Acute lumbosacral radiculopathy: Pathophysiology, clinical features, and diagnosis

Authors: Philip S Hsu, MD, Carmel Armon, MD, MHS, Kerry Levin, MD
Section Editor: Jeremy M Shefner, MD, PhD
Deputy Editor: Richard P Goddeau, Jr, DO, FAHA

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: Mar 2023. | **This topic last updated:** Aug 27, 2021.

INTRODUCTION

Lumbosacral radiculopathy is a condition in which a disease process affects the function of one or more lumbosacral nerve roots [1]. The clinical aspects of lumbosacral radiculopathy will be reviewed here.

The treatment of lumbosacral radiculopathy and other disorders of the lower spine are discussed separately. (See "Acute lumbosacral radiculopathy: Treatment and prognosis" and "Lumbar spinal stenosis: Pathophysiology, clinical features, and diagnosis" and "Evaluation of low back pain in adults" and "Subacute and chronic low back pain: Nonpharmacologic and pharmacologic treatment" and "Subacute and chronic low back pain: Nonsurgical interventional treatment" and "Subacute and chronic low back pain: Surgical treatment".)

ANATOMY

The lumbar spine consists of five movable lumbar vertebral bodies, numbered L1 to L5 (figure 1). The sacrum is made up of five developmentally fused vertebral levels (S1 to S5), followed by a terminal bony prominence, the coccyx. The entire region is commonly described as the lumbosacral spine.

Directly beneath each lumbar and sacral vertebra, there is a pair of neural foramina with the same number designation, such that the L1 neural foramina are located just below the L1

vertebral body. Neural foramina are bounded superiorly and inferiorly by pedicles, anteriorly by the intervertebral disc and vertebral body, and posteriorly by facet joints (figure 1).

Through each neural foramen passes the same numbered spinal nerve root, recurrent meningeal nerves, and radicular blood vessels. On each side there are five lumbar, five sacral, and one coccygeal spinal nerve roots.

All lumbar and sacral spinal nerve roots originate at the T10 to L1 vertebral level, where the spinal cord terminates at the conus medullaris. A dorsal (somatic sensory) root from the posterolateral aspect of the spinal cord and a ventral (somatic motor) root from the anterolateral aspect of the cord join in the spinal canal to form the spinal nerve root (figure 1). The roots then course down through the intraspinal canal, forming the cauda equina, until they exit at their respective neural (intervertebral) foramina. Thus, the lumbosacral nerve roots exit the spinal canal at a lower level than where they arise. A potential consequence of this arrangement is that intraspinal pathology may affect roots at higher levels than the level where the roots exit [2,3].

Cell bodies of the motor nerve fibers are located in the ventral (anterior) horns of the spinal cord, while those of the sensory nerve fibers are in a dorsal root ganglion (DRG) at each lumbar and sacral level. DRG tend to be located within the neural foramina, and are therefore not strictly speaking intraspinal (ie, within the lumbar canal). However, at the low lumbar and sacral levels there is a tendency for DRG to reside proximal to the neural foramina, within the intraspinal canal, as found in 11 to 38 percent of cases at L5 and 71 percent at S1 [4,5]. The DRG are attached to the vertebral body on the transverse process [6]. Compressive radicular disease typically occurs proximal to this.

As noted above, the spinal cord normally terminates at the conus medullaris within the lumbar intraspinal canal between the T10 and L1 vertebral levels. Exceptions include patients with congenital spinal deformities known as spina bifida, in which the fetal conus is tethered to ligamentous or bony structures, causing lengthening of the spinal cord during development. In such patients, the conus medullaris can be displaced downward to the middle or lower lumbar spine.

Rami — Just distal to the neural foramen, the nerve root divides in two, forming the dorsal and ventral primary rami.

- The small dorsal (posterior) primary ramus supplies motor innervation to the paraspinal muscles and cutaneous innervation to the skin of the trunk and back

- The large ventral (anterior) primary ramus supplies motor and sensory innervation to the legs and trunk, including abdominal wall muscles

The dorsal rami of the spinal nerves also supply the apophyseal joints and the paraspinal muscles. They innervate structures both above and below the level of the nerve. Clinical evaluation of injury to the dorsal rami is difficult because of the overlap in areas innervated by these nerves and because of the limited ability to clinically evaluate individual paraspinal muscles. However, electromyography (EMG) can be helpful in determining the distribution of disease.

The ventral rami innervate the extremities and the trunk. These branches can be evaluated by assessing the motor and sensory functions of the different myotomes and dermatomes, respectively. However, variability to the dermatomal and myotomal distribution of innervation exists [7,8].

Myotomes and dermatomes — The collection of muscles with significant innervation from a single root is called a myotome. Similarly, the sensory distribution of a single root is labeled a dermatome.

The primary manifestations of lumbosacral root disease can be broken up into dysfunction of two distinct systems: motor and sensory. Motor dysfunction from a root lesion may cause weakness in some or all muscles innervated by that root. However, many muscles have innervation from multiple roots, which may result in preserved strength despite significant involvement of a single root.

Although there are classic descriptions for the distributions of myotomes and dermatomes, substantial variability exists in these distributions from person to person [7,8]. Sensory fields have considerable overlap, but there are areas that are exclusively served by individual nerves. These areas are called autonomous zones. The most important of these in the evaluation of lumbosacral radiculopathies include the sole of the foot (S1), dorsum of the foot (L5), medial calf (L4), and anterior thigh (L2 and 3) [9].

Lumbosacral myotomes are listed in the table (table 1).

Sinuvertebral nerves — The sinuvertebral nerves are sensory nerves that innervate various structures within the spine, such as ligamentous structures, the dura, periosteum, and blood vessels [10]. They originate distal to the DRG and extend to communicate with branches from radicular levels both above and below the level of entry, as well as the contralateral side, making it difficult to localize pain from involvement of these nerves.

Irritation of the sinuvertebral nerves may result in low back pain. Because they arise distal to the nerve root, however, involvement of sinuvertebral nerves or their branches without involvement of the rest of the nerve root is not considered to be radicular in nature.

PATHOPHYSIOLOGY AND ETIOLOGY

The most common etiology of lumbosacral radiculopathy is nerve root compression caused by a disc herniation or spondylosis, which is narrowing of the intraspinal (central) canal, the lateral recess, or the neural foramen due to degenerative arthritis affecting the spine. The lateral recess is the most lateral space in the lumbar canal's triangular configuration, just proximal to the intervertebral foramen. It is bounded laterally by the pedicle, posteriorly by the superior articular facet and ligamentum flavum, and anteriorly by the vertebral body, endplate edge, or disk edge.

Additional etiologies include nonskeletal causes of nerve root compression and noncompressive mechanisms such as infection, inflammation, neoplasm, and vascular disease.

Pain generators in the lumbosacral spine — A number of tissues making up the low back contain nerve fibers with pain receptors. These are listed in the table (table 2). Of note, intervertebral disc material does not contain significant numbers of pain fibers.

- Classic radiating pain from spinal nerve injury is mediated through proximal spinal nerves. Compression of a spinal nerve root by disc or arthritic spur leads to local edema, ischemia, and inflammation. These factors contribute to production of pain impulses through the spinal nerves. The pain from acute disc herniation or spondylotic spinal nerve entrapment reflects a combination of pain generation within the nerve root itself, as well as the pain from neighboring tissues whose pain fibers are activated by the effect of disc herniation on dura, ligaments, and surrounding vasculature.

- Localized lumbosacral pain is thought to arise from intraspinal structures. These pain impulses arise from the blood vessels, dura mater, and longitudinal ligaments, and travel in the sinuvertebral nerves through the neural foramina, connecting via rami communicantes with the extraspinal sympathetic chain.

- Nonlocalized, nonradiating pain is thought to arise from muscle, bone, and ligament outside the spinal canal. Interconnected ventral and dorsal nerve plexuses surround the vertebral column [11]. The ventral nerve plexus serves the anterior longitudinal ligament and has bilateral innervation. Many branches from the sympathetic trunk, rami communicantes, and perivascular nerve plexuses join to form the ventral nerve plexus.

The dorsal nerve plexus arises from the sinuvertebral nerves and serves the posterior longitudinal ligament.

- Referred spine pain may arise from the abdominal viscera that share the same spinal level of innervation. Organs that can potentially refer pain to the spine include the aorta, pancreas, duodenum, colon, rectum, kidney, ureter, bladder, and pelvic organs. Systemic illness can refer pain to the bony spine or can produce bony disease that generates spine pain. The major categories and clues to their diagnosis are listed in the table (table 3).

Degenerative changes — Damage to spinal nerve roots occurs as the result of degenerative change involving three main structures, which are:

- The intervertebral discs
- The uncovertebral joints (cervical spine only)
- The zygapophyseal (facet) joints

Resulting bony overgrowth (osteophytes) or disc herniation at these points may directly impinge on spinal nerve roots or the spinal cord, or their effect may be primarily to produce instability and misalignment of the spine (ie, degenerative spondylolisthesis) that in turn produces pain and neurologic deficits (figure 2). It is not known whether changes in these different structures are causally related or occur independently.

Degenerative spondylotic changes are common with aging and usually do not result in radiculopathy. Low back pain associated with degenerative changes is distinct from radicular pain and is much more frequent. Given the inability to ascertain the exact cause, it is commonly referred to as nonspecific low back pain.

One school of thought suggests that degenerative spondylotic change is led by age-related change in the nucleus pulposus of the disc. With age there is gradual narrowing of the disc space coincident with changes in disc proteoglycan composition. Later, cracks develop in the disc, and deposits of gas and calcification may form. Eventually the disc material becomes desiccated and friable.

- A disc bulge involves >90 degrees of the total circumference of a disc and does not extend beyond the boundaries of the annulus fibrosus.

- A disc herniation is a rupture of disc material beyond the annulus fibrosus. There a two broad categories, protrusion and extrusion. Protrusion refers to a rupture of disc material in which the base is broader than the dome. In general, this does not extend above or

below the disc space. An extrusion is a disc rupture in which the dome is wider than the base, with a dumbbell appearance. Extrusions may extend above or below the disc space.

- A free fragment is a displacement of disc material that loses its connection with the main disc material. Such fragments can migrate from the disc space.

Age-related changes also occur in the annulus fibrosus (figure 1), which becomes more fibrotic and less elastic. Fissures develop and calcium is deposited. As the disc shrinks and the intervertebral disc space narrows, the annulus tends to buckle out. A tear or rupture of the annulus fibrosus is a degenerative loss of integrity of the annular ring surrounding the disc material involving single or multiple layers without disc displacement through the ring. These are not usually apparent on routine magnetic resonance imaging (MRI) studies.

Accompanying this disc degeneration are changes at the vertebral body endplates adjacent to the disc. The marrow undergoes fibrovascular change or fatty marrow replacement. Finally, endplate sclerosis develops. Osteophyte formation occurs at the margins of the vertebral bodies. What triggers osteophyte formation is unclear, although spinal movement at ligamentous attachment sites and loss of buffering tissues between bony surfaces likely play roles. Osteophyte production appears to slow as advancing spondylosis leads to decreasing spinal movement.

