NORMAL LUMBAR SPINE

The lumbar spine consists of five lumbar segments (vertebral bodies), five (fused) sacral segments, and the coccyx. Each intervertebral disk is composed of a central gelatinous core (the nucleus pulposus, which is high signal intensity on T2-weighted images) surrounded by dense fibrous tissue (the annulus fibrosus, which is low signal intensity on T1-weighted images). The bony elements of the lumbar spine include the pedicles, transverse processes, articular pillars (pars interarticularis), superior and inferior articular facets, laminae, spinous processes, and vertebral bodies. The facet joints are diarthrodial (synovial lined) and richly innervated. On axial imaging, the superior articular facet forms a “cap” anterolaterally with the inferior articular facet postero-medial and connecting to the lamina. The ligamentum flavum extends from the anterior aspect of the upper lamina to the posterior aspect of the lower lamina. The epidural venous plexus is prominent in the lumbar spine. In regard to important dermatomes (for clinical diagnosis with a disk herniation), L4 innervates the medial big toe, L5 the midfoot, and S1 the little toe.

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

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

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

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

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

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

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

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

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

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**CONGENITAL DISEASE (INCLUDING STRUCTURAL ANOMALIES)**

**Transitional Vertebrae**

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

**Spina Bifida Occulta (Occult Spinal Dysraphism)**

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

**Caudal Regression (Sacro Agenesis)**

In caudal regression, there is absence of sacrococcygeal vertebrae with or without lumbar involvement. The
level of regression is below L1 in most cases. Agenesis is limited to the sacrum in about half of all cases (Fig. 8–1). Associated anomalies include cord tethering, renal dysplasia, pulmonary hypoplasia, and neuromuscular weakness or paralysis. Caudal regression is associated with maternal diabetes. On MRI, a wedge-shaped cord terminus is seen in about half of patients, with the dorsal aspect extending further caudally than the ventral aspect. MRI clearly depicts the level of regression, presence of stenosis (in the area of vertebral absence), and associated structural anomalies.

Myelomeningocele

Spina bifida is defined as incomplete closure of the posterior bony elements. The contents of the spinal canal can extend through this defect (with tethering of the cord). A meningocele contains dura and arachnoid. Neurologic deficits are uncommon with a simple meningocele. A myelomeningocele contains neural tissue within the expanded posterior subarachnoid space (Fig. 8–2). On intrauterine ultrasonography, the neural arch is open and the posterior elements are flared. There is an associated Chiari type II malformation in almost all cases. MRI is usually obtained postoperatively. A wide dysraphic defect is typically seen, together with a cerebrospinal fluid (CSF)-filled sac covered by skin. There is often retethering of the cord. MRI is the modality of choice for evaluation of the soft tissue elements in suspected spinal dysraphism.

Anterior Sacral Meningocele

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

Diastematomyelia

In diastematomyelia, the spinal cord is split into two hemicords, each invested by pia (Fig. 8–3). Each
Figure 8–2. Meningomyelocele with tethered cord. The midline sagittal T1-weighted scan reveals a sac filled with cerebrospinal fluid located posteriorly in the lower lumbar region. The sac communicates with the normal thecal space. The spinal cord extends at least to the lumbosacral junction. The posterior bony elements are dysraphic from L4 to S1. Abundant fatty tissue is present immediately below the defect. Note also the distinct signal intensity and configuration of the vertebral bodies and intervertebral disks, normal for the patient's age. This newborn presented with a normal neurologic exam and a low lumbosacral mass covered by skin. At the time of surgery for repair of this defect, a single nerve (not seen on magnetic resonance imaging or computed tomography) was identified within the fluid-filled sac.

Lipomyelomeningocele

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

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

Dorsal Dermal Sinus

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

Tethered Cord

A tethered cord is a congenital anomaly in which the conus is held at an abnormally low position. Causes include a short (tight) filum terminale, an intradural lumbosacral lipoma (Fig. 8–5), diastematomyelia, and a delayed consequence of myelomeningocele repair. With a tight filum, the age of presentation is variable. Adults present frequently with radiculopathy. The normal filum should be 2 mm or less in diameter. Caution should be exercised when interpreting postoperative cases (after myelomeningocele repair) because not all patients with evidence of tethering on imaging are symptomatic.

Clinical symptoms are due to cord ischemia caused...
Figure 8–3. Diastematomyelia. A, The midline sagittal T₂-weighted scan demonstrates a segmentation anomaly (block vertebrae) at L2-3. A low-signal-intensity band spans the thecal sac at the L2-3 level. A central region of high signal intensity, consistent with a small syrinx (hydromyelia), is present within the lower thoracic spinal cord. B, The T₁-weighted axial image at the L2 level reveals splitting of the spinal cord by the previously noted band or spur. Bony dysraphism is noted posteriorly. C, An additional T₁-weighted axial image at a slightly higher level more clearly depicts the separation of the cord into two hemicords. This 18-month-old infant presented clinically with lower extremity spasticity.
FIGURE 8–4. Dorsal dermal sinus. Sagittal T2- (A) and T1-weighted (B) images of the lumbar spine reveal a sinus tract (A, arrows) coursing from the skin to the thecal sac. This abnormality is less apparent on the T1-weighted scan because of the high signal intensity of fat posteriorly, accentuated by the proximity to the surface coil. The conus lies at L2. A portion of tract (arrow) is also visualized on the axial T1-weighted image (C).
FIGURE 8–5. Tethered spinal cord with lipomyelomeningocele. A, The midline sagittal T₂-weighted scan demonstrates spinal dysraphism at L4-5, a capacious lumbar thecal sac, and a large abnormal fat pad posteriorly. B, The corresponding T₁-weighted scan reveals the cord to be low lying and tethered to a lipoma at the L4-5 level. C, On axial imaging, the low lying cord is seen in cross-section with a separate, but adjacent, intrathecal lipoma. D, On a lower axial section, the cord is tethered posteriorly and attached to a large lipoma that extends into both the thecal sac and the posterior soft tissues. This 2-month-old infant presented at birth with a posterior lumbar mass that subsequently increased in size. Motion and strength of the lower extremities were normal.
by traction. The typical patient is a young child with progressive neurologic dysfunction. Symptoms include gait difficulty, motor and sensory loss in the lower extremities, and bladder dysfunction. On imaging, the cord is seen to extend without change in caliber to the lumbosacral region, where it is tethered posteriorly. Also commonly present is a lipoma and dysraphism of the posterior spinal elements. Hydromyelia may be present as well. When small, this is usually not symptomatic. T1-weighted scans in all three orthogonal planes are important for depiction of the abnormal anatomy. The coronal plane is superior to the sagittal plane for determination of the level of the conus. On sagittal images, differentiation between the conus and cauda equina can be difficult. For the diagnosis of retethering, the presence of adhesions is a good criterion.

The aim of surgical therapy is to untether the cord and thus arrest symptom progression. Early diagnosis and surgery can prevent urinary incontinence. The associated lipoma is typically removed as completely as possible, with attention to release of the tether. The use of synthetic dural grafts decreases the incidence of retethering. After surgery, the level of the cord termination does not change.

A terminal myelocystocele is a rare congenital cystic dilatation of the caudal central spinal canal with an associated posterior bony defect. There is a trumpet-like flaring of the distal central canal, which is a pia-lined CSF space and may be larger than the accompanying surrounding meningocele. Associated anomalies of the gastrointestinal tract, genitourinary tract, and vertebral bodies are common.

