## Chapter 17: Back and Neck Pain

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# INTRODUCTION

The importance of back and neck pain in our society is underscored by the following: (1) the cost of chronic back pain in the United States is estimated at more than $200 billion annually; approximately one-third of this cost is due to direct health care expenses and two-thirds are indirect costs resulting from loss of wages and productivity; (2) back symptoms are the most common cause of disability in individuals <45 years of age; (3) low back pain (LBP) is the second most common reason for visiting a physician in the United States; and (4) more than four out of five people will experience significant back pain at some point in their lives.

# ANATOMY OF THE SPINE

The anterior spine consists of cylindrical vertebral bodies separated by intervertebral disks and stabilized 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 fibrosis. Disks are responsible for 25% of spinal column length and allow the bony vertebrae to move easily upon each other **(Figs. 17-1 and 17-2)**. Desiccation of the nucleus pulposus and degeneration of the annulus fibrosus worsen with age, resulting in loss of disk height. The disks are largest in the cervical and lumbar regions where movements of the spine are greatest. The anterior spine absorbs the shock of bodily movements such as walking and running, and with the posterior spine protects the spinal cord and nerve roots in the spinal canal.

FIGURE 17-1

**Vertebral anatomy.** ***A.*** Vertebral body—axial view; ***B.*** vertebral column—sagittal view. *(Reproduced with permission from A Gauthier Cornuelle, DH Gronefeld: Radiographic Anatomy Positioning. New York, McGraw-Hill, 1998.)*

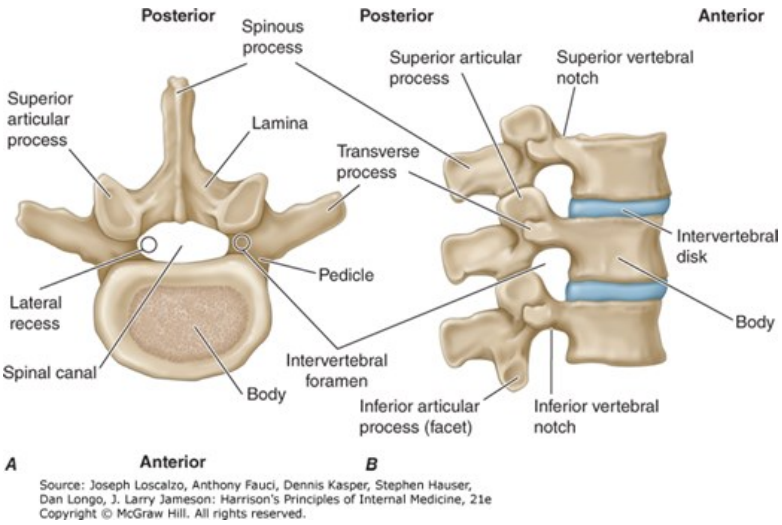
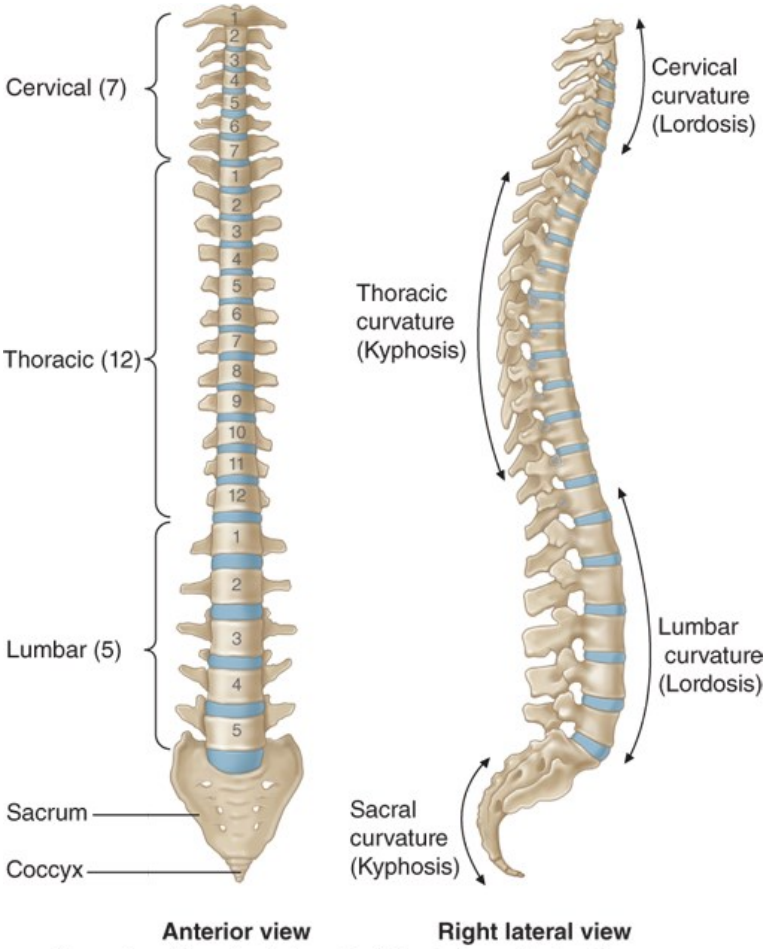





FIGURE 17-2

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



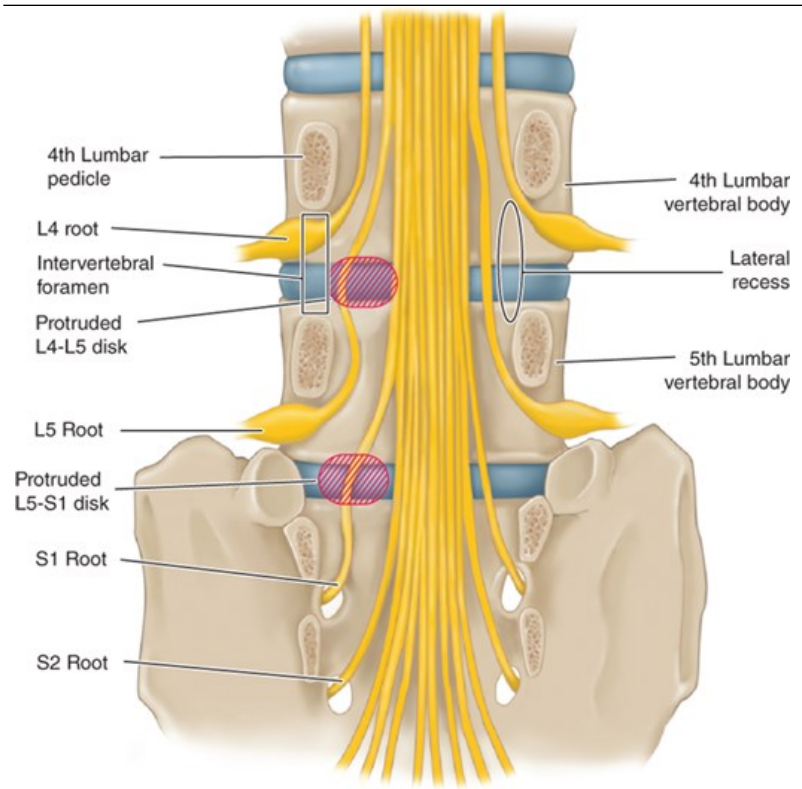



The posterior spine consists of the vertebral arches and processes. Each arch consists of paired cylindrical pedicles anteriorly and paired lamina posteriorly. The vertebral arch also gives rise to two transverse processes laterally, one spinous process posteriorly, plus two superior and two inferior articular facets. The apposition of a superior and inferior facet constitutes a *facet joint*. The posterior spine provides an anchor for the attachment of muscles and ligaments. The contraction of muscles attached to the spinous and transverse processes and lamina works like a system of pulleys and levers producing flexion, extension, rotation, and lateral bending movements of the spine.

Nerve root injury (*radiculopathy*) is a common cause of pain in the neck and arm, or low back and buttock, or leg **(see dermatomes in Figs. 25-2 and 25-3)**. Each nerve root exits just above its corresponding vertebral body in the cervical region (e.g., the C7 nerve root exits at the C6-C7 level), and just below the vertebral body in the thoracic and lumbar spine (e.g., the T1 nerve root exits at the T1-T2 level). The cervical nerve roots follow a short intraspinal course before exiting. In contrast, because the spinal cord ends at the L1 or L2 vertebral level, the lumbar nerve roots follow a long intraspinal course and can be injured anywhere along its path. For example, disk herniation at the L4-L5 level can produce L4 root compression laterally, but more often compression of the traversing L5 nerve root occurs **(Fig. 17-3)**. The lumbar nerve roots are mobile in the spinal canal, but eventually pass through the narrow *lateral recess* of the spinal canal and *intervertebral foramen* (Figs. 17-2 and 17-3). When imaging the spine, both sagittal and axial views are needed to assess possible compression at these sites.

