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Low Back Pain

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KEYWORDS

• Acute low back pain • Chronic low back pain • Risk factors • Cause • Diagnosis • Imaging • Treatment • Sciatica

KEY POINTS

- Low back pain is a common, frequently recurring condition that often has a nonspecific cause.
- History and physical examination should focus on evaluation for evidence of systemic or pathologic causes.
- Imaging is only indicated when there is evidence of neurologic deficits or red flags to suggest fracture, malignancy, infection, or other systemic disease, or when symptoms do not improve after 4 to 6 weeks.
- Most nonspecific low back pain will improve within several weeks with or without treatment.
- Back pain that radiates to the lower extremities, occurs episodically with walking or standing erect, and is relieved by sitting or forward spine flexion is typical of neuroclaudication and suggests central spinal stenosis.
- All patients with acute or chronic low back pain should be advised to remain active.
- The treatment of chronic nonspecific low back pain involves a multidisciplinary approach targeted at preserving function and preventing disability.
- Urgent surgical referral is indicated in the presence of severe or progressive neurologic deficits or signs and symptoms of cauda equina syndrome.

INTRODUCTION

Low back pain affects a significant proportion of the population.[1–5] The precise incidence and prevalence of low back pain are difficult to characterize due to significant heterogeneity in the epidemiologic studies. In a survey of Saskatchewan adults, 84% of participants reported experiencing at least one episode of back pain in their lifetime.[6] A 2002 US National Health Interview Study found that 26.4% of the 30,000 participants had experienced at least one full day of back pain in the past 3 months.[7] A 2010 review article reported 1-year incidences of first time, any time, and recurrent

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low back pain episodes as ranging from 1.5% to 80%, and the 1-year prevalence of low back pain ranging from 0.8% to 82.5%.[8] These findings are summarized in Table 1.

The incidence of low back pain peaks in the third decade of life. The prevalence increases until age 60 to 65 and then gradually declines.

Commonly reported risk factors for low back pain include physical, psychological, social, and occupational factors and are summarized in Table 2.[2,6]

Low back pain has an enormous social and economic impact. It is a leading cause of work absenteeism globally and the second most common cause of missed work days in the United States.[9,10] Direct medical costs attributed to the evaluation and treatment of low back pain are estimated to exceed $33 billion annually in the United States. When the indirect costs of missed work and decreased productivity are added, the total costs exceed $100 billion each year.[2]

Primary care providers play a key role in the evaluation and treatment of low back pain. Indeed, low back pain is the chief complaint in about 2.3% of all ambulatory physician visits, representing about 15 million office visits per year, and is second only to upper respiratory symptoms as a symptom prompting office evaluation.[7]

PATHOPHYSIOLOGY

Anatomy

There are 5 lumbar vertebrae, each of which is composed of a vertebral body, 2 pedicles, 2 lamina, 4 articular facets, and a spinous process. Between each pair of vertebrae are the foramina, openings through which pass the spinal nerves, radicular blood vessels, and sinuvertebral nerves. The spinal canal is formed anteriorly by the posterior surface of the vertebral bodies, intervertebral discs, and posterior longitudinal ligament, laterally by the pedicles, and posteriorly by the ligamentum flavum and lamina (Fig. 1).

In the normal spine, the anterior structures including the vertebral bodies and intervertebral discs perform weight-bearing and shock-absorbing functions. The posterolateral structures, including the vertebral arches, lamina, transverse, and spinous processes, provide protection for the spinal cord and nerve roots. Balance, flexibility, and stability are provided by the facet joints and paraspinous muscles and ligaments.

Physiology

Low back pain is often characterized in terms of radiologic findings (spondylosis, spondylolisthesis, spondylolysis) and clinical and neurologic findings (lordosis, kyphosis, radiculopathy, sciatica). These terms are defined in Table 3.