**Spinal Meningeal Cysts**

Spinal meningeal cysts are diverticula of the meningeal sac, nerve root sheath, or arachnoid. Most cysts are congenital in origin. There are three types. Type I cysts are extradural in location and do not contain nerve roots. This group includes arachnoid cysts and sacral meningoceles. Type II cysts are extradural in location and contain nerve roots. This group includes spinal nerve root diverticula and Tarlov cysts. The latter are not infrequently seen in clinical practice. More correctly known as Tarlov perineural cysts, these lesions are simply nerve root sleeve cysts (focal dilatation of the nerve root sleeves). The nerves may be in the cyst or in the wall. The cyst communicates freely with the thecal sac (Fig. 8–6). Type III cysts are intradural in location and are simply intradural arachnoid cysts.

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

**Lumbosacral Nerve Root Anomalies**

Lumbosacral nerve root anomalies occur in 1% to 3% of the population. These usually involve the L5 and S1 roots unilaterally. There are three types of lumbosacral nerve root anomalies. The first, type I, is a simple conjoined root (Fig. 8–7). This is the most common anomaly. Two roots arise from a single root sleeve but exit separately (in the appropriate foramina). In type II, two roots exit through a single foramen (and there may be only one foramen without a root). In type III, an anastomotic root connects two adjacent roots. Lumbosacral nerve root anomalies are asymptomatic. However, it is important to recognize their presence and report this in the dictation. For lumbar disk surgery to be successful, in the presence of a nerve root anomaly, adequate decompression is required. On computed tomography (CT) without intrathecal contrast, a nerve root anomaly can be mistaken for a herniated disk.

**Fatty Filum Terminale**

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

**Achondroplasia**

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

**SPINAL STENOSIS**

**Congenital**

In congenital spinal stenosis, both the anteroposterior and transverse dimensions of the canal are decreased. The pedicles are typically short and thick with a decreased interpediculate distance. The spinal canal tapers in the lumbar region (Fig. 8–8). This is the opposite of normal, in which the canal is usually equal in size to or greater (in anteroposterior dimension) than that in the thoracic region. The lateral recesses and neural foramina may also be narrowed. The lower limit of normal for the anteroposterior canal dimension is 11.5 mm, and the normal lateral recess should be 5 mm. The L4-5 level is the most common site for canal stenosis and tends to be the most severely affected level when the canal is diffusely narrowed. Congenital spinal stenosis predisposes the patient to early degenerative disk disease. Clinical presentation typically includes myelopathic symptoms. Radicular symptoms may be present as a result of nerve root impingement.
FIGURE 8–6. Tarlov cysts. A and B, Precontrast T₁-weighted sagittal images reveal two oval areas of low signal intensity posterior to the S1 and S2 vertebral bodies, both to the right and left of midline. There is erosion and scalloping of the adjacent sacral segments. On the proton density (C and D) and heavily T₂-weighted (E and F) sagittal images, the signal intensity of these cysts follows that of cerebrospinal fluid. Chemical shift artifact is noted at the interface between the lesions and the adjacent fatty marrow of the sacrum. There was no abnormal contrast enhancement (images not shown). G, The axial T₁-weighted image through S2 reveals both cysts, which occupy (albeit markedly enlarged) the expected location of the nerve root sleeves.
FIGURE 8–7. Conjoined nerve root. A–E, Axial postcontrast T₁-weighted scans are depicted from the middle of the L5 vertebral body to the middle of S1. On the first scan, the right L5 nerve root has already exited from the thecal sac (and is normal). A large abnormal nerve root sleeve is seen on the left, having not yet separated from the sac. On the next scan, two separate nerve roots are noted adjacent to one another on the left. On the third scan, at the level of the L5-S1 foramen, a nerve root (S1) is identified on the left medial to the enhancing dorsal root ganglion of L5. On the last two scans, the left S1 nerve root is seen to remain within the bony canal to descend to a position more symmetrical and normal relative to the right S1 nerve root.
Degenerative (Acquired)

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

Ligamentum flavum hypertrophy is one cause of degenerative spinal stenosis. The ligamentum flavum is a paired, thick, fibroelastic band. The normal thickness is 3 mm in the lumbar spine. The ligamentum flavum connects the lamina of adjacent vertebral bodies and is situated posterolaterally in the canal. It extends from the anteroinferior aspect of the superior lamina to the posterosuperior aspect of the inferior lamina. Anterolaterally, the ligamentum flavum is contiguous with the capsule of the facet joint. With degenerative spine disease, the ligamentum flavum becomes fibrotic, visibly thickened (Fig. 8-9), and buckled. It narrows the posterolateral canal and thus the lateral recess. It may also narrow the central canal and/or the neural foramina.

Facet joint hypertrophy is another cause of degenerative spinal stenosis (Fig. 8–10). Hypertrophy of the superior articular facet is a primary cause of lateral recess stenosis. Failure to recognize lateral recess stenosis is a major cause of persistent symptoms after lumbar discectomy.

A third cause of degenerative spinal stenosis is neural foraminal degenerative disease. The neural foramen is bounded by the pedicles superiorly and inferiorly, the vertebral body and disk anteriorly, and the facets posteriorly. In the lumbar spine, the nerve root exits from the lateral recess and enters the neural foramen. Stenosis of the neural foramen is most common at L4-5 and L5-S1. Degenerative disease of the disk, end plates, and posterior elements (facets) all contribute to foraminal stenosis (Fig. 8–11). The most common cause is hypertrophy of the superior facet. The stenosis is accentuated if the disk is narrowed. Foraminal stenosis causes radicular symptoms as a result of nerve root compression. Pain
Figure 8–9. Spinal stenosis with marked thickening of the ligamentum flavum. A, The sagittal T₁-weighted scan just to the right of midline demonstrates narrowing of the thecal sac at L4-5, with indentation posteriorly by intermediate-signal-intensity soft tissue: the thickened ligamentum flavum (arrow). Less marked findings are present on the midline sagittal T₁-weighted scan (B). These two sagittal images also reveal disk degeneration at L4-5 with disk space narrowing, a disk bulge with associated spurs, end plate irregularities, and adjacent degenerative end plate disease. C, The axial T₁-weighted image at the L4-5 disk level demonstrates severe central stenosis of the spinal canal. The thecal sac is very small and triangular in shape, narrowed anteriorly by the disk bulge and spurs and posteriorly by the markedly thickened ligamentum flavum (extending along the posterolateral margins of the thecal sac). The thickened ligaments (measuring 6 mm in cross-section) and facet hypertrophy have obliterated the lateral recesses. A tiny amount of epidural fat is seen in the posterior canal.
Severe spinal stenosis and lateral recess stenosis at L4-5 resulting from facet joint hypertrophy. 

A. The midline sagittal postcontrast T₁-weighted image reveals prominent narrowing of the lumbar canal at the L4-5 level. The canal stenosis is also well seen on the fast spin echo T₂-weighted sagittal image (B). C. A T₁-weighted sagittal image in the plane of the left lumbar facet joints reveals hypertrophy and sclerosis of the L4-5 facet (arrow). The neural foramen remains patent at this level. A similar appearance was present at the right facet joint of L4-5 (not shown). D. A T₁-weighted axial image at the L4-5 level confirms the marked facet hypertrophic changes, left greater than right. The superior articulating facet of L5 is particularly affected. The facet hypertrophy results in bilateral lateral recess stenosis, more severe on the left, where the lateral recess is less than 3 mm in width. Sclerosis of the facet joints is also apparent. The spinal canal is narrowed, measuring 11 mm in anteroposterior dimension. Even more striking is the degree of narrowing of the thecal sac, which measures 4 mm in anteroposterior diameter.
Degenerative foraminal stenosis is that of chronic pain in the lower back and buttocks. There may be paresthesias (abnormal sensation) or pain in the posterolateral leg. Standing and walking aggravate the pain, and resting (sitting or lying down) relieves it. This is the opposite of clinical symptoms for an acute disk herniation, in which the pain is aggravated by sitting. Neurologic deficits are minimal with degenerative spinal stenosis. The pathogenesis is nerve root ischemia.