FIGURE 17-3

**Compression of L5 and S1 roots by herniated disks.** *(Reproduced with permission from AH Ropper, MA Samuels: Adams and Victor's Principles of Neurology, 9th ed. New York, McGraw-Hill, 2009.)*




Source: Joseph Loscalzo, Anthony Fauci, Dennis Kasper, Stephen Hauser, Dan Longo, J. Larry Jameson: Harrison's Principles of Internal Medicine, 21e


Beginning at the C3 level, each cervical (and the first thoracic) vertebral body projects a lateral bony process upward—the uncinate process. The uncinate process articulates with the cervical vertebral body above via the uncovertebral joint. The uncovertebral joint can hypertrophy with age and contribute to neural foraminal narrowing and cervical radiculopathy.

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 longitudinal ligaments. Disease of these diverse structures may explain many cases of back pain without nerve root compression. Under normal circumstances, the nucleus pulposus of the intervertebral disk is not pain sensitive.

## APPROACH TO THE PATIENT WITH BACK PAIN

### Types of Back Pain

Delineating the type of pain reported by the patient is the essential first step. Attention is also focused on identifying risk factors for a serious underlying etiology. The most frequent serious causes of back pain are radiculopathy, fracture, tumor, infection, or referred pain from visceral structures **(Table 17-1)**.

TABLE 17-1

**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 (especially lung, urinary tract, skin, poor dentition) |
| 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 |
| Focal palpation/percussion tenderness over the midline spine |
| Abdominal, rectal, or pelvic mass |
| Internal/external rotation of the leg at the hip |
| Straight-leg or reverse straight-leg raising signs |
| Progressive focal neurologic deficit |

*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, accompanied by back pain, and usually unaffected by posture. The patient may occasionally complain of back pain only.

*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, calves, or feet. Referred pain often explains pain syndromes that cross multiple dermatomes without evidence of nerve or nerve root injury.

*Radicular 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 or worsen the radiating pain.

The pain may also 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 sciatic 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 referred pain and radiculopathy, although a burning or electric quality favors radiculopathy.

*Pain associated with muscle spasm* is commonly associated with many spine disorders. The spasms may be accompanied by an abnormal posture, tense 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

A complete physical examination including vital signs, heart and lungs, abdomen and rectum, and limbs 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 of the back may reveal a lateral curvature of the spine (scoliosis). A midline hair tuft, skin dimpling or pigmentation, or a sinus tract may indicate a congenital spine anomaly. Asymmetry in the prominence of the paraspinal muscles suggests muscle spasm. Palpation over the spinous process transmits force to the entire vertebrae and suggests vertebral pathology.

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 passive internal and external rotation at the hip with the knee and hip in flexion or by percussing the heel with the examiner's palm with the leg extended (heel percussion sign).

The *straight-leg raising (SLR)* maneuver is a simple bedside test for nerve root disease. With the patient supine, passive straight-leg flexion at the hip stretches the L5 and S1 nerve roots and the sciatic nerve; dorsiflexion of the foot during the maneuver adds to the stretch. In healthy individuals, flexion to at least 80° is normally possible without causing pain, although a tight, stretching sensation in the hamstring muscles is common. The SLR test is positive if the maneuver reproduces the patient's usual back or limb pain. Eliciting the SLR sign in both the supine and sitting positions 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 present when flexion of one leg reproduces the usual pain in the opposite leg or buttocks. In disk herniation, the crossed SLR sign is less sensitive but more specific than the SLR sign. 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. For all of these tests, the nerve or nerve root lesion is always on the side of the pain. Examination of the unaffected leg first provides a control test, ensures mutual understanding of test parameters, and enhances test utility.

The neurologic examination includes a search for focal weakness or muscle atrophy, localized 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 fluctuations in the maximum power generated during muscle testing. Breakaway weakness may be due to pain, inattention, or a combination of pain and underlying true weakness. Breakaway weakness without pain is usually due to a lack of effort. In uncertain cases, electromyography (EMG) can determine if true weakness due to nerve tissue injury is present. Findings with specific lumbosacral nerve root lesions are shown in **Table 17-2** and are discussed below.

TABLE 17-2

**Lumbosacral Radiculopathy: Neurologic Features**

| LUMBOSACRAL NERVE ROOT | EXAMINATION FINDINGS | | | PAIN DISTRIBUTION |
|---|---|---|---|---|
| | REFLEX | SENSORY | MOTOR | |
| L2[a] | — | Upper anterior thigh | Psoas (hip flexors) | Anterior thigh |
| L3[a] | — | Lower anterior thigh | Psoas (hip flexors) | Anterior thigh, knee |
| | | Anterior knee | Quadriceps (knee extensors) | |
| | | | Thigh adductors | |
| L4[a] | Quadriceps (knee) | Medial calf | Quadriceps (knee extensors)[b] | Knee, medial calf |
| | | | Thigh adductors | Anterolateral thigh |
| L5[c] | — | Dorsal surface—foot | Peronei (foot evertors)[b] | Lateral calf, dorsal foot, posterolateral thigh, buttocks |
| | | Lateral calf | Tibialis anterior (foot dorsiflexors) | |
| | | | Gluteus medius (leg abductors) | |
| | | | Toe dorsiflexors | |
| S1[c] | Gastrocnemius/soleus (ankle) | Plantar surface—foot | Gastrocnemius/soleus (foot plantar flexors)[b] | Bottom foot, posterior calf, posterior thigh, buttocks |
| | | Lateral aspect—foot | Abductor hallucis (toe flexors)[b] | |
| | | | Gluteus maximus (leg extensors) | |

[a]Reverse straight-leg raising sign may be present—see "Examination of the Back."

[b]These muscles receive the majority of innervation from this root.

[c]Straight-leg raising sign may be present—see "Examination of the Back."

### Laboratory, Imaging, and EMG Studies

Laboratory studies are rarely needed for the initial evaluation of nonspecific acute (<3 months duration) low back pain (ALBP). Risk factors for a serious underlying cause and for infection, tumor, or fracture in particular should be sought by history and examination. If risk factors are present (Table 17-1), then laboratory studies (complete blood count [CBC], erythrocyte sedimentation rate [ESR], urinalysis) are indicated. If risk factors are

absent, then management is conservative (see “Treatment,” below).

CT scanning is used as a primary screening modality for acute trauma that is moderate to severe. CT is superior to x-rays for detection of fractures involving posterior spine structures, craniocervical and cervicothoracic junctions, C1 and C2 vertebrae, bone fragments in the spinal canal, or misalignment. MRI or CT myelography is the radiologic test 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, defines bony abnormalities, and is tolerated by claustrophobic patients.

Population surveys in the United States suggest that patients with back pain report greater functional limitations in recent years, despite rapid increases in spine imaging, opioid prescribing, injections, and spine surgery. This suggests that more selective use of diagnostic and treatment modalities may be reasonable for many patients. One prospective case-control study found that older adults with back pain of less than 6 weeks duration who received spine imaging as part of a primary care visit had no better outcomes than the control group.

Spine imaging often reveals abnormalities of dubious clinical relevance that may alarm clinicians and patients alike and prompt further testing and unnecessary therapy. When imaging tests are reviewed, it is important to remember that degenerative findings are common in normal, pain-free individuals. Randomized trials and observational studies have suggested that imaging can have a “cascade effect,” creating a gateway to other unnecessary care. Interventions have included physician education and computerized decision support within the electronic medical record to require specific indications for approval of imaging tests. Other strategies have included audit and feedback of individual practitioners’ rates of ordering, more rapid access to physical therapy, or consultation with spine experts for patients without imaging indications.

Educational tools created by the America College of Physicians for patients and the public have included “Five Things Physicians and Patients Should Question”: (1) Do not recommend advanced imaging (e.g., MRI) of the spine within the first 6 weeks in patients with nonspecific ALBP in the absence of red flags. (2) Do not perform elective spinal injections without imaging guidance, unless contraindicated. (3) Do not use bone morphogenetic protein (BMP) for routine anterior cervical spine fusion surgery. (4) Do not use EMG and nerve conduction studies (NCSs) to determine the cause of purely midline lumbar, thoracic, or cervical spine pain. (5) Do not recommend bed rest for >48 h when treating LBP. In an observational study, application of this strategy was associated with lower rates of repeat imaging, opioid use, and referrals for physical therapy.

Electrodiagnostic studies can be used to assess the functional integrity of the peripheral nervous system **(Chap. 446)**. Sensory NCSs are normal when focal sensory loss confirmed by examination is due to nerve root damage because the nerve roots are proximal to the 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. Needle EMG complements NCSs 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 clinical evaluation of weakness is limited by pain or poor effort. EMG and NCSs will be normal when sensory nerve root injury or irritation is the pain source.

The COVID-19 pandemic has disrupted and complicated the care of patients with LBP. Paraspinal myalgias may result in LBP. The sedentary lifestyle resulting from quarantine is associated with an increased frequency or severity of LBP. Fear of infection risk has also prevented many patients from seeking needed care. Video-telemedicine visits can help identify patients with underlying risks for a serious cause and inform appropriate next steps in management.