Facet joint degeneration may not be directly related to spondylotic changes, but often coexists. Disc degeneration is likely to put additional weight-bearing strains on facet joints, which are not weight-bearing structures. With unnatural movement of the spine, the synovial joint bears more structural burden, degenerates, and develops osteophytes. These osteophytes grow into the posterior aspect of the neural foramen (image 1).

Disc protrusion and level of injury — Disc protrusion can give rise to different anatomic levels of nerve root compression depending on the orientation of spinal nerve roots as they exit from the spinal cord. As noted earlier, all lumbar and sacral spinal nerve roots are constituted at the T12-L1 vertebral level, where the spinal cord ends as the conus medullaris. The roots then course down the canal as the cauda equina, until they exit at their respective neural foramina.

Depending upon the nature and location of intraspinal compression, roots may be injured at any disc level, from the L1-2 level to the level of their exit into their neural foramina. For example, the L5 root can be compressed by a central disc protrusion at the L2-3 or L3-4 level, a posterolateral (paracentral) disc protrusion at the L4-5 level (image 2), or far lateral disc protrusion into the foramen at the L5-S1 level (image 3). Because of the presence of multiple spinal nerve roots in the cauda equina, there is increased likelihood of multiple, bilateral simultaneous nerve root compressions.

Nerve root injury may be complete or only partial and thus may involve all or only a subset of root fibers. With partial injury, incomplete myotomal involvement may result, making the distinction between a radiculopathy and a peripheral nerve injury more challenging.

The lumbosacral spine is susceptible to disc herniations because of its mobility from flexion, extension, and torsion. Seventy-five percent of flexion and extension occurs at the lumbosacral joint [12]. This level, on the other hand, has limited torsion. Twenty percent of flexion and extension occurs at L4-L5. The remaining 5 percent occurs between L1 and L3 [12]. As the L4-L5 and L5-S1 levels are most susceptible to injuries from routine movements of the spine, approximately 90 to 95 percent of compressive radiculopathies occur at these levels [13]. The incidence of radiculopathies is split somewhat evenly between L4-L5 and L5-S1, as the lack of torsion at L5-S1 helps to increase its stability despite its higher degree of flexion and extension [14]. Next in frequency is L4. Other levels are uncommon.

Far lateral herniations are seen more often at the L2-4 levels. They may affect the rostral root. This is rare, as only 10 percent of far lateral herniations will result in nerve root compression [5]. Pain from the far lateral disc herniations may be more severe due to compression of the dorsal root ganglia (DRG) [15].

Other skeletal causes — Congenital abnormalities of the bony spinal column or its contents occur in 25 to 50 percent of the general population [16]. Congenital narrowing of the canal is not uncommon as a substrate upon which spondylosis may result in neurologic disease; this is especially true of younger adults. Other developmental abnormalities include tethered cord or diastematomyelia and spina bifida. They may lead to radicular dysfunction due to injuries resulting from traction of the root.

Root avulsion is a rare cause of lumbosacral radiculopathy that may occur with fractures of the sacroiliac joint or with diastasis of the symphysis pubis or the pubic rami [17].

Nonskeletal causes — Infection, inflammation, neoplasm, and vascular disease are less common causes of radiculopathy and polyradiculopathy (table 4) [18-21]. Specific etiologies include the following:

- Diabetes mellitus, a common cause of peripheral neuropathy and polyneuropathy
- Infectious diseases
  - *Borrelia burgdorferi*
  - Cytomegalovirus
  - Epidural abscess

- Epstein-Barr virus
- Herpes simplex virus
- Human immunodeficiency virus (HIV)
- Mycobacterium
- Mycoplasma
- Syphilis
- Varicella zoster virus (herpes zoster or shingles)

- Inflammatory conditions
  - Acute inflammatory demyelinating polyradiculoneuropathy (AIDP; Guillain-Barré syndrome)
  - Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)
  - Arachnoiditis related to postsurgical changes
  - Chemical radiculitis
  - Sarcoidosis

- Mass lesion or malignancy
  - Metastasis (most common)
  - Epidural abscess
  - Intradural tumor, particularly meningioma, neurofibroma, and ependymoma [18]
  - Lymphoma
  - Myeloma
  - Root sleeve cyst, such as a Tarlov or perineural cyst (though most are asymptomatic and found incidentally on MRI) [22-25]
  - Epidural lipomatosis [26]

- Vascular
  - Arteriovenous malformation
  - Vasculitis (nerve root infarction)
  - Radiation-induced vascular occlusion

There is generally complete involvement of the root with radiculitis caused by leptomeningeal carcinomatosis, cytomegalovirus, or herpes zoster.

With leptomeningeal carcinomatosis, the source may be metastases from breast cancer, lung cancer and melanoma, non-Hodgkin lymphoma, or leukemia [20]. Nerve roots may be encased by tumor cells causing compression and ischemia.

Cytomegalovirus radiculitis occurs in immunosuppressed patients with very low CD4 counts. It is characterized by severe paresthesia and sensory loss and weakness. Both small and large nerve fibers are involved. There may also be bowel and bladder dysfunction from diminished sphincter tone. Cerebrospinal fluid (CSF) analysis may reveal increased protein, decreased glucose, and a polymorphonuclear pleocytosis. Polymerase chain reaction may be positive for cytomegalovirus [19].

Herpes zoster colonizes the DRG and may remain latent for years. Reactivation is associated with a hemorrhagic lymphocytic infiltration of the ventral roots. Patients present with an erythematous vesicular maculopapular rash in the dermatome of the affected root that lasts for three to five days. Sensory changes characterized by marked allodynia often follow [27]. When the motor nerve root is involved (so-called segmental zoster) a clinical picture of radiculopathy with weakness occurs in affected myotomes.

Root infarction may occur from either large-vessel or small-vessel disease. It is most commonly seen in diabetics where the microvasculature is affected. Such a process may be one of the causes of the clinical syndrome known as diabetic amyotrophy [28]. Diabetic amyotrophy involves the L2, L3, and L4 roots and presents with weakness, pain, and dysesthesia primarily in the proximal leg that evolves over days to weeks. This condition is discussed separately. (See "Diabetic amyotrophy and idiopathic lumbosacral radiculoplexus neuropathy".)

Radiculopathy may also arise in the absence of obvious compression, leading to the hypothesis that the injury may be due to a chemical radiculitis, perhaps caused by the rupture of the annulus fibrosus with subsequent release of inflammatory mediators tracking along the nerve root sheath [29-31]. Electromyography (EMG) may be abnormal even though compression is not observed on imaging studies.

Fiber size — Like most peripheral nerves, lumbosacral nerve roots are composed of both large and small fibers. Different fiber types play different roles by virtue of the information they carry. Injury to these fibers may result in focal demyelination as seen initially with compression, or axonal damage as seen with infarction or more severe compressive injury.

Large fibers carry motor efferent and sensory information involved with vibratory sense and conscious and unconscious proprioception. The typical clinical findings of large-fiber involvement in radiculopathies are weakness and reflex abnormalities. Deep tendon reflexes may be diminished or lost. Clinical testing for proprioception and vibratory sense tends to extend across different dermatomes and thus is often not as helpful in the evaluation of lumbosacral radiculopathies as small-fiber modalities, as discussed below.

The smaller myelinated A-delta fibers and unmyelinated C fibers carry pain and temperature information. A-delta fibers transmit cold sensation, while C fibers subserve warm sensation. Isolated injury to these fibers is not common in lumbosacral radiculopathies. However, their dermatomal distributions tend to be more specific than those of the large fibers, increasing their importance in the clinical evaluation of radiculopathies.

In addition, dysfunction of A-delta and C fibers even in the absence of overt nerve root compression may play a significant role in the symptomatology of radiculopathies. A study of warm and cold sensory thresholds in 32 patients with unilateral L5 or S1 radiculopathy and disc herniation found that warm thresholds (C fiber function) were impaired to a greater extent than cold thresholds (A-delta fibers) [32]. Since it is thought that unmyelinated axons, such as C fibers, are less affected by compression than myelinated ones, this finding suggests that the generation of lumbar radicular pain is mediated by inflammation more than by nerve root compression [32].

Central pain sensitization may result from nerve root compression. Supporting evidence comes from a small study of patients with L5 or S1 radiculopathy that demonstrated a lowered pain threshold in the contralateral root, perhaps mediated by pre- and postsynaptic modulation of opioidergic interneurons [33].

EPIDEMIOLOGY

Lumbosacral radiculopathy is one of the most common problems seen in neurologic consultation. Although data are limited, the estimated lifetime prevalence is approximately 3 to 5 percent for adults, with equal rates among males and females [1].

CLINICAL PRESENTATIONS

The clinical presentations of lumbosacral radiculopathy vary according to the level of nerve root or roots involved. The most frequent are the L5 and S1 radiculopathies. All lumbosacral nerve roots exit the spinal canal at the neural foramina below their respective vertebrae and are most commonly compressed as they descend in the lateral recess **above** the exiting level. As an example, the L5 nerve roots exit via the neural foramina at the L5/S1 disc space level but are usually compressed by posterolateral (paracentral) disc herniation of the L4/L5 disc. (See 'Disc protrusion and level of injury' above.)

L1 radiculopathy — Lumbar disc herniation at the L1 level is rare, and thus L1 radiculopathy is uncommon. Symptoms on presentation generally involve pain, paresthesia, and sensory loss in

the inguinal region [1]. Rarely, minor hip flexion weakness is present.

L2/L3/L4 radiculopathy — There is marked overlap of the L2, L3, and L4 innervation of the anterior thigh muscles, making it difficult to differentiate these spinal nerve root levels based on symptoms, neurologic examination, or electrodiagnostic testing. Thus, these radiculopathies are generally considered as a group. These nerve roots are most commonly involved in older patients with symptoms of spinal stenosis.

Acute back pain is the most common presenting complaint, often radiating around the anterior aspect of the thigh down into the knee and occasionally down the medial aspect of the lower leg as far as the arch of the foot. On examination, there may be weakness of hip flexion, knee extension, and hip adduction. Higher lesions may result in greater weakness of the hip flexors. Sensation may be reduced over the anterior thigh down to the medial aspect of the lower leg. A reduced knee reflex is common in the presence of moderate weakness.

Electromyography (EMG) and nerve conduction studies (NCS) may reveal abnormalities confined to muscles of the affected root(s), including the quadriceps, leg adductors, and iliopsoas, with associated paraspinal abnormalities. Saphenous sensory response remains normal even if sensory loss is prominent in the distal leg.

L5 radiculopathy — L5 radiculopathy is the most common radiculopathy affecting the lumbosacral spine. It often presents with back pain that radiates down the lateral aspect of the leg into the foot. On examination, strength can be reduced in foot dorsiflexion, toe extension, foot inversion, and foot eversion. Weakness of leg abduction may also be evident in severe cases due to involvement of gluteus minimus and medius. Atrophy may be present in the extensor digitorum brevis muscle of the foot and the tibialis anterior muscle of the lower leg. In severe cases, there may be "tibial ridging," a condition in which the normal convexity of the anterior compartment of the leg is lost because of atrophy, leaving a prominent sharp contour of the medial aspect of the tibial bone.