INFECTION AND INFLAMMATORY DISEASE

Disk Space Infection

Disk space infection can be either hematogenous (Fig. 8–12) or postoperative (Fig. 8–13) in origin. In children, with hematogenous seeding, the disk serves as the initial site of infection (because it is richly vascularized). In adults, the initial site of infection (with hematogenous seeding) is the vertebral body (subchondral portion) or soft tissue. Patients with postoperative disk space infection present clinically with severe back pain 1 to 4 weeks after surgery. Disk space infection is seen in 1% to 3% of all back surgery patients. *Staphylococcus aureus* is the most common organism. Delays in diagnosis are common. Fever, wound infection, and elevation of white blood cell count are seen in only a minority of patients.

On lumbar spine x-ray films, disk space narrowing, poorly defined end plates, and sclerosis of the adjacent vertebrae may be seen. On CT, disk space narrowing, cortical bone loss (from the end plate), and abnormal paraspinous soft tissue may be seen. All are late changes. Radionuclide bone scans are sensitive but nonspecific in disk space infection.

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

Arachnoiditis

In arachnoiditis, there is clumping and thickening of nerve roots on the imaging exam regardless of modality.
FIGURE 8–12. Hematogenous diskitis. A, On the T₂-weighted scan, the L2-3 disk is high signal intensity (which by itself could be normal), yet irregular in contour. There is absence of the normal intranuclear cleft. The thecal sac is narrowed at the L2-3 level. B, On the precontrast T₁-weighted scan, both the L2 and L3 vertebral bodies are of abnormal low signal intensity. There is loss of definition between the L2-3 disk and the adjacent vertebral end plates. C, On the postcontrast T₁-weighted scan, there is enhancement of the L2 and L3 marrow space, with irregular enhancement along the disk margin and residual low-signal-intensity (noneshancing) soft tissue within the disk space. The latter corresponds in position to the high signal intensity noted on the T₂-weighted scan and represents inflammatory exudates. The basis for thecal sac narrowing is now evident, with abnormal paraspinal enhancing the soft tissue. In the adult patient, noniatrogenic disk space infection is usually the result of hematogenous seeding to the soft tissue or to the subchondral portion of the vertebral body.
FIGURE 8–13. Postoperative disk space infection. A, Precontrast on the T2-weighted sagittal scan, diffuse abnormal high signal intensity (SI) is noted within the marrow of the L4 and L5 vertebral bodies. The disk is reduced in height, irregular, and of abnormal high SI. B, On the precontrast T1-weighted scan, the L4-5 disk is difficult to identify. Also noted is abnormal low SI within the lower half of L4 and the upper half of L5, paralleling the disk. C, Postcontrast, abnormal enhancement of the disk space is noted, together with a soft tissue mass that compresses the thecal sac. Comparison of pre- (D) and postcontrast (E) T1-weighted axial scans at the disk level reveals a paraspinal mass with enhancement. There is abnormal enhancement of the disk as well, permitting identification of fluid pockets that remain low SI (arrows).
Inflammation initially elicits only a minimal cellular response, which then progresses to collagenous adhesions. The pathogenesis includes infection, which is uncommon today, previous surgery, hemorrhage within the thecal sac, and prior myelography with Pantopaque.

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

The nerve roots and abnormalities thereof are clearly seen on MRI. Several common patterns of nerve root involvement in arachnoiditis are subsequently described. In mild disease, nerve roots can be clumped and lie centrally within the sac (Fig. 8–14). Alternatively, individual nerve roots may be adherent to the periphery of the sac. With severe disease, abnormal soft tissue can fill the majority of the thecal sac, with no discernible individual nerve roots. With acute infection (viral or bacterial meningitis), the nerve roots themselves enhance (Fig. 8–15). Care should be exercised in the diag-

**Figure 8–14.** Arachnoiditis. Midline sagittal $T_2$- (A) and $T_1$-weighted (B) images suggest clumping of nerve roots along the posterior margin of the thecal sac. No individual nerve roots are visualized; rather a single thick strand is seen. The clumping of nerve roots is confirmed on the axial $T_2$- (C) and $T_1$-weighted (D) images.
FIGURE 8–15. Spinal meningitis with progression to arachnoiditis. Postcontrast sagittal (A) and axial (B) images reveal prominent enhancement (white arrows) of the lumbar nerve roots. These also appear mildly thickened but retain their usual position within the dependent portion of the thecal sac. The patient returned for follow-up after 1 month of antibiotic therapy. Her back pain remained severe at this time. C, The postcontrast T1-weighted sagittal image reveals persistent enhancement of the lumbar nerve roots. The nerve roots now lie anteriorly within the thecal sac. D, A postcontrast axial image at L3-4 demonstrates the enhancing nerve roots to be clumped anteriorly. Cultures in this patient revealed *Staphylococcus aureus* as the causative organism.
nosis of arachnoiditis when spinal stenosis is present. Spinal stenosis can lead to a false impression of nerve root clumping.

**NEOPLASTIC DISEASE**

**Benign Neoplasms of Bone**

**Vertebral Body Hemangioma**

Vertebral body hemangiomas are a common incidental finding on MRI. This benign neoplasm can be found, on autopsy, in more than 10% of the population. Solitary lesions are most common, although multiple lesions are not uncommon. The size is variable, ranging from small to large, involving the entire vertebral body. Posterior extension can cause canal compromise. A large lesion can weaken the vertebral body and lead to fracture. Histologically, vertebral hemangiomas are composed of a mixture of adipose and angiomatous tissue with prominent bony trabeculae. The coarse vertical trabeculation can be seen on plain film and CT, which also depict the lesion as generally lucent. On MRI, vertebral hemangiomas are classically high signal intensity on both T₁- and T₂-weighted scans (Fig. 8–16). Also commonly noted is a reticular pattern of low signal intensity (prominent vertically) corresponding to the thickened trabeculae. The major differential diagnosis on MRI is that of focal fat (within the vertebral bodies). The latter is a common finding, particularly with increasing age. Focal fat deposition will be seen to follow the signal intensity of fat on all pulse sequences.

**Osteoid Osteoma**

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

**Giant Cell Tumor**

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

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**Figure 8–16.** Vertebral hemangioma. A round, mottled area of increased signal intensity is seen in the central portion of the L3 vertebral body on the sagittal T₁-weighted image (A). The postcontrast T₁-weighted image (not shown) revealed mild enhancement. The lesion also exhibits high signal intensity on the sagittal T₂-weighted image (B). Mottled signal intensity is demonstrated with interspersed areas of very low signal intensity on all imaging sequences corresponding to prominent trabeculae.
elements, less lobular), aneurysmal bone cyst (younger age group), and metastatic disease.

Malignant Neoplasms of Bone

Lumbar Metastases

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

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

MRI is the modality of choice for detecting and assessing vertebral metastatic disease. MRI is more sensitive (as well as more specific) than bone scintigraphy for detecting vertebral metastases. We now know, because of MRI, that the vertebral body is nearly always the initial site of involvement. Sagittal scans provide screening of the area of interest. These should be supplemented with axial scans in areas where canal compromise is questioned. Imaging of the entire spine in the body coil is not recommended for lesion detection. Although STIR images are predominantly T1-weighted, the gray scale is reversed compared with spin echo images, and metastases appear as hyperintense vertebral body lesions. On spin echo T1-weighted scans, contrast administration is not helpful for detecting bone metastases. Most metastases enhance postcontrast to near isointensity with normal marrow, decreasing their conspicuity. Contrast enhancement is, however, useful for improved depiction of epidural and soft tissue extent of metastatic disease and for the detection of leptomeningeal metastases (Fig. 8–19). T1-weighted scans are not of great use in the evaluation of metastatic disease to the vertebral column, although they are routinely acquired (Fig. 8–20). Many bony metastases will have abnormal high signal intensity on T1-weighted scans, but many will also be isointense. Osteoblastic metastases, which are common with prostate carcinoma, deserve special comment. These are typically low signal intensity on both T1- and T2-weighted scans. When both osteoblastic and lytic lesions are present, it is commonly observed that the blastic lesions are substantially lower in signal intensity on T1-weighted scans than the lytic lesions. Metastatic lesions in lung and breast carcinoma are typically lytic but may be osteoblastic when treated. Bony sclerosis is seen, of course, on plain film with osteoblastic metastases.