## CAUSES OF BACK PAIN

### (TABLE 17-3)

TABLE 17-3

**Causes of Back or Neck Pain**

| |
|---|
| **Lumbar or Cervical Disk Disease** |
| **Degenerative Spine Disease** |

| |
|---|
| Lumbar spinal stenosis without or with neurogenic claudication |
| Intervertebral foraminal or lateral recess narrowing |
| Disk-osteophyte complex |
| Facet or uncovertebral joint hypertrophy |
| Lateral disk protrusion |
| Spondylosis (osteoarthritis), spondylolisthesis, or spondylolysis |
| **Spine Infection** |
| Vertebral osteomyelitis |
| Spinal epidural abscess |
| Septic disk (diskitis) |
| Meningitis |
| Lumbar arachnoiditis |
| **Neoplasms** |
| Metastatic with/without pathologic fracture |
| Primary Nervous System: Meningioma, neurofibroma, schwannoma |
| Primary Bone: chordoma, osteoma |
| **Trauma** |
| Strain or sprain |
| Whiplash injury |
| Trauma/falls, motor vehicle accidents |
| **Metabolic Spine Disease** |
| Osteoporosis with/without pathologic fracture—hyperparathyroidism, immobility |
| Osteosclerosis (e.g., Paget's disease) |
| **Congenital/Developmental** |
| Spondylolysis |
| Kyphoscoliosis |
| Spina bifida occulta |
| Tethered spinal cord |
| **Autoimmune Inflammatory Arthritis** |
| **Other Causes of Back Pain** |
| Referred pain from visceral disease (e.g., abdominal aortic aneurysm) |
| Postural |
| Psychiatric, malingering, chronic pain syndromes |

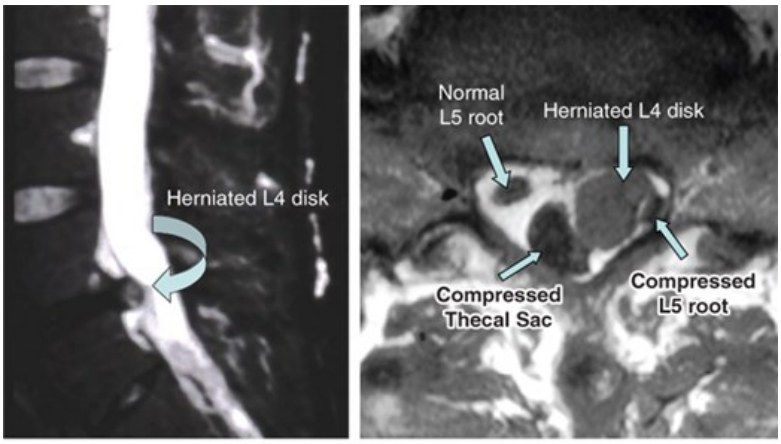
## LUMBAR DISK DISEASE

Lumbar disk disease is a common cause of acute, chronic, or recurrent low back and leg pain **(Figs. 17-3 and 17-4)**. Disk disease is most likely to

occur at the L4-L5 or L5-S1 levels, but upper lumbar levels can also be involved. The cause is often unknown, but the risk is increased in overweight individuals. Disk herniation is unusual prior to age 20 years and is rare in the fibrotic disks of the elderly. Complex genetic factors may play a role in predisposition. 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 can protrude through the annulus (herniation) or become extruded to lie as a free fragment in the spinal canal.

FIGURE 17-4

**Disk herniation.** ***A***. Sagittal T2-weighted image on the left side of the spinal canal reveals disk herniation at the L4-L5 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. (*Source: JL Jameson, AS Fauci, DL Kasper, SL Hauser, DL Longo, J Loscalzo: Harrison's Principles of Internal Medicine, 20th Edition Copyright © McGraw Hill Education. All rights reserved.*)






The mechanism by which intervertebral disk injury causes back pain is uncertain. The inner annulus fibrosus and nucleus pulposus are normally devoid of innervation. Inflammation and production of proinflammatory cytokines within a ruptured nucleus pulposus may trigger or perpetuate back pain. Ingrowth of nociceptive (pain) nerve fibers into the nucleus pulposus of a diseased disk may be responsible for some cases of chronic "diskogenic" pain. Nerve root injury (radiculopathy) from disk herniation is usually due to inflammation, but lateral herniation may produce compression in the lateral recess or intervertebral foramen.

A ruptured disk may be asymptomatic or cause back pain, limited spine motion (particularly flexion), a focal neurologic deficit, or radicular pain. A dermatomal pattern of sensory loss or a reduced or absent deep tendon reflex is more suggestive of a specific root 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 herniations that involve roots bilaterally or cause inflammation of nerve roots within the spinal canal. Clinical manifestations of specific nerve root lesions are summarized in Table 17-2.

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 myelopathy suggest an etiology other than lumbar disk disease. Absent ankle reflexes can be a normal finding in persons >60 years or a sign of bilateral S1 radiculopathies. An absent deep tendon reflex or focal sensory loss may indicate injury to a nerve root, but other sites of injury along the nerve must also be considered. As examples, an absent knee reflex may be due to a femoral neuropathy or an L4 nerve root injury; 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 may reflect injury to the anterior horn cells of the spinal cord, a nerve root, peripheral nerve, or disuse.

A lumbar spine MRI scan or CT myelogram can often confirm the location and type of pathology. Spine MRIs yield exquisite views of intraspinal and adjacent soft tissue anatomy, whereas bony lesions of the lateral recess or intervertebral foramen are optimally visualized by CT myelography. The correlation of neuroradiologic findings to clinical symptoms, 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 radiologic findings. Entirely asymptomatic disk protrusions are also common, occurring in up to one-third of adults, and these may also 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 hypertrophic facet joints 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. There is often good correlation between CT and EMG findings for localization of nerve root injury.

Management of lumbar disk disease is discussed below. *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-L2. LBP, weakness and areflexia in the legs, saddle anesthesia, or loss of bladder function may occur. The problem must be distinguished from disorders of the lower spinal cord (conus medullaris syndrome), acute transverse myelitis **(Chap. 442)**, and Guillain-Barré syndrome **(Chap. 447)**. Combined involvement of the conus medullaris and cauda equina can occur. CES is most commonly due to a large ruptured lumbosacral intervertebral disk, but other causes include lumbosacral spine fracture, hematoma within the spinal canal (sometimes following lumbar puncture in patients with coagulopathy), and tumor or other compressive mass lesions. Treatment is usually surgical decompression, sometimes on an urgent basis in an attempt to restore or preserve motor or sphincter function, or radiotherapy for metastatic tumors **(Chap. 90)**.

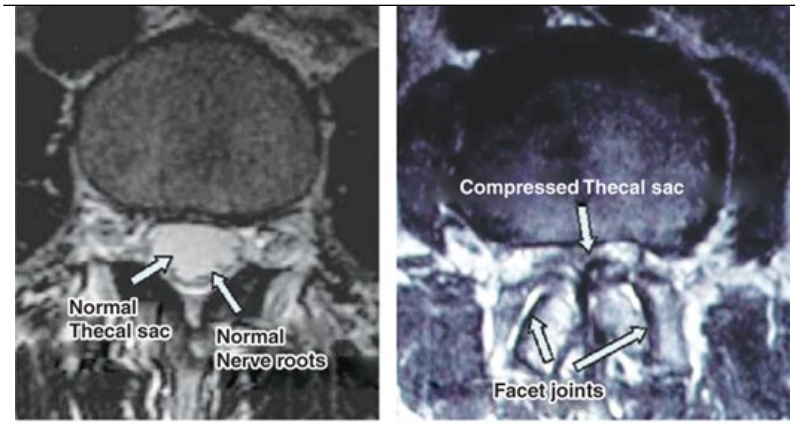
## DEGENERATIVE CONDITIONS

*Lumbar spinal stenosis* (*LSS*) describes a narrowed lumbar spinal canal. *Neurogenic claudication* consists of pain, typically in the back and buttocks or legs, that is brought on by walking or standing and relieved by sitting. Unlike vascular claudication, symptoms are often provoked by standing without walking. Unlike lumbar disk disease, symptoms are usually relieved by sitting. Patients with neurogenic claudication can often walk much farther when leaning over a shopping cart and can pedal a stationary bike with ease while sitting. These flexed positions increase the anteroposterior spinal canal diameter and reduce intraspinal venous hypertension, producing pain relief. Focal weakness, sensory loss, or reflex changes may occur when spinal stenosis is associated with neural foraminal narrowing and radiculopathy. Severe neurologic deficits, including paralysis and urinary incontinence, occur only rarely.

LSS by itself is common (6–7% of adults) and is usually asymptomatic. Symptoms are correlated with severe spinal canal stenosis. LSS is most often acquired (75%) but can also be congenital or due to a mixture of both etiologies. Congenital forms (achondroplasia and 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, and scoliosis), trauma, spine surgery, metabolic or endocrine disorders (epidural lipomatosis, osteoporosis, acromegaly, renal osteodystrophy, and hypoparathyroidism), and Paget's disease. MRI provides the best definition of the abnormal anatomy **(Fig. 17-5)**.

