal intensity seen on magnetic resonance imaging makes acute trauma very unlikely, with substantial edema otherwise anticipated. E, The lateral radiograph confirms the nonunion of the superior dens with its base. The superior fragment (arrow) is well corticated.
(neurofibromas and meningiomas) and intramedullary lesions (ependymomas and low-grade astrocytomas) (Fig. 6–9). The presence of bilateral acoustic neuromas on imaging of the head is considered pathognomonic of NF2. These patients may also have schwannomas, meningiomas, gliomas, and hamartomas of the brain. Peripheral nerve lesions, either solitary or involving multiple nerves in plexiform manner, are considered hallmarks of NF, but these are less commonly seen on MRI because of the focus of the exam being the brain or spine.

**FIGURE 6–9.** Neurofibromatosis 2. A, The midline sagittal T2-weighted image reveals two intramedullary lesions (likely either ependymomas or low-grade astrocytomas) with abnormal high signal intensity. One lesion is at the level of C3 and the other at the cervicomedullary junction. A third lesion, at C2, was better seen on adjacent slices. B, The midline sagittal T1-weighted image reveals an extramedullary mass with soft tissue signal intensity along the posterior margin of the thecal sac anterior to the posterior arch of the C1. The mass causes mild deformity of the upper cervical cord. C, On the postcontrast sagittal T1-weighted image, two foci of abnormal intramedullary enhancement are seen (corresponding to the abnormalities noted on the T2-weighted scan): one within the cervical spinal cord and one at the cervicomedullary junction. The cord shows mild enlargement at the lesion sites. An intradural extramedullary enhancing mass (a meningioma) with a broad base abutting the dura is also seen along the posterior thecal sac just below the margin of the foramen magnum. D, An axial contrast-enhanced T1-weighted image through the posterior fossa reveals bilateral (enhancing) acoustic schwannomas. Other images through the brain (not shown) demonstrated multiple meningiomas.

**INFECTION AND INFLAMMATORY DISEASE**

**Epidural Abscess**

Causes for an epidural abscess include hematogenous spread, direct extension, and penetrating trauma. *Staphylococcus aureus* is the most common organism. On MRI, thickened inflamed soft tissue is seen initially, which progresses to a frank abscess with a liquid center (Fig. 6–10). Depending on the stage of disease, the enhance-
FIGURE 6–10. Epidural abscess. A cervical epidural catheter had previously been placed (now removed) for management of chronic left upper extremity pain. A. The T₂-weighted axial scan reveals anterior displacement of the thecal sac. B. The T₁-weighted scan raises the question of a posterior soft tissue mass. C. Postcontrast, an epidural fluid collection (arrow) is noted, with prominent enhancement of surrounding soft tissue. These findings are confirmed on the sagittal T₂ (D), T₁ (E), and postcontrast T₁-weighted (F) scans. Contrast use permits identification of the fluid pocket, with surrounding inflammatory change (F, arrow), indicating the diagnosis of infection.

ment on MRI after contrast administration can be homogeneous or rimlike with central low signal intensity (pus). An epidural abscess may cause cord compression as a result of the presence of inflammation, granulation tissue, or pus.

Sarcoidosis
Sarcoidosis is a noncaseating granulomatous disease of unknown cause. The CNS is involved clinically in 5% of patients. The basal leptomeninges and floor of the third ventricle are the most common sites of involvement. Spinal cord involvement is much less common. MRI findings in sarcoidosis of the spine include fusiform cord enlargement, nodular parenchymal enhancement (broad based along the cord surface), and thin pial enhancement. Treatment is with steroids. Follow-up scans may demonstrate a return to normal appearance.

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

the atlas and dens (with instability), erosion of the dens (by surrounding inflammatory pannus), a retrodental soft tissue mass (resulting from involvement of the transverse ligament), and settling of the skull on the atlas. Rheumatoid arthritis, with involvement of the atlas and dens, can lead to cord compression.

BENIGN FOCAL LESIONS
Osteochondroma

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

Aneurysmal Bone Cyst

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

**Eosinophilic Granuloma**

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

**Cavernous Angioma**

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

The typical cavernous angioma is small and smoothly marginated on imaging studies (Fig. 6–12). The border or rim of the lesion is markedly hypointense on T2-weighted scans as a result of hemosiderin and ferritin deposition within macrophages after hemorrhage. Centrally, a cavernous angioma contains a honeycomb of vascular spaces separated by fibrous stands, which appears as a mixture of high and low signal intensity on T2-weighted scans.