Chordoma

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

Plasma Cell Myeloma

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

Intraspinal Neoplasms

Intradural Lipoma

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

Text continued on page 198
FIGURE 8–17. Lumbar vertebral metastatic disease. Sagittal (A–C) and axial (D and E) precontrast T1-weighted images reveal multiple low-signal-intensity vertebral body lesions. These involve T12, L1, L4, and S1. The metastases are in general round and well demarcated, occasionally extending to the cortex of the vertebral body. Incidental note is made of a lumbarized S1 vertebral body.
FIGURE 8–18. Sacral metastases with epidural tumor causing right S1 nerve root compression. A, The T1-weighted midline sagittal image demonstrates abnormally decreased signal intensity throughout the sacrum, most prominent at the S1 level. Epidural soft tissue involvement is apparent posterior to both S1 and S2. These abnormalities are increased signal intensity on the corresponding T2-weighted sagittal image (B). Irregular enhancement of the sacrum is apparent on the postcontrast T1-weighted sagittal image (C). The epidural disease demonstrates homogeneous enhancement. D, A precontrast T1-weighted axial image at the S1 level confirms the abnormal low signal intensity within the sacrum. The epidural soft tissue mass distorts the thecal sac and severely compresses the right S1 nerve root. The normal left S1 nerve root (arrow) is unaffected. Expansion of the right sacral ala with paraspinal extension is also apparent. This 79-year-old patient with lung cancer presented clinically with a right S1 radiculopathy.
Vertebral body and leptomeningeal metastases. The patient is 35 years old, has breast cancer, and presents with increasing pain and numbness in the legs. A, On the precontrast T1-weighted midline sagittal scan, vertebral body metastases with low signal intensity relative to normal marrow are noted in L1, L3, and L4. The vertebral body lesions are less apparent on the corresponding T2-weighted scan (B). The lesions in L1 and L4 do demonstrate slight hyperintensity relative to normal marrow. Posterior within the thecal sac, a questionable area of abnormal hyperintensity is noted at the L2 level. C, Postcontrast on the T1-weighted scan, the vertebral body lesions demonstrate enhancement to near isointensity with normal marrow. Partial collapse of L4 is now evident. Critical for prognosis and treatment is, however, the identification of two enhancing nodules (small arrows) within the thecal sac, consistent with leptomeningeal tumor spread.
FIGURE 8–20. Expansile L3 vertebral body metastasis. A, The T₂-weighted scan reveals abnormal high signal intensity within the L3 vertebral body. This vertebral body also has an abnormal configuration, consistent with a compression fracture. The posterior margin has a convex outward curvature, compressing the thecal sac. The L3 vertebral body is low signal intensity on the precontrast T₁-weighted sagittal scan (B) and enhances postcontrast (C). Of the axial scans—T₂-weighted (D), precontrast T₁-weighted, (E), and postcontrast T₁-weighted (F)—the postcontrast scan best delineates the thecal sac (arrow), which is severely compressed. The patient, who had nonsquamous cell lung carcinoma, presented clinically with pain radiating into the right lower extremity.
roots can in some cases be identified coursing through the lesion (Fig. 8–21).

Care should be exercised in the diagnosis of a lipoma. The lesion should be of the exact same signal intensity as that of fat on all pulse sequences. The presence of septations, a slight difference in signal intensity from fat, or contrast enhancement make it very unlikely that a fatty lesion is a lipoma (Fig. 8–22).

**Dermoid and Epidermoid**

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

**Teratoma**

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

**Lymphangioma**

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

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**Figure 8–21.** Intradural lipoma. An intradural, high-signal-intensity soft tissue mass is noted at the L1-2 level on the sagittal T1-weighted scan (A). B, On the axial T1-weighted scan, nerve roots (with lower signal intensity) are noted to course through the lesion. The mass is isointense with fat on the intermediate T2-weighted scan (C). This was also the case on all other pulse sequences. An artifactual low-signal-intensity line is noted at the inferior margin of the mass, at the interface with cerebrospinal fluid. This dark band occurs in the direction of the readout gradient and is caused by chemical shift artifact, with the image being acquired at 1.5 T.
Angiolipomas are rare benign tumors composed of lipocytes and abnormal blood vessels. These tumors are epidural in location, occur most commonly in the midthoracic region, and can cause cord compression. Lymphangiomias are typically asymptomatic and treated by surgical resection. These lesions have fluid signal intensity, low on T1- and high on T2-weighted scans. Septa and fat may be present between the fluid spaces.

**Ependymoma**

Ependymomas are slow-growing, well-circumscribed, benign tumors. Complete surgical resection is possible. Ependymomas make up 60% to 70% of all spinal cord tumors. They occur in the third to sixth decades of life. Most arise in the conus, cauda equina, or filum terminale. The cervical cord is the most common site for an intramedullary ependymoma. The clinical presentation is nonspecific and can include motor and sensory deficits and sphincter dysfunction. On MRI, focal cord enlargement limited to two or three levels favors the diagnosis of an ependymoma over an astrocytoma. Virtually all ependymomas enhance strongly after contrast administration (Fig. 8–23).

**Neurofibroma and Schwannoma**

Neurofibromas (Fig. 8–24) and schwannomas (Fig. 8–25) are the most common of the nerve root sheath tumors. Most are intradural extramedullary in location. One third are extradural. A foraminal lesion may be mistaken for a herniated disk (Fig. 8–26). Enhancement postcontrast allows differentiation.

