

Low Back Pain

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KEYWORDS

- Sciatica • Low back pain • Acute low back pain
- Chronic back pain • Spine • Pain

LOW BACK PAIN

General internists and family practitioners play an important role in the initial evaluation and treatment of acute low back pain and chronic low back pain. In the present managed care environment, it is doubtful that this role will decrease, and indeed it should not, because a large percentage of patients who have low back pain can be managed by the primary care physician without referral to a specialist. According to one study, the primary care physician is the initial provider of care for acute low back pain approximately 65% of the time and frequently is the sole provider of care.¹ Given the limited time in the present medical system the primary care physician often has available to evaluate a patient who has acute or chronic low back pain, it is imperative that the generalist have an understanding of the salient points in the history, the essentials of the physical/neurologic examination, the diagnostic testing options, and the effectiveness (or lack of effectiveness) of available treatments.

WHAT IS THE INCIDENCE AND PREVALENCE OF LOW BACK PAIN?

Epidemiologic studies of low back pain are an interpretive challenge because of the inconsistency and lack of standardization used to define back pain in different studies and the variability of criteria from one study to the next.² Both the prevalence and incidence of low back pain are high. Hart and colleagues³ estimated the total number of annual adult visits for low back pain to a physician in the United States to be 15 million, making back pain the fifth most common reason for a physician office visit at the time of this study. The lifetime prevalence of an episode of significant low back pain is 60% to 90%.^{2,4,5} Deyo and Tsui-Wu⁶ reported a 13.8% lifetime prevalence of an episode of low back pain of 2 or more weeks' duration. About 1.6% of the same population (12% of the patients who had low back pain) reported sciatica.⁶ An example of the interpretive challenge of the epidemiology literature is that the reported the annual incidence of developing an episode of low back pain ranges from 4% to 93%, depending on the study.² In one well-designed epidemiologic survey of adults age 20 to 69 years, 19% of 318 patients who did not have a history of back pain over a period of 6 months

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before entry into the study developed an episode of low back pain (usually mild in intensity) over the 1-year study period.⁷

Low back pain has a major social impact in the industrialized world. In the United States, back pain is the second leading cause of work absenteeism. Spine symptoms are the reason for approximately 25% of all lost work days.⁵ In 1988, approximately 175.8 million restricted activity days were caused by spine-related disorders.^{4,8,9} At any one time an estimated 1% of adults in the United States are disabled temporarily, and 1% are disabled chronically as a result of low back pain. Approximately 400,000 compensated back injuries occur each year.^{4,8,9}

WHAT ARE THE RISK FACTORS FOR LOW BACK PAIN?

Commonly accepted risk factors for acute low back pain (and often for chronic low back pain) include^{5,10}

- Increasing age
- Heavy physical work (particularly involving long periods of static work postures, heavy lifting, twisting, and vibration)
- Psychosocial factors, including work dissatisfaction and monotonous work
- Depression
- Obesity (body mass index > 30%; possibly a more significant factor in women than in men)
- Smoking
- Severe (> 80%) scoliosis
- Drug abuse
- History of headache

Many other factors that commonly, but probably erroneously, are thought to increase the risk of low back pain include⁵

- Anthropometric status (height and body build)
- Posture, including kyphosis, lordosis, and modest scoliosis (< 80%)
- Leg-length differences
- State of physical fitness (Although physical fitness is not a predictor of acute low back pain, fit individuals have a lower incidence of chronic low back pain and tend to recover more quickly from episodes of acute low back pain than unfit individuals.)

WHAT ARE THE ANATOMIC ESSENTIALS THAT A PRIMARY CARE PHYSICIANS NEED TO KNOW HOW TO DIAGNOSIS AND TREAT THE CAUSES OF LOW BACK PAIN?

There are five lumbar vertebrae. Each is composed of a body, two pedicles, two laminae, four articular facets, and a spinous process. Between each pair of vertebrae are two openings, the foramina, through which pass a spinal nerve (the nerve “root”), radicular blood vessels, and the sinuvertebral nerves (**Fig. 1**). The spinal canal itself is formed posterolaterally by the laminae and ligamentum flavum, anterolaterally by the pedicles, and anteriorly by the posterior surface of the vertebral bodies and intervertebral disks. The midsagittal diameter of the lumbar canal averages about 18 mm. Narrowing as the result of spondylotic degeneration with superimposed additional narrowing secondary to extension of the trunk can compress the cauda equina. The conus medullaris, the tip of the spinal cord, is at the level of L1-L2.

The facets (the zygapophyseal joints) are true synovial joints. These joints, like all synovial joints, are subject to degenerative and inflammatory changes with resultant

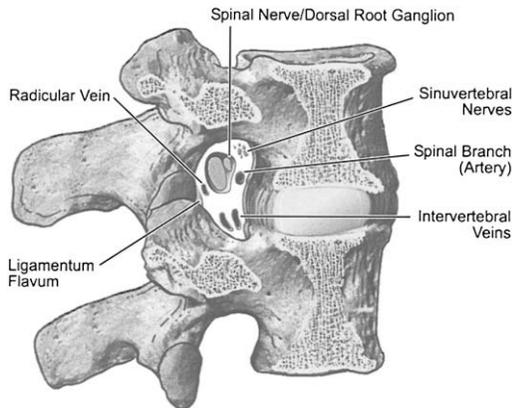


Fig. 1. The foramen. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. Continuum: Lifelong Learning Neurol 2001;7:9; with permission.)

enlargement that, in association with thickening of the ligamentum flavum, can contribute to canal stenosis as a component of spondylosis. The exact role of the facet joint in the production of spine pain, particularly low back pain, remains somewhat controversial.^{11,12}

The intervertebral disk has the dual role of providing the primary support for the column of vertebral bones while maintaining elasticity to permit the required mobility of the spine. Each disk is composed of a ring of elastic collagen, the annulus fibrosus, surrounding the gelatinous nucleus pulposus (**Fig. 2**). The aging or chronically injured disk contains increasing amounts of fibrous tissue that gradually replaces the highly elastic collagen fibers comprising the annulus fibrosus of the young disk. The older disk is less elastic, and its hydraulic recall mechanism is weakened. By age 50 years, the annulus becomes fissured, and ultimately the disk deteriorates into a desiccated, fragmented, and frayed annulus fibrosus surrounding a fibrotic nucleus pulposus. The intervertebral disk is avascular by age 20 years. The nucleus pulposus has no nerve

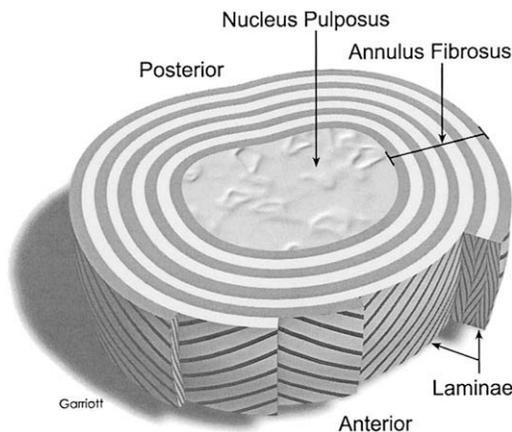


Fig. 2. The intervertebral disk. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. Continuum: Lifelong Learning Neurol 2001;7:11; with permission.)

supply, but the outer lamellae of the annulus contains nerve endings derived from the sinuvertebral nerves (recurrent meningeal nerves).^{5,13-18}

There is debate regarding the nociceptive nerve supply to the intervertebral disk and the role the disk plays as a generator of back pain. Korkala and colleagues¹³ showed that the nerve endings entering the annulus fibrosus do not contain substance P and are not nociceptors. The authors noted that nociceptive nerve endings are located in the posterior ligament adjacent to the disk. Palmgren and colleagues,¹⁹ in a study of normal human lumbar intervertebral disk tissue, demonstrated that nerve endings could be found at a depth of a few millimeters, whereas neuropeptide markers (eg, substance P) revealed nociceptive nerves only in the outermost layers of the annulus fibrosus. This study supports the concept that the normal intervertebral disk is almost without nociceptive innervation.