**NEOPLASTIC DISEASE**

**Astrocytoma**

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

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

**Hemangioblastoma/Von Hippel-Lindau Disease**

Hemangioblastomas of the spinal cord can be solid, with surrounding cord edema, or cystic, with an enhancing mural nodule. If the lesion is cystic, the fluid contents, although similar, will be differentiable from CSF on some pulse sequences. Hemangioblastomas are highly vascular lesions and thus enhance prominently on MRI. Their appearance on x-ray angiography is distinctive because of the tumor blush and enlarged feeding arteries and draining veins. Hemangioblastomas are most frequently found in the posterior fossa. They are much less common in the cord, but when in this location have an equal incidence in the cervical and thoracic spine. Spinal cord hemangioblastomas can be solitary or multiple, the latter pathognomonic of von Hippel-Lindau disease. Von Hippel-Lindau disease is an autosomal-dominant syndrome. Features of this disease outside the CNS include renal cell carcinoma, pheochromocytoma, and cysts of the kidney and pancreas. In regard to neurologic disease, these patients present with hemangioblastomas of the cerebellum or spinal cord (Fig. 6–16).

**Meningioma**

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

**Metastases to Bone**

Vertebral metastases are a major source of morbidity in cancer patients. The spinal column is involved in up to 40% of patients dying of metastatic disease. Bone expansion, pathologic fractures, and cord compression are not uncommon. Plain x-ray films are insensitive for lesion detection; at least 50% of the bone needs to be destroyed in order for the lesion to be seen. Bone scans have high sensitivity but low specificity. Reasons for false-positive results on bone scan include infection, trauma, and degenerative disease. Computed tomography (CT) is typically limited in the extent of coverage and offers poor soft tissue contrast. With myelography, cord compression can be evaluated, but lesions are inferred (not directly visualized). MRI offers high sensitivity and specificity, excellent anatomic coverage, and

Text continued on page 134
Figure 6–12. Cavernous angioma. The patient is a 34-year-old woman with right and left hand, arm, and neck pain and numbness. On T₂-weighted scans using fast spin echo (A) and gradient echo (B) technique, a high-signal-intensity abnormality is noted within the cord at the T1 level, with a circumferential rim of hypointensity. The lesion is well margined from surrounding tissue. Although the central high-signal-intensity portion of the lesion is clearly seen on the fast T₂ scan, the rim of hypointensity is much less evident. Gradient echo scans, because of their sensitivity to susceptibility effects, clearly depict the presence of hemosiderin and ferritin. Fast T₂-weighted scans are inferior in this regard because of the acquisition of closely spaced spin echoes and thus relative insensitivity to susceptibility effects. The lesion is not well seen on the sagittal noncontrast T₁-weighted image (C). D. The axial gradient echo scan demonstrates the central hyperintense fluid collection (methemoglobin), together with the smooth peripheral rim of hypointensity (hemosiderin/ferritin). A second lesion with similar characteristics was present within the medulla (not shown).
FIGURE 6–13. Cervical cord astrocytoma. A 10-year-old presented with arm weakness. A, On the precontrast T₂-weighted sagittal scan, a hyperintense cord lesion is noted, which extends from C3 to C7. B, The precontrast T₁-weighted scan reveals marked cord enlargement. C, Postcontrast, there is mottled abnormal enhancement within portions of the lesion. Although not all cord astrocytomas demonstrate enhancement postcontrast on magnetic resonance imaging, this finding, when present, improves differential diagnosis and provides guidance for biopsy. Administration of contrast is particularly important in the presence of a syrinx if a neoplastic origin is in question.
FIGURE 6–14. Recurrent astrocytoma. The magnetic resonance imaging (MRI) exam was performed several years after an extensive laminectomy for resection of a spinal cord astrocytoma. Examining the sagittal precontrast T₂- (A) and T₁- weighted (B) scans, a syrinx cavity is noted, which expands the cord and extends from C2 to T2. The signal intensity characteristics of the syrinx differ from that of cerebrospinal fluid, suggesting a neoplastic origin. On the sagittal (C) and axial (D) postcontrast T₁-weighted scans, there is abnormal enhancement of a large soft tissue nidus within the syrinx at the C5–6 level. This finding was new from the prior MRI exam and represents recurrent tumor. Postcontrast scans in the spine are particularly valuable for detecting recurrent intramedullary neoplastic disease. Such lesions are often difficult to detect without contrast administration because of the distortion of normal structures and the isointensity of the lesion with surrounding soft tissue.
FIGURE 6–15. Cord ischemia (resulting from therapeutic radiation). This 7-year-old became quadriplegic after spinal axis radiation for acute lymphocytic leukemia. Biopsy revealed gliosis. A cervical spine magnetic resonance imaging (MRI) scan obtained before treatment was normal. A. On the sagittal T2-weighted scan, there is abnormal hyperintensity within the cervical cord and lower brainstem. Enlargement of the upper cervical cord is best visualized on the precontrast T1-weighted scan (B). Comparison of pre- (B) and postcontrast (C) sagittal T1-weighted scans reveals marked abnormal enhancement within the upper cervical cord. The MRI exam was repeated 5 months later with a sagittal T1-weighted scan (D). At that time, only atrophy of the upper cervical cord was noted. There was no abnormal contrast enhancement.
Figure 6–16. Spinal cord hemangioblastoma. A 42-year-old with known von Hippel-Lindau disease presented clinically with increasing gait disturbance. A cord syrinx is noted, with high signal intensity on the T₂-weighted scan (A) and low signal intensity on the T₁-weighted scan (B), which extends from the medulla to C2–3. There is secondary expansion of the spinal cord. No abnormal soft tissue mass is noted precontrast. The cerebellar tonsil is globular in shape and normal in position, ruling out a Chiari type I malformation. After contrast administration (C, sagittal; D, axial), an enhancing nodule is identified along the posterior wall of the upper portion of the syrinx. Hemangioblastomas are relatively rare benign epithelial tumors. In von Hippel-Lindau disease (with which there is an association), these tumors may be multiple.