**FIGURE 17-5**

**Spinal stenosis.** ***A***. An axial T2-weighted image of the normal lumbar spine shows a normal thecal sac within the lumbar spinal canal. The thecal sac is bright. The lumbar roots are seen as dark punctate dots located posteriorly in the thecal sac. ***B***. The thecal sac is not well visualized due to severe lumbar spinal canal stenosis, partially the result of hypertrophic facet joints. *(Source: JL Jameson, AS Fauci, DL Kasper, SL Hauser, DL Longo, J Loscalzo: Harrison's Principles of Internal Medicine, 20th Edition Copyright © McGraw Hill Education. All rights reserved.)*





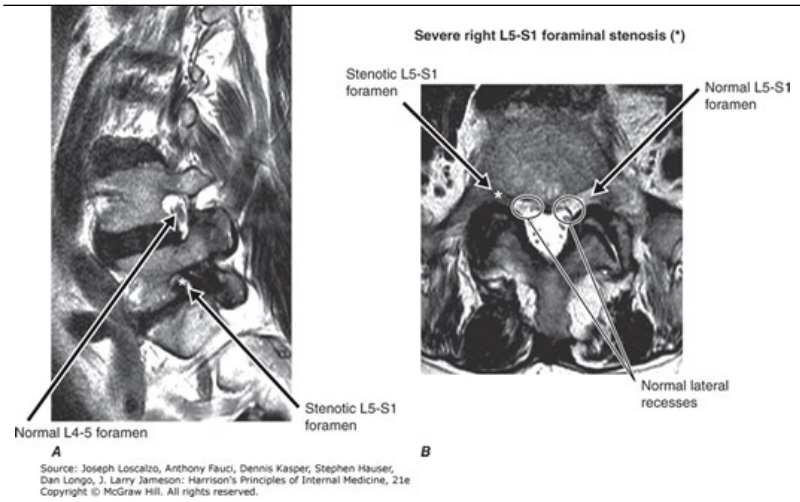

LSS accompanied by neurogenic claudication responds to surgical decompression of the stenotic segments. The same processes leading to LSS may cause lumbar foraminal or lateral recess narrowing resulting in coincident lumbar radiculopathy that may require treatment as well.

Conservative treatment of symptomatic LSS can include nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, exercise programs, and symptomatic treatment of acute pain episodes. There is insufficient evidence to support the routine use of epidural glucocorticoid injections. Surgery is considered when medical therapy does not relieve symptoms sufficiently to allow for resumption of activities of daily living or when focal neurologic signs are present. Most patients with neurogenic claudication who are treated medically do not improve over time. Surgical management with laminectomy, which increases the spinal canal diameter and reduces venous hypertension, can produce significant relief of exertional back and leg pain, leading to less disability and improved functional outcomes. Laminectomy and fusion is usually reserved for patients with LSS and spondylolisthesis. Predictors of a poor surgical outcome include impaired walking preoperatively, depression, cardiovascular disease, and scoliosis. Up to one-quarter of surgically treated patients develop recurrent stenosis at the same or an adjacent spinal level within 7–10 years; recurrent symptoms usually respond to a second surgical decompression.

*Neural foraminal narrowing or lateral recess stenosis with radiculopathy* is a common consequence of osteoarthritic processes that cause LSS **(Figs. 17-1 and 17-6)**, including osteophytes, lateral disk protrusion, calcified disk-osteophytes, facet joint hypertrophy, uncovertebral joint hypertrophy (in the cervical spine), congenitally shortened pedicles, or, frequently, a combination of these processes. Neoplasms (primary or metastatic), fractures, infections (epidural abscess), or hematomas are less frequent causes. Most common is bony foraminal narrowing leading to nerve root ischemia and persistent symptoms, in contrast to inflammation that is associated with a paracentral herniated disk and radiculopathy. These conditions can produce unilateral nerve root symptoms or signs due to compression at the intervertebral foramen or in the lateral recess; symptoms are indistinguishable from disk-related radiculopathy, but treatment may differ depending on the etiology. The history and neurologic examination alone cannot distinguish between these possibilities. Neuroimaging (CT or MRI) is required to identify the anatomic cause. Neurologic findings from the examination and EMG can help direct the attention of the radiologist to specific nerve roots, especially on axial images. For *facet joint hypertrophy with foraminal stenosis,* surgical foraminotomy produces long-term relief of leg and back pain in 80–90% of patients. Facet joint or medial branch blocks for back or neck pain are sometimes used to help determine the anatomic origin of back pain or for treatment, but there is a lack of clinical data to support their utility. Medical causes of lumbar or cervical radiculopathy unrelated to primary spine disease include infections (e.g., herpes zoster and Lyme disease), carcinomatous meningitis, diabetes, and root avulsion or traction (trauma).

**FIGURE 17-6**

**Foraminal stenosis.** ***A***. Sagittal T2-weighted image reveals normal high signal around the exiting right L4 nerve root in the right neural foramen at L4-L5; effacement of the high signal is noted one level below at L5-S1, due to severe foraminal stenosis. ***B***. Axial T2-weighted image at the L5-S1 level demonstrates normal lateral recesses bilaterally, a normal intervertebral foramen on the left, but a severely stenotic foramen (*) on the right. (*Source: JL Jameson, AS Fauci, DL Kasper, SL Hauser, DL Longo, J Loscalzo: Harrison's Principles of Internal Medicine, 20th Edition Copyright © McGraw Hill Education. All rights reserved.)*





## SPONDYLOSIS AND SPONDYLOLISTHESIS

*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 increases with movement, is associated with stiffness, and is better with inactivity. The relationship between clinical symptoms and radiologic findings is usually not straightforward. Pain may be prominent when MRI, CT, or x-ray findings are minimal, and prominent degenerative spine disease can be seen in asymptomatic patients. Osteophytes, combined disk-osteophytes, or a thickened ligamentum flavum may cause or contribute to central spinal canal stenosis, lateral recess stenosis, or neural foraminal narrowing.

*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 interarticularis (e.g., infection, osteoporosis, tumor, trauma, earlier surgery). The slippage may be asymptomatic or may cause LBP, nerve root injury (the L5 root most frequently), symptomatic spinal stenosis, or CES in rare severe cases. A “step-off” on palpation or tenderness may be elicited near the segment that has “slipped” (most often L4 on L5 or occasionally L5 on S1). Focal anterolisthesis or retrolisthesis can occur at any cervical or lumbar level and be the source of neck or LBP. Plain x-rays of the low back or neck in flexion and extension will reveal movement at the abnormal spinal segment. Surgery is performed for spinal instability (slippage 5–8 mm) and considered for pain symptoms that do not respond to conservative measures (e.g., rest, physical therapy), cases with a progressive neurologic deficit, or scoliosis.

## NEOPLASMS

Back pain is the most common neurologic symptom in patients with systemic cancer and is the presenting symptom in 20%. The cause is usually vertebral body metastasis (85–90%) but can also result from spread of cancer through the intervertebral foramen (especially with lymphoma), carcinomatous meningitis, or metastasis to the spinal cord. The thoracic spine is most often affected. Cancer-related back pain tends to be constant, dull, unrelieved by rest, and worse at night. By contrast, mechanical causes of LBP usually improve with rest. MRI, CT, and CT myelography are the studies of choice when spinal metastasis is suspected. Once a metastasis is found, imaging of the entire spine is essential, as it reveals additional tumor deposits in one-third of patients. MRI is preferred for soft tissue definition, but the most rapidly available imaging modality is best because the patient’s condition may worsen quickly without intervention. Early diagnosis is crucial. A strong predictor of outcome is baseline neurologic function prior to diagnosis. Half to three-quarters of patients are nonambulatory at the time of diagnosis and few regain the ability to walk. **The management of spinal metastasis is discussed in detail in Chap. 90.**

## INFECTIONS/INFLAMMATION

*Vertebral osteomyelitis* is most often caused by hematogenous seeding of staphylococci, but other bacteria or tuberculosis (Pott’s disease) may be responsible. The primary source of infection is usually the skin or urinary tract. Other common sources of bacteremia are IV drug use, poor dentition, endocarditis, lung abscess, IV catheters, or postoperative wound sites. Back pain at rest, tenderness over the involved vertebra, and an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) 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. The intervertebral disk can

also be affected by infection (diskitis) and almost never by tumor. Extension of the infection posteriorly from the vertebral body can produce a spinal epidural abscess.

*Spinal epidural abscess* **(Chap. 442)** presents with back pain (aggravated by movement or palpation of the spinous process), fever, radiculopathy, or signs of spinal cord compression. The subacute development of two or more of these findings should increase suspicion for spinal epidural abscess. The abscess is best delineated by spine MRI and may track over multiple spinal levels.