This finding leads to the question of the mechanism of primary diskogenic pain, particularly in the lumbar spine. Damage to the intervertebral disk can produce pain, but no consensus exists on the mechanisms responsible. Radial tears and fissures in the annulus fibrosus occur as the disk ages. This change has been linked to the ingrowth of blood vessels and nerve fibers, leading to the concept that the ingrowth of these nerve endings may be the pathoanatomic basis for diskogenic pain.^{16,20} If the ingrowth of nociceptive nerve fibers into the intervertebral disk is the neuroanatomic substrate for diskogenic pain, however, why are most degenerative disks not a source of pain? For example, diskography of degenerative disks does not uniformly induce pain.²⁰ Because disk degeneration alone is not the basis for diskogenic pain, contributing factors must be at play. Possibly a combination of focal damage to the annulus fibrosus, inflammation, neoinnervation, and nociceptor sensitization is necessary to induce diskogenic pain.²¹

Many ligaments lash the vertebrae together and, along with the paraspinal muscles, help control and limit spinal column motion. From a clinical perspective, some of the ligaments are more important than others. The posterior longitudinal ligament stretches from the axis (the tectorial membrane in the "high" cervical spine) to the sacrum and forms the anterior wall of the spinal canal. It is broad throughout the cervical and thoracic portions of the spine. At the L1 vertebral level, however, it begins to narrow, and at L5 it is one half its original width. It is attached firmly to each intervertebral disk and hyaline cartilage endplate, but only in the midline of the vertebral body by a septum to the periosteum. The open space between the posterior longitudinal ligament and the vertebral body is the anterior epidural space, which is important in the process of disk herniation. The narrowing of the ligament in the lumbar spine inadequately reinforces the lumbar disk, creating an inherent structural weakness. This narrowing, coupled with the great static and kinetic stress placed on the lumbar disks, contributes to their susceptibility to injury and herniation.

The ligamenta flava is composed of a series of strong, paired elastic ligaments that span the space between the laminae and are attached to the anterior inferior surface of the laminae above and the posterior superior margin of the laminae below. Each component stretches laterally, joining the facet joint capsule. The ligament stretches under tension, permitting flexion of the spine. It contains few, if any, nociceptive nerve fibers. It can be clinically important, because it can thicken with age and, along with other spondyloytic degenerative changes, can contribute to canal stenosis that can produce myelopathy in the cervical spine and compression of the cauda equina in the lumbar spine.

With the exception of the atlas and axis, the range and type of movement in each segment of the spine is determined by the facet (zygapophyseal) joints, but spine stability and control of spine movement depends on muscles and ligaments. The movement itself, of course, is generated by muscle.

The spinal muscles are arranged in layers. The deeper layers comprise the intrinsic true back muscles, as defined by their position and innervation by the posterior rami of the spinal nerves. The more superficial extrinsic muscles insert on the bones of the upper limbs and are innervated by anterior rami of the spinal nerves.

The intrinsic muscles also are divided into superficial and deep groups. The superficial layer is comprised of the paraspinous erector spinae group, which spans the entire length of the spine from the occiput to the sacrum, and the splenius muscles of the upper back and neck. This superficial group functions collectively primarily to maintain erect posture. Deep to the erector spinae is the transversospinalis muscle group, which is composed of muscles made up of several smaller muscles that run obliquely and longitudinally. In essence, they form a system of guy ropes that provide lateral stability to the spine, contribute to maintenance of an erect posture, and rotate the spine. Deepest of all are the interspinal and intertransverse muscles, which are composed of numerous small muscles involved in the maintenance of posture.

The multiple subdivisions of muscle mass, numerous connective tissue planes, and multiple attachments of tendons over small areas of vertebral periosteum help explain the prevalence of neck and back pain and also the difficulty in localizing the source of that pain precisely. Given this difficulty in identifying muscle and tendon injury as the source of pain and the fact that there are other generators of low back pain besides muscles (eg, fascia, ligaments, facet joint, intervertebral disks), it is no wonder that, according to Deyo and colleagues,⁹ the source of acute low back pain cannot be identified in 85% of patients. Also, when muscle is the source of pain, the pathophysiologic pain-generating process is unclear. In the clinic, muscle spasm often is the diagnosis offered. "Muscle spasm" generally is defined as a contraction of muscle that cannot be released voluntarily and is associated with electromyographic activity. Johnson²² and Mense and Simons²³ have taken issue with increased muscle activity as a source of paraspinous pain, noting a lack of electromyographic evidence indicative of muscle spasm.

Localized spine pain is mediated through two peripheral nerve systems: the posterior rami of the spinal nerves and the sinuvertebral nerves. The sinuvertebral nerves supply structures within the spinal canal. These nerves arise from the rami communicantes and enter the spinal canal by way of the intervertebral foramina.¹⁴ Branches ascend and descend one or more levels interconnecting with the sinuvertebral nerves from other levels and innervating the anterior and posterior longitudinal ligaments, the anterior and posterior portion of the dura mater, and blood vessels, among other structures (**Fig. 3**). This system also may supply nociceptive branches to degenerated intervertebral disks. Branches of the posterior rami of the spinal nerves provide nociceptive fibers to the fascia, ligaments, periosteum, and facet joints. The source of deep somatic neck and low back pain therefore can be the vertebral column itself, the surrounding muscle tendons, ligaments fascia, or a combination thereof.

Radicular pain, unlike spondylogenic pain, is mediated by the proximal spinal nerves rather than by the sinuvertebral nerves or the anterior or posterior rami of the spinal nerves.

WHAT TYPES OF PAIN ARE GENERATED BY LUMBAR SPINE DISORDER?

There are three major categories of pain related to the spine: localized, radiating, and referred.