Figure 6–17. Foramen magnum meningioma. On the precontrast T₂- (A) and T₁-weighted (B) axial images, a mass is seen at the level of the foramen magnum. There is substantial deformity of the medulla. Axial (C) and coronal (D) postcontrast images demonstrate intense lesion enhancement (D, arrow). The dural-based origin of the lesion, questioned on the basis of precontrast scans, is confirmed postcontrast.
excellent soft tissue lesion detection. MRI is universally accepted as the modality of choice for the detection and assessment of metastases involving the spinal column. High cervical vertebral metastases, in particular, can be a cause of great morbidity (Fig. 6–18). Sensory and motor deficits from such lesions can be extensive. Cranial neuropathies can occur as a result of spread to the skull base. Compression of the cervical cord above C3 can lead to death by respiratory embarrassment. In regard to tumor type, involvement of cervical spine and skull base by squamous cell carcinoma of neck is not uncommon. This tumor generally spreads by local invasion. Cervical vertebral metastases also commonly arise from a distant primary, with prostate, lung, and breast carcinoma common causes.

Metastases to a vertebra, regardless of location, appear
on MRI as low signal intensity lesions on T1-weighted scans because of the replacement of normal high signal intensity fatty marrow. Metastases are often high signal intensity on T2-weighted scans. The appearance on T1-weighted scans is, however, variable. Blastic metastases are often low signal intensity on T1-weighted scans. Thus, most MRI sites use precontrast T1-weighted scans for detection of vertebral metastases. After intravenous contrast administration, vertebral metastases often enhance to isointensity with normal surrounding marrow. Postcontrast scans, particularly as commonly used without fat saturation, are poor for detection of lesions within the bones of the spinal column. However, contrast enhancement generally improves the depiction of the epidural soft tissue extent of metastatic disease.

**Figure 6–19.** Leptomeningeal metastases from pineoblastoma. Two years before the current exam, this 9-year-old boy presented with persistent headaches and vomiting. Imaging revealed obstructive hydrocephalus, with a mass in the pineal region which proved (by subtotal resection) to be a pineoblastoma. The patient subsequently received brain and spinal axis radiation as well as chemotherapy. At this time, he presents with intractable vomiting, ataxia, and back pain. A bulky soft tissue mass is noted at the C1–2 level on sagittal T2- (A) and T1-weighted (B) scans, causing marked cord compression. The T2-weighted scan identifies an additional lesion at the C6 level, which is poorly seen on the T1-weighted exam. A portion of the larger lesion at C1–2 is of low signal intensity on the T2-weighted scan, suggesting tumor hemorrhage. In the midthoracic region, multiple additional soft tissue masses were seen within the thecal sac (not shown). These were immediately adjacent to the cord and produced an irregular surface contour (C, D). Head magnetic resonance imaging obtained 2 weeks later reveals intracranial metastases. Two low signal intensity foci (arrows) can be identified precontrast on the T2-weighted exam (C). At least two enhancing lesions (arrows) are identified postcontrast on the T1-weighted exam (D). Pineoblastomas are primitive tumors of pinealocyte origin (as opposed to the more differentiated pineocytomas) that present in the first decade of life and are more common in males. Dissemination via the cerebrospinal fluid (CSF) is common. Another pediatric tumor with a propensity for early CSF spread is medulloblastoma.

**Leptomeningeal and Spinal Cord Metastases**

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

Leptomeningeal metastases in the cervical region can present on imaging as soft tissue nodules within the thecal sac (Fig. 6–19), irregularity of the cord surface contour (tumor adherent to or encasing the cord) (Fig.
Leptomeningeal ("drop") metastases from medulloblastoma. The patient is a 4-year-old who had headaches for 9 months and now presents with diminished coordination. Head magnetic resonance imaging (not shown) revealed a midline enhancing posterior fossa mass with obstructive hydrocephalus. On the midline sagittal T₁-weighted cervical image, multiple large soft tissue nodules are noted adjacent to the cervical cord. These demonstrated only very slight enhancement postcontrast (not shown). The posterior fossa mass was resected, followed by whole brain and spinal axis radiation. The follow-up scan 2 months later (not shown) revealed a normal thecal sac and spinal cord.

