

Back and Neck Pain
Chapter 17

John W. Engstrom, MD
Betty Anker Fife Distinguished Professor of Neurology
University of California, San Francisco

Richard A. Deyo, MD, MPH
Kaiser Permanente Professor of Evidence-Based Family Medicine
Oregon Health and Science University

The importance of back and neck pain in our society is underscored by the following: (1) the cost of back pain in the United States exceeds ~\$100 billion annually, including one third in direct health care expenses plus two thirds due to indirect costs due to loss of wages and productivity; (2) back symptoms are the most common cause of disability in those <45 years; (3) low back pain is the second most common reason for visiting a physician in the United States; and (4) ~1% of the U.S. population is chronically disabled because of back pain.

Anatomy of the Spine

The anterior portion of the spine consists of cylindrical vertebral bodies separated by intervertebral disks and held together by the anterior and posterior longitudinal ligaments. The intervertebral disks are composed of a central gelatinous nucleus pulposus surrounded by a tough cartilaginous ring, the annulus fibrosus. Disks are responsible for 25% of spinal column length and allow the bony vertebrae to move easily upon each other. Desiccation with age results in loss of height (**Figs. 17-1** and **17-2**). The disks are largest in the cervical and lumbar regions where movements of the spine are greatest. The functions of the anterior spine are to absorb the shock of body movements such as walking and running, and protect the contents of the spinal canal.

The posterior portion of the spine consists of the vertebral arches and several processes. Each arch consists of paired cylindrical pedicles anteriorly and paired laminae posteriorly. The vertebral arch also gives rise to two transverse processes laterally, one spinous process posteriorly, plus two superior and two inferior articular facets. The apposition of a superior and inferior facet constitutes a *facet joint*. The functions of the posterior spine are to protect the spinal cord and nerves within the spinal canal and to provide an anchor for the attachment of muscles and ligaments. The contraction of muscles attached to the spinous and transverse processes and laminae works like a system of pulleys and levers that results in flexion, extension, and lateral bending movements of the spine. The laminae and facets posteriorly are joined to the vertebral body by a pedicle (Figure 17-1). The lateral portion of the spinal canal medial to the foramen for the nerve root is the lateral recess.

Nerve root injury (*radiculopathy*) is a common cause of neck, arm, low back, buttock, and leg pain (Figs. 25-2 and 25-3). The nerve roots exit at a level above their respective vertebral bodies in the cervical region (e.g.- C7 nerve root exits at the C6-C7 level) and below their respective vertebral bodies in the thoracic and lumbar regions (e.g. - T1 nerve root exits at the T1-T2 level). The cervical nerve roots follow a short intraspinal course before exiting. The spinal cord ends at the L1 or L2 level of the bony spine; the lumbar nerve roots follow a long intraspinal course and can be injured anywhere from the upper lumbar spine to their exit at the intervertebral foramen. For example, lumbar disk herniation is most commonly paracentral. As a result, disk herniation at the at the L4-L5 level typically produces compression of the traversing L5 nerve root (Fig. 17-3).

Pain-sensitive structures of the spine include the periosteum of the vertebrae, dura, facet joints, annulus fibrosus of the intervertebral disk, epidural veins and arteries, and the posterior longitudinal ligament. Disease of these diverse structures may explain many cases of back pain without nerve root compression. The nucleus pulposus of the intervertebral disk is not pain-sensitive under normal circumstances. Pain sensation from within the spinal canal is conveyed partially by the sinuvertebral nerve that arises from the spinal nerve at each spine segment and reenters the spinal canal through the intervertebral foramen at the same level. The lumbar and cervical spine possess the greatest potential for normal movement and are most prone to injury.

APPROACH TO THE PATIENT:

Back Pain

TYPES OF BACK PAIN

Understanding the types of pain reported by patients is essential. Attention is also focused on identification of risk factors for serious underlying diseases; the majority of these are due to radiculopathy, fracture, tumor, or infection (**Table 17-1**).

Local pain is caused by injury to pain-sensitive structures that compress or irritate sensory nerve endings. The site of the pain is near the affected part of the back.

Pain referred to the back may arise from abdominal or pelvic viscera. The pain is usually described as primarily abdominal or pelvic but is accompanied by back pain and usually unaffected by posture. The patient may occasionally complain of back pain only (e.g.-abdominal aortic aneurysm).

Pain of spine origin may be located in the back or referred to the buttocks or legs. Diseases affecting the upper lumbar spine tend to refer pain to the lumbar region, groin, or anterior thighs. Diseases affecting the lower lumbar spine tend to produce pain referred to the buttocks, posterior thighs, or rarely the calves or feet. Referred or "sclerotomal" pain may explain instances where the leg pain crosses multiple dermatomes (e.g.-the anterior thigh) without evidence of nerve root compression.

Radicular back pain is typically sharp and radiates from the low back to a leg within the territory of a nerve root (see "Lumbar Disk Disease," below). Coughing, sneezing, or voluntary contraction of abdominal muscles (lifting heavy objects or straining at stool) may elicit the radiating pain. The pain may increase in postures that stretch the nerves and nerve roots. Sitting with the leg outstretched places traction on the sciatic nerve and L5 and S1 roots because the nerve passes posterior to the hip. The femoral nerve (L2, L3, and L4 roots) passes anterior to the hip and is not stretched by sitting. The description of the pain alone often fails to distinguish between sclerotomal pain and radiculopathy.

Pain associated with muscle spasm, although of obscure origin, is commonly associated with many spine disorders. The spasms are accompanied by abnormal posture, tense palpable paraspinal muscles, and dull or achy pain in the paraspinal region.

Knowledge of the circumstances associated with the onset of back pain is important when weighing possible serious underlying causes for the pain. Some patients involved in accidents or work-related injuries may exaggerate their pain for the purpose of compensation or for psychological reasons.

EXAMINATION OF THE BACK

A physical examination that includes the abdomen and rectum is advisable. Back pain referred from visceral organs may be reproduced during palpation of the abdomen [pancreatitis, abdominal aortic aneurysm (AAA)] or percussion over the costovertebral angles (pyelonephritis).

The normal spine has a cervical and lumbar lordosis, and a thoracic kyphosis. Exaggeration of these normal alignments may result in hyperkyphosis of the thoracic spine or hyperlordosis of the lumbar spine. Inspection may reveal a lateral curvature of the spine (scoliosis) or an asymmetry in the prominence of the paraspinal muscles, suggesting muscle spasm. Back pain of bony spine origin is often reproduced by palpation or percussion over the midline spinous processes of the affected vertebrae.

Forward bending is often limited by paraspinal muscle spasm; the latter may flatten the usual lumbar lordosis. Flexion at the hips is normal in patients with lumbar spine disease, but flexion of the lumbar spine is limited and sometimes painful. Lateral bending to the side opposite the injured spinal element may stretch the damaged tissues, worsen pain, and limit motion. Hyperextension of the spine (with the patient prone or standing) is limited when nerve root compression, facet joint pathology, or other bony spine disease is present.

Pain from hip disease may mimic the pain of lumbar spine disease. Hip pain can be reproduced by internal and external rotation at the hip with the knee and hip in flexion (Patrick sign) and by percussing the heel with the examiner's palm while the leg is extended.

With the patient supine, passive flexion of the extended leg at the hip stretches the L5 and S1 nerve roots and the sciatic nerve (straight leg-raising maneuver). Passive dorsiflexion of the foot during the maneuver adds to the stretch. While flexion to at least 80° is normally possible without causing pain, many patients normally report a tight, stretching sensation in the hamstring muscles unrelated to back pain.. The *straight leg-raising (SLR)* test is positive if the maneuver reproduces the patient's usual back or limb pain. Eliciting the SLR sign in the sitting position can help determine if the finding is reproducible. The patient may describe pain in the low back, buttocks, posterior thigh, or lower leg, but the *key feature is reproduction of the patient's usual pain*. The *crossed SLR sign* is positive when flexion of one leg reproduces the usual pain in the opposite leg or buttocks. The crossed SLR sign is less sensitive but more specific for disk herniation than the SLR sign. The nerve or nerve root lesion is always on the side of the pain. The *reverse SLR sign* is elicited by standing the patient next to the examination table and passively extending each leg with the knee fully extended. This maneuver, which stretches the L2-L4 nerve roots, lumbosacral plexus, and femoral nerve, is considered positive if the patient's usual back or limb pain is reproduced.

The neurologic examination includes a search for focal weakness or muscle atrophy, focal reflex changes, diminished sensation in the legs, or signs of spinal cord injury. The examiner should be alert to the possibility of breakaway weakness, defined as fluctuating strength during muscle testing. Breakaway weakness with pain may be due to pain during muscle strength testing, lack of effort, or underlying true weakness or a combination of these factors. Breakaway weakness without pain is almost always due to a lack of effort. In uncertain cases, electromyography (EMG) can determine whether or not true weakness due to nerve tissue injury is present. Findings with specific nerve lumbosacral nerve root lesions are shown in Table 17-2 and are discussed below.

LABORATORY, IMAGING, AND EMG STUDIES

Routine laboratory studies are rarely needed for the initial evaluation of nonspecific acute (<3 months duration) low back pain (ALBP). If risk factors for a serious underlying cause are present (Table 17-1), then laboratory studies [complete blood count (CBC), erythrocyte sedimentation rate (ESR), urinalysis] are indicated.