Sensory loss is confined to the lateral aspect of the lower leg and dorsum of the foot, but may be obvious only when testing sharp sensation in the web space between the first and second digits. Reflexes are generally normal, although the internal hamstring reflex may be diminished on the symptomatic side.

EMG and NCS typically show abnormalities in the L5 muscles, including the gluteus medius, tensor fascia lata, semitendinosus, tibialis anterior, tibialis posterior, and the L5 paraspinals. Sensory studies (sural and superficial peroneal responses) are normal since the lesion is almost always proximal to the dorsal root ganglion (DRG).

S1 radiculopathy — In S1 radiculopathy, pain radiates down the posterior aspect of the leg into the foot from the back. On examination, weakness of plantar flexion (gastrocnemius muscle) is specific. There may also be weakness of leg extension (gluteus maximus) and knee flexion (biceps femoris long and short heads). Sensation is generally reduced on the posterior aspect of the leg and the lateral edge of the foot. Ankle reflex loss is typical.

EMG and NCS reveal abnormalities in S1 innervated muscles, including the gluteus maximus, long and short heads of the biceps femoris, gastrocnemius and soleus muscles, and the S1 paraspinals, with intact sensory responses (sural generally tested) [8]. Soleus H reflex is usually absent in patients with significant S1 radiculopathy. Testing should be performed bilaterally to identify asymmetric amplitude loss or latency prolongation on the affected side. (See "Overview of nerve conduction studies", section on 'H reflex'.)

S2/S3/S4 radiculopathy — Structural radiculopathies at these lower levels are less common than other lumbosacral radiculopathies, but may be caused by a large central disc compressing the nerve roots intrathecally at a higher level (eg, L5). Patients can present with sacral or buttock pain that radiates down the posterior aspect of the leg or into the perineum. Weakness may be minimal, with urinary and fecal incontinence as well as sexual dysfunction.

The utility of EMG and NCS of the legs for the evaluation of S2, S3, and S4 radiculopathy is limited. However, EMG investigation of the low paraspinal muscles may be helpful. In addition, abnormalities may be identified in gluteus maximus and gastrocnemius. Sensory studies are normal. Prolongations in latency when performing electrical bulbocavernosus reflex testing may indicate a lesion in the region, although this is not specific for radiculopathy.

Severity — Acute lumbosacral radiculopathy can be separated into three general categories from least to most severe:

- Pure sensory/painful radicular pattern, characterized by radicular pain and a segmental pattern of sensory dysfunction but no other neurologic deficits
- Mild motor deficit pattern, characterized by radicular pain, sensory dysfunction, and mild nonprogressive segmental motor weakness and/or reflex change
- Marked motor deficit pattern, characterized by radicular pain and sensory dysfunction with severe or worsening motor deficits

EVALUATION AND DIAGNOSIS

The diagnosis of a lumbosacral radiculopathy is clinical, and can usually be made based upon compatible symptoms and examination findings.

Disc herniation and foraminal stenosis due to spondylotic degeneration are the most common etiologies for lumbosacral radiculopathy (figure 2), and clinical symptoms are self-limited in most cases. Thus, immediate diagnostic testing is not necessary for patients with suspected radiculopathy who are neurologically intact and at low risk for neoplastic, infectious, or inflammatory etiologies (algorithm 1). Nevertheless, testing is suggested to confirm the diagnosis and etiology for patients who have persistent symptoms that are not adequately controlled with conservative therapy and who are candidates for invasive treatment options.

We recommend urgent neuroimaging in the initial assessment for patients with any of the following conditions [1]:

- Acute radiculopathy with progressive neurologic deficits
- Radiculopathy with urinary retention, saddle anesthesia, or bilateral neurologic symptoms or signs
- Suspected neoplasm
- Suspected epidural abscess

If imaging is negative or equivocal, nerve conduction studies (NCS) and electromyography (EMG) may be useful three weeks or more after symptom onset to identify nonstructural causes of lumbosacral radiculopathy and lumbosacral polyradiculopathy.

A lumbar puncture and cerebrospinal fluid (CSF) analysis can be diagnostic for patients with a suspected inflammatory or infectious cause of lumbosacral radiculopathy if the etiology cannot be determined in other ways. However, there are theoretical risks with lumbar puncture in the setting of epidural abscess or other epidural infectious processes. One is that lumbar puncture in the presence of complete canal obstruction by the infectious mass could cause a differential in CSF pressure above and below the obstruction, leading to stretching of neural elements (ie, herniation). Another is that infection could be inadvertently carried into the intrathecal space by the lumbar puncture needle. In addition to these concerns, the diagnostic yield of lumbar puncture for epidural abscess is low, as discussed separately. (See "Spinal epidural abscess", section on 'Diagnosis'.)

The most useful modalities in the evaluation of lumbosacral radiculopathy are magnetic resonance imaging (MRI), computed tomography (CT), NCS, and EMG. An MRI scan of the lumbosacral spine will identify most pathologic states that may benefit from surgical intervention. CT or MRI myelography can assess the anatomy of the root sleeve. The sensitivities of CT and MRI are similar for compressive radiculopathies [34]. NCS and EMG have

a high diagnostic accuracy for radiculopathy when neurologic weakness is present for at least three weeks.

Plain radiographs of the lumbar spine are of limited value in the evaluation of lumbosacral radiculopathy. They can visualize bony structures but do not detect herniated discs. Thus, plain radiographs are not recommended in the work-up of lumbosacral radiculopathy unless there is a need to evaluate for infection, fracture, malignancy, spondylolisthesis, degenerative changes, disc space narrowing, or prior surgery. However, normal plain films do not exclude malignancy or infection in patients with a suspicious history. (See "Evaluation of low back pain in adults", section on 'Modalities'.)

History — Pain and sensory symptoms such as paresthesia, dysesthesia, hyperesthesia, or anaesthesia that involve a specific lumbosacral dermatome are suggestive of a radicular process. Paresthesia occurs in 63 to 72 percent of cases, radiating pain in approximately 35 percent, and numbness in approximately 27 percent [35,36].

Similarly, weakness confined to the muscles in a particular lumbosacral myotome should raise suspicion for radiculopathy, though few people spontaneously report such specific complaints. Inability to get up from a chair suggests iliopsoas or quadriceps weakness, while buckling of the knee is consistent with quadriceps weakness, and dragging of the toe points to tibialis anterior weakness.

Elucidation of triggering and alleviating factors may also be helpful. Radicular pain that worsens with Valsalva or improves while lying down suggests a compressive etiology. Conversely, radicular pain that worsens with lying down may suggest an inflammatory or neoplastic etiology. However, such symptoms have not been shown to be sensitive or specific for these conditions.

The acute onset of symptoms with bending, lifting, or trauma may herald a radiculopathy. In large series, approximately 30 to 50 percent of patients identify an inciting event, most commonly nonlifting activities (eg, bending, slipping without falling, vacuuming, recreational sports), heavy lifting, falls, and motor vehicle accidents [37,38]. However, none of these factors are specific for radiculopathy.

Physical examination — Evaluation for lumbosacral radiculopathy requires a careful neurologic examination. The localization of pain, sensory loss, weakness, and reflex loss can suggest involvement of specific nerve root levels (table 5).

Additional procedures (eg, straight leg raising, reverse straight leg raising) may be useful, but their specificity, sensitivity, and reproducibility are variably limited. Vertebral tenderness is

suggestive of infection but is not specific enough to be clinically useful [13]. The spine should be evaluated for any cutaneous abnormalities, such as tufts of hair, nevi, or pores that may be suggestive of a neural tube developmental abnormality.

Muscle stretch reflexes should be assessed at both the quadriceps (L2/L3/L4) and Achilles (S1). The internal hamstring reflex can be useful in assessing a suspected L5 radiculopathy [39], but its correct performance requires experience. It is sometimes difficult to elicit in heavyset people. An experienced clinician may try to elicit this reflex by tapping the semitendinosus and semimembranosus (internal hamstring muscles) tendons just proximal to the popliteal fossa. Side-to-side asymmetries are usually significant.

The sensory examination is more subjective than other parts of the neurologic examination and is further confounded by dermatomal overlap and variability. However, it can provide important information for localization. Thus, we suggest testing the relevant lumbosacral dermatomes for light touch, pain, and temperature. Vibratory sensation and proprioception are usually not useful for evaluating radiculopathies.

Maneuvers — Specific maneuvers can be helpful in determining whether symptoms are radicular in nature. These include the straight leg raise, the contralateral straight leg raise, and the reverse straight leg raise (also referred to as femoral stretch).

- The straight leg raise test is done with the patient supine. The examiner raises the patient's extended leg on the symptomatic side with the foot dorsiflexed, being careful that the patient is not actively "helping" in lifting the leg. Straight leg raising results in an increase in dural tension in the low lumbar and high sacral levels.

  - Lasègue's sign is the presence or worsening of radicular pain (not just low back pain or hamstring pain) with the straight leg maneuver (ie, hip flexion with the leg extended at the knee). By contrast, radicular pain is absent or unchanged when the hip is flexed with the leg in flexion at the knee. A positive Lasègue's sign usually occurs when hip flexion is between 30 and 60 degrees, though positive tests can also occur at smaller and larger degrees of hip flexion.

  - The bowstring sign refers to the relief of radicular pain when the knee is flexed during a positive straight leg raise.

  The straight leg raise test is more sensitive but less specific than the contralateral straight leg raise for the diagnosis of radiculopathy due to disc herniation [40]. It is most helpful in the evaluation of radiculopathy at the L5 and S1 levels. In a prospective study of 100 patients, Lasègue's sign was positive in 83 percent [41]. One study of patients with acute

low back pain found that the combination of radiating pain into the leg, sensory loss in the foot, and a positive straight leg raise test was predictive of a herniated disc with nerve root compression [42].

- The contralateral (crossed) straight leg raise test refers to passive elevation of the unaffected leg by the examiner. The test is positive when lifting the unaffected leg reproduces radicular pain in the affected leg. It is relatively specific for radiculopathy due to disc herniation, but has poor sensitivity [42,43].

- The reverse straight leg raise (femoral stretch) test is accomplished by placing the patient prone on the table and passively extending the hip and leg straight up off the plane of the table. This maneuver is most useful for evaluating the L2, L3, and L4 roots. However, the value of this test is limited by inadequate information on its sensitivity and specificity.

- Patrick's test is a maneuver during which the hip is externally rotated with the ipsilateral knee flexed at 90 degrees and placed on the opposite knee. The test is positive if it elicits hip or buttock pain. A positive test raises suspicion for hip or sacroiliac disease. However, it is nonspecific for a radicular process. (See "Musculoskeletal examination of the hip and groin", section on 'Tests for acetabular pathology'.)