Diffuse subarachnoid spread of tumor can cause coating and encasement (with deformity) of the spinal cord, leading to an appearance on gross exam resembling "icing." Most leptomeningeal metastatic disease enhances postcontrast on MRI; contrast administration is highly recommended for diagnosis. The entire spinal axis (cervical, thoracic, and lumbar) should be studied to rule out leptomeningeal metastases, with attention to the lumbar region (because of the effect of gravity). MRI, performed with and without contrast enhancement, has been consistently demonstrated in published studies to be superior to CT myelography for the detection of leptomeningeal metastases. This is particularly true for small tumor nodules and coating of the spinal cord by tumor. CT myelography is also not sensitive to intramedullary tumor involvement.

HYDROSYRINGOMYELIA

According to terminology developed for histopathology, syringomyelia is defined as an abnormal cavity within bone marrow. The patient is a 45-year-old woman with a clinical history of radical neck dissection and radiation therapy for squamous cell carcinoma of the mouth. The midline sagittal T₁-weighted image demonstrates diffuse homogeneous high signal intensity throughout the marrow spaces of the C2-4 vertebral bodies. The high signal intensity in the marrow spaces shows an abrupt transition to the normal marrow signal intensity of the adjacent lower cervical vertebral bodies. Detection of this change was aided by comparison with the previous exam (not shown) and inspection of the relative signal intensity of the cord, disk spaces, and marrow. The increase in marrow signal intensity and uniformity of signal is due to radiation therapy with resultant replacement of normal red marrow by fat.

RADIATION THERAPY

The changes encountered with radiation therapy can at times be readily identified because of the confinement to the treatment area or port. After therapeutic radiation, there is uniform fatty replacement of bone marrow. This occurs as early as 2 weeks after initiation of therapy, with temporal progression. Imaging in the sagittal plane with T₁-weighted scans is recommended. Vertebral bodies within the port will have substantially higher signal intensity on such scans (Fig. 6–21), assuming that the choice of time to echo and time to repetition has been made appropriately to obtain moderate to heavy T₁-weighting.
Hydrodynamic myelopathy should be used. On imaging, hydrodynamic myelopathy is seen as a longitudinally oriented fluid cavity (with CSF signal intensity on all pulse sequences) within the spinal cord.

Of special note in the cervical spine is syringobulbia, which is simply extension of a syrinx into the brainstem (Fig. 6–23). This lesion is caused by obstruction of CSF flow at the foramen magnum, usually because of the presence of a Chiari type II malformation. Extension of the syrinx superiorly to involve the brainstem, with a tubular or saccular configuration, is thought to be the result of episodes of increased intra-abdominal pressure (as a result of coughing or sneezing). Symptoms of syringobulbia include facial pain and numbness, dysphagia, vertigo, loss of taste, and respiratory problems (in severe cases).

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

Clinical symptoms of a cervical syrinx include progressive upper extremity weakness, muscle wasting, decreased upper extremity reflexes, and loss of pain and temperature sensation (with preservation of light touch and proprioception). A syrinx that enlarges in the post-traumatic patient can cause clinically significant neurologic deterioration. Large symptomatic syrinxes are treated surgically by shunting into the subarachnoid, pleural, or peritoneal spaces (Fig. 6–24).
FIGURE 6–23. Syringobulbia. This 31-year-old is status postcervical fusion 8 years ago for multiple fractures. A, The T₅-weighted midline sagittal image reveals postoperative changes at C₅–₆. The C₅ and C₆ vertebral bodies have been surgically fused, with loss of the normal intervening disk space. A large amount of metallic artifact is present in the region of the posterior elements compatible with known stainless steel fixation wires. A fluid collection, which is noted to be septated on the T₁-weighted image (B), is identified within the spinal cord above the site of fusion, extending superiorly to near the inferior extent of the fourth ventricle. The abnormality (a posttraumatic syrinx) is isointense with cerebrospinal fluid with on T₁- and T₂-weighted images.
FIGURE 6–24. Posttraumatic hydrosyringomyelia with interval shunting and collapse. This 44-year-old man suffered a fracture of T9 that was treated by laminectomy and fusion 12 years ago. The patient now presents with delayed, progressive neurologic deficits. A and B, The preoperative study demonstrates fluid signal extending down the central portion of the spinal cord from the cervicomedullary junction through the visualized lower cervical region. This is seen as high signal intensity within the cord on the sagittal T2-weighted image (A) and low signal intensity on the postcontrast T1-weighted image (B). No abnormal enhancement is noted. The lesion is a posttraumatic syrinx secondary to a severe wedge compression fracture of T9 (not shown). The spinal cord is expanded with effacement of the surrounding subarachnoid space. The patient underwent a thoracic laminectomy with placement of a syringoperitoneal shunt. C, The postoperative sagittal T1-weighted image of the cervical spine demonstrates collapse of the syrinx. The spinal cord is mildly atrophic, with cerebrospinal fluid now present surrounding the cord. Postoperatively, the patient’s muscle strength and sensation improved.
TRAUMA