CT scans are used as a primary screening modality for moderate to severe spine trauma. In the setting of acute trauma, CT scanning is superior to routine x-rays for the detection of fractures involving posterior spine structures, craniocervical and craniothoracic junctions, C1 and C2 vertebrae, bone fragments within the spinal canal, or misalignment; In the absence of risk factors, these imaging studies are rarely helpful in nonspecific ALBP. MRI and CT-myelography are the radiologic tests of choice for evaluation of most serious diseases involving the spine. MRI is superior for the definition of soft tissue structures, whereas CT-myelography provides optimal imaging of the lateral recess of the spinal canal, bony lesions, and is better tolerated by claustrophobic patients. When soft tissue imaging is crucial, conscious sedation with a benzodiazepine (e.g.-lorazepam) prior to the scan may enable the patient to tolerate the MRI. While the added diagnostic value of modern neuroimaging is significant, there is concern that these studies may be overutilized in patients with benign ALBP.

Electromyography studies can be used to assess the functional integrity of the peripheral nervous system. In the setting of a focal sensory deficits, sensory nerve conduction studies are normal if there is nerve root damage because the nerve roots are proximal to the sensory nerve cell bodies in the dorsal root ganglia. Injury to nerve tissue distal to the dorsal root ganglion (e.g.-plexus or peripheral nerve). results in reduced sensory nerve signals. The needle EMG complements the nerve conduction studies by detecting denervation or reinnervation changes in a

myotomal (segmental) distribution. Multiple muscles supplied by different nerve roots and nerves are sampled; the pattern of muscle involvement indicates the nerve root(s) responsible for the injury. Needle EMG provides objective information about motor nerve fiber injury when the clinical evaluation of weakness is limited by pain or poor effort. EMG and nerve conduction studies will be normal when only limb or back pain unassociated with motor nerve root injury is present, or only sensory nerve root injury or irritation is present. EMG studies are normal in the presence of isolated sclerotomal pain.

CAUSES OF BACK PAIN

(Table 17-3)

Congenital Anomalies of the Lumbar Spine

Spondylolysis is a bony defect in the pars interarticularis (a segment near the junction of the pedicle with the lamina) of the vertebra; the etiology is usually congenital and reflects multiple microfractures within the weakened abnormal bony segment. It occurs in up to 6% of adolescents. The defect (usually bilateral) is best visualized on plain x-rays, CT scan, or bone scan and is frequently asymptomatic. Symptoms may occur in the setting of a single injury, repeated minor injuries, or growth. Athletic adolescents are particularly prone to symptoms. Spondylolysis is the most common cause of persistent low back pain in adolescents.

Spondylolisthesis is the anterior slippage of the vertebral body, pedicles, and superior articular facets, leaving the posterior elements behind. Spondylolisthesis can be associated with spondylolysis, congenital anomalies, degenerative spine disease, or other causes of mechanical weakness of the pars (e.g.-infection, osteoporosis, tumor, trauma, prior surgery). The slippage may be asymptomatic or may cause low back pain and hamstring tightness, nerve root injury (L5 root most frequently), symptomatic spinal stenosis, or cauda equina syndrome (CES) in severe cases. Tenderness may be reported or elicited near the segment that has “slipped” forward (most often L4 on L5 or occasionally L5 on S1). A “step” may be present on deep palpation of the posterior elements of the segment above the spondylolisthetic joint. The trunk may be shortened and the abdomen protuberant as a result. Anterolisthesis or retrolisthesis can occur at other cervical or lumbar levels in adults and be the source of neck or low back pain. Plain x-rays with the neck or low back in flexion and extension will reveal the movement at the abnormal spinal segment. (Surgery is considered for pain symptoms that do not respond to conservative measures (e.g., rest, physical therapy), or cases with progressive neurologic deficit, abnormal gait or postural deformity, slippage > 50%, or scoliosis.

Spina bifida occulta is a failure of closure of one or several vertebral arches posteriorly; the meninges and spinal cord are normal. A dimple or small lipoma may overlie the defect. Most cases are asymptomatic and discovered incidentally during an evaluation for back pain.

Tethered cord syndrome usually presents as a progressive cauda equina disorder (see below), although myelopathy may also be the initial manifestation. The patient is often a young adult who complains of perineal or perianal pain, sometimes following minor trauma. Magnetic resonance imaging studies reveal a low-lying conus (below L1-L2) and a short, thickened fatty filum terminale.

Trauma

A patient complaining of back pain and inability to move the legs in the setting of trauma may have a spinal fracture or dislocation, and with fractures above L1, spinal cord compression. Care must be taken to avoid further damage to the spinal cord or nerve roots by immobilizing the back pending the results of x-rays.

Sprains and Strains

The terms *low back sprain*, *strain*, or *mechanically induced muscle spasm* refer to minor, self-limited injuries associated with lifting a heavy object, a fall, or a sudden deceleration such as in an automobile accident. These terms are used loosely by patients and health care providers and do not clearly describe a specific anatomic lesion or mechanism of injury. The pain is usually

confined to the lower back, and there is no radiation to the buttocks or legs. Patients with paraspinal muscle spasm often assume unusual postures.

Traumatic Vertebral Fractures

Most traumatic fractures of the lumbar vertebral bodies result from injuries producing anterior wedging or compression. With severe trauma, the patient may sustain a fracture-dislocation or a “burst” fracture involving the vertebral body and posterior elements. Traumatic vertebral fractures are caused by falls from a height (a pars interarticularis fracture of the L5 vertebra is common), sudden deceleration in an automobile accident, or direct injury. Neurologic impairment is common, and early surgical treatment is indicated. In victims of blunt trauma, CT scans of the chest, abdomen, or pelvis can be reformatted to detect associated vertebral fractures.

Lumbar Disk Disease

This is a common cause of chronic or recurrent low back and leg pain (Figs. 16-3 and 16-4). Disk disease is most likely to occur at the L4-L5 or L5-S1 levels, but upper lumbar levels are involved occasionally. The cause is often unknown; the risk is increased in overweight individuals. Disk herniation is unusual prior to age 20 and is rare in the elderly. Dessiccation of the nucleus pulposus and degeneration of the annulus fibrosus increases with age. Genetic factors may play a role in predisposing some patients to disk disease. The pain may be located in the low back only or referred to a leg, buttock, or hip. A sneeze, cough, or trivial movement may cause the nucleus pulposus to prolapse, pushing the frayed and weakened annulus posteriorly. With severe disk disease, the nucleus may protrude through the annulus (herniation) or become extruded to lie as a free fragment in the spinal canal.

The mechanism by which intervertebral disk injury causes back pain is controversial. The inner annulus fibrosus and nucleus pulposus are normally devoid of innervation. Inflammation and production of proinflammatory cytokines within the protruding or ruptured disk may trigger or perpetuate inflammation and back pain. Ingrowth of nociceptive (pain) nerve fibers into inner portions of a diseased disk may be responsible for chronic “diskogenic” pain. Nerve root injury (radiculopathy) from disk herniation may be due to compression, inflammation, or both; pathologically, demyelination or axonal loss are usually present.

A ruptured disk may be asymptomatic or cause back pain, abnormal posture, limitation of spine motion (particularly flexion), focal neurologic deficit or radicular pain. A dermatomal pattern of sensory loss, reduced or absent deep tendon reflex or focal weakness is more suggestive of a specific nerve root or nerve lesion than is the pattern of pain. Motor findings (focal weakness, muscle atrophy, or fasciculations) occur less frequently than focal sensory or reflex changes. Symptoms and signs are usually unilateral, but bilateral involvement does occur with large central disk herniation or multiple herniations that compress multiple roots or cause inflammation of nerve roots within the spinal canal. Clinical manifestations of specific nerve root lesions are summarized in Table 16-2. There is suggestive evidence that lumbar disk herniation with a nonprogressive nerve root deficit can be managed nonsurgically.

The differential diagnosis covers a variety of serious and treatable conditions, including epidural abscess, hematoma, fracture, or tumor. Fever, constant pain uninfluenced by position, sphincter abnormalities, or signs of spinal cord disease suggest an etiology other than lumbar disk disease. Bilateral absence of ankle reflexes can be a normal finding in persons over 60 years old or a sign of bilateral S1 radiculopathy. An absent deep tendon reflex or focal sensory loss may indicate injury to a nerve root or peripheral nerve. For example, an absent knee reflex may be due to a femoral neuropathy or an L4 nerve root injury. A loss of sensation over the foot and lateral lower calf may result from a peroneal or lateral sciatic neuropathy or an L5 nerve root injury. Focal muscle atrophy is nonspecific and may reflect a nerve root, peripheral nerve, or anterior horn cell disease, or disuse.

A lumbar spine MRI scan or CT-myelogram is necessary to establish the location and type of pathology. Spine MRIs yield exquisite views of intraspinal and adjacent soft tissue

anatomy. Bony lesions of the lateral recess or intervertebral foramen are optimally visualized by CT-myelography. The correlation of neuroradiologic findings with focal neurologic examination findings helps pinpoint the etiology of the pain in a significant minority of cases. The correlation of neuroradiologic findings to symptoms only, particularly pain, is not simple. Contrast-enhancing tears in the annulus fibrosus or disk protrusions are widely accepted as common sources of back pain; however, studies have found that many asymptomatic adults have similar findings. Asymptomatic disk protrusions are also common and may enhance with contrast. *Furthermore, in patients with known disk herniation treated either medically or surgically, persistence of the herniation 10 years later had no relationship to the clinical outcome.* In summary, MRI findings of disk protrusion, tears in the annulus fibrosus, or contrast enhancement are common incidental findings that, by themselves, should not dictate management decisions for patients with back pain.

The diagnosis of nerve root injury is most secure when the history, examination, results of imaging studies, and the EMG are concordant. The correlation between CT and EMG for localization of nerve root injury is between 65 and 73%. Up to one-third of asymptomatic adults have a disk protrusion detected by CT or MRI scans. Thus, surgical intervention based solely upon radiologic findings increases the likelihood of an unsuccessful outcome..