*Lumbar adhesive arachnoiditis* with radiculopathy is due to fibrosis following inflammation within the subarachnoid space. The fibrosis results in nerve root adhesions and presents as back and leg pain associated with multifocal motor, sensory, or reflex changes. Causes of arachnoiditis include multiple lumbar operations (most common in the United States), chronic spinal infections (especially tuberculosis in the developing world), spinal cord injury, intrathecal hemorrhage, myelography (rare), intrathecal injections (glucocorticoids, anesthetics, or other agents), and foreign bodies. The MRI shows clumped nerve roots on axial views or loculations of cerebrospinal fluid within the thecal sac. Clumped nerve roots should be distinguished from enlarged nerve roots seen with demyelinating polyneuropathy or neoplastic infiltration. Treatment is usually unsatisfactory. Microsurgical lysis of adhesions, dorsal rhizotomy, dorsal root ganglionectomy, and epidural glucocorticoids have been tried, but outcomes have been poor. Dorsal column stimulation for pain relief has produced varying results.

## TRAUMA

A patient complaining of back pain and an inability to move the legs may have a spine fracture or dislocation; fractures above L1 place the spinal cord at risk for compression. Care must be taken to avoid further damage to the spinal cord or nerve roots by immobilizing the back or neck pending the results of radiologic studies. Vertebral fractures frequently occur in the absence of trauma in association with osteoporosis, glucocorticoid use, osteomyelitis, or neoplastic infiltration.

### Sprains and Strains

The terms *low back sprain*, *strain*, and *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 and do not correlate with specific underlying pathologies. The pain is usually confined to the lower back. 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, 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. Rules have been developed to avoid unnecessary spine imaging associated with low-risk trauma, but these studies typically exclude patients aged >65 years—a group that can sustain fractures with minor trauma.

## METABOLIC CAUSES

### Osteoporosis and Osteosclerosis

Immobilization, osteomalacia, the postmenopausal state, renal disease, multiple myeloma, hyperparathyroidism, hyperthyroidism, metastatic carcinoma, or glucocorticoid use may accelerate osteoporosis and weaken the vertebral body, leading to compression fractures and pain. Up to two-thirds of compression fractures seen on radiologic imaging are asymptomatic. The most common nontraumatic vertebral body fractures are due to a postmenopausal cause, or to osteoporosis in adults >75 years old **(Chap. 411)**. The risk of an additional vertebral fracture 1 year following a first vertebral fracture is 20%. The presence of fever, weight loss, fracture at a level above T4, any fracture in a young adult, or the predisposing conditions described above should increase suspicion for a cause other than typical osteoporosis. The sole manifestations of a compression fracture may be localized back or radicular pain exacerbated by movement and often reproduced by palpation over the spinous process of the affected vertebra.

Relief of acute pain can often be achieved with acetaminophen, NSAIDs, opioids, or a combination of these medications. Both pain and disability are improved with bracing. Antiresorptive drugs are not recommended in the setting of acute pain but are the preferred treatment to prevent additional

fractures. Less than one-third of patients with prior compression fractures are adequately treated for osteoporosis despite the increased risk for future fractures; even fewer at-risk patients without a history of fracture are adequately treated. The literature for percutaneous vertebroplasty (PVP) or kyphoplasty for osteoporotic compression fractures associated with debilitating pain does not support their use.

Osteosclerosis, an abnormally increased bone density often due to Paget's disease, is readily identifiable on routine x-ray studies and can sometimes be a source of back pain. It may be associated with an isolated increase in alkaline phosphatase in an otherwise healthy older person. Spinal cord or nerve root compression can result from bony encroachment. The diagnosis of Paget's disease as the cause of a patient's back pain is a diagnosis of exclusion.

**For further discussion of these bone disorders, see Chaps. 410, 411, and 412.**

## AUTOIMMUNE INFLAMMATORY ARTHRITIS

Autoimmune inflammatory disease of the spine can present with the insidious onset of low back, buttock, or neck pain. Examples include rheumatoid arthritis (RA) **(Chap. 358)**, ankylosing spondylitis, reactive arthritis and psoriatic arthritis **(Chap. 355)**, or inflammatory bowel disease **(Chap. 326)**.

## CONGENITAL ANOMALIES OF THE LUMBAR SPINE

*Spondylolysis* is a bony defect in the vertebral pars interarticularis (a segment near the junction of the pedicle with the lamina), a finding present in up to 6% of adolescents. The cause is usually a stress microfracture in a congenitally abnormal segment. Multislice CT with multiplanar reformation is the most accurate modality for detecting spondylolysis in adults. Symptoms may occur in the setting of a single injury, repeated minor injuries, or during a growth spurt. Spondylolysis is the most common cause of persistent LBP in adolescents and is often associated with sports-related activities.

*Scoliosis* refers to an abnormal curvature in the coronal (lateral) plane of the spine. With *kyphoscoliosis,* there is, in addition, a forward curvature of the spine. The abnormal curvature may be congenital, due to abnormal spine development, acquired in adulthood due to degenerative spine disease, or progressive due to paraspinal neuromuscular disease. The deformity can progress until ambulation or pulmonary function is compromised.

Spina bifida occulta (closed spinal dysraphism) 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, but the skin is intact. Most cases are asymptomatic and discovered incidentally during a physical examination 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 child or young adult who complains of perineal or perianal pain, sometimes following minor trauma. MRI studies typically reveal a low-lying conus (below L1 and L2) and a short and thickened filum terminale. The MRI findings also occur as incidental findings, sometimes during evaluation of unrelated LBP in adults.

## REFERRED PAIN FROM VISCERAL DISEASE

Diseases of the thorax, abdomen, or pelvis may refer pain to 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 midlumbar 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.

### Low Thoracic or Lumbar Pain with Abdominal Disease

Tumors of the posterior wall of the stomach or duodenum typically produce epigastric pain **(Chaps. 80 and 324)**, but back pain may occur if retroperitoneal extension is present. Fatty foods occasionally induce back pain associated with biliary or pancreatic disease. Pathology in retroperitoneal structures (hemorrhage, tumors, and 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 testicle. The sudden appearance of lumbar pain in a patient receiving anticoagulants should prompt consideration of retroperitoneal hemorrhage.

Isolated LBP occurs in some patients with a contained rupture of an AAA. The classic clinical triad of abdominal pain, shock, and back pain occurs in <20% of patients. The diagnosis may be 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 50–75% of patients) is an important physical finding. Patients with suspected AAA should be evaluated with abdominal ultrasound, CT, or MRI **(Chap. 280)**.

### Sacral Pain with Gynecologic and Urologic Disease

Pelvic organs rarely cause isolated LBP. Uterine malposition (retroversion, descensus, and prolapse) may cause traction on the uterosacral ligament. The pain is referred to the sacral region, sometimes appearing after prolonged standing. Endometriosis or uterine cancers can invade the uterosacral ligaments. Pain associated with endometriosis is typically premenstrual and often continues until it merges with menstrual pain.

Menstrual pain with poorly localized, cramping pain can radiate down the legs. LBP that radiates into one or both thighs is common in the last weeks of pregnancy. Continuous and worsening pain unrelieved by rest or at night may be due to neoplastic infiltration of nerves or nerve roots.

Urologic sources of lumbosacral back pain include chronic prostatitis, prostate cancer with spinal metastasis **(Chap. 87)**, and diseases of the kidney or ureter. 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. Exercises to strengthen the paraspinal and abdominal muscles are sometimes helpful. 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. 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 LBP occasionally remains unclear. Some patients have had multiple operations for disk disease. 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 on neurologic signs, psychological factors, physiologic studies, and imaging studies have been devised to minimize the likelihood of unsuccessful surgery.

## GLOBAL CONSIDERATIONS

While many of the history and examination features described in this chapter apply to all patients, information regarding the global epidemiology and prevalence of LBP is limited. The Global Burden of Diseases Study 2019 reported that LBP represented the #1 cause overall for total years lived with disability (YLD), and #9 overall as a cause of disability-related life years (DALYs). These numbers increased substantially from 1990 estimates, and with the aging of the population worldwide, the numbers of individuals suffering from LBP are expected to increase further in the future. Although rankings for LBP generally were higher in developed regions, a high burden exists in every part of the world. An area of uncertainty is the degree to which regional differences exist in terms of the specific etiologies of LBP and how these are managed. For example, the most common cause of arachnoiditis in developing countries is a prior spinal infection, but in developed countries the most frequent cause is multiple lumbar spine surgeries.

## TREATMENT OF BACK PAIN

Management is considered separately for acute and chronic low back pain syndromes without radiculopathy, and for back pain with radiculopathy.

## ACUTE LOW BACK PAIN WITHOUT RADICULOPATHY

This is defined as pain of <12 weeks duration. Full recovery can be expected in >85% of adults with ALBP without leg pain. Most have purely “mechanical” symptoms (i.e., pain that is aggravated by motion and relieved by rest).