Table 1
Incidence and prevalence of low back pain episodes

Low Back Pain (LBP) Episode	Incidence or Prevalence
1-y incidence of first ever LBP episode	6.3%–15.4%
1-y incidence of any LBP episode	1.5%–36%
1-y incidence of recurrent LBP episode	24%–80%
Point prevalence of LBP episodes	1.0%–58.1% (mean 18.1%, median 15.0%)
1-y prevalence of LBP episodes	0.8%–82.5% (mean 38.1%, median 37.4%)

Data from Hoy D, Brookes P, Blyth F, et al. The epidemiology of low back pain. Best Pract Res Clin Rheumatol 2010;24(6):769–81.

Table 2
Risk factors for development of low back pain

Physical Factors	Psychological Factors	Social Factors	Occupational Factors
Older age	Depression	Low educational achievement	Physically or psychologically strenuous work
Female gender Obesity Smoking	Anxiety Somatization disorder	Increased life stress	Sedentary work Whole body vibration Low social support in the workplace Job dissatisfaction Workers compensation insurance

Experimental studies indicate that mechanical low back pain can originate in one or more of the many structures of the spine, including ligaments, facet joints, intervertebral discs, paravertebral musculature and fascia, and spinal nerve roots.

Acute Low Back Pain

Acute low back pain occurring after physical activity most likely results from increased paraspinous muscle tension with resultant avulsion of tendinous attachments between the muscles and bone, or tearing of muscle fibers/sheaths. Persistent muscle overuse, particularly of untrained or poorly conditioned muscles, can cause tonic contraction (spasms).[11] Ligament sprains are another common cause of acute low back pain and occur when the ligament is stretched beyond its physiologic range.

Chronic Low Back Pain

In chronic low back pain, the most common source of pain is thought to be degenerative changes of the bony structures and ligaments. That said, arthritis of the spine, termed "spondylosis," seems to be a naturally occurring process. By age 49 years, 60% of women and 80% of men have osteophytes and other changes that indicate early spondylosis; by age 79, nearly all individuals have evidence of spondylosis on plain radiographs.[12,13] In addition, there is poor correlation between the presence of spondylosis, including disc herniation, on imaging studies, and clinical pain syndromes (Fig. 2).[12,13]

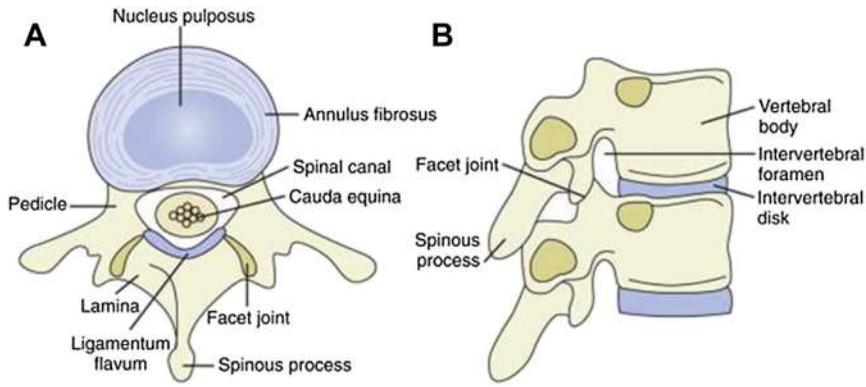


Fig. 1. Anatomy of the lumbar spine. (*A*) Cross-sectional view through a lumbar vertebra. (*B*) Lateral view of the lumbar spine. (*From* Firestein GS, Budd RC, Gabriel SE, et al. Kelley's textbook of rheumatology. Philadelphia: Saunders; 2013. p. 666; with permission.)