Cauda equina syndrome (CES) signifies an injury of multiple lumbosacral nerve roots within the spinal canal distal to the termination of the spinal cord at L1-2. Low back pain, weakness and areflexia in the legs, saddle anesthesia, or loss of bladder function may occur. CES must be distinguished from disorders of the lower spinal cord (conus medullaris syndrome), acute transverse myelitis (Chap. 372), and Guillain-Barré syndrome (Chap. 380). Combined involvement of the conus medullaris and cauda equina can occur. CES is commonly due to a ruptured lumbosacral intervertebral disk, lumbosacral spine fracture, hematoma within the spinal canal (e.g., following lumbar puncture in patients with coagulopathy), compressive tumor, or other mass lesion. Treatment options include surgical decompression, sometimes urgently, in an attempt to restore or preserve motor or sphincter function. (For radiotherapy of metastatic tumors see Chap. 374).

Degenerative Conditions

Lumbar spinal stenosis (LSS) describes a narrowed lumbar spinal canal and is frequently asymptomatic. *Neurogenic claudication* is back and buttock or leg pain induced by walking or standing, relieved by sitting, and occurring in the setting of severe LSS. Pain in the legs with walking is the most common symptom. Unlike vascular claudication, symptoms are often provoked by standing without walking. Patients with neurogenic claudication can walk much farther if leaning over a shopping cart and can pedal a stationary bike while sitting with ease. These flexed positions increase anteroposterior spinal canal diameter and relieve intraspinal venous hypertension, resulting in pain relief. Unlike lumbar disk disease, symptoms are relieved by sitting. Focal weakness, sensory loss, or reflex changes may occur when spinal stenosis is associated with neural foraminal narrowing that causes radiculopathy or, very rarely, with severe lumbar spinal canal narrowing. It is impressive how often severe spinal canal narrowing is asymptomatic. Severe neurologic deficits, including paralysis and urinary incontinence, also occur rarely. LSS can be acquired (75%), congenital, or due to a combination of these factors. Congenital forms (achondroplasia, idiopathic) are characterized by short, thick pedicles that produce both spinal canal and lateral recess stenosis. Acquired factors that contribute to spinal stenosis include degenerative diseases (spondylosis, spondylolisthesis, scoliosis), trauma, spine surgery with scar tissue formation, metabolic or endocrine disorders (epidural lipomatosis, osteoporosis, acromegaly, renal osteodystrophy, hypoparathyroidism), and Paget's disease. MRI provides the best definition of the abnormal anatomy (**Fig. 17-5**).

Conservative treatment of symptomatic LSS includes nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, exercise programs during lumbar flexion, and symptomatic

treatment of acute pain episodes. There is lack of evidence for epidural steroid injections as treatment in this setting. Surgical therapy is considered when medical therapy does not relieve symptoms sufficiently to allow for activities of daily living or when significant focal neurologic signs are present. Most patients with neurogenic claudication treated surgically experience significant relief of back and leg pain as early as 6 weeks post-operatively. Pain relief persists for at least two years. Patients treated non-operatively improve uncommonly. Reoperation at the same spinal level or an adjacent level occurs 7-10 years after the initial surgery in 10-23% of patients.

Neural foraminal narrowing with radiculopathy is a common degenerative disorder most often caused by the same processes that cause lumbar spinal stenosis (Fig 17-1, 17-6), including osteophytes, lateral disc protrusion, calcified disk-osteophytes, facet joint hypertrophy, uncovertebral joint hypertrophy (cervical spine), congenitally shortened pedicles, or (frequently) a combination of these processes. Neoplasms (primary or metastatic), fractures (traumatic or pathologic) infection (epidural abscess) or hematomas can also cause radiculopathy at the intervertebral foramina. The history and neurologic examination alone cannot distinguish between these possibilities. Examination findings of radiculopathy must be followed-up with spine neuroimaging (CT or MRI) to identify the underlying cause. The course of the lumbar nerve roots, arising as the ventral and dorsal rootlets from the spinal cord, pass down the lumbar spinal canal from the distal spinal cord (conus medullaris), and exit through the lateral recess and full diameter of the intervertebral foramen. These structures are best seen in axial images that should be obtained as part of routine spine neuroimaging. The same principle applies to cervical roots, except that the intraspinal course of the cervical root is very short. Focal neurologic deficits by neurologic exam or EMG findings, when present, should be used to direct the attention of the radiologist to the anatomic course of a specific nerve or root..

Facet joint hypertrophy or osteophytes can produce unilateral nerve root symptoms or signs due to bony compression at the intervertebral foramen or lateral recess; symptoms are indistinguishable from disk-related radiculopathy, but treatment may be different depending upon the specific etiology. Stretch signs, focal motor weakness, hyporeflexia, or dermatomal sensory loss may be present. Hypertrophic superior or inferior facets that narrow the intervertebral foramen can be visualized by CT or MRI. Surgical foraminotomy results in long-term relief of leg and back pain in 80–90% of these patients. The usefulness of therapeutic facet joint blocks for pain has not been rigorously studied.

Arthritis

Spondylosis, or osteoarthritic spine disease, typically occurs in later life and primarily involves the cervical and lumbosacral spine. Patients often complain of back pain that is increased with movement and associated with stiffness. The relationship between clinical symptoms and radiologic findings is usually not straightforward. Pain may be prominent when x-ray, CT, or MRI findings are minimal, and prominent degenerative spine disease can be seen in asymptomatic patients. Osteophytes or combined disk-osteophytes may cause or contribute to central spinal canal stenosis, lateral recess stenosis, or neural foraminal narrowing.

Ankylosing Spondylitis

(See also Chap. 318) This distinctive arthritic spine disease typically presents with the insidious onset of low back and buttock pain with a peak incidence in the 20s and 30s in men and women. Associated features include morning back stiffness, nocturnal pain, pain unrelieved by rest, an elevated ESR, and the histocompatibility antigen HLA-B27. Onset at a young age and back pain improving with exercise are characteristic. Loss of the normal lumbar lordosis and exaggeration of thoracic kyphosis develop as the disease progresses. Inflammation followed by ossification and bony growth that bridges adjacent vertebral bodies and reduces spine mobility in all planes. MRI has been used to assess the presence of inflammation in joints as well as response to

treatment and is more sensitive than plain x-rays. In later stages, plain x-rays reveal bridging of vertebral bodies to produce the fused “bamboo spine.”

Stress fractures after minimal or no trauma can occur through the spontaneously ankylosed posterior bony elements of the rigid, osteoporotic spine resulting in focal pain, spinal instability, spinal cord compression, or CES. Atlantoaxial subluxation with spinal cord compression can occur in up to 20% of patients over time. Ankylosis of the ribs to the spine and a decrease in the height of the thoracic spine may compromise respiratory function. Therapy with anti-tumor necrosis factor agents is effective in reducing disease activity and improving patient function. Similar to ankylosing spondylitis, restricted movements may accompany Reiter’s syndrome, psoriatic arthritis, and chronic inflammatory bowel disease.

Neoplasms

(See also Chap. 374) Back pain is the most common neurologic symptom in patients with systemic cancer and is the presenting symptom in 20%. Delays in identification of spine metastases are common, including early signs of spinal cord compression. Fewer than 5% of patients who are nonambulatory at the time of diagnosis ever regain the ability to walk, thus early diagnosis is crucial. The cause is usually vertebral body metastasis (Figure 1), and posterior extension of the tumor into the spinal canal resulting in spinal cord or nerve root compression. Other spine related sources of tumor include the posterior bony spine elements and the subarachnoid space (carcinomatous meningitis). Metastatic carcinoma (breast, lung, prostate, thyroid, renal, gastrointestinal tract), multiple myeloma, and non-Hodgkin’s and Hodgkin’s lymphomas frequently involve the spine. Cancer-related back pain tends to be constant, dull, unrelieved by rest, and worse at night. By contrast, mechanical low back pain usually improves with rest. MRI, CT, or CT-myelography are the studies of choice when spinal metastasis is suspected. Once a metastasis is found, full imaging of the entire spine will reveal additional tumor deposits in 1/3 of patients; these may be included in plans for treatment. MRI is preferred for soft tissue definition, but the most rapidly available procedure is best because the patient’s condition may worsen quickly without intervention.

Infections/Inflammation

Vertebral osteomyelitis is often caused by staphylococci, but other bacteria or tuberculosis (Pott’s disease) may be responsible. The primary source of infection is usually hematogenous spread of chronic infection of the urinary tract, skin, or lungs. Intravenous drug use is a well-recognized risk factor. Whenever pyogenic osteomyelitis is found, the possibility of bacterial endocarditis should be considered. Back pain unrelieved by rest, spine tenderness over the involved spine segment, and an elevated ESR are the most common findings in vertebral osteomyelitis. Fever or an elevated white blood cell count is found in a minority of patients. MRI and CT are sensitive and specific for early detection of osteomyelitis; CT may be more readily available in emergency settings and better tolerated by some patients with severe back pain. The intervertebral disk is commonly affected by infection (discitis), and very rarely by tumor.

Spinal epidural abscess (Chap. 372) presents with back pain (aggravated by movement or on palpation), fever, radicular pain, or new signs of nerve root injury or spinal cord compression.) The subacute development of two or more of these findings should increase the index of suspicion for spinal epidural abscess. The abscess may track over multiple spinal levels and is best delineated by spine MRI.