Deyo and colleagues⁹ noted that a definitive diagnosis of low back pain cannot be established in 85% of patients because of the weak association between the symptoms, pathologic changes, and imaging results. Nonetheless, it is widely assumed

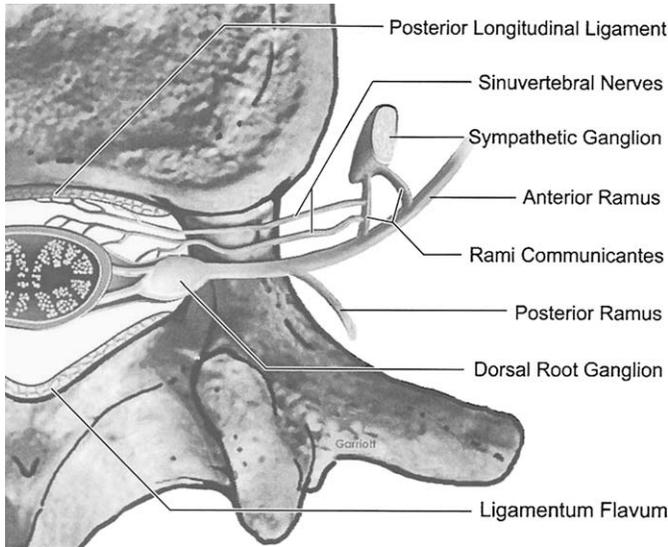


Fig. 3. The sinuvertebral nerves. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. *Continuum: Lifelong Learning Neurol* 2001;7:13; with permission.)

that much nonradiating low back pain is secondary to musculoligamentous injury, degenerative changes in the spine, or a combination of the two. As already noted, local spine pain is mediated primarily through the posterior rami of the spinal nerves and the sinuvertebral nerves.

The source of acute low back pain is most likely the result of increased tension of the paraspinal muscles related to physical activity, such as lifting, with resultant avulsion of tendinous attachments of the muscles to bony structures and/or rupture of the muscle fibers and/or tearing of muscle sheaths. Persistent overuse of a muscle group in the absence of muscle injury can result in pain and potential tonic contraction (spasm), probably reflecting increased metabolic activity and the production of chemical by-products stimulating unmyelinated nerve fibers. This development is particularly likely with the persistent use of untrained, poorly conditioned muscles (as previously noted there is controversy regarding the existence of spasm).^{22,23} The role of paraspinal muscles as a generator of chronic low back pain is uncertain but is likely to be much smaller than in acute low back pain.

As already stated, radiating or radicular pain is mediated not by the sinuvertebral nerves or the posterior rami of the spinal nerves but rather by the proximal spinal nerves. This radiating pain is the generator of "sciatica" (radicular pain into one or both lower extremities distal to the knee). At times, particularly in the case of high lumbar radiculopathies (L2 and L3), the radiating pain may be less well defined, with referral only into the thigh. Two major factors are involved in the generation of radicular-based radiating pain: compression and inflammation.

Compression of the nerve root produces local ischemia with a possible alteration in axoplasmic transport and resultant edema affecting the large mechanoreceptor fibers, resulting in loss of inhibition of pain and impulses carried by the unmyelinated fibers with resultant increased nociceptive input into the spinal cord (Gait theory).⁵ Inflammation, which may be neurogenic or immunologic, develops at the site of compression, probably secondary to the previously isolated nuclear pulposus being herniated

through the annulus fibrosus and exposed to the immune system for the first time, contributing to the generation of pain. The simultaneous involvement of the posterior ramus, the sinuvertebral nerves, and the compressed inflamed spinal nerve can result in the combination of low back pain and radiating pain into a lower extremity subserved by the anterior ramus.

Multiple processes affecting most organs in the abdomen as well as retroperitoneal structures (eg, dissecting aortic aneurysm, renal colic, tumors of many abdominal organs) can present with low back pain. Therefore, the examination of the abdomen is an important part of the initial assessment of a patient presenting with acute low pain.

WHAT ARE THE MOST COMMON CAUSES OF ACUTE NONRADIATING LOW BACK PAIN?

The exact generator of acute low back pain usually is not specifically diagnosable, as suggested by the lack of precision and specificity in the names given the syndromes: “back strain,” “musculoskeletal pain syndrome,” “back spasms,” “myofascial pain syndrome,” and, if pain is more widespread, “fibromyalgia.” Tendon, ligament, muscle, and facet joints have all been implicated as sources of pain, but none can be readily documented in any given patient.

Muscle strain probably is the most common cause of neck and acute low back pain. Immediate or delayed-onset muscular strain often results from some type of physical activity. This type of injury occurs when the muscles in question undergo forceful elongation, usually while being activated. Tearing of muscle fibers occurs primarily at the musculotendinous interface but can occur in the belly of the muscle as well.¹²

Ligamentous sprains are caused by stretching the ligament beyond its physiologic range. The pain of muscle strain and ligamentous sprain is described variously as aching, sharp, or dull and can be mild to severe. The key to the diagnosis in these patients is the absence of any significant radiating pain into either lower extremity and the absence of abnormalities on the neurologic and general examination.²⁴

In this setting back pain generally is reduced significantly when the patient is recumbent. If such is the case, and there are no neurologic findings, an immediate diagnostic work-up usually is unnecessary. If pain does persist in recumbency, however, infections of the spinal column (osteomyelitis/epidural abscess) and metastatic cancer should be considered. An immediate diagnostic work-up in this setting usually is warranted.

HOW BIG A ROLE DOES DEGENERATIVE SPINE DISEASE PLAY IN THE GENERATION OF LOW BACK PAIN?

“Spondylosis” is the more correct term for degenerative changes that occur throughout the spine. These changes occur to the greatest degree in the lumbar and cervical portions of the spine, because these portions of the spine are more mobile (and hence subject to more “wear and tear”) than the thoracic spine. “Spondylosis” is a more accurate term than “arthritis of the spine,” because much of the degenerative change is not primarily inflammatory.⁵ Spondylosis is a naturally occurring process in the aging spine, particularly the lumbar spine. By 79 years of age, nearly all individuals have some degree of spondylosis. By age 49 years, 60% of women and 80% of men have osteophytes and other changes indicative of early spondylosis. There is a poor correlation between the presence of spondylosis on radiographs of the lumbar spine and back pain. Some patients may have marked degenerative change with little or no pain. Disk herniation, a component of spondylosis, is a common finding in the absence of pain.^{25,26} Other patients have significant low back pain in the absence of significant degenerative change. As with acute low back pain, it often is difficult to localize the

source of nonradiating chronic low back pain, and lumbar spine surgery for nonradiating chronic low back pain often is unsuccessful. Progressive spondylosis with osteophyte formation, thickening of the ligaments (particularly the ligament of flavum), and bulging/transverse bar formation of aging lumbar intervertebral disks can lead to lumbar canal stenosis and ultimately to compression of individual nerve roots or of the cauda equina as a whole.

In summary, in dealing with patients who have low back pain and radiologic evidence of lumbar spondylosis, it is important to treat the patient and not the results of imaging procedures. Surgical treatment for chronic low back pain in patients who have a normal neurologic examination but with spondylosis (even severe spondylosis) on neuroimaging procedures often has a poor outcome.

WHAT IS THE DEFINITION OF SCIATICA AND WHAT CAUSES IT?