Neuroimaging — For imaging of the lumbar spine, MRI, CT, and CT myelography (CT scan after intrathecal administration of contrast media) are equally sensitive for the diagnosis of disc herniation [34]. For routine initial assessment, an MRI (image 1 and image 2 and image 3) is more informative than CT because it can identify soft tissue abnormalities. In addition, MRI is not associated with ionizing radiation and is less invasive than CT myelography. MRI with intravenous contrast is performed to help identify other intraspinal pathologies, including inflammatory, malignant, and vascular disorders.

However, there is a high prevalence of abnormal neuroimaging findings in asymptomatic individuals, including some who have what appears to be frank nerve root compression by MRI [44]. As an example, one study of 98 people without back pain found MRI evidence of disc herniation in 27 percent [45]. Furthermore, lumbar spine abnormalities on MRI in asymptomatic patients do not appear to be predictive for the future development or duration of low back pain [46].

Although rarely indicated, CT myelography can visualize spinal nerve roots and their trajectory through the neural foramina. It is useful for patients with intolerance of or contraindications to MRI (eg, implanted electrical devices such as cardiac pacemakers or defibrillators) when standard CT fails to define the anatomic correlates of the clinical presentation. In addition, CT

myelography is preferred for patients who have surgically placed spinal hardware that produces magnetic artifacts.

A CT scan can assess osseous structures better than either plain radiography or MRI and is therefore helpful in assessing for bony disease. However, CT alone is unable to visualize nerve roots, so it is not helpful in the direct imaging of a radicular process.

Electrodiagnosis — The primary electrodiagnostic procedures for lumbosacral radiculopathy are NCS and EMG. In combination, the information provided gives insights into the integrity of spinal nerve roots and their connection with the muscles they innervate. These tests are most commonly considered in patients with persistent disabling symptoms of radiculopathy in whom neuroimaging findings are not consistent with the clinical presentation, and are most useful in the setting of neuromuscular weakness (algorithm 1).

In a systematic review of evidence published through mid-2006, the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) assessed the utility of electrodiagnostic testing for patients with lumbosacral radiculopathy [47]. The AANEM noted that the available data are limited by the lack of a universally accepted "gold standard" definition for the diagnosis of lumbosacral radiculopathy, thus preventing comparison of sensitivities and specificities reported by the included studies. With this caveat in mind, the AANEM concluded that the following tests probably aid the clinical diagnosis:

- Peripheral myotomal limb EMG
- H reflex in S1 radiculopathy (see "Overview of nerve conduction studies", section on 'H reflex')

The AANEM observed that the available evidence suggests a low sensitivity for peroneal and posterior tibial F waves [47].

Nerve conduction studies and electromyography — NCS and EMG have a high diagnostic utility for radiculopathy when neurologic weakness is present for at least three weeks, which is why these studies are most often considered for those with persistent unexplained symptoms. The yield is lower in patients with only pain or sensory loss as the manifestation of radiculopathy. For patients with nonspecific spine pain, EMG can help to distinguish pain-related reduced muscular effort from true neurogenic weakness.

In patients with weakness due to radiculopathy, NCS and EMG together can localize the specific spinal nerve root that is damaged, distinguish between old and new axon loss nerve damage, and provide indirect support for the presence of demyelinating conduction block at the root level [8,48]. Electrodiagnostic testing also can identify conditions that mimic radiculopathy, such

as mononeuropathies of the leg or lumbosacral plexopathy. In the absence of structural abnormalities on neuroimaging, NCS and EMG can assess for the presence of nonstructural disorders of spinal nerve roots and peripheral neuropathies that mimic root disease.

From studies of the relative value of electrodiagnostic testing in the diagnosis of lumbosacral radiculopathy, the following observations have been made:

- EMG and imaging studies have a comparable diagnostic sensitivity, varying between 50 to 85 percent, depending on the patient population [49-51]. In a retrospective comparison of 47 patients who had a clinical history suggestive of either cervical or lumbosacral radiculopathy, there was congruence between EMG and MRI findings in 60 percent of patients [49]. Agreement between EMG and MRI was highest for patients with clearly abnormal examination findings consistent with radiculopathy.

- EMG provides physiologic information, which complements the anatomic information provided by MRI [49,52].

Thus, these tests have overlapping diagnostic sensitivity but provide different kinds of information. When precision is needed to decide whether the neuroimaging findings are etiologically related to specific neurologic deficits, EMG is useful to support or refute this relationship. In particular, EMG may provide objective evidence of denervation when there is no motor deficit or uncertain motor deficit. In addition, EMG findings may help determine the timing of the denervation (eg, distant past versus ongoing). This is particularly useful in patients with past surgery and residual pain.

In acute radiculopathy (ie, the first three weeks), EMG and NCS provide limited but potentially important information. The needle EMG examination is more sensitive and provides better localizing information than NCS, but is not likely to show prominent features of acute axon loss until three or more weeks after symptom onset. This is due to a two- to three-week delay in the development of fibrillation potentials after acute motor axon loss. In the face of marked weakness from acute radiculopathy, NCS can show loss of amplitude of compound muscle action potentials by day 8 after injury.

NCS are carried out by applying an electrical stimulus to the skin overlying a nerve trunk, followed by the recording of the generated electrical response over either the nerve trunk or muscle innervated by it, at some distance from the stimulation. EMG records the electrical potentials generated in a muscle belly through a needle electrode inserted in the muscle. Both methods are discussed in detail elsewhere. (See "Overview of electromyography" and "Overview of nerve conduction studies".)

Somatosensory evoked potentials — Somatosensory evoked potentials (SEPs) are performed by repetitively stimulating the tibial nerve at the ankle and recording the propagated sensory potentials as they ascend up the leg, into the cauda equina, through central sensory pathways in the spinal cord and brain, and then to the sensory cortex. However, the available evidence is conflicting regarding the ability of SEPs to localize specific nerve root compression [53-55]. As noted above, a systematic review found insufficient evidence to reach a conclusion about the utility of dermatomal/segmental SEPs of the L5 or S1 dermatomes [47]. Thus, the clinical value of SEPs for the diagnosis of lumbosacral radiculopathy is uncertain, and they are not part of the routine evaluation of radiculopathy.

Cerebrospinal fluid analysis — A lumbar puncture for CSF evaluation is indicated if there is suspicion for a neoplastic or infectious cause for radiculopathy and the etiology cannot be determined by neuroimaging or other methods (algorithm 1). However, lumbar puncture is usually **not** obtained when there is suspicion for epidural abscess (see "Spinal epidural abscess", section on 'Diagnosis'). In the presence of a known primary tumor, evaluation of the CSF is usually indicated only if contrast-enhanced MRI is nondiagnostic. However, CSF sampling should be done in cases without a known primary cancer when neuroimaging is nondiagnostic, particularly in patients who fail to improve, have progressive neurologic deficits, and/or have involvement of multiple roots.

CSF cytology can be diagnostic in patients with leptomeningeal carcinomatosis, but it may be negative in 10 percent of cases [21]. In such patients, CSF testing should be repeated if there is clinical suspicion for cancer. Other abnormalities in the CSF include a monocytic pleocytosis, decreased glucose, and increased protein.

Radiculopathy may result from infectious causes, including Lyme disease, cytomegalovirus, and herpes zoster. CSF sampling will provide the diagnosis in the first two cases, and may be needed in the third when there is no characteristic rash.

Discography — Discography is a controversial technique of uncertain utility that involves the injection of contrast under fluoroscopy into the nucleus of a disc thought to be the cause of a patient's low back pain. The test is considered positive if it demonstrates an annular disruption and reproduces the patient's usual low back pain symptoms. It is not helpful in the evaluation of lumbosacral radiculopathy.

DIFFERENTIAL DIAGNOSIS

The most common cause of lumbosacral radiculopathy is a herniated disc, though the differential diagnosis includes nonradicular back pain, lumbar spinal stenosis, cauda equina syndrome, diabetic amyotrophy, lumbosacral plexopathy, and mononeuropathies of the leg such as femoral, sciatic, peroneal, and tibial nerve lesions.

Low back pain — Low back pain is one of the most common symptoms causing patients to seek medical attention. Most patients with lumbosacral radiculopathy will also have symptoms of low back pain. However, the majority of cases of low back pain are not due to a primary neurologic cause. Disc disease proves to be the underlying etiology in <5 percent of patients [56]. Furthermore, a specific etiology cannot be reliably established for most patients with low back pain. Thus, while lumbosacral radiculopathy is one possible underlying etiology, it is not the most common.

Since the treatment of radicular and nonradicular low back pain may differ, it is important to distinguish the two.

- Radicular low back pain is generally associated with pain that radiates down one or both legs. There may be associated nerve root dysfunction resulting in sensory loss and/or weakness that may or may not be overtly symptomatic. If the appropriate roots are involved, there may also be a loss or diminution of reflexes.

- Nonradicular low back pain is not associated with other neurologic symptoms or signs. Symptoms are generally localized to the spine and paraspinal regions and may or may not radiate into the leg. However, some patients have pain that radiates from the lower back into the buttocks and upper leg in a manner that mimics radiculopathy even though it is not due to nerve root impingement. Pain may be caused by injury of non-neurologic structures such as bone, muscle, tendon, ligament, or fascia without affecting the roots.

  - A history of osteoporosis should raise suspicion for vertebral body fracture as a cause for acute back pain. (See "Osteoporotic thoracolumbar vertebral compression fractures: Clinical manifestations and treatment".)

  - Back pain associated with fever should raise suspicion for an infection of the spine, such as discitis or epidural abscess. (See "Vertebral osteomyelitis and discitis in adults" and "Spinal epidural abscess".)

  - Back pain associated with progressive weakness, gait disorder, and/or bowel-bladder dysfunction should raise suspicion for spinal cord or cauda equina compression. (See "Disorders affecting the spinal cord" and "Clinical features and diagnosis of neoplastic epidural spinal cord compression".)

- Nonradicular back pain may be the result of a primary inflammatory process (spondyloarthropathy), such as ankylosing spondylitis; the condition needs to be distinguished from low back pain due to musculoskeletal causes, as specific treatment is required. (See "Clinical manifestations of axial spondyloarthritis (ankylosing spondylitis and nonradiographic axial spondyloarthritis) in adults" and "Treatment of axial spondyloarthritis (ankylosing spondylitis and nonradiographic axial spondyloarthritis) in adults".)

- Nonradicular low back pain may be referred from other sites, such as the retroperitoneum. Symptoms that should raise suspicion include history of malignancy, weight loss, pain at night, unvarying pain, and inability to find a position that lessens pain.

Lumbosacral radiculopathies are often misdiagnosed in cases of nonradicular low back pain. This confusion is reinforced by the use of nonspecific nomenclature such as the term "sciatica." Sciatica is often applied to conditions involving low back pain with or without radiation into the leg regardless the cause. Because of its nonspecific use, the term "sciatica" should be avoided if possible when referring to radicular pain.

Diagnostic testing for low back pain is often equivocal. In such cases, the diagnosis is typically that of nonspecific low back pain. Lumbosacral magnetic resonance imaging (MRI) may reveal findings suggestive of root compression, though the patient may not have specific radicular signs or symptoms. Persistence of findings from remote root injury and intercurrent processes such as diabetic neuropathy may also be noted on electromyography (EMG), confounding the evaluation for a more acute problem.