Lumbar adhesive arachnoiditis with radiculopathy is due to fibrosis and inflammation within the subarachnoid space. The fibrosis results in nerve root adhesions, and presents as back and leg pain associated with motor, sensory, or reflex changes. Causes of arachnoiditis include multiple lumbar operations, chronic spinal infections (especially TB in the developing world), spinal cord injury, intrathecal hemorrhage, myelography (rare), intrathecal injection of glucocorticoids, anesthetics, other medications, and foreign bodies. The MRI in the axial plane shows clumped nerve roots or loculations of cerebrospinal fluid within the thecal sac. Clumped

nerve roots may also occur with demyelinating polyneuropathy or neoplastic infiltration. Symptomatic treatment is usually unsatisfactory. Microsurgical lysis of adhesions, dorsal rhizotomy, dorsal root ganglionectomy, and epidural steroids have been tried, but outcomes have been poor. Dorsal column stimulation for pain relief has produced varying results.

Metabolic Causes

Osteoporosis and Osteosclerosis

Immobilization or underlying conditions such as osteomalacia, renal disease, multiple myeloma, hyperparathyroidism, hyperthyroidism, metastatic carcinoma, post-menopausal state, or glucocorticoid use may accelerate osteoporosis and weaken the vertebral body, leading to compression fractures and pain. Up to 2/3 of compression fractures seen on radiologic imaging are asymptomatic. The most common nontraumatic vertebral body fractures are due to postmenopausal or senile osteoporosis (Chap. 348). The risk of an additional vertebral fracture at one year following a first vertebral fracture is 20%. The presence of fever, weight loss, fracture at a level above T4, or other conditions described above should increase the suspicion for a cause other than senile osteoporosis. If tumor is suspected, a bone biopsy or diagnostic search for a primary tumor is indicated. The sole manifestation of a compression fracture may be localized back pain or radicular pain exacerbated by movement and often reproduced by palpation over the spinous process of the affected vertebra. The clinical context, neurologic signs, and radiologic appearance of the spine establish the diagnosis.

Acute pain relief with acetaminophen or a combination of opioids and acetaminophen is often helpful. The role of NSAIDs is controversial. Both pain and disability are better with bracing in the short term. Antiresorptive drugs especially bisphosphonates (e.g., alendronate), have been shown to reduce the risk of osteoporotic fractures and are the preferred treatment. Fewer than one-third of patients with prior compression fractures are adequately treated for osteoporosis despite the increased risk for future fractures; rates of primary prevention among individuals at risk, but without a history of fracture, are even less. Interventions [percutaneous vertebroplasty (PVP), kyphoplasty] exist for acute-subacute osteoporotic compression fractures associated with debilitating pain. Controlled studies suggest a benefit for pain reduction acutely, but not at 2 months when compared with conservative care. Other studies give conflicting results. Relief of pain following PVP has also been reported in patients with vertebral metastases, myeloma, or hemangiomas. **For a complete discussion of diagnosis and management of osteoporosis and other bone disorders, see Chaps. 347-349.**

The osteosclerosis of Paget's disease results in an abnormally increased bone density readily identifiable on routine x-ray studies, often suggested as an isolated increase in alkaline phosphatase in healthy older persons, and may or may not produce back pain. Spinal cord or nerve root compression may result from bony encroachment. It should not be assumed that Paget's disease is the cause of a patient's back pain until other etiologies have been carefully considered.

Referred Pain from Visceral Disease

Diseases of the thorax, abdomen, or pelvis may refer pain to the posterior portion of the spinal segment that innervates the diseased organ. Occasionally, back pain may be the first and only manifestation. Upper abdominal diseases generally refer pain to the lower thoracic or upper lumbar region (eighth thoracic to the first and second lumbar vertebrae), lower abdominal diseases to the mid-lumbar region (second to fourth lumbar vertebrae), and pelvic diseases to the sacral region. Local signs (pain with spine palpation, paraspinal muscle spasm) are absent, and little or no pain accompanies routine movements of the spine.

Low Thoracic or Lumbar Pain with Abdominal Disease Tumors of the posterior wall of the stomach or duodenum typically produce epigastric pain (Chaps. 87 and 287), but midline back or paraspinal pain may occur if retroperitoneal extension is present. Fatty foods occasionally induce back pain associated with biliary disease. Diseases of the pancreas can produce right paraspinal back pain (head of the pancreas involved) or left paraspinal pain (body or tail involved). Pathology in retroperitoneal structures (hemorrhage, tumors, pyelonephritis) can produce paraspinal pain that radiates to the lower abdomen, groin, or anterior thighs. A mass in the iliopsoas region can produce unilateral lumbar pain with radiation toward the groin, labia, or testicles. The sudden appearance of lumbar pain in a patient receiving anticoagulants should trigger consideration of retroperitoneal hemorrhage. Combined back and abdominal pain are common. Pain arising from the descending colon, ascending colon, kidney, ureter, ovary, or gallbladder result in abdominal pain on the same side as the pathology.

Isolated low back pain occurs in some patients with a contained rupture of an abdominal aortic aneurysm (AAA). The classic clinical triad of abdominal pain, shock, and back pain occurs in < 20% of patients. The typical patient at risk is an elderly male smoker with back pain. Frequently, the diagnosis is initially missed because the symptoms and signs can be nonspecific. Misdiagnoses include nonspecific back pain, diverticulitis, renal colic, sepsis, and myocardial infarction. A careful abdominal examination revealing a pulsatile mass (present in 30–75% of patients) is an important physical finding. Patients with suspected AAA should be evaluated with abdominal ultrasound, CT, or MRI (Chap. 242).

Sacral Pain with Gynecologic and Urologic Disease

Pelvic organs rarely cause low back pain, except for gynecologic disorders involving the uterosacral ligaments. The pain is referred to the sacral region. Endometriosis or uterine cancers may invade the uterosacral ligaments. Pain associated with endometriosis is typically premenstrual and often continues until it merges with menstrual pain. Uterine malposition may cause uterosacral ligament traction (retroversion, descensus, and prolapse) or produce sacral pain after prolonged standing.

Menstrual pain may be felt in the sacral region. Poorly localized, cramping pain can radiate down the legs. Pain due to neoplastic infiltration of nerves is typically continuous, progressive in severity, and unrelieved by rest at night. Less commonly, radiation therapy of pelvic tumors may produce sacral pain from late radiation necrosis of tissue. Low back pain that radiates into one or both thighs is common in the last weeks of pregnancy.

Urologic sources of lumbosacral back pain include chronic prostatitis, prostate cancer with spinal metastasis (Chap. 91), and diseases of the kidney or ureter. Lesions of the bladder and testes do not often produce back pain. Infectious, inflammatory, or neoplastic renal diseases may produce ipsilateral lumbosacral pain, as can renal artery or vein thrombosis. Paraspinal lumbar pain may be a symptom of ureteral obstruction due to nephrolithiasis.

Other Causes Of Back Pain

Postural Back Pain

There is a group of patients with nonspecific chronic low back pain (CLBP) in whom no specific anatomic lesion can be found despite exhaustive investigation. These individuals complain of vague, diffuse back pain with prolonged sitting or standing that is relieved by rest. Exercises to strengthen the paraspinal and abdominal muscles are sometimes helpful.

Psychiatric Disease

CLBP may be encountered in patients who seek financial compensation; in malingerers; or in those with concurrent substance abuse. Many patients with CLBP have a history of psychiatric illness (depression, anxiety states), or childhood trauma (physical or sexual abuse) that antedates the onset of back pain. Over half of patients with back pain and 9 or more common somatic symptoms have a mood disorder. Preoperative psychological assessment has been used to

exclude patients with marked psychological impairments that predict a poor surgical outcome from spine surgery.

Idiopathic

The cause of low back pain occasionally remains unclear. Some patients have had multiple operations for disk disease but have persistent pain and disability. The original indications for surgery may have been questionable, with back pain only, no definite neurologic signs, or a minor disk bulge noted on CT or MRI. Scoring systems based upon neurologic signs, psychological factors, physiologic studies, and imaging studies have been devised to minimize the likelihood of unsuccessful surgery.

BACK PAIN

ACUTE LOW BACK PAIN (ALBP) WITHOUT RADICULOPATHY

ALBP is defined as pain of <3 months' duration. Full recovery can be expected in 85% of adults with ALBP without leg pain. Most have purely "mechanical" symptoms (e.g.- pain that is aggravated by motion and relieved by rest).

The initial assessment excludes serious causes of spine pathology that require urgent intervention, including infection, cancer, or trauma. Risk factors for a serious cause of ALBP are shown in **Table 17-1**. Laboratory and imaging studies are unnecessary if risk factors are absent. Plain spine films or CT are rarely indicated in the first month of symptoms unless a spine fracture is suspected. The prognosis of acute low back pain is generally excellent. Many patients do not seek medical care and apparently improve on their own. Even among those seen in primary care, two-thirds report being substantially improved after seven weeks. This spontaneous improvement can mislead clinicians and researchers about the efficacy of treatment interventions. Perhaps as a result, many ineffective treatments have become widespread in the past, such as bed rest, lumbar traction, sacroiliac fusion, and coccygectomy.

Clinicians should reassure patients that improvement is very likely, and instruct them in self-care. Education is an important part of treatment. Satisfaction and the likelihood of follow-up increase when patients are educated about prognosis, treatment methods, activity modifications, and strategies to prevent future exacerbations. In one study, patients who felt they did not receive an adequate explanation for their symptoms wanted further diagnostic tests. In general, bed rest should be avoided, or kept to a day or two at most, for relief of severe symptoms. Several randomized trials suggest that bed rest does not accelerate the pace of recovery. In general, the best activity recommendation is for walking and early resumption of normal physical activity, avoiding only strenuous manual labor. Possible advantages of early ambulation for acute back pain include maintenance of cardiovascular conditioning, improved disk and cartilage nutrition, improved bone and muscle strength, and increased endorphin levels. Specific back exercises or early vigorous exercise have not shown benefits for acute back pain, but may be useful for chronic pain. Application of heat by heating pads or heated blankets is sometimes helpful.