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

**Leptomeningeal Metastases**

The presence of leptomeningeal metastases portends a poor prognosis. One third of all patients with metastases to the brain or spine will eventually acquire leptomeningeal metastatic disease. Breast and lung carcinomas are the most common visceral neoplasms to spread to the subarachnoid space. In the lumbar region on MRI, leptomeningeal metastases can take on several different appearances. There can be large or small nodules or a...
FIGURE 8–23. Mixed papillary ependymoma of the conus medullaris. A, On the sagittal T₂-weighted scan, an intradural extramedullary soft tissue mass is noted. The spinal cord is displaced anteriorly and flattened. B, On the axial T₂-weighted scan, the mass is seen posteriorly and to the right, with severe compression of the cord. C, On the postcontrast T₁-weighted scan, there is heterogeneous enhancement of the mass (arrows), greater peripherally and less centrally. The cord itself is thinned and lies anterior and slightly to the left. The patient presented with slowly increasing low back pain and left lower extremity weakness. Virtually all ependymomas demonstrate strong enhancement after intravenous contrast injection on magnetic resonance imaging.
FIGURE 8–24. Neurofibroma. Pre- (A) and postcontrast (B) sagittal T1-weighted scans reveal a large enhancing soft tissue mass in the left L3-4 neural foramen. The mass is of high signal intensity on the T2-weighted scan (C). Comparison of pre- (D) and postcontrast (E) axial T1-weighted scans reveals a smoothly marginated enhancing lesion, which has expanded the foramen. Contrast enhancement of the mass favors a neural origin and improves lesion demarcation from surrounding soft tissue. Schwannomas tend to enhance in a heterogeneous fashion, often more intense peripherally. Neurofibromas typically demonstrate homogeneous contrast enhancement. The patient is a 66-year-old veteran with neurofibromatosis.
FIGURE 8–25. Lumbar nerve root schwannoma. A, On the T₂-weighted scan, a small round lesion with intermediate signal intensity is noted within the thecal sac at the L5 level. The lesion appears to be immediately adjacent to or part of the L5 nerve root. The lesion is nearly isointense with cerebrospinal fluid on the precontrast T₁-weighted scan (B) and demonstrates prominent enhancement (arrow) postcontrast (C). The lesion, a schwannoma, was confirmed on subsequent surgery performed for lumbar disk disease. Incidental note is made of an L3 vertebral body hemangioma.
FIGURE 8–26. Neurofibroma, mimicking a free disk fragment. Parasagittal T2- (A) and T1-weighted (B) images reveal a soft tissue mass (B, arrow) in the left L4-5 neural foramen. The L4 nerve root is not identified. Comparison of pre- (C) and postcontrast (D) axial T1-weighted scans reveals homogeneous enhancement of the mass (D, arrow). Contrast enhancement in this instance provides important information for differential diagnosis, eliminating from consideration a free disk fragment.
FIGURE 8–27. Leptomeningeal (“drop”) metastases from medulloblastoma. The midline sagittal T2-weighted scan reveals multiple large soft tissue nodules adjacent to the conus, adherent to the cauda equina, and near the termination of the thecal sac. The size and extent of these intrathecal metastases lead to their excellent visualization on the T2-weighted scan in this instance. The patient, a 4-year-old with metastatic medulloblastoma, presented clinically with diminished coordination.

combination (Fig. 8–27). Alternatively (or concurrently), there can be (smooth) coating of nerve roots and the cord (Fig. 8–28). The nerve roots can also appear “beaded” as a result of nodular metastatic deposits (Fig. 8–29). Intramedullary extension of leptomeningeal metastatic disease, although rare, can occur. Contrast-enhanced MRI is markedly superior to CT myelography for detection. The differential diagnosis should include meningeal infection (in immunosuppressed patients), toxoplasmosis, and sarcoidosis. In the latter disease, cord involvement usually dominates.

EFFECT OF TRAUMA
Flexion Injury

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

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

Osteoporotic Compression Fracture

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

Pathologic Compression Fracture

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

Spondylolysis and Spondylolisthesis

In spondylolysis, there is interruption of the pars interarticularis. This may be unilateral or bilateral. Bilateral
FIGURE 8–28. Leptomeningeal metastases. The presence of an intradural soft tissue mass at T12-L1 is questioned on the basis of precontrast sagittal T2- (A) and T1-weighted (B) scans. C, Postcontrast, the lesion is confirmed because of intense enhancement (white arrow). Also noted postcontrast is an enhancing nerve root within the filum terminale and a second smaller mass within the thecal sac at the L2 level (black arrow). Leptomeningeal metastases are best identified postcontrast; enhancement in this case permits diagnosis. This elderly individual with lung carcinoma presented 6 months before the current exam with brain metastases.

FIGURE 8–29. Leptomeningeal metastases. A, On the midline sagittal T2-weighted image, there is diffuse disk degeneration, with narrowing of the thecal sac at multiple levels on the basis of degenerative disease. The lumbar nerves within the thecal sac appear prominent (suggesting nerve root thickening) on both the T2- and precontrast T1-weighted (B) images. C, Postcontrast, there is striking abnormal enhancement of the cauda equina and lumbar nerves, which now also appear somewhat “beaded.” Head computed tomography (not shown) revealed multiple brain metastases. This 83-year-old patient was diagnosed with and treated for small cell carcinoma of the lung 1 year before the current exam. The patient is now admitted with a 2-week history of low back pain, leg weakness, and mental status changes.
involvement allows motion of the posterior elements relative to the adjacent vertebrae. The superior and inferior facets at the involved level can move independently. The superior facet remains attached to the vertebral body. The inferior facet articulates and moves with the more inferior vertebral body. On axial CT, the defects are seen as lucent clefts, oriented in the coronal plane. On axial MRI, the discontinuity of bone may be difficult to visualize. One key to diagnosis is the presence of a “continuous facet” sign from the disk space above to the disk space below. The bony defect is often clearly seen on sagittal MRI.

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

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

With degenerative spondylolisthesis, the midline sagittal image demonstrates narrowing of the spinal canal (Fig. 8–30). The posterior elements are contiguous with and, therefore, move anteriorly with the displaced vertebral body. When spondylolisthesis occurs in combination with spondylolysis, the canal is typically not narrowed because the posterior elements move independently from the vertebral body (Fig. 8–31). The adjacent posterior elements remain in alignment, and the spinal canal may widen in this situation.

Retrolisthesis

A retrolisthesis is a posterior subluxation of a vertebral body relative to the adjacent lower body. This is caused
by disk degeneration with preservation of the facet joints. A retrolisthesis can occur after surgery or other intervention (Fig. 8–32), with resultant neural foraminal narrowing (and nerve root impingement). This is one cause of the failed back syndrome. Retrolisthesis is most common in the lumbar and cervical spine. In the lumbar spine, L3-4 and L4-5 are the most frequently involved levels. Disk bulges and spurs commonly accompany a retrolisthesis. Central canal stenosis is uncommon, but neural foraminal narrowing is common.

**Pseudomeningocele**

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

**Postoperative Lumbar Spine**

The recurrence of symptoms after lumbar surgery, which occurs in 10% to 40% of patients, defines the failed back syndrome. Causative factors include recurrent disk herniation, spinal stenosis, arachnoiditis, and epidural fibrosis (scar). MRI plays an extremely valuable role in the evaluation of the patient with recurrent pain after lumbar spine surgery. On postcontrast scans, postoperative scar can be differentiated from a recurrent or residual disk herniation; the distinction is critical for the therapeutic decision-making process (Fig. 8–33). This use of intravenous contrast accounts for a substantial amount of the contrast used overall in spine MRI.

In the postoperative back, postcontrast scans should
FIGURE 8–32. Retrolisthesis. A, The sagittal T₁-weighted image near the midline shows disk space narrowing at L4-5 and mild posterior displacement of the L4 vertebra on L5. A small disk bulge is present at L4-5 with thin, high-signal-intensity type II end plate changes adjacent to the L4-5 and L3-4 disks. B, The parasagittal T₁-weighted image through the right neural foramina demonstrates narrowing of the L4-5 bony foramen. The inferior aspect of the foramen is obliterated by the posteriorly displaced L4 vertebra and the associated disk bulge. The right L4 nerve root exits under the L4 pedicle with a small amount of surrounding high signal intensity fat. The superior articular facet of L5 (arrow) has moved in an anterior and cephalad direction, obliterating the inferior aspect and narrowing the superior aspect of the neural foramen. C, The sagittal T₁-weighted image after intravenous contrast administration confirms the retrolisthesis at L4-5. The malalignment is more easily detected because of enhancement of the epidural venous plexus along the posterior margin of the vertebrae. D, The comparable sagittal, intermediate T₂-weighted image again demonstrates the malalignment at L4-5. Decreased T₂ signal intensity in the L3-4 and L4-5 disks is due to disk degeneration at these levels. The patient, 41 years old, presents with recurrent low back and bilateral leg pain after L4-5 disk surgery.