“Sciatica” refers to pain radiating into either lower extremity distal to the knee. It is associated most commonly with L5 and S1 radiculopathies, which together comprise more than 90% of all lumbosacral radiculopathies. L5 and S1 radiculopathies may result from a variety of pathophysiologic processes compromising the spinal nerve root/roots, including acute disk herniation and spondylosis. Less common causes include compression by a benign or occasionally malignant tumor or epidural abscess. Although L5 and S1 radiculopathies combined are the most common cause of sciatica, there are other potential causes of radiating pain distal to the knee, including lesions involving the pelvic plexus or sciatic nerve (eg, the piriformis syndrome).

WHAT ARE THE ESSENTIALS IN THE HISTORY THAT HELP TO DEFINE THE NATURE OF LOW BACK PAIN?

The history of low back pain is of critical importance in assessing patients who have symptoms believed to be secondary to lumbar spine disorders, especially patients who have a nonfocal neurologic examination. The differential diagnosis frequently is based solely on the history in these patients. As with most pain syndromes, it is essential to establish a pain profile.^{5,18}

Onset

In most instances, patients who present with acute-onset low back pain have a history of preceding pain, often for weeks, months, or longer. This is also true for patients who present with the acute onset of radicular pain. The onset of lumbosacral radicular pain in the absence of any prior history of low back pain is the exception rather than the rule. Radicular distribution pain into a lower extremity (“sciatica”) often is the result of disk herniation. Slowly progressing pain is more typical of degenerative disorders of the spine and of slow-growing tumors.

Location

Musculoskeletal pain usually is localized to the paraspinal regions, spreading at time to the flanks and into the buttocks. In the case of lumbosacral radiculopathy, the pain usually radiates into a lower extremity. Occasionally, the distribution of the pain can point to the specific root involved (**Table 1**). For example, high lumbar (L2, L3) radiculopathic pain does not radiate distal to the knee, whereas the pain of an L4 radiculopathy can radiate distal to the knee to the medial leg. L5 and S1 radiculopathies tend to produce pain that radiates into the posterolateral thigh and posterolateral leg, often involving the foot. Pain may

Root	Pain Distribution	Dermatomal Sensory Distribution	Weakness	Affected Reflex
L1	Inguinal region	Inguinal region	Hip flexion	Cremasteric
L2	Inguinal region and anterior thigh	Anterior thigh	Hip flexion Hip adduction	Cremasteric thigh adductor
L3	Anterior thigh and knee	Distal anteromedial thigh including knee	Knee extension Hip flexion Hip adduction	Patellar thigh adductor
L4	Anterior thigh, medial aspect leg	Medial leg	Knee extension Hip flexion Hip adduction	Patellar
L5	Posterolateral thigh Lateral leg Medial foot	Lateral leg, dorsal foot and great toe	Dorsiflexion foot/toes Knee flexion Hip adduction	—
S1	Posterior thigh and leg, and lateral foot	Posterolateral leg and lateral aspect of foot	Plantar flexion foot/toes Knee flexion Hip extension	Achilles

Data from Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. *Continuum: Lifelong Learning Neurol* 2001;7:16.

be maximum in the medial (L5 radiculopathy) or lateral (S1 radiculopathy) aspect of the foot.

Duration

Mechanical low back pain generally has a duration of days to weeks. Radicular pain often resolves more gradually over 6 to 8 weeks, if it resolves at all. A patient who presents with a history of chronic low back pain may not require a neurodiagnostic evaluation, but a careful history is required to rule out a new problem producing pain superimposed on chronic symptoms. These patients are a challenge even to the most experienced clinician, because such a patient, even with a history of chronic pain, may prove to be an exception and require an immediate neurodiagnostic evaluation.

Severity

As all clinicians recognize, the severity of pain often is difficult to interpret, because it can be colored by several factors, including the patient's personality. Nonradiating musculoskeletal pain may range from mild to incapacitating. Radicular pain secondary to a herniated disk often is severe but may not be. Severe low back pain not relieved when the patient is recumbent suggests metastatic cancer, pathologic vertebral fracture, or infection of a vertebra, disk, or in the epidural space.

Time of Day

In the absence of a history of trauma, new-onset of lumbar radiculopathy frequently is present upon awakening in the morning. Nonradiating pain that tends to be dull during the day often is the result of mechanical disorders (eg, muscle strain,

degenerative disk disease/spondylosis). Tumors of the spine and spinal cord often produce pain that persists and occasionally increases in the supine position, sometimes increasing in bed at night.

Associated Symptoms

Paresthesia, weakness in one or both lower extremities, and bladder/bowel dysfunction may accompany low back pain, particularly radicular pain. Many patients who have radiating pain into a lower extremity complain of weakness. Distinguishing guarding secondary to pain from weakness caused by nerve root compromise can be challenging. Also, the patient should be questioned about abdominal pain and intestinal or genitourinary symptoms, because lesions in various abdominal organs may present with referred low back pain. Recognizing the presence of neurologic symptoms/signs is important and may lead to an expedited diagnostic work-up.

Triggers

Valsalva maneuvers (eg, coughing, sneezing, and bearing down at stool) often transiently aggravate lumbosacral radicular pain. Low back radicular pain generally is worse when the patient is seated or standing and often is relieved by lying in a supine position. If pain persists or increases in the supine position, the possibility of metastatic cancer or an infection must be considered. In the case of lumbar canal stenosis, the symptoms of neurogenic claudication often can be aggravated by standing erect (which tends to reduce the mid sagittal diameter of the lumbar canal) and by walking.

Motor Symptoms

In the face of pain, distinguishing between weakness and guarding by the history alone can be difficult. In the case of low back and lower extremity pain, however, weakness is suggested by the history of a foot slap when walking or falls secondary to a lower extremity “giving way.” Although weakness usually is best appreciated on the neurologic examination, the history is a useful adjunct in helping distinguish weakness from guarding secondary to pain.

Sensory Disturbances

Patients who have radiculopathy often report numbness, tingling, and even coolness in the involved extremity. At times symptoms may suggest dysesthesia and allodynia. The distribution of a sensory disturbance by history, particularly numbness and tingling, sometimes may be more useful than the sensory examination itself in determining the presence and localization of radiculopathy.

Bladder and Bowel Disturbances

Sphincter disturbances in association with low back pain syndromes are uncommon, but a neurogenic bladder can be a component of a cauda equina syndrome. Disturbances of bowel and bladder control should serve as a warning of the need for an urgent neurodiagnostic work-up and, very probably, surgical intervention.

WHAT ARE THE ESSENTIALS OF THE PHYSICAL EXAMINATION?

General Examination

The necessity for a general physical assessment in a patient who complains of back pain cannot be overstated. The presence of low-grade fever, for example, may signal

infection that involves the vertebral column, the epidural space, or the surrounding muscle (eg, psoas abscess). Inspection of the skin for lesions may yield diagnostic information (**Box 1**).

The rectal examination for sphincter tone, anal “wink,” and the bulbocavernosus reflex may reflect changes in the spinal cord or cauda equina. An abnormal prostate examination may lead to a diagnosis of prostate cancer with spinal metastases. The abdominal examination is particularly important. In a patient who has low back pain. The presence of abdominal tenderness, organomegaly, or a pulsatile abdominal mass with a bruit signals the need for an urgent diagnostic evaluation that may lead to a potentially life-saving discovery, such as a leaking abdominal aneurysm. In patients who have low back pain and claudication, evaluation of the peripheral pulses in both lower extremities is essential to help distinguish neurogenic claudication from vascular claudication.