Spinal stenosis — Lumbar spinal stenosis is a common entity, often asymptomatic, that can be caused by a variety of congenital or acquired conditions. The most common etiology is degenerative spondylosis. The major manifestation is neurogenic claudication, a syndrome of bilateral, often asymmetric pain, sensory loss, and/or weakness affecting the legs. The symptoms are produced or exacerbated by walking or prolonged standing in an erect posture. These symptoms represent intermittent mechanical and/or ischemic disruption of lumbosacral nerve root function. (See "Lumbar spinal stenosis: Pathophysiology, clinical features, and diagnosis".)

In a few patients with lumbar spinal stenosis, more fixed nerve root injury may occur, causing lumbosacral radiculopathy, and rarely cauda equina syndrome or conus medullaris syndrome.

Cauda equina syndrome — The cauda equina syndrome is caused by an intraspinal lesion caudal to the conus that injures 2 or more of the 18 nerve roots constituting the cauda equina

within the lumbar spinal canal. (See "Anatomy and localization of spinal cord disorders", section on 'Cauda equina syndrome'.)

The cauda equina syndrome is typically associated with marked neurologic disability. The clinical presentation is dominated by bilateral leg weakness in multiple root distributions (L3-S1), and may be associated with perineal sensory symptoms as well as bowel, bladder, and sexual dysfunction due to involvement of the S2-4 spinal nerve roots.

Potential etiologies include developmental abnormalities such as neural tube defects, infectious or inflammatory conditions, or mass lesions such as tumors. (See "Clinical features and diagnosis of neoplastic epidural spinal cord compression".)

The cauda equina syndrome is a rare complication of lumbar spinal stenosis. (See "Lumbar spinal stenosis: Pathophysiology, clinical features, and diagnosis".)

Diabetic amyotrophy — Diabetic amyotrophy generally involves the L2, L3, and L4 roots, at least at onset, and presents with weakness, pain, and dysesthesia primarily in the proximal leg that evolves over days to weeks. This condition is discussed separately. (See "Diabetic amyotrophy and idiopathic lumbosacral radiculoplexus neuropathy".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Lower spine disorders" and "Society guideline links: Radiculopathy".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Herniated disc (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Anatomy** – Lumbar (L1 to L5) and sacral spinal (S1 to S5) nerve roots originate at the T10 to L1 vertebral level, where the spinal cord ends. The roots then course down through the intraspinal canal, forming the cauda equina, until they exit at their respective neural (intervertebral) foramina (figure 1). Spinal nerve roots within the same numbered neural foramina pass directly below each numbered vertebrae. (See 'Anatomy' above.)

- **Etiologies** – Disc herniation and neural foraminal stenosis are the most common etiologies of lumbosacral radiculopathy. Other causes include tethered cord or diastematomyelia, spina bifida, and nonskeletal conditions such as infection, inflammation, neoplasm, and vascular disease. (See 'Pathophysiology and etiology' above.)

- **Specific radicular presentations** – Lumbar radiculopathy most commonly involves either the L5 or S1 root.

  - L1 radiculopathy is rare. Symptoms involve pain, paresthesia, and sensory loss in the inguinal region. (See 'L1 radiculopathy' above.)

  - L2, L3, and L4 radiculopathies are most often seen in older patients with spinal stenosis. Acute back pain is the most common presenting complaint, often radiating around the anterior aspect of the thigh down into the knee. (See 'L2/L3/L4 radiculopathy' above.)

  - L5 radiculopathy often presents with back pain that radiates down the lateral aspect of the leg into the foot. Strength can be reduced in foot dorsiflexion, toe extension, foot inversion, and foot eversion. Reflexes are generally normal. (See 'L5 radiculopathy' above.)

  - In S1 radiculopathy, pain radiates down the posterior aspect of the leg into the foot from the back. Strength may be reduced in leg extension (gluteus maximus) and plantar flexion. Ankle reflex loss is typical. (See 'S1 radiculopathy' above.)

- S2, S3, and/or S4 radiculopathies present with sacral or buttock pain that radiates down the posterior aspect of the leg or into the perineum. Weakness may be minimal, but urinary and fecal incontinence as well as sexual dysfunction may be present. (See 'S2/S3/S4 radiculopathy' above.)

- **Diagnosis** – For most patients, the clinical diagnosis of a lumbosacral radiculopathy can be made based upon compatible symptoms and examination findings. Diagnostic testing is warranted for other patients with specific associated features (algorithm 1) (see 'Evaluation and diagnosis' above):

  - We recommend neuroimaging for patients with bilateral radicular signs, urinary retention, or saddle anesthesia, those with a suspected high-risk mechanism (eg, neoplasm, epidural abscess, or hematoma). We suggest neuroimaging for patients with severe or progressive symptoms and those whose symptoms persist despite initial conservative treatment. (See 'Neuroimaging' above.)

  - For patients with persistent or severe findings in whom the etiology is not confirmed on neuroimaging, we suggest electromyography and nerve conduction studies. (See 'Electrodiagnosis' above.)

  - A lumbar puncture may be useful for patients with a suspected inflammatory or infectious cause. However, we **avoid** lumbar puncture when there is suspicion for epidural abscess. (See 'Cerebrospinal fluid analysis' above.)

REFERENCES

1. Tarulli AW, Raynor EM. Lumbosacral radiculopathy. Neurol Clin 2007; 25:387.
2. Ranson SW. The structure of the spinal ganglia and of the spinal nerves. J Comp Neurol 1912; 22:159.
3. Hay MC. Anatomy of the lumbar spine. Med J Aust 1976; 1:874.
4. Hamanishi C, Tanaka S. Dorsal root ganglia in the lumbosacral region observed from the axial views of MRI. Spine (Phila Pa 1976) 1993; 18:1753.
5. Kikuchi S, Sato K, Konno S, Hasue M. Anatomic and radiographic study of dorsal root ganglia. Spine (Phila Pa 1976) 1994; 19:6.
6. Levin KH. Electrodiagnostic approach to the patient with suspected radiculopathy. Neurol Clin 2002; 20:397.

7. Lee MW, McPhee RW, Stringer MD. An evidence-based approach to human dermatomes. Clin Anat 2008; 21:363.
8. Tsao BE, Levin KH, Bodner RA. Comparison of surgical and electrodiagnostic findings in single root lumbosacral radiculopathies. Muscle Nerve 2003; 27:60.
9. Foerster O. The dermatomes in man. Brain 1933; 56:1.
10. Bogduk N. The anatomy of the lumbar intervertebral disc syndrome. Med J Aust 1976; 1:878.
11. Groen GJ, Baljet B, Drukker J. Nerves and nerve plexuses of the human vertebral column. Am J Anat 1990; 188:282.
12. Weinstein PR. Anatomy of the lumbar spine. In: Lumbar Disc Disease, 2nd ed, Hardy RW (E d), Raven Press, New York 1993. p.5.
13. Deyo RA, Weinstein JN. Low back pain. N Engl J Med 2001; 344:363.
14. Ljunggren AE. Natural history and clinical role of the herniated disc. In: The Lumbar Spine, 2nd ed, Wiesel SW, Weinstein JN, Herkowitz H, et al (Eds), WB Saunders, Philadelphia 1996. Vol 1, p.473.
15. Maroon JC, Kopitnik TA, Schulhof LA, et al. Diagnosis and microsurgical approach to far-lateral disc herniation in the lumbar spine. J Neurosurg 1990; 72:378.
16. Keim HA, Kirkaldy-Willis WH. Low back pain. Clin Symp 1980; 32:1.
17. Chin CH, Chew KC. Lumbosacral nerve root avulsion. Injury 1997; 28:674.
18. Love JG. The differential diagnosis of intraspinal tumors and protruded intervertebral disks in their surgical treatment. J Neurosurg 1944; 1:275.
19. Anders HJ, Goebel FD. Cytomegalovirus polyradiculopathy in patients with AIDS. Clin Infect Dis 1998; 27:345.
20. Viali S, Hutchinson DO, Hawkins TE, et al. Presentation of intravascular lymphomatosis as lumbosacral polyradiculopathy. Muscle Nerve 2000; 23:1295.
21. Grossman SA, Krabak MJ. Leptomeningeal carcinomatosis. Cancer Treat Rev 1999; 25:103.
22. Paulsen RD, Call GA, Murtagh FR. Prevalence and percutaneous drainage of cysts of the sacral nerve root sheath (Tarlov cysts). AJNR Am J Neuroradiol 1994; 15:293.
23. Lucantoni C, Than KD, Wang AC, et al. Tarlov cysts: a controversial lesion of the sacral spine. Neurosurg Focus 2011; 31:E14.
24. Acosta FL Jr, Quinones-Hinojosa A, Schmidt MH, Weinstein PR. Diagnosis and management of sacral Tarlov cysts. Case report and review of the literature. Neurosurg Focus 2003; 15:E15.

25. Oaklander AL, Long DM, Larvie M, Davidson CJ. Case records of the Massachusetts General Hospital. Case 7-2013. A 77-year-old woman with long-standing unilateral thoracic pain and incontinence. N Engl J Med 2013; 368:853.
26. Morishita S, Arai Y, Yoshii T, et al. Lumbar epidural lipomatosis is associated with visceral fat and metabolic disorders. Eur Spine J 2018; 27:1653.
27. Chad DA. Disorders of nerve roots and plexuses. In: Neurology in Clinical Practice, 4th ed, B radley WG, Daroff RB, Fenichel GM, et al (Eds), Elsevier, Philadelphia 2004. p.2267.
28. Dyck PJ, Norell JE, Dyck PJ. Microvasculitis and ischemia in diabetic lumbosacral radiculoplexus neuropathy. Neurology 1999; 53:2113.
29. Holmes JM, Sworn BR. Lumbo-sacral Root Pain. Br Med J 1946; 1:946.
30. Marshall LL, Trethewie ER, Curtain CC. Chemical radiculitis. A clinical, physiological and immunological study. Clin Orthop Relat Res 1977; :61.
31. Slipman CW, Isaac Z, Lenrow DA, et al. Clinical evidence of chemical radiculopathy. Pain Physician 2002; 5:260.
32. Zwart JA, Sand T, Unsgaard G. Warm and cold sensory thresholds in patients with unilateral sciatica: C fibers are more severely affected than A-delta fibers. Acta Neurol Scand 1998; 97:41.
33. Strian F, Lautenbacher S, Karlbauer G, Galfe G. Disturbances of C-fibre-mediated sensibility in lumbosacral disc disease. J Neurol Neurosurg Psychiatry 1991; 54:1013.
34. Bischoff RJ, Rodriguez RP, Gupta K, et al. A comparison of computed tomography-myelography, magnetic resonance imaging, and myelography in the diagnosis of herniated nucleus pulposus and spinal stenosis. J Spinal Disord 1993; 6:289.
35. Norlen G. On the value of neurological symptoms in sciatica for localization of a lumbar disc herniation. Acta Chir Sand 1944; 95:7.
36. BROWN HA, PONT ME. DISEASE OF LUMBAR DISCS. TEN YEARS OF SURGICAL TREATMENT. J Neurosurg 1963; 20:410.
37. Suri P, Hunter DJ, Jouve C, et al. Inciting events associated with lumbar disc herniation. Spine J 2010; 10:388.
38. Engle AM, Chen Y, Marascalchi B, et al. Lumbosacral Radiculopathy: Inciting Events and Their Association with Epidural Steroid Injection Outcomes. Pain Med 2019; 20:2360.
39. Esene IN, Meher A, Elzoghby MA, et al. Diagnostic performance of the medial hamstring reflex in L5 radiculopathy. Surg Neurol Int 2012; 3:104.
40. van der Windt DA, Simons E, Riphagen II, et al. Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. Cochrane Database

Syst Rev 2010; :CD007431.