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

Cord hemorrhage after spinal cord injury carries in general a poor prognosis (Fig. 6–25). Cord edema, in the absence of hemorrhage, carries a much better prognosis, often with substantial neurologic recovery. Several patterns of acute spinal cord injury have been described on T1-weighted MRI scans. Type I injury has central hypointensity with a thin rim of hyperintensity (deoxyhemoglobin centrally with methemoglobin at the periphery) and carries a very poor prognosis, with little neurologic recovery anticipated. Type II injury has uniform hyperintensity as a result of spinal cord edema and carries an excellent prognosis, with substantial, often complete, neurologic recovery (Fig. 6–26). Type III injury has an isointense center with a thick rim of hyperintensity, representing a combination of hemorrhage and edema, and follows a variable course; some recovery of function is anticipated.

Myelomalacic changes in the spinal cord after trauma follow a well-known sequence. Early on there is cord edema, with compression and stasis within venules and blood-cord barrier disruption. In this early stage, the area of injury in the cord is high signal intensity on T2-weighted scans because of the presence of vasogenic edema. With progression of time, cystic necrosis occurs within the central gray matter. This has high signal intensity on T1-weighted scans and low signal intensity on T1-weighted scans. In the chronic stage, progressive cystic degeneration centrally may lead to a syrinx. The presence and extent of a syrinx is often best defined on axial T1 images; visualization on sagittal images suffers from partial volume imaging. Cord atrophy may also develop in the chronic period. Cord atrophy is defined by a cord diameter of less than 6 mm in the cervical region and less than 5 mm in the thoracic region.

In the imaging of spine trauma, as previously stated,

![Figure 6–25. Traumatic spinal cord injury, with cord hemorrhage (and edema) and canal compromise. The 34-year-old patient was an unrestrained passenger in a single-vehicle motor accident. A, On the midline sagittal T2-weighted image, there is abnormal high signal intensity within the cord extending from the tip of the dens to below the C7 level. There is compromise of the spinal canal posteriorly at the C6 and C7 levels. Abnormal high signal intensity is noted within the C4–5, C5–6, and C6–7 intervertebral disks, suggesting fluid accumulation or edema (secondary to trauma). B, On the corresponding T1-weighted sagittal image, there is abnormal hyperintensity within the cord from C4 to C6, corresponding to methemoglobin. Posterior compromise of the spinal canal is again noted. C, On the single axial T1-weighted image, the posterior soft tissues are asymmetric, suggesting additional injury, and the lamina on the left appears fractured. Lamina fractures were noted on computed tomography (not shown) at both the C5 and C6 levels on the left. The patient, who was quadriplegic after the accident, died 2 weeks later of multisystem failure.](image-url)
FIGURE 6–26. Cord contusion with a small posterior epidural hematoma. The patient is a 25-year-old man who 12 hours earlier was involved in a motor vehicle accident and now complains of bilateral upper extremity and shoulder pain. A. On the T2-weighted exam, abnormal high signal intensity is identified within the cord from C5 to C6 consistent with edema (E) (cord contusion). There is obliteration from C4 to C6 of the cerebrospinal fluid space that normally surrounds the cord. Posteriorly in the epidural space, abnormal soft tissue (with mixed high and low signal intensity) is identified (A, white arrow), causing thecal sac compression. B. The sagittal T1-weighted exam at first glance appears unremarkable, with perhaps only subtle loss of definition of the superior end plate of C7 (white arrow). Examining closely the epidural space at the C5 and C6 levels, abnormal high signal intensity corresponding to methemoglobin is identified (B, black arrows). Without comparison to the T2-weighted scan, this small extradural hematoma might have been mistaken for normal epidural fat. High signal intensity is identified on the T2-weighted image within the bodies of C6 and C7 as a result of microfractures and resultant narrow edema. This finding is consistent with the poor visualization of the superior end plate of C7 (B), which suggests gross bony damage. Extensive high signal intensity in the soft tissues posteriorly on the T2-weighted exam indicates substantial soft tissue and ligamentous injury.