Neurologic Examination

The experienced clinician knows that the neurologic examination of a patient who has low back pain can be altered by the pain itself. For example, when testing strength, guarding must be taken into account. Tendon reflexes may be suppressed as a result of poor relaxation of the limb as a consequence of pain. Preparing the patient by explaining each step of the examination in advance may reduce anxiety and encourage relaxation, thereby reducing guarding and enhancing the reliability of the examination itself. The neurologic examination should include

Inspection of the low back: The presence of a tuft of hair over the lumbar spine suggests diastematomyelia/spina bifida occulta.

Posture: Splinting with a list away from the painful lower extremity is seen with a lateral lumbar disk herniation, whereas list toward the painful side can be seen with medial herniation. Tilting of the trunk to the side opposite the list by the examiner can cause additional nerve root compression with resultant accentuation of radicular distribution pain. Patients who have neurogenic claudication secondary to compression of the cauda equina may tend to stand and walk with the trunk flexed forward, a posture that reduces compression by widening the anterior-posterior dimension of the lumbar canal.

Percussion: Percussion over the lumbar spine may induce pain over a vertebral malignancy or infection.

Box 1

Skin lesions related to spine pain

Psoriasis—psoriatic arthritis

Erythema nodosum—inflammatory disease, cancer

Café-au-lait spots—neurofibromatosis

Hidradenitis suppurative—epidural abscess

Vesicles—herpes zoster

Needle marks (intravenous drug abuse)—vertebral column infections

Subcutaneous masses—neurofibroma, lymphadenopathy

Lumbar spine mobility: Lumbar spine mobility usually is reduced in patients who have low back pain, but a measurement of the degree of mobility usually is not useful because mobility varies so widely with conditioning and age.

Gait evaluation: An antalgic gait with splinting favors the side of a lumbar radiculopathy. Foot slap (ie, foot drop) secondary to weakness of dorsiflexors of the foot can be seen with an L5 radiculopathy. Heel and toe walking, toe tapping, and hopping on either foot may reveal additional evidence of weakness suggesting radiculopathy.

Motor testing: Individual muscle group testing around each joint in the lower extremity is important to elicit evidence of weakness within a specific myotome (**Table 1**).

Sensory testing: Sensory testing is usually done with a sharp object looking for evidence of sensory loss within a given dermatome (**Table 1**).

Reflexes: See **Table 1**.

Neuromechanical tests: Neuromechanical tests are an important adjunct to the traditional neurologic examination in patients who have low back pain radiating into a lower extremity.

Straight leg-raising test: With the patient in a supine position, the symptomatic lower extremity is elevated slowly off the examining table (**Fig. 4**). The spinal nerve and its dural sleeve, tethered by a herniated disk, are stretched when the lower extremity is elevated between 30° and 70°. The test is positive if radiating pain occurs within this range. Pain generated at less than 30° or more than 70° of elevation is nonspecific.

Lasegue test: A variation of straight leg-raising test. With the patient in a supine position, the symptomatic lower extremity is flexed to 90° at the hip and knee. The knee then is extended slowly, which produces radiating pain as a result of L5 and S1 nerve root compression.

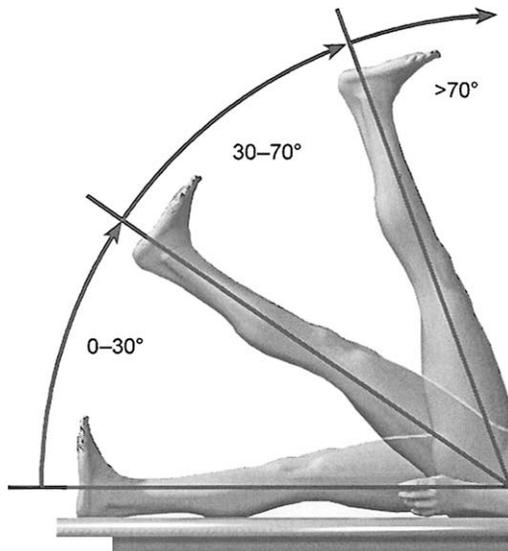


Fig. 4. The straight leg-raising test. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. *Continuum: Lifelong Learning Neurol* 2001;7:20; with permission.)

Bragard's sign (test): If pain is generated by straight leg raising, the symptomatic extremity is lowered until the pain disappears (**Fig. 5**). At that point the foot is dorsiflexed. If this maneuver recreates radiating pain, the test is positive and supports the presence of a low lumbar radiculopathy.

Contralateral ("well") straight leg-raising test: Performed on the asymptomatic lower extremity, this test has specificity but low sensitivity for disk herniation.

Prone straight leg-raising test: With the patient in a prone position, the symptomatic lower extremity is slowly extended at the hip by the examiner (**Fig. 6**). Accentuation of pain in the anterior thigh suggests a high lumbar (L2, L3) radiculopathy.

Valsalva test: The Valsalva maneuver increases intrathecal pressure, which accentuates radicular pain in the presence of spinal nerve compression and inflammation.

Brudzinski test: With the patient supine, the examiner flexes the patient's head. In the presence of spinal compression, this flexion aggravates radicular pain.

Patrick (Faber) test: The lateral malleolus of the symptomatic lower extremity is placed on the patella of the opposite extremity, and the symptomatic extremity is slowly rotated externally (**Fig. 7**). Accentuation of the pain suggests that pain is caused by a lesion in the hip or sacroiliac joint rather than by radiculopathy.

Gaenslen test: With the patient supine and the symptomatic extremity and buttocks extending slightly over the edge of the examination table, the asymptomatic lower extremity is flexed at the hip and knee and brought to the chest (**Fig. 8**). The symptomatic lower extremity is extended at the hip to the floor. Increased nonradiating low back and buttocks pain indicates sacroiliac joint disease.

Waddell test: Excessive sensitivity to light pinching of the skin in the region of low back pain suggests a functional component.

WHAT ARE THE MOST COMMON LUMBOSACRAL RADICULOPATHIES?

L5 radiculopathy is the most common lumbosacral radiculopathy, with S1 radiculopathy a close second. The two combined account for about 90% to 95% of all lumbar radiculopathies. The next most common is L4 radiculopathy (< 5%). High lumbar radiculopathies constitute the remainder. The combination of clinical symptoms and

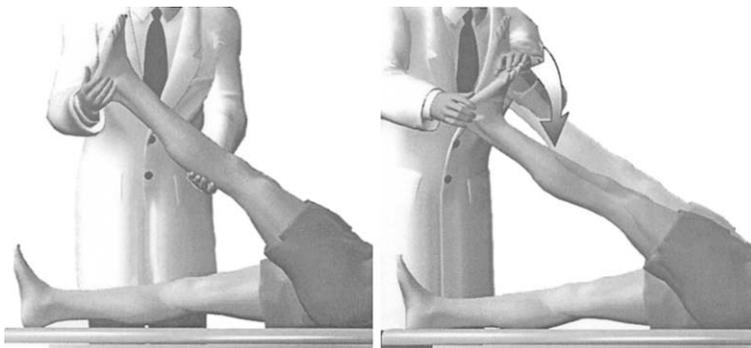


Fig. 5. Bragard's sign. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. Continuum: Lifelong Learning Neurol 2001;7:20; with permission.)