FIGURE 8–33. Postdiskectomy scar tissue. Two months after a right laminectomy and diskectomy, a soft tissue mass is identified anterior and to the right of the thecal sac on the precontrast T₁-weighted axial scan (A). B, Postcontrast, there is uniform enhancement of this abnormal soft tissue (arrow), consistent with scar. The right S₁ nerve root can only be identified postcontrast surrounded by scar.
be obtained within 20 minutes after intravenous contrast administration. After this time, there may be diffusion of contrast from enhancing to nonenhancing tissue, making interpretation difficult. Postoperative scar demonstrates homogeneous enhancement as a result of intrinsic vascularity. However, this is not seen consistently until 3 months after surgery. Scar is one cause of persistent pain after lumbar disk surgery and is in general a contraindication to further surgery. Although the presence of a soft tissue mass favors the diagnosis of a recurrent disk, scar can also have this appearance (Fig. 8–34). Thus, noncontrast scans are not reliable for differentiation. In the patient with recurrent pain and postoperative scar, the fibrosis is often extensive and surrounds an exiting nerve root, presumably the basis for symptoms. A recurrent or residual disk herniation (Fig. 8–35) will not show enhancement on MRI scans obtained after contrast administration (assuming, of course, that these are obtained within 20 minutes of injection). Correct diagnosis on MRI mandates the use of thin sections, 3 mm or less, to avoid partial volume effects. A recurrent disk herniation will be seen as a focal, smooth posterior protrusion of nonenhancing soft tissue (contiguous with the native disk). The disk is commonly circumscribed posteriorly by a thin rim of enhancing soft tissue (Fig. 8–36) corresponding to scar (but in minimal amounts with a normal expected finding).

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

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**ARTHRITIS**

Ankylosing spondylitis is an inflammatory disease of unknown etiology. The sacroiliac joints are involved early in the disease course. Erosion of cortical margins with subchondral bony sclerosis is seen first. Joint space widening, due to bony erosion, follows. The end result is fusion (obliteration) of the sacroiliac joints. In the spine, syndesmophytes are the hallmark of ankylosing spondylitis (Fig. 8–37). These slender, vertical ligamentous calcifications extend from the osseous excrescence of one vertebral body to the next. In the spine, the inflammation associated with ankylosing spondylitis occurs at the junction of the annulus fibrosus and the vertebral body. The outer annular fibers become replaced by bone, or syndesmophytes, which eventually bridge adjacent vertebral bodies. In advanced disease, this leads to the appearance on plain film of a “bamboo spine.” One significant complication of ankylosing spondylitis is bony fracture after minor trauma. In the cervical spine, this can lead to quadriplegia.

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**DEGENERATIVE DISEASE**

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

**Focal Fat Deposition**

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

**Schmorl’s Node**

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

**Synovial Cyst**

In the spine, synovial cysts are associated with degenerative facet disease. When symptomatic, a synovial cyst can present with radicular pain, often sciatic in nature. This can mimic a disk herniation. Large synovial cysts can compress the thecal sac. On CT, the lesion can be hypo- or hyperdense. Synovial cysts may be calcified and are recognized by their location adjacent to a facet joint. On MRI, the signal intensity of the fluid within the cyst is variable; synovial cysts can have any combination of low or high signal intensity on T₁- and T₂-weighted scans. Postcontrast, the cyst capsule and any solid component will demonstrate enhancement (Fig. 8–38). Delayed enhancement of the cyst contents has been observed. Recognition of the relationship to the facet joint is critical for diagnosis.

**Degenerative Disk and End Plate Changes**

There are many signs of disk degeneration on MRI. There can be loss of disk height. Annular tears, with high signal intensity on T₂-weighted scans, may be seen. However, decreased signal intensity of the disk itself on T₂-weighted scans is the most sensitive indicator of early disk degeneration. This finding is often referred to in clinical dictations as disk dehydration or desiccation and occurs with varying degrees and may early on involve only a part of the disk. The actual cause of the decrease in signal intensity is a decrease in proteoglycans and in the ratio of chondroitin sulfate to keratin sulfate. With the exception of trauma, disk herniation without changes

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FIGURE 8–34. Differentiation of scar from disk in the postoperative back. A, The T₁-weighted sagittal image to the right of midline reveals abnormal soft tissue (arrow) projecting posterior to the L5-S1 intervertebral disk. B, After contrast administration, this tissue enhances intensely. Enhancement is also apparent both superior and inferior to the L5-S1 intervertebral disk, at the interface with the adjacent vertebral bodies. C, A T₁-weighted axial view at the inferior L5 level confirms the abnormal soft tissue in the ventral epidural space. The right laminectomy defect is also apparent. D, Postcontrast, the abnormal extradural soft tissue (which is now seen to surround the right S1 nerve root) is noted to enhance. Enhancement within soft tissue posteriorly at the laminectomy site and within the right paraspinal musculature is also noted. The patient presented with continued right leg pain 2 months after diskectomy at L5-S1. The anterior epidural mass in this case, which appears contiguous to the L5-S1 disk, would be suspicious for a recurrent disk herniation on the precontrast scans. The homogeneous enhancement of the abnormality, however, allows confident diagnosis of the lesion as epidural fibrosis (scar).
FIGURE 8–35. Postdisectomy recurrent disk extrusion. T₂- (A) and T₁-weighted (B) midline sagittal scans reveal abnormal soft tissue anterior to the thecal sac at the L4-5 and L5-S1 levels. Two previous percutaneous diskectomies had been performed. C, Postcontrast, the majority of abnormal soft tissue at each level does not enhance. Enhancing soft tissue (C, arrows) above and below the L4-5 disk space level corresponds to a dilated epidural venous plexus. Comparison of pre- (D) and postcontrast (E) T₁-weighted axial scans at the L4-5 level confirms the presence of a recurrent disk herniation (arrow), with a small amount of surrounding enhancing granulation tissue. Lumbar microdiscectomy was subsequently performed.
FIGURE 8–36. Pre- and postdisectomy exams in a patient presenting with a disk extrusion and recurrence after surgery. Also important to the clinical case and surgical approach is the presence of a transitional vertebra. The preoperative exam includes sagittal T2- (A), sagittal precontrast T1- (B), sagittal postcontrast T1- (C), axial precontrast T1- (D) and axial postcontrast T1-weighted (E) images. The postoperative exam, performed 1 year later, includes the same sequences, specifically sagittal T2- (F), sagittal precontrast T1- (G), sagittal postcontrast T1- (H), axial precontrast T1- (I), and axial postcontrast T1-weighted (J) images. It should be recognized first that the patient has a transitional vertebra. The level with significant disease is likely to be L4-5, with L5 being sacralized. This was confirmed by reference to plain radiographs. This patient actually had four subsequent magnetic resonance imaging (MRI) exams, with one reader dictating the level as L5-S1 twice and two other readers dictating the level correctly as L4-5 once each. On the preoperative exam, there is a moderate-size right paracentral disk extrusion. Contrast enhancement provides minimal improvement in demarcation of the abnormal disk. On the MRI scan obtained a year later, with intervening surgery, there is a larger recurrent right paracentral disk extrusion. Postcontrast, there is a thin circumferential rim of enhancing scar tissue, which improves differentiation of the disk from adjacent cerebrospinal fluid. The lack of enhancement of the majority of the soft tissue mass confirms that this represents recurrent disk disease. Postoperative changes caused by the right-sided laminectomy are also noted.
Ankylosing spondylitis. A and B, On parasagittal T1-weighted images of the lumbar spine, there are prominent anterior osteophytes (curved arrows), which appear to bridge the disk space at several levels. C, The anteroposterior plain film of the lumbar spine reveals the sacroiliac joints to be obliterated, with bony bridges (marginal syndesmophytes) connecting adjacent vertebral bodies.
Synovial cyst. Images from two patients with similar symptoms are presented. The first is 60 years old and has experienced increasing left leg pain and intermittent numbness over the last 6 months. A, On the sagittal T2-weighted scan, a low-signal-intensity abnormality is noted within the bony spinal canal, immediately posterior to the L4-5 intervertebral disk. There is displacement and compression of the thecal sac. B, Before contrast administration, the lesion is difficult to identify. C, After contrast injection, there is rim enhancement. On this scan, the lesion (a synovial cyst) appears (correctly) to be extradural. Pre- (D) and postcontrast (E) axial images at the L4-5 level are presented from the second patient’s exam. There is facet hypertrophy bilaterally. D, Precontrast, the question is raised of a left-sided lesion causing compression posteriorly of the thecal sac. E, Postcontrast, there is rim enhancement, which improves the differentiation of the lesion from cerebrospinal fluid within the thecal sac. The lesion appears cystic in nature by signal intensity and enhancement characteristics. The lesion (another synovial cyst) is contiguous with the left facet joint.

of disk degeneration is extremely unusual. This can be very helpful in directing the film reader toward the disk space levels that should be more closely examined (those demonstrating disk desiccation).