41. Weir BK. Prospective study of 100 lumbosacral discectomies. J Neurosurg 1979; 50:283.
42. Deyo RA, Rainville J, Kent DL. What can the history and physical examination tell us about low back pain? JAMA 1992; 268:760.
43. Vroomen PC, de Krom MC, Knottnerus JA. Diagnostic value of history and physical examination in patients suspected of sciatica due to disc herniation: a systematic review. J Neurol 1999; 246:899.
44. Boden SD, Davis DO, Dina TS, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am 1990; 72:403.
45. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994; 331:69.
46. Borenstein DG, O'Mara JW Jr, Boden SD, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects : a seven-year follow-up study. J Bone Joint Surg Am 2001; 83-A:1306.
47. Cho SC, Ferrante MA, Levin KH, et al. Utility of electrodiagnostic testing in evaluating patients with lumbosacral radiculopathy: An evidence-based review. Muscle Nerve 2010; 42:276.
48. Levin KH. Radiculopathy. In: Comprehensive Clinical Neurophysiology, Levin KH, Lüders HO (Eds), WB Saunders, New York 2000. p.627.
49. Nardin RA, Patel MR, Gudas TF, et al. Electromyography and magnetic resonance imaging in the evaluation of radiculopathy. Muscle Nerve 1999; 22:151.
50. Kuruoglu R, Oh SJ, Thompson B. Clinical and electromyographic correlations of lumbosacral radiculopathy. Muscle Nerve 1994; 17:250.
51. Wilbourn AJ, Aminoff MJ. AAEM minimonograph 32: the electrodiagnostic examination in patients with radiculopathies. American Association of Electrodiagnostic Medicine. Muscle Nerve 1998; 21:1612.
52. Albeck MJ, Taher G, Lauritzen M, Trojaborg W. Diagnostic value of electrophysiological tests in patients with sciatica. Acta Neurol Scand 2000; 101:249.
53. Katifi HA, Sedgwick EM. Evaluation of the dermatomal somatosensory evoked potential in the diagnosis of lumbo-sacral root compression. J Neurol Neurosurg Psychiatry 1987; 50:1204.
54. Aminoff MJ, Goodin DS, Barbaro NM, et al. Dermatomal somatosensory evoked potentials in unilateral lumbosacral radiculopathy. Ann Neurol 1985; 17:171.

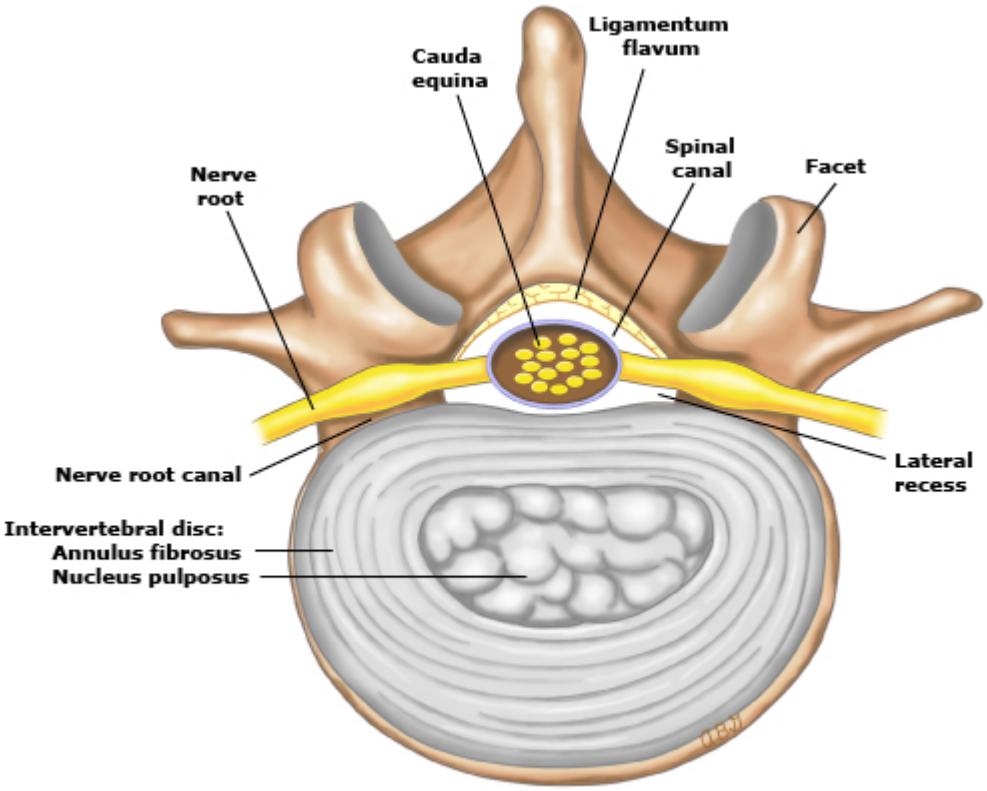
55. Dumitru D, Dreyfuss P. Dermatomal/segmental somatosensory evoked potential evaluation of L5/S1 unilateral/unilevel radiculopathies. Muscle Nerve 1996; 19:442.

56. Bogduk N. The lumbar disc and low back pain. Neurosurg Clin N Am 1991; 2:791.

Topic 5262 Version 37.0

GRAPHICS

Cross section of the lumbar spine



Graphic 66123 Version 4.0

Lumbosacral myotomes

L1
Femoral nerve
Iliopsoas (hip flexion)
L2
Femoral nerve
Iliopsoas (hip flexion)
Quadriceps (knee extension)
Obturator nerve
Hip adductors
L3
Femoral nerve
Iliopsoas (hip flexion)
Quadriceps (knee extension)
Obturator nerve
Hip adductors
L4
Femoral nerve
Iliopsoas (hip flexion)
Quadriceps (knee extension)
Obturator nerve
Hip adductors
L5
Peroneal nerve
Ankle dorsiflexion (tibialis anterior)
Ankle eversion (peroneus muscles)
Tibial nerve
Ankle inversion (tibialis posterior)
Superior gluteal nerve
Hip abduction (gluteus medius)

Leg internal rotation (tensor fascia latae)
S1
Inferior gluteal nerve
Hip extension (gluteus maximus)
Sciatic nerve
Knee flexion (hamstrings)
Tibial nerve
Ankle plantar flexion
S2
Inferior gluteal nerve
Hip extension (Gluteus maximus)
Sciatic nerve
Knee flexion (hamstrings)
Tibial nerve
Ankle plantar flexion

Graphic 64209 Version 2.0

Tissues in the low back that can generate pain

External Tissues
• Skin
• Subcutaneous tissue
• Fat
Joints
• Capsules of facet and sacroiliac joints
Ligaments
• Longitudinal spinal
• Posterior interspinous
• Sacroiliac
• Ligamentum flavum (minimal pain fiber innervation)
Periosteum
• Vertebral bodies and arches
Dura mater
Epidural fibro-adipose tissue
Annulus fibrosus
Arterioles
• Of spinal and sacroiliac joints
• Of vertebral cancellous bone
Veins
• Epidural
• Paravertebral
Paravertebral muscles
• Pain fibers in perivascular connective tissues

Courtesy of Kerry Levin, MD.

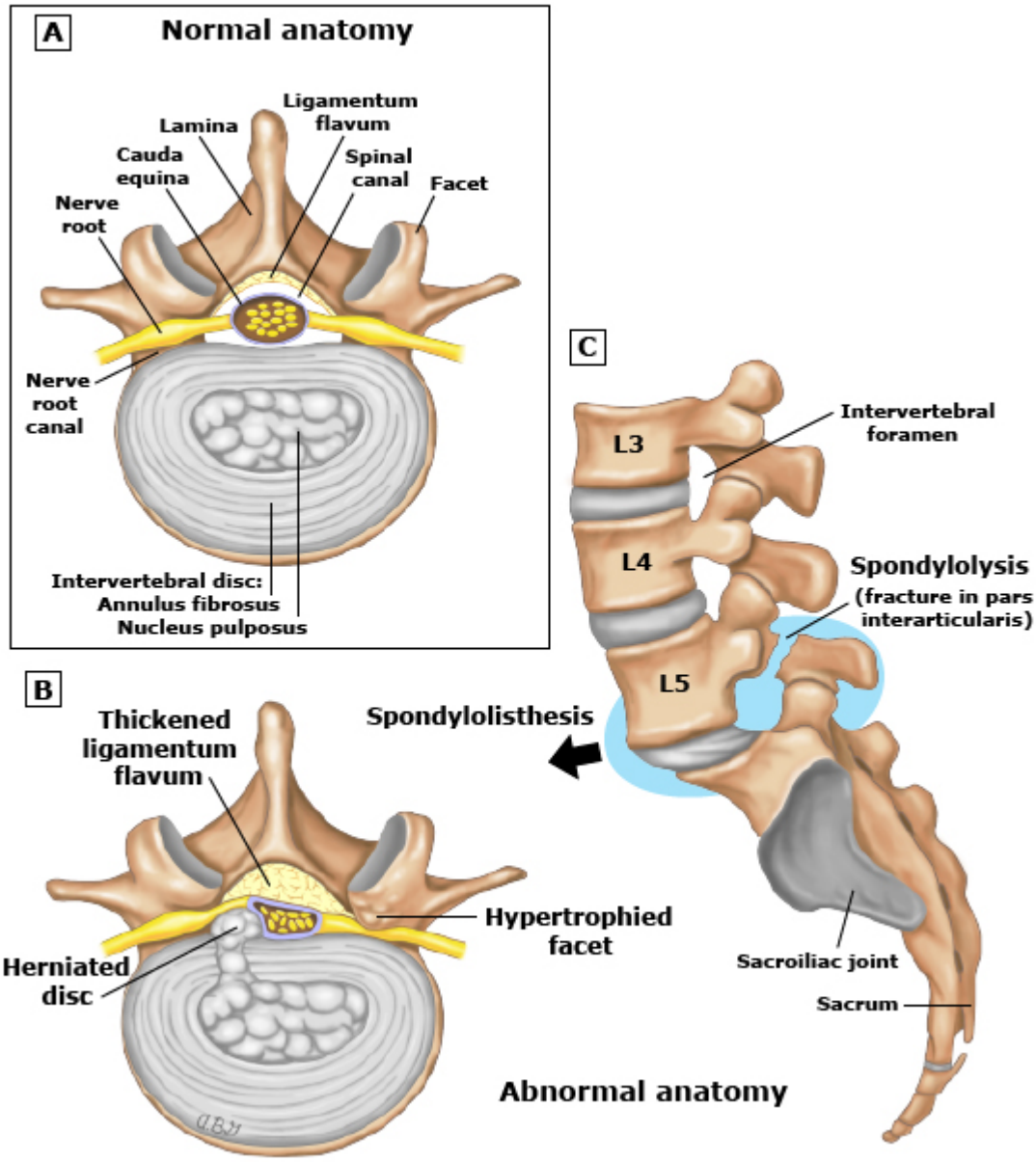
Graphic 76627 Version 1.0

Categories of systemic disease associated with spine pain, and clues to their diagnosis