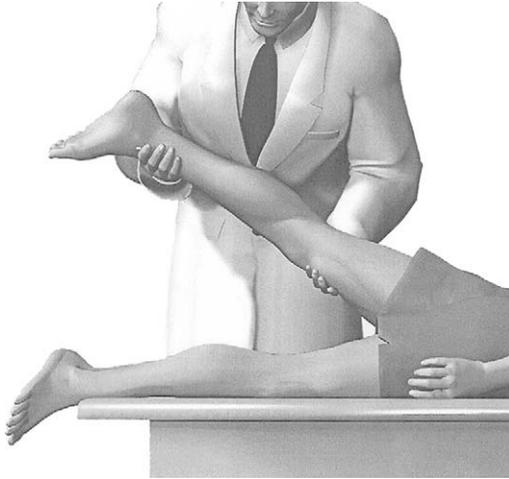


Fig. 6. Prone straight leg-raising test. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. *Continuum: Lifelong Learning Neurol* 2001;7:21; with permission.)

signs (see **Table 1**) usually leads to the correct diagnosis. When appropriate, an MRI, and an electromyogram (EMG) with nerve conductions can give further support to the diagnosis.⁵

WHAT ARE THE MOST COMMON CAUSES OF LUMBAR RADICULOPATHY?

Although there are many potential causes of lumbar radiculopathy (**Box 2**), the most common are acute disk herniation and spondylosis or a combination of the two.^{5,27,28} Most commonly, a herniated disk tethers the root exiting at the level below the disk herniation (L4-L5 disk herniation causing S1 radiculopathy).



Fig. 7. Faber maneuver. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. *Continuum: Lifelong Learning Neurol* 2001;7:22; with permission.)

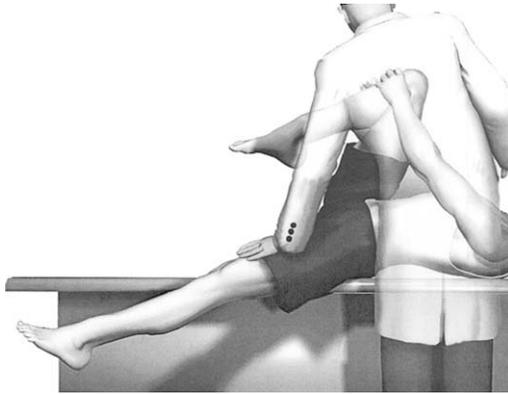


Fig. 8. Gaenslen test. (From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. *Continuum: Lifelong Learning Neurol* 2001;7:22; with permission.)

In the case of disk herniation, the distribution of symptoms and signs depends on the level of herniation, the location of the herniation (midline, paramedian, lateral), and the size of the herniation. Disk herniation is common in asymptomatic patients.^{25,26} Therefore in a symptomatic patient the presence of a herniated disk on MRI may be coincidental. It is imperative to link the symptoms with the identified herniated disk by a careful examination and additional tests (eg, EMG) where appropriate.

WHAT ARE THE CLINICAL MANIFESTATIONS OF LUMBAR CANAL STENOSIS?

Lumbar spinal stenosis can be caused by many conditions (**Box 3**).

Lumbar spinal stenosis is caused most commonly by spondylosis, at times superimposed over other causes of stenosis, particularly congenital. Spondylotic spinal stenosis often is asymptomatic. The presence and type of clinical manifestations reflect the degree of degenerative change and the location of the stenosis, affecting either the central canal of the spinal column or the lateral recesses of the canal. Symptomatic lateral canal stenosis typically presents as a radiculopathy. Although symptoms often are progressive at onset, they can appear suddenly as a result of an acute change, such as disk herniation superimposed over lateral stenosis.

Symptomatic central canal stenosis typically presents as neurogenic claudication/pseudoclaudication,^{5,27} This condition is characterized by aching pain, often associated with paresthesia in the lower extremities precipitated by walking or standing erect. Sitting or flexing the trunk while standing often relieves the symptoms. As is the case with vascular claudication, the symptoms dissipate after a period of rest, and the patient can walk again before the symptoms recur.

Neurogenic claudication can be confused with vascular claudication of the lower extremities. Together, the presence of paresthesia (and occasionally motor weakness), reflex changes, relief of symptoms with trunk flexion, and preservation of the distal pulses can help distinguish neurogenic claudication from vascular claudication. Both types of claudication are seen most often in the elderly, and the two can occur together.

Treatment includes counseling and avoidance of activity that brings on symptoms. In severe cases surgical decompression may be necessary.

Box 2**Causes of lumbosacral radiculopathy***Degenerative*

Disk herniation

Spondylosis

Neoplastic

Primary tumors

Metastatic tumors

Carcinomatous meningitis

Congenital/developmental

Arachnoid cyst

Synovial cyst

Spondylolisthesis

Infectious

Osteomyelitis

Epidural abscess

Tuberculosis

Herpes zoster

Lyme disease

HIV

Inflammatory

Sarcoidosis

Vasculitis

Endocrinologic/metabolic

Diabetic radiculopathy

Osteoporosis with vertebral fractures

Paget's disease

Acromegaly

Traumatic

Disk herniation

Vertebral fracture

Nerve root injury from spinal anesthesia

Arteriovenous malformation

Dural

Intradural

WHAT SHOULD BE THE APPROACH TO VERTEBRAL COMPRESSION FRACTURES?

Vertebral compression fracture is a common clinical problem in the elderly, usually secondary to osteoporosis and less frequently to osteolytic metastatic tumors and multiple myeloma.⁵ There are approximately 700,000 vertebral compression fractures annually in the United States.²⁹ Vertebral compression fractures are most common in

Box 3**Lumbar spinal stenosis**

Congenital

Acquired

Degenerative

Spondylosis

Spondylolisthesis

Adult scoliosis

Calcification of ligamentum flavum

Intraspinal synovial cysts

Spinal dysraphism

Metabolic/endocrinologic

Osteoporosis with fracture

Acromegaly

Renal osteodystrophy

Hypoparathyroidism

Epidural lipomatosis

Postoperative

Postlaminectomy

Postfusion

Postdiscectomy

Traumatic

Fracture

Spondylolisthesis

Miscellaneous

Paget's disease

Diffuse idiopathic skeletal hyperostosis

Amyloid

From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. Continuum: Lifelong Learning Neurol 2001;7:34; with permission.

the thoracolumbar region (T12, L1), but they can occur throughout the thoracic and lumbar spine. A precipitating event, such as a fall or an automobile accident, occurs in perhaps 10% to 15% of patients who have osteoporosis. In most cases, however, there is no definable event or activity. The result of a fracture often is disabling pain. Although pain may be acute in onset and severe even in the absence of a precipitating event, the pain also may have a subacute onset, building gradually over days. The pain usually is localized over the fractured vertebra but occasionally can radiate in a radicular distribution. The severe pain generally lasts weeks but can last for months, often followed by low-grade discomfort that may persist for additional months. The pain usually is relieved by lying supine and often is aggravated by sitting, standing, or bodily

motion. The diagnosis often is established by spine radiographs, although MRI and a technetium bone scan may be necessary to distinguish a pathologic fracture secondary to tumor from other causes.