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

Degenerative vertebral body end plate changes are a common finding on MRI of the lumbar spine. A change in signal intensity of the marrow space adjacent to the end plate is by far the most clear indicator of degenerative end plate disease (Fig. 8–39). These changes are parallel and directly adjacent to the disk space. Such changes typically involve the entirety of both end plates (surrounding a degenerated disk), although involvement of just one end plate (and even just a portion of one) can occur.

Type I end plate changes reflect increased water content and are low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Type I end plate changes enhance after contrast administration, often to isointensity with marrow fat. Type I end plate changes can be mimicked by two other disease entities; differential diagnosis is critical. Metastatic disease can at times resemble type I end plate changes. However, typically, there are multiple additional lesions. Isolated involvement of the end plate by metastatic disease is uncommon. Disk space infection and adjacent osteomy-
FIGURE 8–39. Degenerative end plate changes. A–C, Type I end plate changes histologically show vascular infiltration, fibrosis, and granulation tissue between thickened bony trabeculae. Increased water content results in both $T_1$ and $T_2$ lengthening. On magnetic resonance imaging (MRI) the end plates show increased signal intensity on $T_2$-weighted scans (A) and decreased signal intensity on $T_1$-weighted scans (B). The signal is usually parallel to the end plates and directly adjacent to the intervertebral disk. C, The affected end plates commonly enhance (to isointensity with normal marrow) after intravenous contrast administration. D, Type II end plate changes show fatty infiltration interposed between thickened trabeculae histologically. MRI reveals increased signal intensity on both $T_1$- (D) and $T_2$-weighted scans (not shown) compared with normal marrow. E and F, Another end plate pattern, Type III, consists of sclerotic changes. On MRI, the end plates are low signal intensity on both $T_1$- (E) and $T_2$-weighted (F) scans. These areas correspond with sclerosis on plain x-ray films.
elitis can also resemble type I end plate changes. However, with infection, the disk should be grossly abnormal, the demarcation between disk and body lost, and a paraspinous mass often present.

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

**DISK HERNIATION**

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

Tears of the annulus fibrosus are classified into three types. Concentric, or type I, is parallel to the curvature of the outer disk. Radial, or type II, involves all the layers of the annulus from the nucleus pulposus to the surface. Transverse, or type III, involves the insertion of Sharpey's fibers into the ring apophysis. Tears of the annulus fibrosus are high signal intensity on T2-weighted scans. A tear will also enhance after intravenous gadolinium chelate administration (Fig. 8–40). Contrast enhancement is due to the presence of fibrovascular (granulation) tissue, a result of the body's normal reparative process.

A disk or annular bulge is an extension of the posterior disk beyond the margin of the adjacent vertebral end plates but without focal disk protrusion (Fig. 8–41). The posterior disk margin forms a smooth curvilinear contour. A disk bulge by definition is broad based and circumferential. A disk bulge occurs as a result of laxity of and tears within the annulus fibrosus. It is a sign of early disk degeneration. A disk bulge can, however, narrow the spinal canal and the inferior neural foramen.

A disk protrusion is a herniation of the nucleus through a (small) tear in the annulus but still contained by outer fibers of the annulus. A disk protrusion is differentiated from a bulge by axial imaging, with demonstration of focal extension of disk material beyond the margin of the vertebral end plates (Fig. 8–42). Although disk protrusion and extrusion are distinct entities, differentiated by the degree of rupture of the annulus, this can rarely be appreciated on MRI. In common usage, the term disk protrusion is reserved for a small herniation and disk extrusion for a large herniation of disk material through the ruptured annulus.

A disk extrusion is a herniation of the nucleus through the ruptured annulus with no intact remaining annular fibers. It is important to specify, when interpreting MRI exams, whether a disk extrusion (or protrusion) is central (Fig. 8–43), paracentral (Fig. 8–44), foraminal (Fig. 8–45), or lateral (Fig. 8–46) in location. A disk extrusion, when combined with lateral stenosis, can cause nerve root ischemia with eventual fibrosis, leading to irreversible axonal damage. In an extrusion, the disk material remains in contiguity with the parent disk. This distinction differentiates a disk extrusion from a free fragment. Even without surgery, granulation tissue forms around

**FIGURE 8–40.** Annular tear. T1-weighted pre- (A) and postcontrast (B) axial images demonstrate a mild, focal, asymmetrical extension of the posterior disk margin. The disk abuts, but does not significantly displace, the right L5 nerve root sleeve. The postcontrast image reveals a curvilinear area of high signal intensity (arrow) paralleling the posterior disk margin because of enhancement of a concentric tear in the outer fibers of the annulus fibrosus. A T1-weighted image was not acquired in this patient in the axial plane. Such an image would have also clearly depicted the tear, with abnormal hyperintensity.