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

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

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

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

Figure 6–30. Type II dens fracture. The patient is a 19-year-old woman who is being scanned 1 month after an unrestrained motor vehicle accident. Sagittal T₂- (A) and postcontrast T₁-weighted (B) images demonstrate a fracture through the base of the dens. Mild anterior slippage of the superior fracture fragment relative to the C3 vertebral body is also present. A small amount of enhancing granulation tissue or venous plexus is identified posterior to the dens on the T₁-weighted scan. No evidence for cord compression or contusion is seen. The dens fracture and the offset of the C2 and C3 vertebral bodies were confirmed on a lateral x-ray film of the cervical spine (not shown).
FIGURE 6–31. Flexion-rotation injury of the cervical spine. The patient is a 28-year-old man with central cord syndrome who is being imaged 3 days after a motor vehicle accident. A, The midline sagittal T₂-weighted image demonstrates mild increased signal intensity within the C3 and C4 vertebral bodies suggestive of microfractures. Increased signal is present within the spinal cord at the C3–4 level consistent with edema and cord contusion. There is also abnormal high signal intensity within the posterior musculature, as a result of edema. B, The corresponding precontrast T₁-weighted image demonstrates a small central disk herniation at the C3–4 level with resultant canal compromise. No abnormal signal intensity is present in the cord to suggest hemorrhage. The vertebral bodies are grossly normal in height and alignment. C, The precontrast T₁-weighted parasagittal image reveals discontinuity and deformity of the left C3 pedicle consistent with a fracture. The pre- (D) and postcontrast (E) T₁-weighted axial images demonstrate asymmetric abnormal enhancement within the injured right paraspinal muscles. Plain cervical spine films (not shown) revealed a fracture-dislocation of C3–4 with approximately 3 mm anterior slippage of C3 on C4. No vertebral fracture was detected. Computed tomography (not shown) revealed a linear fracture through the left pedicle at C3. Mild anterior slippage of C3 on C4 was present with 20% compromise of the spinal canal but no direct impingement on the spinal cord. Traction was applied before the magnetic resonance imaging exam accounting for the normal alignment on this study.
CERVICAL SPINE

ligaments occurs with flexion. Unilateral involvement suggests a rotational component.

Perched Facet

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

Brachial Plexus Injury

Injury to the brachial plexus can lead to a posttraumatic neuroma, fibrosis, or meningocele (with or without nerve root avulsion). On MRI, meningoceles caused by brachial plexus injury are clearly seen. The lesion will follow the course of the nerve root in the foramen and manifest CSF signal intensity on all pulse sequences (Fig. 6–33). Nerve root avulsions per se are best evaluated by myelography.

DISK HERNIATION

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

Clinical symptoms of a cervical disk herniation depend on its location. Large central herniations cause myelopathic symptoms (Fig. 6–34). Posterolateral or foraminal herniations can compress the exiting nerve root and cause radicular symptoms (Fig. 6–35).

Thin-section (less than 2 to 3 mm) images should be acquired in both the sagittal and axial planes on MRI when a disk herniation is suspected. T₁-weighted spin echo and T₂-weighted fast spin echo images are typically

**FIGURE 6–32.** C2 teardrop fracture and unilateral perched facet at C6–7. The patient presented 6 months after a motor vehicle accident with persistent left arm pain. Midline sagittal T₁- (A) and T₂-weighted (B) images demonstrate a teardrop fracture at the base (anteriorly) of C2, which had been noted on previous diagnostic exams. Mild anterior slippage of C6 on C7 is also apparent. The T₁-weighted sagittal image (C) to the left of midline reveals a facet dislocation at C6–7 (arrow). The alignment of the facets on the right was normal (not shown).
FIGURE 6–33. Meningocele secondary to birth trauma. The patient is a 15-month-old infant with left upper extremity spasticity since birth. A, The T₁-weighted left parasagittal image of the cervical spine reveals two low signal intensity extradural fluid collections within the C6–7 and C7–T1 neural foramina, respectively. These two abnormalities remained isointense with cerebrospinal fluid on T₂-weighted scans (not shown). B, An axial T₁-weighted spin echo scan confirms, at one level, the extradural location of the lesion and association with the exiting nerve root sleeve.