Table 3
Commonly used terms in low back pain

Term	Definition
Spondylosis	Osteoarthritis of the spine; evidenced by disc space narrowing and/or arthritic changes of the facet joints on radiographs
Spondylolisthesis	Anterior displacement of a vertebra in relation to the one beneath it. Displacement is graded 1–IV as follows: Grade I: 1%–25% slip; generally nonsurgical Grade II: 26%–50% slip; generally nonsurgical Grade III: 51%–75% slip; may be surgical Grade IV: 76%–100% slip; may be surgical
Spondylolysis	Fracture in the pars interarticularis of the vertebral arch (the joining of the vertebral body to the posterior structures), usually at L5. This is a congenital variant in 3%–6% of people
Spinal stenosis	Local, segmental, or generalized narrowing of the central spinal canal by bone or soft tissue elements, usually bony hypertrophy of the facet joints or thickening of the ligamentum flavum
Radiculopathy	Pain, sensory, and/or motor deficits resulting from compression of a spinal nerve root
Sciatica	Pain, numbness, or tingling in the sciatic nerve distribution, radiating down the posterior or lateral aspect of the leg often to the foot, due to compression of the sciatic nerve or its component nerve roots
Cauda equina syndrome	Loss of bowel or bladder control, numbness in the groin or saddle region of the perineum, and lower extremity weakness caused by compression of the inferior-most part of the spinal cord or spinal nerve roots due to canal stenosis or a large herniated disc
Kyphosis	Outward (convex) curve of the spine; there is a normal small thoracic kyphosis (at the level of the ribs)
Lordosis	Inward (concave) curve of the spine; there is a normal small lumbar lordosis
Scoliosis	Sideways (lateral) curve of the spine, always abnormal

The facet joints are true synovial joints and therefore are subject to develop degenerative or inflammatory changes. The resultant bony enlargement of these joints is thought to cause facet-mediated arthritic pain and can contribute to canal stenosis along with thickening of the ligamentum flavum.[14]

There is some debate about the role of internal disc degeneration or disruption, referring to degenerative changes of the annulus fibrosis (elastic collagen ring) and nucleus pulposus (gelatinous inner contents of the disc, surrounded by the annular fibrosis). Internal disc degeneration has been proposed to cause primary discogenic back pain. However, the nucleus pulposus has no nerve supply, and the nerve endings that enter the annulus fibrosis do not contain substance P and are not considered nociceptors,[15] leaving uncertainty regarding the pathophysiology of disc-related pain. Some have observed that new nerves and blood vessels can grow into the damaged annulosis fibrosis and propose that this neogrowth may be the source of discogenic pain.[16] Provocative discography, a procedure in which pain level is assessed while contrast material is injected into a disc, has been used to diagnose primary discogenic pain. However, this procedure can cause pain in people with normal discs and does not induce pain in all people with degenerated discs, leaving further questions regarding the clinical significance of internal disc degeneration and source of discogenic pain.[17]