Four treatment options for acute vertebral compression fractures are available:³⁰

Medical management consists primarily of bed rest, pain management, and mobilization as soon as feasible to reduce muscle wasting.

Open surgery, often with placement of hardware to stabilize the spine, has a limited role.

Vertebroplasty is a percutaneous injection of a bone-filler material to stabilize the fractured vertebra. This treatment addresses the pain but not the vertebral deformity and resultant spinal deformity.

Balloon kyphoplasty is a promising, minimally invasive procedure in which an inflatable bone tamp (balloon) is inserted into the vertebra and inflated, reducing the compression fracture. The device is removed, and a bone filler is injected into the cavity created by the inflated balloon, stabilizing the vertebra. This treatment has the advantage of reducing the vertebral and spine deformity, pain, and length of hospital stay.

WHAT IS SPONDYLOLISTHESIS, AND WHAT IS ITS ROLE IN LOW BACK PAIN?

Spondylolisthesis, from the Greek “spondylous” (spine) and “olisthesis” (slip), is a condition in which a vertebra slips generally anteriorly with respect to the inferior vertebra. This slippage is termed an “anterolisthesis.” When a vertebra slips posteriorly, the slippage is referred to as a “retrolisthesis.”¹² The degree of slippage often is described as one of four grades:

Grade I: 0 to 25%

Grade II: 26% to 50%

Grade III: 51% to 75%

Grade IV: more than 75%

The lower lumbar vertebrae (L4-L5, L5-S1) are the usual sites of spondylolisthesis.

Spondylolysis is related to spondylolisthesis. It is a defect in the pars interarticularis without slippage. It is a common finding, present in more than 5% of individuals older than age 7 years.³¹ It probably results from the combination of an inherited potential deficit in the pars and a stress fracture related to increased activity in later childhood. Spondylolysis is especially common in young athletes.³²

Five types of spondylolisthesis have been described (**Box 4**).^{5,33}

The most common type is isthmic spondylolisthesis, which is secondary to an acute fracture or deformity of the pars interarticularis. It has its greatest clinical impact in adults older than age 50 years. Other types of spondylolisthesis are caused by congenital disorders, degenerative changes, trauma, and pathologic fractures resulting from bone disease.

Although spondylolisthesis is a common cause of low back pain, it may remain asymptomatic for years. The onset of pain in a patient who has longstanding asymptomatic spondylolisthesis often is associated with an injury. Along with low back pain, spondylolisthesis may produce radiating pain into one or both lower extremities as a reflection of a build-up of a fibrocartilaginous mass at the site of the defect, with resultant nerve root compression at the level of the foramen.

Spondylolisthesis in the absence of neurologic compromise is treated much the same as nonspecific backaches from other causes. Acute low back pain secondary

Box 4**Types of spondylolisthesis**

Dysplastic: congenital defect of L5-S1 facets with resultant slippage

Isthmic

Abnormality in pars interarticularis (most common variety)

Fatigue fracture of the pars interarticularis

Elongated pars interarticularis

Acute fracture of the pars interarticularis

Degenerative: secondary to longstanding intersegmental instability

Traumatic: secondary to acute fracture of "bony hook" (pedicle, lamina, facets)

Pathologic: secondary to structural weakness of bones caused by localized or generalized disease

From Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. Continuum: Lifelong Learning Neurol 2001;7:28; with permission.

to spondylolisthesis can be treated with rest and anti-inflammatory medications. Spondylolisthesis may be one condition in which brace therapy can be justified, although this treatment is debated. Surgical decompression with fusion may prove necessary in patients who develop chronic pain and in particular neurologic compromise (radiculopathy, cauda equina syndrome).

CAN A DAMAGED INTERVERTEBRAL DISK PRODUCE PAIN IN THE ABSENCE OF HERNIATION?

The condition referred to as "internal disk disruption" (IDD) is a controversial disorder. Many believe that degenerative disk disease may lead to IDD and resultant localized spine pain in the absence of deformity of the disk as seen with disk herniation/protrusion. With regard to this entity, two things must be kept in mind: healthy young disks may not contain nociceptive nerve endings, and patients who have severe degenerative disk disease by MRI scan may have no back pain at all. IDD most commonly affects the L4-L5 and L5-S1 intervertebral disks.^{12,34} The pathophysiology underlying IDD is thought to be progressive annular deterioration and fissuring, ultimately involving the outer portions of the disk containing an ingrowth of nociceptive nerve endings.^{12,19,35} There are no specific symptoms or findings on general and neurologic examinations to distinguish IDD from other causes of nonradiating low back pain.

The controversial diagnosis of IDD is established by the also controversial procedure, diskography. Diskography is a diagnostic test in which contrast material is injected under fluoroscopy into the disk thought to be the cause of low back pain. This evaluation often is followed by a CT scan. If the injection reproduces the low back pain and the disk architecture is disrupted by annular tears, the test is considered positive, and the diagnosis of IDD is established. As already stated, there is a significant amount of controversy surrounding provocative diskography and its value in predicting the outcome of spinal fusion to alleviate low back pain.^{36,37}

WHEN IS DIAGNOSTIC TESTING INDICATED IN PATIENTS WHO HAVE LOW BACK PAIN, AND WHICH TESTS SHOULD BE PERFORMED?

Determining which diagnostic tests to perform and when to conduct them are among the more difficult decisions in the management of low back pain. As already stated, findings and diagnostic tests in a patient who has back pain may be misleading.^{5,25,26}

A safe generalization is that initially patients who have “uncomplicated” low back pain with or without radiating pain into a lower extremity in the absence of physical findings on examination can be observed without diagnostic testing.³⁸ If pain persists for several weeks to a month, and particularly if the pain worsens, baseline diagnostic tests may be indicated even in the absence of physical findings.

Blood tests may be indicated, depending on the clinical setting, and can include a complete blood cell count, erythrocyte sedimentation rate, antinuclear antibody, prostate-specific antigen, and a metabolic panel. For patients who have persistent nonradiating low back pain, spine radiographs ultimately may be indicated, although generally the yield is low.^{5,39} Nonetheless, spondylolisthesis, vertebral compression fractures, osteomyelitis/diskitis, and subtle evidence of metastatic cancer may be uncovered. In patients who have low back pain and neurologic findings, MRI has supplanted all other imaging techniques. In patients who have radiating pain, EMG and nerve conduction testing in skilled hands can prove useful, particularly when attempting to draw a correlation between symptoms, signs, and a specific abnormality on an MRI. Other diagnostic modalities, including bone scanning, CT scan/myelography, and possibly diskography (although this test remains controversial) may have a role; however, the primary care provider usually has called in a specialist before these tests prove necessary.