The initial assessment is focused on excluding 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. CT, MRI, or plain spine films are rarely indicated in the first month of symptoms unless a spine fracture, tumor, or infection is suspected.

The prognosis of ALBP is generally excellent; however, episodes tend to recur, and as many as two-thirds of patients will experience a second episode within 1 year. Most patients do not seek medical care and improve on their own. Even among those seen in primary care, two-thirds report substantial improvement after 7 weeks. This high likelihood of spontaneous improvement can mislead clinicians and patients about the efficacy of treatment interventions, highlighting the importance of rigorous prospective trials. Many treatments commonly used in the past are now known to be ineffective, including bed rest and lumbar traction.

Clinicians should reassure and educate patients that improvement is very likely and instruct them in self-care. Satisfaction and the likelihood of follow-up increase when patients are educated about prognosis, evidence-based treatments, appropriate activity modifications, and strategies to prevent future exacerbations. Counseling patients about the risks of overtreatment is another important part of the discussion. Patients who report that they did not receive an adequate explanation for their symptoms are likely to request further diagnostic tests.

In general, bed rest should be avoided for relief of severe symptoms or limited to a day or two at most. Several randomized trials suggest that bed rest does not hasten the pace of recovery. In general, early resumption of normal daily physical activity should be encouraged, avoiding only strenuous manual labor. Advantages of early ambulation for ALBP also include maintenance of cardiovascular conditioning; improved bone, cartilage, and muscle strength; and increased endorphin levels. Specific back exercises or early vigorous exercise have not shown benefits for acute back pain. Empiric use of heating pads or blankets is sometimes helpful.

### NSAIDs and Acetaminophen

Evidence-based guidelines recommend over-the-counter medicines such as NSAIDs and acetaminophen as first-line options for treatment of ALBP. In otherwise healthy patients, a trial of NSAIDs can be followed by acetaminophen for time-limited periods. In theory, the anti-inflammatory effects of NSAIDs might provide an advantage over acetaminophen to suppress inflammation that accompanies many causes of ALBP, but in practice there is no clinical evidence to support the superiority of NSAIDs. The risk of renal and gastrointestinal toxicity with NSAIDs is increased in patients with preexisting medical comorbidities (e.g., renal insufficiency, cirrhosis, prior gastrointestinal hemorrhage, use of anticoagulants or glucocorticoids, heart failure). Some patients elect to take acetaminophen and an NSAID together in hopes of a more rapid benefit.

### Muscle Relaxants

Skeletal muscle relaxants, such as cyclobenzaprine or methocarbamol, may be useful, but sedation is a common side effect. Limiting the use of muscle relaxants to nighttime only may be an option for patients with back pain that interferes with sleep.

### Opioids

There is no good evidence to support the use of opioid analgesics or tramadol as first-line therapy for ALBP. Their use is best reserved for patients who cannot tolerate acetaminophen or NSAIDs and for those with severe refractory pain. Also, the duration of opioid treatment for ALBP should be strictly limited to 3–7 days. As with muscle relaxants, these drugs are often sedating, so it may be useful to prescribe them at nighttime only. Side effects of short-term opioid use include nausea, constipation, and pruritus; risks of long-term opioid use include hypersensitivity to pain, hypogonadism, and dependency. Falls, fractures, driving accidents, and fecal impaction are other risks. The clinical efficacy of opioids for chronic pain beyond 16 weeks of use is unproven.

Mounting evidence of morbidity from long-term opioid therapy (including overdose, dependency, addiction, falls, fractures, accident risk, and sexual dysfunction) has prompted efforts to reduce its use for chronic pain, including back pain **(Chap. 13)**. When used, safety may be improved with automated notices for high doses, early refills, prescriptions from multiple pharmacies, overlapping opioid and benzodiazepine prescriptions, and in the United States by state-based prescription drug monitoring programs (PDMPs). A recent study indicated that most patients with opioid use disorder presenting to emergency departments had no prescriptions recorded in the PDMP, reflecting other methods used to obtain opioids. Greater access to alternative treatments for chronic pain, such as tailored exercise programs and cognitive behavioral therapy (CBT), may also reduce opioid prescribing.

### Other Approaches

There is no evidence to support use of oral or injected glucocorticoids, antiepileptics, antidepressants, or therapies for neuropathic pain such as gabapentin or herbal therapies. Commonly used nonpharmacologic treatments for ALBP are also of unproven benefit, including spinal manipulation, physical therapy, massage, acupuncture, laser therapy, therapeutic ultrasound, corsets, transcutaneous electrical nerve stimulation (TENS), special mattresses, or lumbar traction. Although important for chronic pain, use of back exercises for ALBP are generally not supported by clinical evidence. There is no convincing evidence regarding the value of ice or heat applications for ABLP; however, many patients report temporary symptomatic relief from ice or frozen gel packs just before sleep, and heat may produce a short-term reduction in pain after the first week. A recent randomized study suggested that a multidisciplinary biopsychosocial intervention or an individualized postural therapy approach each led to a small but significant reduction of pain at 3 months. Patients often report improved satisfaction with the care that they receive when they actively participate in the selection of symptomatic approaches.

## CHRONIC LOW BACK PAIN WITHOUT RADICULOPATHY

Back pain is considered chronic when the symptoms last >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 ALBP can be useful for patients with CLBP. In this setting, however, the benefit of opioid therapy or muscle relaxants is less clear. In general, improved activity tolerance is the primary goal, while pain relief is secondary.

Some observers have raised concerns that CLBP may often be overtreated. For CLBP without radiculopathy, multiple guidelines explicitly recommend against use of SSRIs, any type of injection, TENS, lumbar supports, traction, radiofrequency facet joint denervation, intradiskal electrothermal therapy, or intradiskal radiofrequency thermocoagulation. On the other hand, exercise therapy and treatment of depression appear to be useful and underused.

### Exercise Programs

Evidence supports the use of exercise therapy to alleviate pain symptoms and improve function. Exercise can be one of the mainstays of treatment for CLBP. Effective regimens have generally included a combination of core-strengthening exercises, stretching, and gradually increasing aerobic exercise. A program of supervised exercise can improve compliance. 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.

Intensive multidisciplinary rehabilitation programs can include daily or frequent physical therapy, exercise, CBT, a workplace evaluation, and other interventions. For patients who have not responded to other approaches, such programs appear to offer some benefit. Systematic reviews, however, suggest that the evidence and benefits are limited.

### Nonopioid Medications

Medications for CLBP may include short courses of NSAIDs or acetaminophen. Duloxetine is approved for the treatment of CLBP (60 mg daily) and may also treat coincident depression. Tricyclic antidepressants can provide modest pain relief for some patients without evidence of depression. Depression is common among patients with chronic pain and should be appropriately treated.

### Cognitive Behavioral Therapy

CBT is based on evidence that psychological and social factors, as well as somatic pathology, are important in the genesis of chronic pain and disability; CBT focuses on efforts to identify and modify patients’ thinking about their condition. In one randomized trial, CBT reduced disability and pain in patients with CLBP. Such behavioral treatments appear to provide benefits similar in magnitude to exercise therapy.

### Complementary Medicine

Back pain is the most frequent reason for seeking complementary and alternative treatments. Spinal manipulation or massage therapy may provide short-term relief, but long-term benefit is unproven. Biofeedback has not been studied rigorously. There is no convincing evidence that either TENS,

laser therapy, or ultrasound are effective in treating CLBP. Rigorous 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 provided even by sham acupuncture is uncertain.

### Injections and Other Interventions

Various injections, including epidural glucocorticoid injections, facet joint injections, and trigger point injections, have been used for treating CLBP. However, in the absence of radiculopathy, there is no clear evidence that these approaches are sustainably effective.

Injection studies are sometimes used diagnostically to help determine the anatomic source of back pain. Pain relief following a glucocorticoid and anesthetic injection into a facet or medial branch block are used as evidence that the facet joint is the pain source; however, the possibility that the response was a placebo effect or due to systemic absorption of the glucocorticoids is difficult to exclude.

Another category of intervention for CLBP is electrothermal and radiofrequency therapy. Intradiskal therapy has been proposed using energy to thermocoagulate and destroy nerves in the intervertebral disk, using specially designed catheters or electrodes. Current evidence does not support the use of discography to identify a specific disk as the pain source, or the use of intradiskal electrothermal or radiofrequency therapy for CLBP.

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 disk (ramus communicans), and radicular back pain (dorsal root ganglia). These interventional therapies have not been studied in sufficient detail to draw firm conclusions regarding their value for CLBP.

### Surgery

Surgical intervention for CLBP without radiculopathy has been evaluated in a number of randomized trials. The case for fusion surgery for CLBP without radiculopathy is weak. While some studies have shown modest benefit, there has been no benefit when compared to an active medical treatment arm, often including highly structured, rigorous rehabilitation combined with CBT. The use of bone matrix protein (BMP) instead of iliac crest graft for the fusion was shown to increase hospital costs and length of stay but not improve clinical outcomes.