FIGURE 6–34. Large central disk herniation at C4–5. The 42-year-old patient presented with recurrent neck pain. The clinical history is significant for a prior diskectomy and fusion at C5–6. A, The T₁-weighted midline sagittal image reveals a prominent anterior extradural soft tissue mass contiguous with and posterior to the C4–5 intervertebral disk. The abnormality is of relatively low signal intensity, similar to the intervertebral disks, on this T₁-weighted scan. On T₁-weighted images (not shown), the abnormality remained isointense to disk material. The C5–6 disk space is narrowed and indistinct, compatible with the prior anterior diskectomy and fusion. B, A T₁-weighted axial view through the C4–5 disk confirms the extradural soft tissue mass, with resultant central cord compression. The lesion is again noted to be contiguous with and isointense to the C4–5 disk. C, The corresponding axial gradient echo scan depicts this central disk herniation as high signal intensity.
FIGURE 6–35. Right foraminal disk herniation at C6–7. This 49-year-old patient presents with excruciating right arm pain. A, A midline T₁-weighted sagittal image demonstrates small spurs and end plate degenerative changes at the C5–6 and C6–7 levels. B, A T₁-weighted sagittal image to the right of midline demonstrates abnormal soft tissue (white arrow) extending posterior to the vertebral bodies at the C6–7 level. This abnormality was isointense to disk material and contiguous with the C6–7 disk on both this image and the corresponding T₂-weighted scan (not shown). C, An axial T₁-weighted image at the C6–7 level does not clearly demonstrate the abnormality. However, the spinal cord does appear mildly shifted to the left. D, After contrast administration, the soft tissue abnormality (black arrow) is highlighted by the enhancement of the epidural venous plexus within the neural foramen. The disk herniation fills the right neural foramen at the C6–7 level.
acquired in the sagittal plane. T2-weighted gradient echo images are of high value in the axial plane. On the latter, a thin rim of low signal intensity often outlines the high-signal-intensity disk herniation (along its posterior aspect). The low-signal-intensity rim corresponds to the dura and posterior longitudinal ligament. An acute disk herniation is seen on sagittal and axial images as an anterior epidural soft tissue mass. The abnormal soft tissue will be contiguous with the disk space unless a disk fragment is present. The signal intensity of the herniated material is similar to the native disk on both T1- and T2-weighted scans. A decade ago, postcontrast T1-weighted images were also commonly acquired. These can be very useful in diagnosis, but cost constraints led to their elimination in most clinical practices for the study of cervical disk disease. On postcontrast T1-weighted scans, the dilated epidural venous plexus surrounding a disk herniation will enhance, outlining the disk material and improving visualization of the neural foramina (Fig. 6–36). Without contrast administration, the epidural venous plexus is isointense with and cannot be distinguished from disk material on T1-weighted scans.

A “hard” disk is the result of a long-standing herniation (Fig. 6–37). A chronic disk herniation is covered above and below by bony spurs from the end plates. These form as a result of bone remodeling; elevation of the periosteum by the disk herniation leads to bone deposition at the site. Myelopathic symptoms are more common with chronic disk herniations as opposed to radicular symptoms, which are more common with acute disk herniations. Damage to the blood–spinal cord barrier, on the basis of chronic repetitive trauma at the level, can lead to enhancement within the cord at the level of a hard disk herniation (see Fig. 6–37). This is rarely visualized in current clinical practice because of the nonuse of contrast in the setting of chronic degenerative disease.

**Figure 6–36.** C3–4 right paracentral disk herniation. This 35-year-old patient presents with neck and right arm pain after a motor vehicle accident. Precontrast axial and sagittal T2- (A and D) and T1-weighted (B and E) scans reveal abnormal soft tissue at the C3–4 disk level anterior and to the right of the thecal sac, causing mild cord deformity. The lesion is difficult to separate from the contents of the right neural foramen. Postcontrast (C and F), the disk herniation itself (white arrow) can be differentiated from dilated epidural venous plexus (black arrows) because of prominent enhancement of the latter. There is no foraminal component.
FIGURE 6–37. Early compressive myelomalacia secondary to a large “hard” disk herniation at C3–4. This 44-year-old presented clinically with increasing pain and numbness in the upper extremities. A, On the midline fast spin echo T2-weighted sagittal image, abnormal increased signal intensity (likely a combination of edema and gliosis) is seen within the cord, extending from mid C3 to C4–5. At the C4–5 level, prominent osteophytes obliterate the cerebrospinal fluid space anterior to the cord. Mild retrolisthesis of C3 on C4 is identified on the midline sagittal T1-weighted image (B). The C3–4 intervertebral disk is narrowed, and abnormal soft tissue extends posterior to the disk, causing deformity of the cervical cord. C, The corresponding postcontrast T1-weighted image reveals prominent enhancement within the flattened cervical cord. Abnormal enhancement resulting from de novo scar and dilated epidural venous plexus is also apparent about the C3–4 disk.