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Disk bulge. A, The sagittal T₂-weighted image demonstrates decreased signal intensity in the L3-4, L4-5, and L5-S1 disk spaces consistent with disk degeneration. The posterior disk margins extend beyond the adjacent vertebral end plates and indent the anterior thecal sac at these levels. The sagittal pre- (B) and postcontrast (C) T₁-weighted images again show mild posterior extension of disk material from L3-4 through L5-S1. The disk margin is better delineated after the administration of intravenous contrast because of enhancement of the epidural venous plexus. D, The axial T₁-weighted image through the L3-4 level reveals a generalized disk bulge with mild convexity of the posterior disk margin. The disk material narrows the lateral recesses bilaterally (arrows). The posterior disk margin has a smooth curvilinear contour with no focal disk protrusion.
FIGURE 8–42. Disk protrusion. Sagittal precontrast T2- (A) and T1-weighted (B) scans reveal extension of disk material beyond the vertebral end plates at L4-5. C, Enhancement of de novo scar and epidural venous plexus postcontrast improves delineation of the disk margin from cerebrospinal fluid on the T1-weighted scan. Comparison of pre- (D) and postcontrast (E) axial T1-weighted scans through the L4-5 disk level reveals the extension of disk material, which is relatively small, to be focal and central in location.
FIGURE 8–43. Central disk extrusion. Midline sagittal precontrast T₂-weighted (A) and postcontrast T₁-weighted (B) scans demonstrate moderate compression of the thecal sac by posterior extension of disk material at the L4-5 level. Loss of the normal high signal intensity (on the T₂-weighted scan) of the intervertebral disks at L4-5 and L5-S1 is compatible with disk degeneration. Axial pre- (C) and postcontrast (D) scans at the L4-5 level demonstrate the disk extrusion to be central in location. At high field, with current software, an alternative imaging approach (driven by cost) is to add an axial fast spin echo T₂-weighted scan and not acquire the postcontrast scans.
Figure 8–44. Right paracentral disk extrusion. Sagittal precontrast T₂-weighted (A) and postcontrast T₁-weighted (B) scans, just to the right of midline, demonstrate substantial compression of the thecal sac by disk material at the L5-S1 level. Axial pre- (C) and postcontrast (D) scans at the L5-S1 level demonstrate this large disk extrusion to be paracentral in location. This 32-year-old patient presented with right leg pain.
Foraminal disk extrusion. Parasagittal precontrast T₂- (A), precontrast T₁- (B), and postcontrast T₁-weighted (C) scans reveal extension of disk material into the inferior portion of the foramen at the L5-S1 level. After contrast administration, there is enhancement of a thin line (presumably scar) separating the disk extrusion from the superior portion of the foramen, which contains the L5 nerve root (surrounded by fat). Axial pre- (D) and postcontrast (E) scans at the L5-S1 level depict very clearly the focal extrusion of disk material within the foramen. The dorsal root ganglion is seen just lateral to the extrusion, with normal enhancement postcontrast. Axial T₂-weighted scans (not shown), although excellent for demonstrating central and paracentral disk disease, are poor for foraminal disease; differentiation of disk material and other foraminal contents is difficult.
FIGURE 8–46. Lateral disk extrusion. Precontrast sagittal T₂- (A) and T₁-weighted (B) scans to the left of midline demonstrate extension of the L₄-₅ disk posteriorly into the left L₄-₅ neural foramen. The exiting left L₄ nerve root is identified just above the disk extrusion. C. The precontrast axial T₁-weighted scan reveals a large focal lateral herniation of disk material (black arrow). The exiting L₄ nerve root (small white arrow) is seen on the right but is obscured by the herniated disk material on the left. D. The postcontrast T₁-weighted axial scan provides clearer delineation of the disk extrusion as a result of enhancement of the epidural venous plexus and foraminal veins. The displaced left L₄ nerve root (D, arrow) can now be distinguished from the nonenhancing extruded disk. Mass effect on the left side of the thecal sac is also more apparent.
the extruded disk, part of the body’s normal reparative process. This tissue enhances postcontrast, forming a thin rim of high signal intensity “wrapping” the extruded disk material on enhanced T1-weighted exams. This appearance can be confusing to radiologists who have experience principally with nonenhanced MRI scans. Scar in the nonoperated back may potentially assist in recovery by limiting the herniation and with contraction decreasing the degree of compression of neural structures. The neurosurgeons of yesteryear were very familiar with the fact that a substantial reduction in size of a disk herniation could be observed with conservative therapy. Thus, follow-up MRI scans can demonstrate a reduction in size of a disk extrusion without intervening surgery (Fig. 8–47).

Ninety percent of lumbar disk extrusions occur at L4-5 or L5-S1. Of the remainder, most occur at L3-4. Central lesions may cause no symptoms, with the exiting nerve roots unaffected. Paracentral lesions cause symptoms as a result of compression of the exiting nerve root. For example, the S1 nerve root will be compressed by a paracentral L5-S1 disk extrusion. Lateral disk extrusions are the least common because the annulus is thinnest posteriorly. Superior migration of lateral fragments is common. A lateral disk extrusion will compress the ganglion or nerve root within the neural foramen. This causes radiculopathy of the nerve root above the interspace. For example, a lateral disk extrusion at the L3-4 level will compress the L3 nerve. Lateral disk extrusions occur beyond the termination of the nerve root sleeve. Thus, myelography is relatively insensitive to lateral disk disease. Myelographic findings with a disk extrusion include displacement of the contrast-filled sac, elevation, displacement, or amputation of the nerve root sleeve, and nerve root enlargement (as a result of edema). When a nerve is acutely compressed by a disk extrusion, edema of the nerve root in question can occasionally be seen within the thecal sac on MRI (with nerve root enlargement and abnormal high signal intensity on the T2-weighted exam). Lumbar nerve root

**FIGURE 8–47. Resolution of L4-5 paracentral disk protrusion with conservative therapy.** Precontrast sagittal (A) and postcontrast axial (B) T1-weighted scans from the patient’s initial clinical presentation are compared with scans obtained 1 year later (C and D). At presentation, disk material protrudes posteriorly on the sagittal image at the L4-5 level (A). The protrusion (B, arrow) is well delineated by a thin rim of enhancement on the postcontrast axial scan and is noted to be paracentral in location. On the follow-up exam obtained 1 year later, there is no abnormal posterior extension of disk material on the sagittal scan (C). Enhancing scar tissue is noted on the postcontrast axial T1-weighted scan (D) but without compression of the thecal sac.
FIGURE 8–48. Enhancing nerve root resulting from compression by a large free fragment. Comparison of pre- (A) and postcontrast (B) T₁-weighted axial scans at the L5-S1 level reveals intense enhancement of the left S1 nerve root (arrow) within the thecal sac. This is confirmed on the postcontrast T₁-weighted sagittal scan (C, arrow), which also identifies nerve root compression by a large disk fragment. The patient was referred for a magnetic resonance imaging scan because of recent onset of a left S1 radiculopathy.
FIGURE 8–49. Free disk fragment. A, On the T₂-weighted sagittal scan, a soft tissue mass with abnormal high signal intensity is identified posterior to S1. This mass (white arrow) is isointense with the remaining disk material at the L5-S1 level on the T₁-weighted sagittal scan (B). C, Postcontrast, the periphery of the mass enhances. Inspection of pre- (D) and postcontrast (E) axial scans through the S1 vertebral body confirms the presence of a free disk fragment. The fragment is “wrapped” by enhancing scar (arrows), deforms the thecal sac, and compresses the left S1 nerve root.
Figure 8–50. Free disk fragment. A, The sagittal T₁-weighted scan reveals only mild disk degeneration at L4-5. B, On the precontrast T₁-weighted sagittal scan, abnormal soft tissue (arrow) is noted partly contiguous with the L4-5 disk but posterior to the L5 vertebral body. C, Postcontrast, the lesion (a free fragment) is better delineated because of the surrounding rim of enhancing tissue. Examining the T₁-weighted scan in retrospect, the lesion is noted to be of high signal intensity and thus difficult to differentiate from cerebrospinal fluid. The free fragment (D, white arrow), which has migrated inferiorly, is well demonstrated on pre- (D) and postcontrast (E) axial T₁-weighted scans, which also reveal compression of the right L5 nerve root (E, black arrow). The patient presented 5 days after injury with low back and right leg pain.
Figure 8–51. Free disk fragment within the L4-5 foramen. A, Abnormal soft tissue is noted within the left neural foramen on the precontrast axial T₁-weighted scan. B, After contrast administration, a thin rim of enhancement better delineates the lesion, which otherwise does not change in signal intensity. Contrast administration in this instance permits the differentiation of a neural origin tumor within the foramen (which would enhance; see Fig. 8–26) from a migrated free fragment (which, as illustrated by this case, does not enhance).

enhancement is not uncommon with acute disk extrusions, although many radiologists are unfamiliar with this appearance. Their unfamiliarity is due to the fact that most screening exams of the lumbar spine for disk disease are performed without contrast enhancement. Lumbar nerve root enhancement occurs as a result of disruption of the blood-nerve root barrier. Its presence supports the clinical significance of a compressive lesion (Fig. 8–48).

With a free fragment or sequestered disk, the herniated disk material is separate from the parent disk. A free fragment may be anterior (contained by) or posterior to the posterior longitudinal ligament. When anterior, a thin midline septum directs the fragment paracentrally away from the midline. Free fragments have characteristic signal intensity on MRI, intermediate to low signal intensity on T₁-weighted scans, and high signal intensity (but less than that of CSF) on T₂-weighted scans (Fig. 8–49). Free fragments can migrate superiorly or inferiorly (Fig. 8–50) within the epidural space or into the neural foramen (Fig. 8–51).