Recent evidence-based guidelines suggest that over-the-counter medicines such as acetaminophen and NSAIDs are generally first-line medication options. Skeletal muscle relaxants, such as cyclobenzaprine or methocarbamol, may be useful, but sedation is a common side effect. For some patients, these medications may be prescribed just at night, to minimize sleep disruption by pain. Some drugs in this category, including benzodiazepines and carisoprodol, carry a risk of abuse. If they are used, short time-limited courses are generally recommended.

It is unclear whether opioid analgesics and tramadol are more effective than NSAID's or acetaminophen for treating low back pain, though most data are for chronic back pain. Their use is best reserved for patients who cannot tolerate acetaminophen or NSAIDs, or those with the most severe pain. As with muscle relaxants, these drugs are often sedating, so it may be useful to prescribe them at night, with acetaminophen and NSAIDs for daytime use. In addition to

common short-term side effects such as nausea, constipation, and itching, there is growing concern for side effects with long-term use of opioids, such as hypersensitivity to pain and hypogonadism. Problems with dependency, addiction, diversion, and even mortality related to prescription opioids are rapidly increasing.

There is no evidence to support the use of oral or injected glucocorticoids for acute low back pain without radiculopathy. Anti-epileptic drugs, such as gabapentin, are not FDA approved for treating low back pain, and there is insufficient evidence to support their use in this setting.

Other nonpharmacologic treatments for acute low back pain include spinal manipulation, physical therapy, massage, acupuncture, transcutaneous electrical nerve stimulation, ultrasound, diathermy, or magnets. Spinal manipulation appears to be roughly equivalent to effective conventional medical treatments and may be a useful alternative for patients who wish to avoid or who cannot tolerate drug therapy. There is little to support the use of physical therapy, massage, or acupuncture in the setting of acute low back pain. Similarly, there is insufficient evidence to support the use of laser therapy, therapeutic ultrasound, magnets, corsets, or lumbar traction. Though important for chronic pain, back exercises for acute back pain are generally not supported by clinical evidence. Evidence regarding the efficacy of using ice acutely is lacking, although many patients report temporary symptomatic relief. Heat may provide a short-term reduction in pain after the first week or so of pain. These latter 2 interventions are optional given the lack of negative evidence, low cost, and low risk.

CHRONIC LOW BACK PAIN WITHOUT RADIULCOPATHY

Chronic low back pain is, defined as pain lasting >12 weeks; it accounts for 50% of total back pain costs. Risk factors include obesity, female gender, older age, prior history of back pain, restricted spinal mobility, pain radiating into a leg, high levels of psychological distress, poor self-rated health, minimal physical activity, smoking, job dissatisfaction, and widespread pain. In general, the same treatments that are recommended for acute low back pain can be useful for patients with chronic low back pain. In this setting, however, the value of opioid therapy or muscle relaxants is less clear.

There is evidence to support the use of exercise therapy, and this may be one of the mainstays of treatment for chronic back pain. Effective regimens have generally included a combination of gradually increasing aerobic exercise, strengthening exercises, and stretching exercises. Motivating patients is sometimes challenging, and supervised exercise is best. This may be a situation in which a supportive physical therapist can be most helpful. In general, activity tolerance is the primary goal, while pain relief is secondary. Exercise programs can reverse atrophy in paraspinal muscles and strengthen extensors of the trunk. Supervised intensive physical exercise or “work hardening” regimens have been effective in returning some patients to work, improving walking distance, and reducing pain. In addition, some forms of yoga have been evaluated in randomized trials, and may be helpful for patients who are interested.

Medications for chronic low back pain may include acetaminophen, NSAIDs, and tricyclic antidepressants. Trials of the latter suggest some benefit even for patients without evidence of depression. Trials do not support the efficacy of selective serotonin reuptake inhibitors in this condition. However, depression is common among patients with chronic pain, and should be appropriately treated.

Cognitive-behavioral therapy is based on evidence that psychological and social factors, as well as somatic pathology, are important in the genesis of chronic pain and disability. Patient attitudes and beliefs, psychological distress, and patterns of illness behavior may all influence responses to chronic pain. Thus, in addition to addressing possible pathophysiological mechanisms, psychological treatments are aimed at reducing disability by modifying cognitive processes and environmental contingencies. Cognitive-behavioral therapy is one of the most widely used of these approaches. This includes efforts to identify and modify patients’ thinking about their pain and disability by strategies that may involve imagery, attention diversion, or

modifying maladaptive thoughts, feelings, and beliefs. This approach includes educating patients about a multidimensional view of pain, identifying pain-eliciting or pain-aggravating thoughts and feelings, using coping strategies and relaxation techniques, and even hypnosis. A systematic review concluded that such treatments are more effective than a waiting list control group for short-term pain relief, but that long-term results remain unclear. Behavioral treatments may have effects similar in magnitude to exercise therapy.

Back pain is the most common reason for seeking complementary and alternative treatments. The most common of these for back pain are spinal manipulation, acupuncture, and massage. The role of complementary and alternative medicine approaches, aside from spinal manipulation, remains unclear. Biofeedback has not been studied rigorously. As with acute back pain, spinal manipulation may on average offer benefits similar to conventional care. Rigorous recent trials of acupuncture suggest that true acupuncture is not superior to sham acupuncture, but that both may offer an advantage over routine care. Whether this is due entirely to placebo effects or to stimulation provided even by sham acupuncture is uncertain. Some trials of massage therapy have been encouraging, but this has been less well studied than manipulation or acupuncture.

Studies of transcutaneous electrical nerve stimulation (TENS) have reached conflicting conclusions, but a recent evidence-based guideline suggested that there was no convincing evidence for its efficacy in treating chronic back pain.

Various injections, including epidural corticosteroid injections, facet joint injections, and trigger point injections have been used for treating chronic low back pain. However, in the absence of radiculopathy, there is no evidence that epidural steroids are effective for treating chronic back pain. Several randomized trials suggest that facet joint injections are not more effective than saline injections, and recent evidence-based guidelines recommend against their use. Similarly, there is little evidence to support the use of trigger point injections. Injection studies are sometimes used diagnostically to help determine the anatomic source of back pain. Reproduction of the patient's typical pain with discography has been used as evidence that a specific disk is the pain generator. Pain relief following a foraminal nerve root block or glucocorticoid injection into a facet has been similarly used as evidence that the facet joint or nerve root is the source. However, the possibility that the injection response was a placebo effect or due to systemic absorption of the glucocorticoids is often not considered.

Another category of interventions for chronic back pain includes electro thermal and radiofrequency therapies. Intradiscal therapy has been proposed using both types of energy to thermocoagulate and destroy nerves in the intervertebral disc, using specially designed catheters or electrodes. A systematic review has suggested that current evidence does not support the use of these intradiscal therapies.

Radiofrequency denervation is sometimes used to destroy nerves that are thought to mediate pain, and this technique has been used for facet joint pain (with the target nerve being the medial branch of the primary dorsal ramus), for back pain thought to arise from the intervertebral disc (ramus communicans) and radicular back pain (dorsal root ganglia). A small number of small trials has resulted in conflicting results for facet joint pain. The evidence for presumed discogenic pain and for radicular pain is similarly meager. A trial for patients with chronic radicular pain found no difference between radiofrequency denervation of the dorsal root ganglia and sham treatment. Recent systematic reviews have thus concluded that there is insufficient evidence to reliably evaluate these interventional therapies.

Surgical intervention for chronic low back pain in the absence of radiculopathy has been evaluated in a small number of randomized trials, all conducted in Europe. Each of these studies included patients with back pain and a degenerative disc, but no sciatica. Three of the four trials concluded that lumbar fusion surgery was no more effective than highly structured, rigorous rehabilitation combined with cognitive behavioral therapy. The fourth trial found an advantage

of fusion surgery over haphazard “usual care”, which appeared to be less effective than the structured rehabilitation in other trials. Given conflicting evidence, indications for surgery for chronic back pain alone have remained controversial. Both US and British guidelines suggest considering referral for an opinion on spinal fusion for people who have completed an optimal nonsurgical treatment program (including combined physical and psychological treatment) and who have persistent severe back pain for which they would consider surgery.

The newest surgical treatment for degenerated discs with back pain is disc replacement with prosthetic disks. These are generally designed as metal plates with a polyethylene cushion sandwiched in between. The trials that led to approval of these devices compared them to spine fusion, and concluded that the artificial discs were “not inferior”. Serious complications appeared to be somewhat more likely with the artificial disc. This treatment remains controversial for low back pain.

Intensive multidisciplinary rehabilitation programs may involve daily or frequent care and many hours, involving physical therapy, exercise, cognitive behavioral therapy, a workplace evaluation, and other interventions. For patients who have not responded to other interventions, such programs appear to offer some benefit. Systematic reviews suggest that the evidence is fair and effects are moderate.

Some observers have raised concern that chronic back pain may often be overtreated. The use of opioids, epidural steroid injections, facet joint injections, and surgical intervention has increased rapidly in the past decade, without corresponding population-level improvements in pain or functioning among patients with back pain. In each case, randomized trials provide minimal support for these treatments in the setting of chronic back pain without radiculopathy. For low back pain without radiculopathy, new British guidelines explicitly recommend against use of selective serotonin reuptake inhibitors (SSRIs), any type of injection, TENS, lumbar supports, traction, radiofrequency facet joint denervation, intradiscal electrothermal therapy, or intradiscal radiofrequency thermocoagulation. Similarly, these treatments are not recommended in guidelines from the American College of Physicians and the American Pain Society. On the other hand, exercise therapy and treatment of depression appear to be underused.