Guidelines suggest that referral for an opinion on spinal fusion can be considered for patients 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 high cost, wide geographic variations, and rapidly increasing rates of spinal fusion surgery have prompted scrutiny regarding the lack of standardization of appropriate indications. Some insurance carriers have begun to limit coverage for the most controversial indications, such as LBP without radiculopathy.

Lumbar disk replacement with prosthetic disks is US Food and Drug Administration–approved for uncomplicated patients needing single-level surgery at the L3-S1 levels. The disks are generally designed as metal plates with a polyethylene cushion sandwiched in between. The trials that led to approval of these devices were not blinded. When compared to spinal fusion, the artificial disks were “not inferior.” Long-term follow-up is needed to determine device failure rates over time. Serious complications are somewhat more likely with the artificial disk. This treatment remains controversial for CLBP.

## LOW BACK PAIN WITH RADICULOPATHY

A common cause of back pain with radiculopathy is a herniated disk affecting the nerve root and producing back pain with radiation down the leg. The term *sciatica* is used when the leg pain radiates posteriorly in a sciatic or L5/S1 distribution. The prognosis for acute low back and leg pain with radiculopathy due to disk herniation is generally favorable, with most patients showing substantial improvement over months. Serial imaging studies suggest spontaneous regression of the herniated portion of the disk in two-thirds of patients over 6 months. Nonetheless, several important treatment options provide symptomatic relief while the healing process unfolds.

Resumption of normal activity is recommended. Randomized trial evidence suggests that bed rest is ineffective for treating sciatica as well as back pain alone. Acetaminophen and NSAIDs are useful for pain relief, although severe pain may require short courses (3–7 days) of opioid analgesics. Opioids are superior for acute pain relief in the emergency department.

Epidural glucocorticoid injections have a role in providing symptom relief for acute lumbar radiculopathy due to a herniated disk, but do not reduce

the use of subsequent surgical intervention. A brief course of high-dose oral glucocorticoids (methylprednisolone dose pack) for 3 days followed by a rapid taper over 4 more days can be helpful for some patients with acute disk-related radiculopathy, although this specific regimen has not been studied rigorously.

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 effects of systemic absorption.

Urgent surgery is recommended for patients who have evidence of CES or spinal cord compression, generally manifesting as combinations of bowel or bladder dysfunction, diminished sensation in a saddle distribution, a sensory level on the trunk, and bilateral leg weakness or spasticity. Surgical intervention is also indicated for patients with progressive motor weakness due to nerve root injury demonstrated on clinical examination or EMG.

Surgery is also an important option for patients who have disabling radicular pain despite optimal conservative treatment. Because patients with a herniated disk and sciatica generally experience rapid improvement over weeks, most experts do not recommend considering surgery unless the patient has failed to respond to a minimum of 6–8 weeks of nonsurgical management. For patients who have not improved, randomized trials show that surgery results in more rapid pain relief than nonsurgical treatment. However, after 2 years of follow-up, patients appear to have similar pain relief and functional improvement with or without surgery. Thus, both treatment approaches are reasonable, and patient preferences and needs (e.g., rapid return to employment) strongly influence decision-making. Some patients will want the fastest possible relief and find surgical risks acceptable. Others will be more risk-averse and more tolerant of symptoms and will choose watchful waiting, especially if they understand that improvement is likely in the end.

The usual surgical procedure is a partial hemilaminectomy with excision of the prolapsed disk (diskectomy). Minimally invasive techniques have gained in popularity in recent years, but some evidence suggests they may be less effective than standard surgical techniques, with more residual back pain, leg pain, and higher rates of rehospitalization. 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 have increased dramatically in recent years. There are no large prospective, randomized trials comparing fusion to other types of surgical intervention. In one study, patients with persistent LBP despite an initial diskectomy fared no better with spine fusion than with a conservative regimen of cognitive intervention and exercise. Artificial disks, as discussed above, are used in Europe; their utility remains controversial in the United States. The practice of placing an epidural implantable pulse generator to deliver spinal cord burst stimulation post-operatively was demonstrated to not improve back pain-related disability.

## PAIN IN THE NECK AND SHOULDER

Neck pain, which usually arises from diseases of the cervical spine and soft tissues of the neck, is common, typically precipitated by movement, and may be accompanied by focal tenderness and limitation of motion. Many of the earlier comments made regarding causes of LBP also apply to disorders of the cervical spine. The text below will emphasize differences. Pain arising from the brachial plexus, shoulder, or peripheral nerves can be confused with cervical spine disease **(Table 17-4)**, but the history and examination usually identify a more distal origin for the pain. When the site of nerve tissue injury is unclear, EMG studies can localize the lesion. Cervical spine trauma, disk disease, or spondylosis with intervertebral foraminal narrowing may be asymptomatic or painful and can produce a myelopathy, radiculopathy, or both. The same risk factors for serious causes of LBP also apply to neck pain with the additional feature 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 involvement of the cervical spinal cord.

TABLE 17-4

**Cervical Radiculopathy: Neurologic Features**

| CERVICAL NERVE ROOT | EXAMINATION FINDINGS | | | PAIN DISTRIBUTION |
|---|---|---|---|---|
| | REFLEX | SENSORY | MOTOR | |
| C5 | Biceps | Lateral deltoid | Rhomboids[a] (elbow extends backward with hand on hip) | Lateral arm, medial scapula |
| | | | Infraspinatus[a] (arm rotates externally with elbow flexed at the side) | |
| | | | Deltoid[a] (arm raised laterally 30°–45° from the side) | |
| C6 | Biceps | Palmar thumb/index finger | Biceps[a] (arm flexed at the elbow in supination) | Lateral forearm, thumb/index fingers |
| | | Dorsal hand/lateral forearm | Pronator teres (forearm pronated) | |
| C7 | Triceps | Middle finger | Triceps[a] (forearm extension, flexed at elbow) | Posterior arm, dorsal forearm, dorsal hand |
| | | Dorsal forearm | Wrist/finger extensors[a] | |
| C8 | Finger flexors | Palmar surface of little finger | Abductor pollicis brevis (abduction of thumb) | Fourth and fifth fingers, medial hand and forearm |
| | | Medial hand and forearm | First dorsal interosseous (abduction of index finger) | |
| | | | Abductor digiti minimi (abduction of little finger) | |
| T1 | Finger flexors | Axilla, medial arm, anteromedial forearm | Abductor pollicis brevis (abduction of thumb) | Medial arm, axilla |
| | | | First dorsal interosseous (abduction of index finger) | |
| | | | Abductor digiti minimi (abduction of little finger) | |

[a]These muscles receive the majority of innervation from this root.

## TRAUMA TO THE CERVICAL SPINE

Trauma (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. 442)**. Immediate immobilization of the neck is essential to minimize further spinal cord injury from movement of

unstable cervical spine segments. A CT scan is the diagnostic procedure of choice for detection of acute fractures following severe trauma; plain x-rays are used for lesser degrees of trauma or in settings where CT is unavailable. When traumatic injury to the vertebral arteries or cervical spinal cord is suspected, visualization by MRI with magnetic resonance angiography is preferred.

The decision to obtain imaging should be based on the clinical context of the injury. The National Emergency X-Radiography Utilization Study (NEXUS) low-risk criteria established that normally alert patients without palpation tenderness in the midline; intoxication; neurologic deficits; or painful distracting injuries were very unlikely to have sustained a clinically significant traumatic injury to the cervical spine. The Canadian C-spine rule recommends that imaging should be obtained following neck region trauma if the patient is >65 years old or has limb paresthesias or if there was a dangerous mechanism for the injury (e.g., bicycle collision with tree or parked car, fall from height >3 ft or five stairs, diving accident). These guidelines are helpful but must be tailored to individual circumstances; for example, patients with advanced osteoporosis, glucocorticoid use, or cancer may warrant imaging after even mild trauma.

*Whiplash injury* is due to rapid flexion and extension of the neck, usually from automobile accidents. The likely mechanism involves injury to the facet joints. 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 1 year later. When personal compensation for pain and suffering was removed from the Australian health care system, the prognosis for recovery at 1 year improved. 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

Degenerative cervical disk disease is very common and usually asymptomatic. Herniation of a lower cervical disk is a common cause of pain or tingling in the neck, shoulder, arm, or hand. Neck pain, stiffness, and a range of motion limited by pain are the usual manifestations. Herniated cervical disks are responsible for ~25% of cervical radiculopathies. Extension and lateral rotation of the neck narrow the ipsilateral intervertebral foramen and may reproduce radicular symptoms (Spurling's sign). In young adults, 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 cervical nerve root lesions are summarized in Table 17-4. Although the classic patterns are clinically helpful, there are numerous exceptions because (1) there is overlap in sensory function between adjacent nerve roots, (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; these causes together account 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 and produce signs of myelopathy alone or radiculopathy with myelopathy (myeloradiculopathy). When little or no neck pain accompanies cervical cord involvement, other diagnoses to be considered include amyotrophic lateral sclerosis **(Chap. 437)**, multiple sclerosis **(Chap. 444)**, spinal cord tumors, or syringomyelia **(Chap. 442)**. Cervical spondylotic myelopathy should be considered even when the patient presents with symptoms or spinal cord signs in the legs only. MRI is the study of choice to define soft tissues in the cervical region including the spinal cord, whereas plain CT is optimal to identify bone pathology including foraminal, lateral recess, OPLL, or spinal canal stenosis. In spondylotic myelopathy, focal enhancement by MRI, sometimes in a characteristic "pancake pattern," may be present at the site of maximal cord compression.