HYPERTROPHIC END PLATE SPURS

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

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

SURGERY FOR CERVICAL SPONDYLOSIS

Damage to the spinal cord in cervical spondylosis is the result of ischemia from chronic compression. The aim of surgery is to prevent further deterioration. An anterior surgical approach is used for one- to two-level stenosis (Figs. 6–39 and 6–40), and is the most common neurosurgical procedure in cervical disk disease. The disk is resected (using an anterior approach) and a bone graft placed between the two adjacent vertebral bodies to achieve a stable fusion. Portions of the adjacent vertebral bodies may or may not be removed. The signal intensity characteristics of the graft are variable. After more than 2 years, continuous marrow signal intensity is typically seen at the site of fusion, with no evidence of bone graft or native disk. There is a propensity over the long term for new disk herniations to develop above and below the site of fusion. The posterior approach, which is less common, involves a laminectomy and is used for congenital narrowing or extensive contiguous disease (multiple levels). MRI can be diagnostic in postoperative cases despite the presence of substantial metal hardware. Artifacts from metal will be greatest in general on gradient echo scans (because of the lack of a 180-degree
FIGURE 6–38. Hypertrophic osteophytic end plate spurs. A 57-year-old woman presented with right-sided neck and arm pain. Midline sagittal T2-weighted gradient echo (A) and T1-weighted spin echo images before (B) and after (C) contrast administration demonstrate a ventral extradural defect along the anterior margin of the thecal sac at the C5–6 level. A. The sagittal T2-weighted gradient echo image demonstrates, in addition to the low signal intensity spurs at C5–6, smaller spurs at C4–5 and C6–7 that partially efface the ventral subarachnoid space. B. The precontrast sagittal T1-weighted image shows pointed extensions of bone marrow signal intensity along the posterior end plates adjacent to the C5–6 disk. C. Postcontrast, enhancement of dilated venous plexus is noted immediately above and below the C5–6 level. Mild irregularities of the posterior margin of the vertebral end plate are present on the axial T1-weighted gradient echo image (D). The signal intensity of these projections is very low, indicative of cortical bone. These findings are consistent with osteophytes extending from the posterior vertebral end plates.
Figure 6-39. Normal late appearance of anterior cervical diskectomy and fusion. The patient has continued left arm pain 4 months after anterior diskectomy and fusion for a C5–6 disk herniation. A, On the midline sagittal T2-weighted image, the C5–6 intervertebral disk is not seen. Small spurs with mild compromise of the thecal sac are noted at the levels above and below (C4–5 and C6–7). B, The sagittal T1-weighted image (obtained after contrast administration) demonstrates fusion of the C5 and C6 vertebral bodies. Mild decreased signal intensity is evident within the central portion of the fusion. The alignment of the cervical spine is normal.
FIGURE 6–40. Normal appearance after anterior diskectomy and titanium plate fusion at C6–7. This 48-year-old patient presented clinically with continued neck pain after surgery for a C6–7 disk herniation. A, The T₁-weighted midline sagittal image reveals prominent metallic artifact anterior to and within the C6 and C7 vertebral bodies. The alignment of the cervical vertebral bodies is normal. No significant canal stenosis is present. The artifact is again present, but less apparent, on the fast spin echo T₂-weighted sagittal image (B). A small osteophyte causes effacement of the ventral subarachnoid space at C4–5. C, On the axial gradient echo image at C6–7, the metal artifact is more extensive, with artifactual mild effacement of the anterior thecal sac. A lateral radiograph of the cervical spine (not shown) demonstrated a metallic plate and three screws that fused the C6 and C7 vertebral bodies.
Multiple sclerosis, with active spinal cord plaques. The patient is a 28-year-old white woman with new onset 2 months ago of numbness below the waist, now involving the left arm. Several episodes of blurred vision in one eye have also occurred during the past 2 years. 

A, On the T2-weighted midline sagittal scan, two intramedullary lesions are noted, at C2–3 and C5–6, with the latter larger and exhibiting a flamelike pattern of edema extending superiorly and inferiorly.

B, On the postcontrast T1-weighted midline sagittal scan, faint lesion enhancement (arrows) is identified at both levels. Axial T2-weighted gradient echo (C) and postcontrast T1-weighted spin echo (D) scans confirm the lower lesion, which is eccentrically located, causes focal cord enlargement, and demonstrates prominent enhancement.
FIGURE 6–42. Multiple sclerosis (MS) (inactive or chronic disease). The patient is a 52-year-old white man with long-standing neurologic problems. He ambulates with a cane. Bowel function is intact; however, there is bladder incontinence. Heavily T2-weighted midline sagittal images of the cervical (A) and thoracic (B) spine are presented. A single hyperintense intrinsic cord abnormality is noted in the cervical spine at the C2 level, suggesting cord atrophy. Two thoracic cord lesions are also seen, both somewhat elongated in appearance. Incidental note is made of an osteophyte situated between the two thoracic cord lesions, causing anterior compression of the thecal sac. The lesions (all chronic MS plaques) do not cause cord enlargement, and there was no abnormal contrast enhancement (not shown).

refocusing pulse), moderate on spin echo scans, and least on fast spin echo scans (as a result of the short interecho interval). High signal intensity within the cord postoperatively on T2-weighted scans is seen occasionally and can be due to gliosis (present preoperatively) or postoperative complications (such as cord contusion and infarction).

MULTIPLE SCLEROSIS

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

Clinically, MS is characterized by recurrent focal neurologic attacks, progressive deterioration, and ultimately permanent neurologic dysfunction. Symptoms include decreased vibration and position sense, weakness of one or more extremities, and disorders of micturition (urination). Differential diagnosis, based on the results of MRI of the spinal cord, includes transverse myelitis. The presence of multiple cord lesions, combined with characteristic brain lesions, favors the diagnosis of MS.

ACUTE TRANSVERSE MYELITIS

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