LOW BACK PAIN WITH RADICULOPATHY

A common cause of back pain with radiculopathy is a herniated disc with nerve root impingement, resulting in back pain with radiation down the leg. The prognosis for acute low back pain with radiculopathy due to disk herniation (sciatica) is generally favorable, with most patients demonstrating substantial improvement over a matter of months. Serial imaging studies suggest spontaneous regression of the herniated portion of the disc in 2/3 of patients over 6 months. Nonetheless, there are several important treatment options for providing symptom relief while this natural healing process unfolds.

As for back pain without radiculopathy, resumption of normal activity as much as possible is usually the best activity recommendation. Randomized trial evidence suggests that bed rest is ineffective for treating sciatica as well as for back pain alone. Acetaminophen and NSAIDs are appropriate for pain relief, although severe pain may require short courses of opioid analgesics.

Epidural corticosteroid injections have a role in providing temporary symptom relief for sciatica due to a herniated disc. Although randomized trial evidence is conflicting, there appears to be some overall short-term benefit for pain relief of sciatica. However, there does not appear to be an advantage in reducing subsequent surgical interventions. Diagnostic nerve root blocks have been advocated to determine if pain originates from a specific nerve root. However, improvement may result even when the nerve root is not responsible for the pain; this may occur as a placebo effect, from a pain-generating lesion located distally along the peripheral nerve, or from anesthesia of the sinuvertebral nerve. The utility of diagnostic nerve root blocks remains a subject of debate.

Surgical intervention is indicated for patients who have progressive motor weakness, demonstrated on clinical examination or EMG, as a result of nerve root injury. Urgent surgery is recommended for patients who have evidence of the cauda equina syndrome or spinal cord compression, generally suggested by bowel or bladder dysfunction, diminished sensation in a saddle distribution, a sensory level, bilateral leg weakness, or bilateral leg spasticity.

Surgery is also an important option for patients who have disabling radicular pain despite optimal conservative treatment. Sciatica is perhaps the most common reason for recommending spine surgery. Because patients with a herniated disc and sciatica generally experience rapid improvement over a matter of weeks, most experts do not recommend considering surgery unless the patient has failed to respond to 6 to 8 weeks of appropriate nonsurgical management. For the patients who have not improved, randomized trials indicate that, compared to nonsurgical treatment, surgery results in more rapid pain relief. However, after the first year or two of follow-up, patients with sciatica appear to have much the same level of pain relief and functional improvement with or without surgery. Thus, both treatment approaches are reasonable, and patient preferences should play a major role in decision-making. Some patients will want the fastest possible relief and find surgical risks highly acceptable. Others will be more risk-averse and more tolerant of symptoms, and will choose watchful waiting if they understand that improvement is likely in the end.

The usual surgical procedure is a partial hemilaminectomy with excision of the prolapsed disk. Fusion of the involved lumbar segments should be considered only if significant spinal instability is present (i.e., degenerative spondylolisthesis). The costs associated with lumbar interbody fusion increased dramatically between 1999-2003. There are no large prospective, randomized trials comparing fusion to other types of surgical intervention. In one study, patients with persistent low back pain despite an initial discectomy fared no better with spine fusion than with a conservative regimen of cognitive intervention and exercise. Artificial disks have been in use in Europe for the past decade; their utility remains controversial in the United States.

PAIN IN THE NECK AND SHOULDER

(Table 17-4)

Neck pain, which usually arises from diseases of the cervical spine and soft tissues of the neck, is common. Neck pain arising from the cervical spine is typically precipitated by movement and may be accompanied by focal tenderness and limitation of motion. Pain arising from the brachial plexus, shoulder, or peripheral nerves can be confused with cervical spine disease, but the history and examination usually identify a more distal origin for the pain. Cervical spine trauma, disk disease, or spondylosis with intervertebral foraminal narrowing may be asymptomatic or painful and can produce a myelopathy, radiculopathy, or both. There are seven cervical vertebrae and eight cervical nerve roots. The first seven cervical nerve roots exit above their corresponding vertebra. The eighth cervical nerve root exits between the C7 and T1 vertebrae. From the T1 vertebra down, the nerve roots exit below their corresponding vertebra. The same risk factors for a serious cause of low back pain are thought to apply to neck pain with the addition that neurologic signs of myelopathy (incontinence, sensory level, spastic legs) may also occur.

Lhermitte's sign, an electrical shock down the spine with neck flexion, suggests cervical spinal cord involvement from any cause.

Trauma to the Cervical Spine

Trauma to the cervical spine (fractures, subluxation) places the spinal cord at risk for compression. Motor vehicle accidents, violent crimes, or falls account for 87% of cervical spinal cord injuries (Chap. 372). Immediate immobilization of the neck is essential to minimize further spinal cord injury from movement of unstable cervical spine segments. The decision to obtain imaging should be driven by the clinical characteristics of the injury. The NEXUS low risk criteria established that patients with normal alertness, and absence of midline palpation tenderness, intoxication, neurologic deficits, and painful distracting injuries predicted a very low likelihood of a clinically significant cervical spine traumatic injury. The Canadian C-spine rule states that imaging should be obtained in the setting of trauma if the patient is > 65 years old, has limb paresthesias, or a dangerous mechanism for the injury (e.g.-bicycle collision with tree or parked car), fall from height > 3 feet or 5 stairs, diving accident). A CT scan is the diagnostic procedure of choice for detection of acute fractures. When traumatic injury to the vertebral arteries or cervical spinal cord is suspected, visualization by MR imaging is preferred.

Whiplash injury is due to rapid flexion and extension of the neck, usually in automobile accidents, and causes cervical musculoligamentary injury. This diagnosis should not be applied to patients with fractures, disk herniation, head injury, focal neurologic findings, or altered consciousness. Up to 50% of persons reporting whiplash injury acutely have persistent neck pain one year later. Once personal compensation for pain and suffering was removed from the Australian health care system, the prognosis for recovery at one year from whiplash injury improved also. Imaging of the cervical spine is not cost-effective acutely but is useful to detect disk herniations when symptoms persist for >6 weeks following the injury. Severe initial symptoms have been associated with a poor long-term outcome.

Cervical Disk Disease

Herniation of a lower cervical disk is a common cause of neck, shoulder, arm, or hand pain or tingling. Neck pain, stiffness, and a range of motion limited by pain are the usual manifestations. A herniated cervical disk is responsible for ~25% of cervical radiculopathies. Extension and lateral rotation of the neck narrows the ipsilateral intervertebral foramen and may reproduce radicular symptoms (Spurling's sign). In young persons, acute nerve root compression from a ruptured cervical disk is often due to trauma. Cervical disk herniations are usually posterolateral near the lateral recess. Typical patterns of reflex, sensory, and motor changes that accompany specific cervical nerve root lesions are summarized in Table 17-4. The cervical nerve roots most commonly affected are C7 and C6. While the classic patterns are clinically helpful, there are many exceptions for several reasons: (1) overlap in function between adjacent nerve roots is common, (2) symptoms and signs may be evident in only part of the injured nerve root territory, and (3) the location of pain is the most variable of the clinical features.

Cervical Spondylosis

Osteoarthritis of the cervical spine may produce neck pain that radiates into the back of the head, shoulders, or arms, or may be the source of headaches in the posterior occipital region (supplied by the C2-C4 nerve roots). Osteophytes, disk protrusions, or hypertrophic facet or uncovertebral joints may alone or in combination compress one or several nerve roots at the intervertebral foramina (**Fig. 17-6**); this compression accounts for 75% of cervical radiculopathies. The roots most commonly affected are C7 and C6. Narrowing of the spinal canal by osteophytes, ossification of the posterior longitudinal ligament (OPLL), or a large central disk may compress the cervical spinal cord. Combinations of radiculopathy and myelopathy may be present. When little or no neck pain accompanies cord compression, the diagnosis may be confused with amyotrophic lateral sclerosis (Chap. 369), multiple sclerosis (Chap. 375), spinal cord tumors, or syringomyelia (Chap. 372). The possibility of cervical spondylosis should be considered even

when the patient presents with symptoms or signs in the legs only. MRI is the study of choice to define the anatomic abnormalities, but plain CT is adequate to assess bony spurs, foraminal narrowing, lateral recess stenosis, or OPLL. EMG and nerve conduction studies can localize and assess the severity of the nerve root injury.

Other Causes of Neck Pain

Rheumatoid arthritis (RA) (Chap. 314) of the cervical apophyseal joints produces neck pain, stiffness, and limitation of motion. In advanced RA, synovitis of the atlantoaxial joint (C1-C2; Fig. 17-2) may damage the transverse ligament of the atlas, producing ligamentous laxity and forward displacement of the atlas on the axis (atlantoaxial subluxation). Radiologic evidence of atlantoaxial subluxation occurs in 30% of patients with RA. Not surprisingly, the degree of subluxation correlates with the severity of erosive disease. When subluxation is present, careful neurologic assessment is important to identify early signs of myelopathy. Occasional patients develop high spinal cord compression leading to quadriplegia, respiratory insufficiency, and death. Surgery should be considered when myelopathy or spinal instability is present. MRI is the preferred imaging modality as it shows the degree of effacement of the subarachnoid space by pannus, the internal appearance of the spinal cord, and changes in the cervical spine and joints.