Cancer
Prior history of cancer
Older age
Unexplained weight loss
No relief with bed rest
Pain exceeding one month
Failure to improve with spine therapies
Spinal infections
Intravenous drug use
Urinary tract infection
Skin infection
Compression fractures
Older age
Trauma
Prolonged glucocorticoid use
Rheumatologic disorders
Ankylosing spondylitis
Behçet disease
Crohn disease
Psoriatic arthritis
Rheumatoid arthritis
Ulcerative colitis
Whipple disease

Graphic 69498 Version 1.0

Common pathoanatomical conditions of the lumbar spine



(A) A superior view of a normal lumbar vertebra with cauda equina, nerve roots, intervertebral disc and ligamentum flavum.

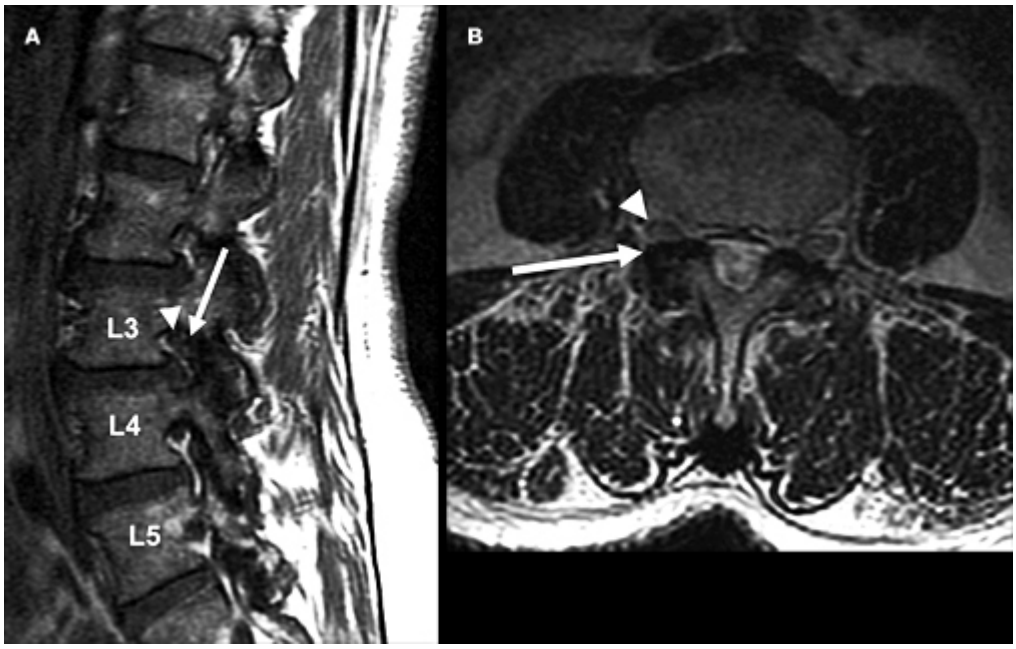
(B) A superior view demonstrating abnormalities including a thickened ligamentum flavum, a hypertrophied facet and a herniated disc. These pathologic structures cause the canal to narrow and can impinge on the cauda equina and nerve roots.

(C) A lateral view of the lumbosacral spine demonstrating spondylolysis and spondylolisthesis. Spondylolysis is a fracture in the pars interarticularis of the vertebra. Spondylolisthesis occurs when this fracture widens and the vertebral body slides forward on the one below it. In older adults, spondylolisthesis can

develop due to degenerative changes in the facet joint and intervertebral disc without an associated spondylolysis fracture.

Graphic 74811 Version 6.0

Lumbar spine MRI showing an osteophyte abutting the exiting L3 nerve root



(A) Sagittal T1-weighted MRI of the lumbar spine shows facet hypertrophic changes with osteophytes (arrow) abutting the exiting L3 nerve root (arrowhead) in the right L3-4 neural foramen. Note the lack of facet hypertrophic changes at the level below, L4-5, where the exiting nerve root is surrounded by hyperintense fat.

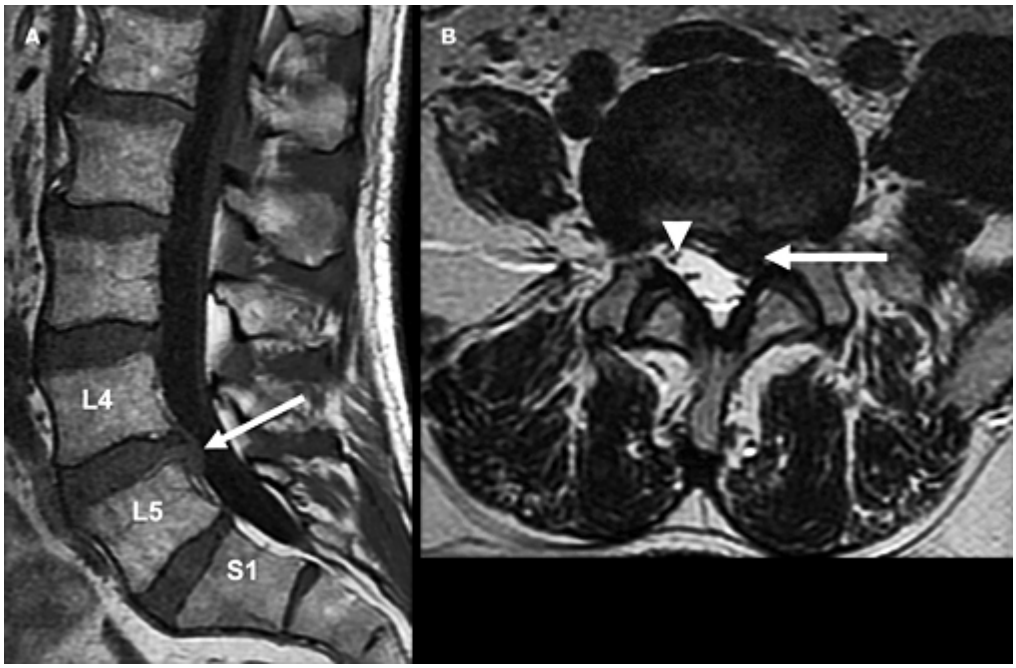
(B) Axial T2-weighted images show the facet osteophytes (arrow) encroaching on the posterior aspect of the neural foramen, adjacent to the exiting L3 nerve root (arrowhead).

MRI: magnetic resonance imaging.

Courtesy of Eric D. Schwartz, MD.

Graphic 77488 Version 4.0

Lumbar spine MRI showing a lateral disc extrusion impinging the L5 nerve root



(A) Sagittal T1-weighted MRI shows a disc extrusion (arrow) at the L4-5 disc level.

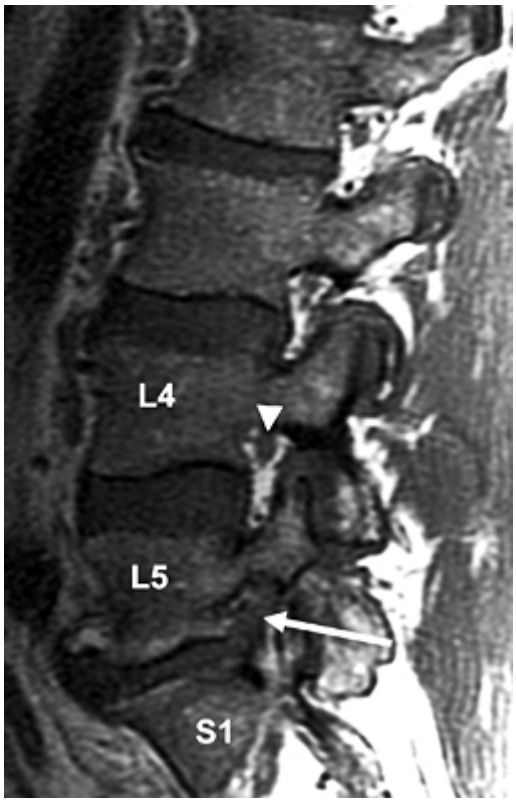
(B) Axial T2-weighted image at the L4-5 disc level shows the disc extrusion to be left paracentral in location (arrow), obliterating the left lateral recess where the descending L5 nerve root would be expected. Note the normal appearing right descending L5 nerve root within the contralateral lateral recess just prior to its exit from the thecal sac (arrowhead).

MRI: magnetic resonance imaging.

Courtesy of Eric D Schwartz, MD.

Graphic 81881 Version 4.0

Lumbar spine MRI showing a foraminal disc extrusion compressing the exiting L5 nerve root



Sagittal T1-weighted MRI of the lumbar spine shows a foraminal disc extrusion filling the L5-S1 neural foramen (arrow), likely compressing the exiting L5 nerve root. Note hyperintense fat surrounding the normal exiting L4 nerve root at the level above (arrowhead).

MRI: magnetic resonance imaging.

Courtesy of Eric D. Schwartz, MD.