An immediate diagnostic work-up without delay usually is indicated when the presentation includes

- Fever
- Neuromuscular weakness
- Significant trauma before the onset of back pain
- Known malignancy
- Pain when recumbent
- Unexplained weight loss
- History of drug and/or alcohol abuse

WHAT IS THE BEST APPROACH TO TREATMENT FOR THE PATIENT WHO HAS ACUTE, NONRADIATING LOW BACK PAIN AND A NORMAL EXAMINATION?

Acute low back pain is an enormously common problem. It usually is self limiting. If the history and physical examination do not reveal any reason to support an early diagnostic work-up, treatment aimed at pain management and early mobilization is the goal.^{5,40} The natural history of acute low back pain favors conservative management.^{5,10,38,41,42} Deyo and colleagues⁹ noted that 50% of all patients resume normal activity in 4 to 6 weeks, and 95% return to normal activity in 6 months.

The literature supports the use of a short course of nonsteroidal antiinflammatory drugs (NSAIDs).⁴³ All NSAIDs seem to be equivalent for acute low back pain. In some patients acetaminophen may prove equally effective.⁴⁴ The analgesic and anti-inflammatory benefits of NSAIDs must be balanced against the potential side effects. Long-term use should be avoided. For severe acute low back pain, the addition of a muscle relaxant may prove helpful but preferably for only a short time.^{45,46} Non-benzodiazepine muscle relaxants such as cyclobenzaprine, methocarbamol, and carisoprodol are the best first choice.⁴⁶ Prolonged bed rest has not been shown to be of value, although bed rest for several days may be unavoidable for the patient who has severe back pain.^{40,47,48} In this case, the patient should be advised to resume physical activity as soon as possible.^{5,40}

Most treatment modalities for acute low back pain (eg, physical therapy,^{38,40,49,50} traction,^{51–53} hot or cold applications,^{52,54} epidural steroid injections,^{5,55}

transcutaneous electrical nerve stimulation^{56,57} and acupuncture⁵⁸) have not been proved conclusively to be effective. Some studies suggest a minor benefit from chiropractic manipulation, but it is controversial and is not cost effective.^{49,59–63} The best and most cost-effective treatments are

Medications: acetaminophen, NSAIDs, a short-term opiate analgesic if necessary, and possibly, at some point, a muscle relaxant

Bed rest: only if necessary because of severe pain, followed as soon as possible by a gradual return to activities of daily living

Hot or cold packs (whichever the patient chooses): sometimes helpful, although not scientifically validated (One should avoid recommending electric heating pads because of the risk of burns.)

Follow-up examinations: As deemed necessary to include psychological support and education

Physical therapy: May be of some value when the acute pain has dissipated

If pain persists, consider diagnostic testing and referral to a specialist when appropriate.

WHAT ARE THE AVAILABLE TREATMENT OPTIONS FOR CHRONIC LOW BACK PAIN?

A detailed description of treatment options is beyond the scope of this article. Management of chronic low back pain is difficult and demanding and generally is best shared by the primary care physician and one or more specialists. Patients who have chronic low back pain often have had one or more unsuccessful lumbar spine surgeries and often are categorized as having “failed back surgery syndrome” (“post lumbar laminectomy syndrome”). Often these patients are best treated in conjunction with a chronic pain management center.⁵ In addition to failed back surgery, these patients frequently have failed courses of epidural blocks and sometimes failed spinal cord stimulation implants.

“Proceduralism” should be avoided or at least controlled as much as possible. Although procedures for chronic low back pain may be well intentioned, they all too often are unsuccessful, sometimes resulting in complications such as chronic arachnoiditis secondary to multiple surgeries, infection, and neurologic impairment. No single treatment approach for chronic low back pain is 100% successful. The best treatment approach includes education, exercise and physical reconditioning, psychotherapy and behavioral modification, and medications.

Medications useful in the management of chronic pain syndromes are serotonin reuptake inhibitors, certain anti-epileptic drugs, and sometimes NSAIDs. One must recognize the risk of potential significant side effects. Long-term administration of opiates, if determined to be necessary, should be done under strict supervision, preferably by a specialist in chronic pain management. Given the risk of addiction as well as analgesic rebound pain, the use of potentially addictive reactive medications should be avoided in the treatment of chronic pain.

Treatment also may include treatment for chemical dependence, if necessary; surgery, if clear-cut indications are uncovered; or the use of a spinal cord stimulator.

WHEN SHOULD A PATIENT WHO HAS LUMBAR RADICULOPATHY SECONDARY TO DISK HERNIATION BE REFERRED FOR SURGERY?

To some extent, social and occupational circumstances continue to play a role in determining whether a patient who has lumbar radiculopathy secondary to disk

herniation requires surgery. A significant percentage of patients who have lumbosacral radiculopathy caused by a herniated disc can improve with medical/nonsurgical treatment.^{5,10,38,64–66} A recent study⁶⁵ demonstrated greater improvement 3 months after surgery in patients who had surgically treated lumbar radiculopathy secondary to a herniated disk than in the medically treated control group. At 1 year, however, there was no difference between groups. The medical treatment for lumbar radiculopathy includes reduced activity with pain avoidance and the judicious use of nonaddictive medications for pain management, including NSAIDs and several different types of glucocorticoid injections. These injections include epidural glucocorticoid injections, facet joint injections, and medial branch blocks. Glucocorticoid injections, although generally safe, are expensive and of limited value. In a recent report, the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology found that epidural steroid injections might result in some short-term improvement in radicular lumbosacral pain but did not recommend their routine use.⁶⁷ Studies supporting facet joint injections and medial branch blocks are small in number and inconclusive.^{68,69}

In the absence of neurologic findings, a rush to surgical treatment for patients who have radiating pain and lumbar disk herniation should be avoided if at all possible to allow time for a potential satisfactory response to medical therapy.^{5,10,38,64–66} Certainly a patient may become a surgical candidate in the following settings:

Low back pain radiating into a lower extremity not responding to conservative therapy

Neurologic deficits, particularly weakness in a radicular distribution (myotome) corresponding to the location/level of a herniated disk on a MRI scan

An EMG revealing active denervation in a radicular (myotome) distribution corresponding to the location/level of a herniated disk on a MRI scan even in the absence of neurologic deficits in a symptomatic patient

The presence of significant weakness in a specific myotome is perhaps the most important factor in the decision to perform a surgical procedure relatively early in the course of a patient who has sciatica. If the weakness is profound, delaying surgery increases the risk of permanent deficit, although all experienced neurologists, neurosurgeons, and orthopedic surgeons have seen occasional spectacular spontaneous recoveries of strength in patients who chose not to have surgery in this setting. Modest weakness often can resolve with conservative therapy. Unfortunately there are no foolproof criteria to guide the physician in the selection of patients for surgery.

To some degree modern neurosurgical techniques have made it easier to recommend surgery. A simple one-level lumbar laminectomy generally requires no more than a 24- to 36-hour hospitalization. This finding is not intended to suggest that surgery should be recommended only because of improvements in surgical technique. Studies suggest that spontaneous recovery without surgery occurs in 75% to 80% of all disk herniations.⁶⁴ Relief of nerve root impingement comes about in part from absorption and shrinkage of the displaced disk material, eliminating the need for surgery in many patients.⁶⁶

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