Ankylosing spondylitis can cause neck pain and less commonly atlantoaxial subluxation; surgery may be required to prevent spinal cord compression. Acute *herpes zoster* presents as acute posterior occipital or neck pain prior to the outbreak of vesicles. *Neoplasms* metastatic to the cervical spine, *infections* (osteomyelitis and epidural abscess), and *metabolic bone diseases* may be the cause of neck pain. Neck pain may also be referred from the heart with coronary artery ischemia (cervical angina syndrome).

Thoracic Outlet

The thoracic outlet contains the first rib, the subclavian artery and vein, the brachial plexus, the clavicle, and the lung apex. Injury to these structures may result in postural or movement-induced pain around the shoulder and supraclavicular region. *Disputed TOS* includes a large number of patients with chronic arm and shoulder pain of unclear cause. The lack of sensitive and specific findings on physical examination or laboratory markers for this condition frequently results in diagnostic uncertainty. The role of surgery in disputed TOS is controversial. Multidisciplinary pain management is a conservative approach, although treatment is often unsuccessful. *True neurogenic thoracic outlet syndrome* (TOS) is an uncommon syndrome resulting from compression of the lower trunk of the brachial plexus or ventral rami of the C8 or T1 nerve roots most often by an anomalous band of tissue connecting an elongate transverse process at C7 with the first rib. Pain is mild or absent. Signs include weakness of intrinsic muscles of the hand and diminished sensation on the palmar aspect of the fourth and fifth digits. An anteroposterior cervical spine x-ray will show the elongate C7 transverse process, and EMG and nerve conduction studies confirm the diagnosis. Treatment consists of surgical resection of the anomalous band. The weakness and wasting of intrinsic hand muscles typically does not improve, but surgery halts the insidious progression of weakness. *Arterial TOS* results from compression of the subclavian artery by a cervical rib resulting in poststenotic dilatation of the artery and thrombus formation. Blood pressure is reduced in the affected limb, and signs of emboli may be present in the hand. Neurologic signs are absent. Ultrasound can confirm the diagnosis noninvasively. Treatment is with thrombolysis or anticoagulation (with or without embolectomy) and surgical excision of the cervical rib compressing the subclavian artery. Venous TOS is due to subclavian thrombosis resulting in swelling of the arm and pain. The vein may be compressed by a cervical rib or anomalous scalene muscle. Venography is the diagnostic test of choice.

Brachial Plexus and Nerves

Pain from injury to the brachial plexus or peripheral nerves of the arm can occasionally mimic pain of cervical spine origin. Neoplastic infiltration of the lower trunk of the brachial plexus may

produce shoulder or supraclavicular pain radiating down the arm, numbness of the fourth and fifth fingers or medial forearm, and weakness of intrinsic hand muscles innervated by the ulnar and median nerves. Delayed radiation injury may produce similar findings, although pain is less often present and almost always less severe. A Pancoast tumor of the lung (Chap. 85) is another cause and should be considered, especially when a Horner's syndrome is present. *Suprascapular neuropathy* may produce severe shoulder pain, weakness, and wasting of the supraspinatus and infraspinatus muscles. *Acute brachial neuritis* is often confused with radiculopathy; the acute onset of severe shoulder or scapular pain is followed typically over days by weakness of the proximal arm and shoulder girdle muscles innervated by the upper brachial plexus. The onset is often preceded by an infection. The long thoracic nerve may be affected; the latter results in a winged scapula. Brachial neuritis may also present as an isolated paralysis of the diaphragm or other single nerve of the arm. Recovery is generally good and may occur slowly over up to three years.

Occasional cases of carpal tunnel syndrome produce pain and paresthesias extending into the forearm, arm, and shoulder resembling a C5 or C6 root lesion. Lesions of the radial or ulnar nerve can mimic a radiculopathy at C7 or C8, respectively. EMG and nerve conduction studies can accurately localize lesions to the nerve roots, brachial plexus, or peripheral nerves.

For further discussion of peripheral nerve disorders, see Chap. 379.

Shoulder

Pain arising from the shoulder can on occasion mimic pain from the spine. If symptoms and signs of radiculopathy are absent, then the differential diagnosis includes mechanical shoulder pain (tendonitis, bursitis, rotator cuff tear, dislocation, adhesive capsulitis, and cuff impingement under the acromion) and referred pain (subdiaphragmatic irritation, angina, Pancoast tumor). Mechanical pain is often worse at night, associated with local shoulder tenderness and aggravated by abduction, internal rotation, or extension of the arm. Pain from shoulder disease may radiate into the arm or hand, but sensory, motor, and reflex changes are absent.

NECK PAIN WITHOUT RADICULOPATHY

In general, the evidence regarding treatments for neck pain is less complete than that for low back pain. As with low back pain, spontaneous improvement is the norm for acute neck pain, and the usual goal of therapy is to provide symptom relief while natural healing processes proceed

A 2007 systematic review of nonsurgical treatments for whiplash-associated disorders concluded that the evidence was generally of poor methodologic quality, with varied comparison treatments and inconsistent results. The authors concluded that the evidence neither supports nor refutes the effectiveness of common treatments used for symptom relief. A subsequent systematic review suggested that gentle mobilization of the cervical spine combined with exercise programs is more beneficial than usual care. Studies of cervical collars, TENS, ultrasound, or diathermy showed no advantage over exercise and mobilization.

Similarly, for patients with axial neck pain unassociated with trauma, supervised exercise, with or without mobilization, appears to be effective. Exercises often include shoulder rolls and neck stretches. There is relatively little evidence specifically for neck pain with regard to medications such as muscle relaxants, analgesics, and NSAIDs. However, many clinicians use these medications in much the same way they are used for low back pain. Again, there is little evidence to support the use of cervical traction, neck collars, TENS, or massage. Systematic reviews suggest that the evidence is insufficient to recommend for or against these approaches. The role of acupuncture for neck pain also remains ambiguous, with poor quality studies and conflicting results. Even systematic reviews have come to conflicting conclusions.

Another controversial therapy is low level laser therapy, typically applied to specific points in the neck identified by tenderness, local acupuncture points, or a grid of predetermined points. The putative benefits could be mediated by anti-inflammatory tissue effects, reduction of

skeletal muscle fatigue, or inhibition of transmission at neuromuscular junctions. These effects have been demonstrated in some human and animal studies. A 2009 meta-analysis suggested that this treatment may provide greater pain relief than sham therapy for both acute and chronic neck pain. Comparison to other conservative treatment measures is needed.

Although some surgical studies have proposed a role for anterior discectomy and fusion in patients with neck pain, these studies have generally had no control groups, failed to report loss to follow-up, lacked standard assessment intervals, had unclear patient selection and screening processes, inadequately described other treatments, included heterogeneous diagnoses, and used non-validated measures of outcome. A systematic review suggested that there was no valid clinical evidence to support either cervical fusion or cervical disc arthroplasty in patients with neck pain but without radiculopathy. Similarly, there is no evidence to support radiofrequency neurotomy or cervical facet injections for neck pain without radiculopathy.

NECK PAIN WITH RADICULOPATHY

The natural history of neck pain even with radiculopathy is favorable, and many patients will improve without specific therapy. Although there are no randomized trials of NSAIDs specifically for neck pain, a course of NSAIDs, with or without muscle relaxants, may be appropriate initial therapy. Other nonsurgical treatments are commonly used, including opioid analgesics, oral corticosteroids, cervical traction, and immobilization with a hard or soft cervical collar. However, there are no randomized trials to establish the effectiveness of these treatments in comparison to natural history alone. Soft cervical collars can be modestly helpful on occasion with pain symptoms by limiting spontaneous and reflex neck movements that exacerbate pain.

As with lumbar radiculopathy, epidural corticosteroids may improve short-term symptom relief in patients with cervical radiculopathy. If cervical radiculopathy is due to bony compression from cervical Spondylosis with foraminal narrowing, then surgical decompression is generally indicated to forestall progression of neurologic signs.

Surgical treatment for neck pain with radiculopathy can produce rapid and substantial symptom relief, although it is unclear whether long-term outcomes are improved over nonsurgical therapy. Reasonable indications for cervical disc surgery include a progressive radicular motor deficit, functionally limiting pain that fails to respond to conservative management, or cervical spinal cord compression.

Surgical treatments include anterior cervical discectomy alone, laminectomy with discectomy, discectomy with fusion, and treatment by disc arthroplasty (implanting an artificial cervical disc). Fusions can be performed with a variety of techniques. The risk of subsequent radiculopathy or myelopathy at cervical segments adjacent to a fusion is ~3% per year and 26% per decade. Although this risk is sometimes portrayed as a late complication of surgery, it may also reflect the natural history of degenerative cervical disk disease. The durability of disc prostheses remains uncertain. Available data do not strongly support one surgical technique over another.

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Table 16-1 Acute Low Back Pain: Risk Factors for an Important Structural Cause

History

- Pain worse at rest or at night
- Prior history of cancer
- History of chronic infection (esp. lung, urinary tract, skin)
- History of trauma
- Incontinence
- Age > 70 years
- Intravenous drug use
- Glucocorticoid use
- History of a rapidly progressive neurologic deficit

Examination

- Unexplained fever
- Unexplained weight loss
- Percussion tenderness over the spine
- Abdominal, rectal, or pelvic mass
- Patrick’s sign or heel percussion sign
- Straight leg or reverse straight-leg raising signs
- Progressive focal neurologic deficit

Table 16-2 Lumbosacral Radiculopathy—Neurologic Features

Lumbosacral Nerve Roots	Examination Findings			Pain Distribution
	Reflex	Sensory	Motor	
L2a	—	Upper anterior thigh	Psoas (hip flexion)	Anterior thigh
L3a	—	Lower anterior thigh	Psoas (hip flexion) Quadriceps (knee extension) Thigh adduction	Anterior thigh, knee
L4a	Quadriceps (knee)	Medial calf	Quadriceps (knee extension) ^b Thigh adduction Tibialis anterior (foot dorsiflexion)	Knee, medial calf Anterolateral thigh
L5c	—	Dorsal surface—foot Lateral calf	Peroneii (foot eversion) ^b Tibialis anterior (foot dorsiflexion) Gluteus medius (hip abduction) Toe dorsiflexors	Lateral calf, dorsal foot, posterolateral thigh, buttocks
S1c	Gastrocnemius/soleus (ankle)	Plantar surface—foot Lateral aspect—foot	Gastrocnemius/solus (foot plantar flexion) ^b Abductor hallucis (toe flexors) ^b Gluteus maximus (hip extension)	Bottom foot, posterior calf, posterior thigh, buttocks

aReverse straight leg-raising sign present—see “Examination of the Back.”

bThese muscles receive the majority of innervation from this root.

cStraight leg-raising sign present—see “Examination of the Back.”