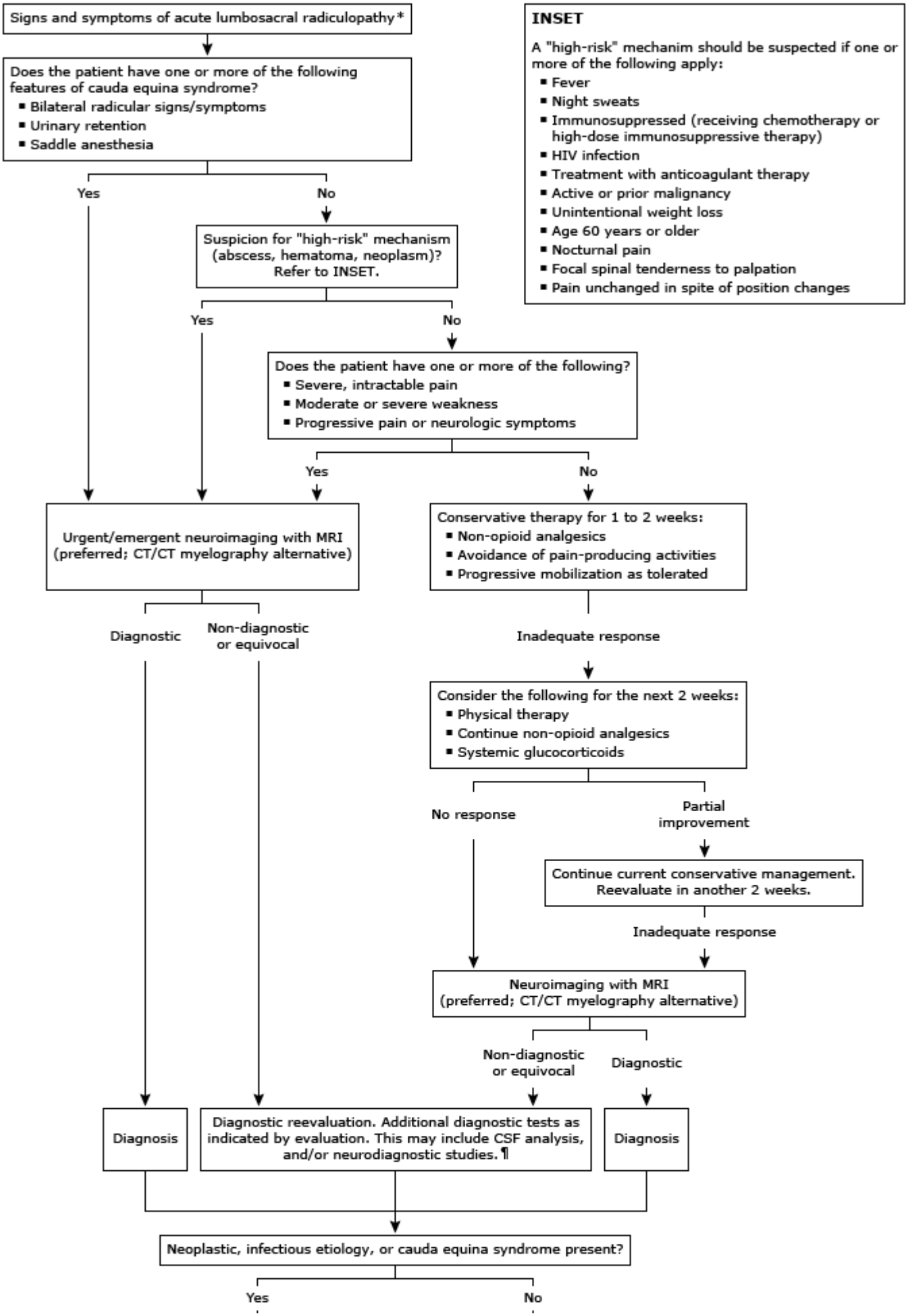
Graphic 79837 Version 4.0

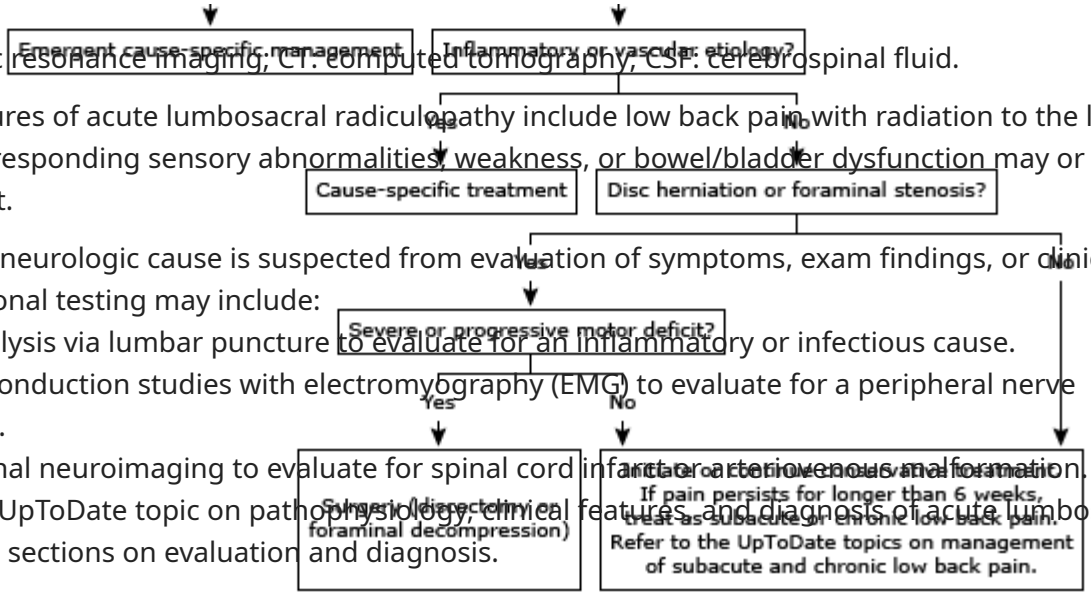
Nonskeletal causes of lumbosacral radiculopathy

Diabetes mellitus
Single or multiple radiculopathies
Inflammatory disorders
Acute inflammatory demyelinating polyradiculoneuropathy (AIDP; Guillain-Barré syndrome)
Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)
Sarcoidosis
Infectious diseases
Borrelia burgdorferi (Lyme disease)
Cytomegalovirus
Epstein-Barr
Herpes simplex virus
Human immunodeficiency virus (HIV)
Mycobacterium
Mycoplasma
Syphilis
Varicella zoster virus (Herpes zoster or shingles)
Malignancy
Lymphoma
Metastasis
Multiple myeloma
Vascular
Arteriovenous malformation
Radiation-induced vascular occlusion
Vasculitis (nerve root infarction)

Graphic 57491 Version 3.0

Approach to acute lumbosacral radiculopathy


Signs and symptoms of acute lumbosacral radiculopathy*
Does the patient have one or more of the following features of cauda equina syndrome?
Bilateral radicular signs/symptoms
Urinary retention
Saddle anesthesia
Yes
No
Suspicion for "high-risk" mechanism (abscess, hematoma, neoplasm)? Refer to INSET.
Yes
No
Does the patient have one or more of the following?
Severe, intractable pain
Moderate or severe weakness
Progressive pain or neurologic symptoms
Yes
No
Urgent/emergent neuroimaging with MRI (preferred; CT/CT myelography alternative)
Conservative therapy for 1 to 2 weeks:
Non-opioid analgesics
Avoidance of pain-producing activities
Progressive mobilization as tolerated
Diagnostic
Non-diagnostic or equivocal
Inadequate response
Consider the following for the next 2 weeks:
Physical therapy
Continue non-opioid analgesics
Systemic glucocorticoids
No response
Partial improvement
Continue current conservative management. Reevaluate in another 2 weeks.
Inadequate response
Neuroimaging with MRI (preferred; CT/CT myelography alternative)
Non-diagnostic or equivocal
Diagnostic
Diagnosis
Diagnostic reevaluation. Additional diagnostic tests as indicated by evaluation. This may include CSF analysis, and/or neurodiagnostic studies. ¶
Diagnosis
Neoplastic, infectious etiology, or cauda equina syndrome present?
Yes
No
INSET
A "high-risk" mechanim should be suspected if one or more of the following apply:
Fever
Night sweats
Immunosuppressed (receiving chemotherapy or high-dose immunosuppressive therapy)
HIV infection
Treatment with anticoagulant therapy
Active or prior malignancy
Unintentional weight loss
Age 60 years or older
Nocturnal pain
Focal spinal tenderness to palpation
Pain unchanged in spite of position changes



MRI: magnetic resonance imaging; CT: computed tomography; CSF: cerebrospinal fluid.

* Clinical features of acute lumbosacral radiculopathy include low back pain with radiation to the leg or the groin; corresponding sensory abnormalities, weakness, or bowel/bladder dysfunction may or may not be present.

¶ If a primary neurologic cause is suspected from evaluation of symptoms, exam findings, or clinical course, additional testing may include:

- CSF analysis via lumbar puncture to evaluate for an inflammatory or infectious cause.
- Nerve conduction studies with electromyography (EMG) to evaluate for a peripheral nerve process.
- Additional neuroimaging to evaluate for spinal cord infarct or arteriovenous malformation.

Refer to the UpToDate topic on pathophysiology, clinical features, and diagnosis of acute lumbosacral radiculopathy, sections on evaluation and diagnosis.

Graphic 101562 Version 3.0

Solitary nerve root lesions of the lumbosacral spine

Root	Pain	Sensory loss	Weakness	Stretch reflex loss
L1	Inguinal region	Inguinal region	Rarely hip flexion	None
L2-L3-L4	Back, radiating into anterior thigh, and at times medial lower leg	Anterior thigh, occasionally medial lower leg	Hip flexion, hip adduction, knee extension	Patellar tendon
L5	Back, radiating into buttock, lateral thigh, lateral calf and dorsum foot, great toe	Lateral calf, dorsum foot, web space between first and second toe	Hip abduction, knee flexion, foot dorsiflexion, toe extension and flexion, foot inversion and eversion	Semitendinosus/semimembranosus (internal hamstrings) tendon
S1	Back, radiating into buttock, lateral or posterior thigh, posterior calf, lateral or plantar foot	Posterior calf, lateral or plantar aspect of foot	Hip extension, knee flexion, plantar flexion of the foot	Achilles tendon
S2-S3-S4	Sacral or buttock pain radiating into the posterior aspect of the leg or the perineum	Medial buttock, perineal, and perianal regions	Weakness may be minimal, with urinary and fecal incontinence as well as sexual dysfunction	Bulbocavernosus, anal wink

Graphic 75645 Version 4.0

Contributor Disclosures

Philip S Hsu, MD No relevant financial relationship(s) with ineligible companies to disclose. **Carmel Armon, MD, MHS** No relevant financial relationship(s) with ineligible companies to disclose. **Kerry Levin, MD** Other Financial Interest: American Academy of Neurology [Editorial board member for Continuum];Elsevier [Royalties as editor for Handbook of Clinical Neurology]. All of the relevant financial relationships listed have been mitigated. **Jeremy M Shefner, MD, PhD** Grant/Research/Clinical Trial Support: AB Sciences [ALS]; Acorda Therapeutics [ALS]; Alector [ALS]; Amylyx [ALS]; Biogen [ALS]; Cytokinetics Incorporated [ALS]; Ionis [ALS]; Mitsubishi Tanabe Pharma America [ALS]; PTC [ALS]; QurAlis [ALS]; Sanofi [ALS]; Wave Life Sciences [ALS]. Consultant/Advisory Boards: AcuraStem [ALS]; Amylyx [ALS]; Apellis [ALS]; Cytokinetics [ALS]; Denali [ALS]; GSK [ALS]; Mitsubishi Tanabe Pharma America [ALS]; Neurosense [ALS]; Novartis [ALS]; Orthogonal [ALS]; Pinteon [ALS]; Revalesio [ALS]; RRD [ALS]; Swanbio [ALS]. All of the relevant financial relationships listed have been mitigated. **Richard P Goddeau, Jr, DO, FAHA** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

→