There is no evidence to support prophylactic surgery for asymptomatic cervical spinal stenosis unaccompanied by myelopathic signs or abnormal spinal cord findings on MRI, except in the setting of *dynamic instability* (see spondylolisthesis above). If the patient has postural neck pain, a prior history of whiplash or other spine/head injury, a Lhermitte sign, or preexisting listhesis at the stenotic segment on cervical MRI or CT, then cervical spine flexion-extension x-rays or MRI are indicated to look for dynamic instability. Surgical intervention is not recommended for patients with listhesis alone, unaccompanied by dynamic instability.

## OTHER CAUSES OF NECK PAIN

Rheumatoid arthritis (RA) **(Chap. 358)** of the cervical facet joints produces neck pain, stiffness, and limitation of motion. Synovitis of the atlantoaxial joint (C1-C2; Fig. 17-2) may damage the transverse ligament of the atlas, producing forward displacement of the atlas on the axis (atlantoaxial subluxation). Radiologic evidence of atlantoaxial subluxation occurs in up to 30% of patients with RA and plain x-ray films of the neck should be routinely performed preoperatively to assess the risk of neck hyperextension in patients requiring intubation. The degree of subluxation correlates with the severity of erosive disease. When subluxation is present, careful assessment is important to identify early signs of myelopathy that could be a harbinger of life-threatening spinal cord compression. Surgery should be considered when myelopathy or spinal instability is present. *Ankylosing spondylitis* is another cause of neck pain and less commonly atlantoaxial subluxation.

Acute *herpes zoster* can present 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, as discussed above. Neck pain may also be referred from the heart with coronary artery ischemia (cervical angina syndrome). Rheumatologic disease should be considered if the neck pain is accompanied by shoulder or hip girdle pain.

## THORACIC OUTLET SYNDROMES

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, classified as follows.

*True neurogenic thoracic outlet syndrome* (*TOS*) is an uncommon disorder resulting from compression of the lower trunk of the brachial plexus or ventral rami of the C8 or T1 nerve roots, caused most often by an anomalous band of cartilaginous tissue connecting an elongate transverse process at C7 with the first rib. Pain is mild or may be absent. Signs include weakness and wasting of intrinsic muscles of the hand and diminished sensation on the palmar aspect of the fifth digit. An anteroposterior cervical spine x-ray will show an elongate C7 transverse process (an anatomic marker for the anomalous cartilaginous band), and EMG and NCSs confirm the diagnosis. Treatment consists of surgical resection of the anomalous band. The weakness and wasting of intrinsic hand muscles typically do 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 in some cases secondary 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 vein 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.

*Disputed TOS* accounts for 95% of patients diagnosed with TOS; chronic arm and shoulder pain are prominent and of unclear cause. The lack of sensitive and specific findings on physical examination or specific markers for this condition results in diagnostic uncertainty. The role of surgery in disputed TOS is controversial. Major depression, chronic symptoms, work-related injury, and diffuse arm symptoms predict poor surgical outcomes. Multidisciplinary pain management is a conservative approach, although treatment is often unsuccessful.

## BRACHIAL PLEXUS AND NERVES

Pain from injury to the brachial plexus or peripheral nerves of the arm can occasionally mimic referred pain of cervical spine origin, including cervical radiculopathy, but the pain typically begins distal to the posterior neck region in the shoulder girdle or upper arm. 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 lower trunk and medial cord of the brachial plexus. Delayed radiation injury may produce weakness in the upper arm or numbness of the lateral forearm or arm due to involvement of the upper trunk and lateral cord of the plexus. Pain is less common and less severe than with neoplastic infiltration. A Pancoast tumor of the lung **(Chap. 78)** is another cause and should be considered, especially when a concurrent Horner's syndrome is present. *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 may be preceded by an infection, vaccination, or minor surgical procedure. The long thoracic nerve may be affected, resulting in a winged scapula. Brachial neuritis may also present as an isolated paralysis of the diaphragm with or without involvement of other nerves of the upper limb. Recovery may take up to 3 years, and full functional recovery can be expected in the majority of patients.

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 also mimic radiculopathy, at C7 or C8, respectively. EMG and NCSs can accurately localize lesions to the nerve roots, brachial plexus, or peripheral nerves.

**For further discussion of peripheral nerve disorders, see Chap. 446.**

## 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 (bicipital tendonitis, frozen shoulder, bursitis, rotator cuff tear, dislocation, adhesive capsulitis, or rotator 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 passive abduction, internal rotation, or extension of the arm. Demonstrating normal passive full range of motion of the arm at the shoulder without worsening the usual pain can help exclude mechanical shoulder pathology as a cause of neck region pain. Pain from shoulder disease may radiate into the arm or hand, but focal neurologic signs (sensory, motor, or reflex changes) are absent.

## GLOBAL CONSIDERATIONS

Many of the considerations described above for LBP also apply to neck pain. The Global Burden of Diseases Study 2019 reported that neck pain ranked second only to back pain as a cause of total years lived with disability (YLD). In general, neck pain rankings were also higher in developed regions of the world.

## TREATMENT OF NECK PAIN WITHOUT RADICULOPATHY

The evidence regarding treatment for neck pain is less comprehensive than that for LBP, but the approach is remarkably similar in many respects. As with LBP, spontaneous improvement is the norm for acute neck pain. The usual goals of therapy are to promote a rapid return to normal function and provide pain relief while healing proceeds.

Acute neck pain is often treated with NSAIDs, acetaminophen, cold packs, or heat, alone or in combination while awaiting recovery. Patients should be specifically educated regarding the favorable natural history of acute neck pain to avoid unrealistic fear and inappropriate requests for imaging and other tests. For patients kept awake by symptoms, cyclobenzaprine (5–10 mg) at night can help relieve muscle spasm and promote drowsiness. For patients with neck pain unassociated with trauma, supervised exercise with or without mobilization appears to be effective. Exercises often include shoulder rolls and neck stretches. The evidence in support of nonsurgical treatments for whiplash-associated disorders is generally of limited quality and neither supports nor refutes the common treatments used for symptom relief. Gentle mobilization of the cervical spine combined with exercise programs may be beneficial. Evidence is insufficient to recommend the use of cervical traction, TENS, ultrasound, trigger point injections, botulinum toxin injections, tricyclic antidepressants, and SSRIs for acute or chronic neck pain. Some patients obtain modest pain relief using a soft neck collar; there is little risk or cost. Massage can produce temporary pain relief.

For patients with chronic neck pain, supervised exercise programs can provide symptom relief and improve function. Acupuncture provided short-term benefit for some patients when compared to a sham procedure and is an option. Spinal manipulation alone has not been shown to be effective and carries a risk for injury. Surgical treatment for chronic neck pain without radiculopathy or spine instability is not recommended.

### Neck Pain With Radiculopathy

The natural history of acute neck pain with radiculopathy due to disk disease is favorable, and many patients will improve without specific therapy. Although there are no randomized trials of NSAIDs for neck pain, a course of NSAIDs, acetaminophen, or both, with or without muscle relaxants, and avoidance of activities that trigger symptoms are reasonable as initial therapy. Gentle supervised exercise and avoidance of inactivity are reasonable as well. A short course of high-dose oral glucocorticoids with a rapid taper, or epidural steroids administered under imaging guidance can be effective for acute or subacute disk-related cervical radicular pain, but have not been subjected to rigorous trials. The risk of injection-related complications is higher in the neck than the low back; vertebral artery dissection, dural puncture, spinal cord injury, and embolism in the vertebral arteries have all been reported. Opioid analgesics can be used in the emergency department and for short courses as an outpatient. Soft cervical collars can be modestly helpful by limiting spontaneous and reflex neck movements that exacerbate pain; hard collars are in general poorly tolerated.

If cervical radiculopathy is due to bony compression from cervical spondylosis with foraminal narrowing, periodic follow-up to assess for progression is indicated and consideration of surgical decompression is reasonable. Surgical treatment can produce rapid pain relief, although it is unclear if long-term functional outcomes are improved over nonsurgical therapy. Indications for cervical disk surgery include a progressive motor deficit due to nerve root compression, functionally limiting pain that fails to respond to conservative management, or spinal cord compression. In other circumstances, clinical improvement over time regardless of therapeutic intervention is common.

Surgical treatments include anterior cervical diskectomy alone, laminectomy with diskectomy, or diskectomy with fusion. 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.

## FURTHER READING

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