Table 16-3 Causes of Back or Neck Pain
Congenital/developmental
Spondylolysis and spondylolisthesis
Kyphoscoliosis
Spina bifida occulta
Tethered spinal cord
Minor trauma
Strain or sprain
Whiplash injury
Fractures
Trauma—falls, motor vehicle accidents
Atraumatic fractures—osteoporosis, neoplastic infiltration, exogenous steroids
Intervertebral disk herniation
Degenerative
Intervertebral Foraminal Narrowing
Disk-osteophyte complex
Internal disk disruption
LSS with neurogenic claudication
Uncovertebral joint disease
Atlantoaxial joint disease (e.g., rheumatoid arthritis)
Arthritis
Spondylosis
Facet or sacroiliac arthropathy
Neoplasms—metastatic, hematologic, primary bone tumors
Infection/inflammation
Vertebral osteomyelitis
Spinal epidural abscess
Septic disk (discitis)
Meningitis
Lumbar arachnoiditis
Autoimmune (e.g. anklyosing spondylitis, Reiter's syndrome)
Metabolic
Osteoporosis—hyperparathyroidism, immobility
Osteosclerosis (e.g., Paget's disease)
Vascular
Abdominal aortic aneurysm
Vertebral artery dissection
Other
Referred pain from visceral disease
Postural
Psychiatric, malingering, chronic pain syndromes

Table 16-4 Cervical Radiculopathy—Neurologic Features				
Cervical Examination Findings				
Nerve	Reflex	Sensory	Motor	Pain Distribution
C5	Biceps	Over lateral deltoid	Supraspinatus ^a (initial arm abduction) Infraspinatus ^a (arm external rotation) Deltoid ^a (arm abduction) Biceps (arm flexion)	Lateral arm, medial scapula
C6	Biceps	Thumb, index fingers Radial hand/forearm	Biceps (arm flexion) Pronator teres (internal forearm rotation)	Lateral forearm, thumb, index finger
C7	Triceps	Middle fingers Dorsum forearm	Triceps ^a (arm extension) Wrist extensors ^a Extensor digitorum ^a (finger extension)	Posterior arm, dorsal forearm, lateral hand
C8	Finger flexors	Little finger Medial hand and forearm	Abductor pollicis brevis (abduction D1) First dorsal interosseous (abduction D2) Abductor digiti minimi (abduction D5)	4th and 5th fingers, medial forearm
T1	Finger flexors	Axilla and medial arm	Abductor pollicis brevis (abduction D1) First dorsal interosseous (abduction D2) Abductor digiti minimi (abduction D5)	Medial arm, axilla

^aThese muscles receive the majority of innervation from this root.

Figure 17-1

Vertebral anatomy. (From A Gauthier Cornuelle, DH Gronefeld: *Radiographic Anatomy Positioning*. New York, McGraw-Hill, 1998; with permission.)

Figure 17-2

Spinal column. (From: A Gauthier Cornuelle, DH Gronefeld: *Radiographic Anatomy Positioning*. New York, McGraw-Hill, 1998; with permission.)

Figure 17-3

Compression of L5 and S1 roots by herniated disks. (From: RD Adams et al: *Principles of Neurology*, 8th ed. New York, McGraw-Hill, 2005; with permission.)

Figure 17-4

Left L5 radiculopathy. a) Sagittal T2-weighted image on the left reveals disk herniation at the L4-5 level. b) Axial T1-weighted image shows paracentral disk herniation with displacement of the thecal sac medially and the left L5 nerve root posteriorly in the left lateral recess.

Figure 17-5 Axial T2-weighted images of the lumbar spine. a) The left image shows a normal thecal sac within the lumbar spinal canal. The thecal sac is bright. The lumbar roots are dark punctuate dots in the posterior thecal sac with the patient supine. b) The thecal sac is not well visualized due to severe lumbar spinal canal stenosis, partially the result of hypertrophic facet joints.

Figure 17-6-Right C7 radiculopathy.

a) Sagittal T2-weighted image shows disk bulging and a mildly narrowed spinal canal, but no visible nerve root compression. b&c) Axial T 2-weighted upper images. The combination of uncinete hypertrophy (upper image) and facet hypertrophy (lower image) narrows the right C 6-7 intervertebral foramen resulting in right C7 nerve root compression (upper image).

Figure 17-4a



Figure 17-4b

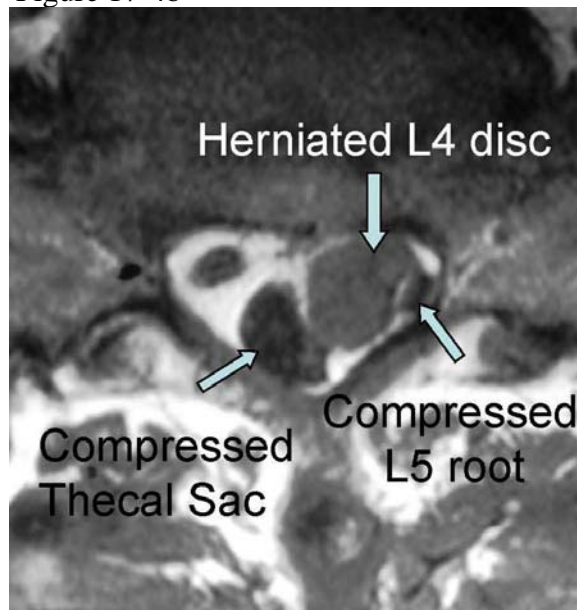


Figure 17-4

Left L5 radiculopathy. a) Sagittal T2-weighted image on the left reveals disk herniation at the L4-5 level. b) Axial T1-weighted image shows paracentral disk herniation with displacement of the thecal sac medially and the left L5 nerve root posteriorly in the left lateral recess.

Figure 17-5a

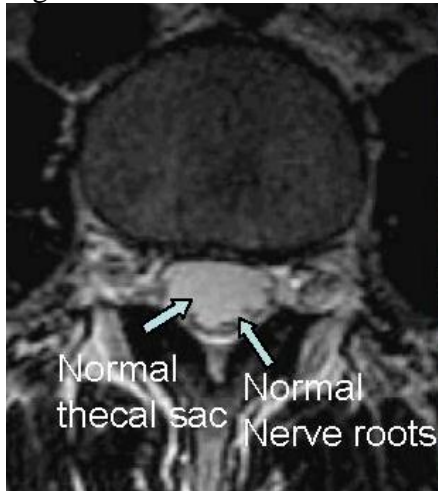


Figure 17-5b

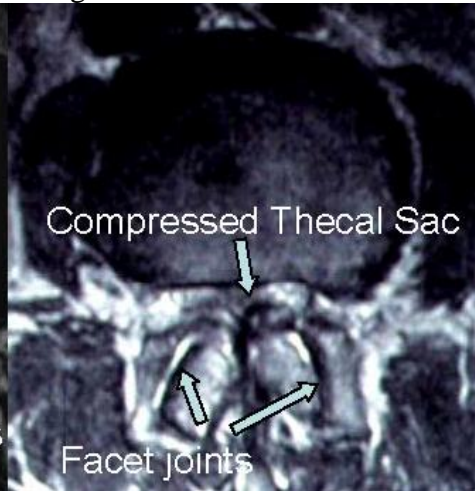


Figure 17-5-Axial T2-weighted images, lumbar spine. a) The left image shows a normal thecal sac within the lumbar spinal canal. The thecal sac is bright. The lumbar roots are dark punctate dots in the posterior and lateral thecal sac with the patient supine. b) Severe spinal stenosis. The thecal sac is obliterated due to severe lumbar spinal canal stenosis from hypertrophic facet joints.

Figure 17-6a



Figure 17-b

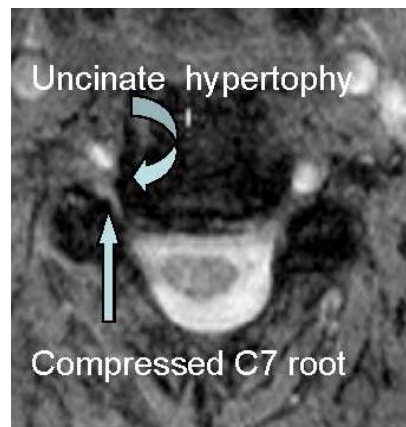


Figure 17-6-Right C7 radiculopathy.

a) Sagittal T2-weighted image shows mild disk bulging at C6-7 and a mildly narrowed spinal canal, but no visible nerve root compression. b) Axial T 2-weighted image. The combination of uncinete hypertrophy and facet hypertrophy (ovoid dark space just lateral to the C7 root) narrows the right C 6-7 intervertebral foramen resulting in right C7 nerve root compression.