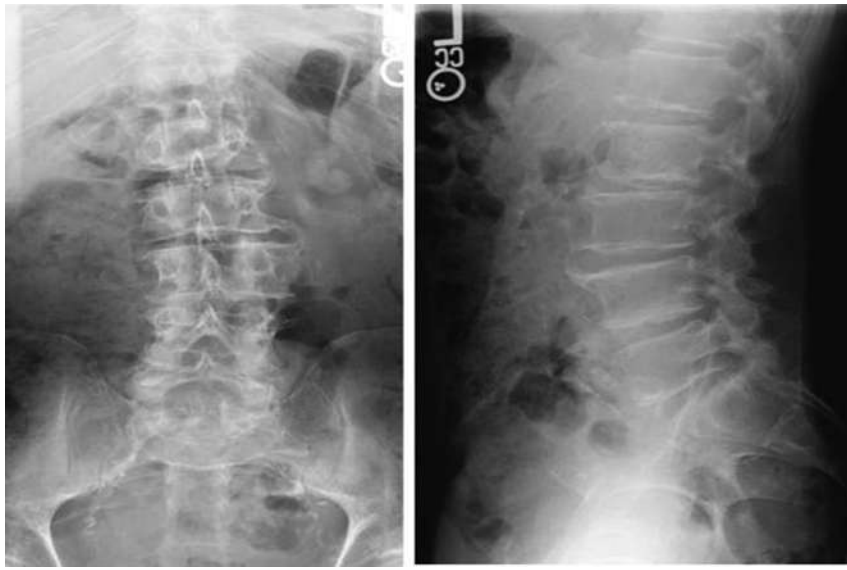


Fig. 2. Spondylosis and scoliosis of the lumbar spine. Anteroposterior and lateral radiographs of the lumbar spine showing mild levoconvex scoliosis with apex L2/3, multilevel disc space narrowing, endplate spurring, and lumbar facet arthropathy.

Radicular low back pain is pain that radiates into the lower extremity and is caused by compression and/or inflammation of a spinal nerve root. Sciatica refers to compression of the sciatic nerve, but is also commonly used to describe radicular back pain radiating into the lower extremities distal to the knee. Spinal nerve compression occurs most commonly from disc herniation or spondylosis, causing foraminal narrowing, and less commonly from benign or malignant tumors or epidural abscesses. The lumbar discs are at higher risk of herniation than cervical and thoracic discs partly because of the increased static and kinetic stress at this level, but also because the posterior longitudinal ligament, which forms the anterior wall of the spinal canal, is only half as wide along the lumbar vertebra as it is more superiorly, thus providing inadequate reinforcement of the lumbar discs. L5 and S1 radiculopathies are most common, comprising more than 90% of lumbosacral radiculopathies (**Figs. 3** and **4**).

Spinal stenosis refers to narrowing of the central spinal canal, most commonly caused by spondylosis, which is often asymptomatic. If symptomatic, the clinical manifestations of spinal stenosis vary by the degree of stenosis and its location. It is most commonly caused by degenerative spondylosis and as a result is usually seen in people over the age of 60. Symptomatic stenosis affecting the lateral aspect of the canal usually presents as a radiculopathy, whereas symptomatic stenosis affecting the central region of the canal presents as neurogenic claudication, also called "pseudoclaudication." This condition is characterized by aching pain or paresthesia in one or both lower extremities that comes on with standing upright or walking and is improved with rest or forward flexion (eg, relieved while pushing a shopping cart). It can be mistaken for vascular claudication, which also improves with rest. The two can be distinguished in that vascular claudication does not improve with forward flexion alone and should not include paresthesias, motor weakness, reflex changes, or intact distal pulses (**Fig. 5**).

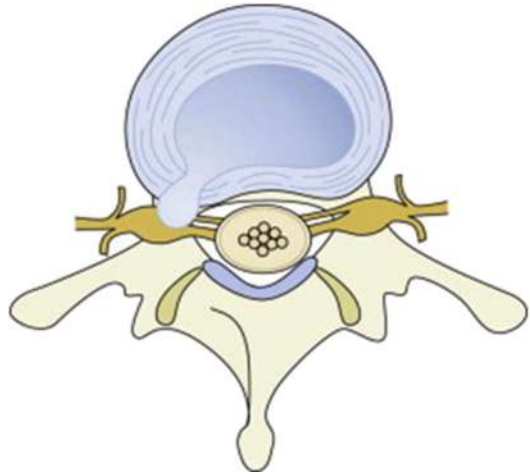


Fig. 3. Schematic drawing showing posterolateral disc herniation resulting in nerve root impingement. (*From* Firestein GS, Budd RC, Gabriel SE, et al. Kelley's textbook of rheumatology. Philadelphia: Saunders; 2013. p. 670; with permission.)

Spondylolisthesis is a condition in which a vertebra slips forward with respect to the vertebra beneath it. It is graded I–IV based on severity, as described in **Table 3**. Spondylolisthesis is caused by fractures or deformities of the pars interarticularis (congenital, traumatic, or pathologic), and degenerative changes. The lower lumbar vertebrae

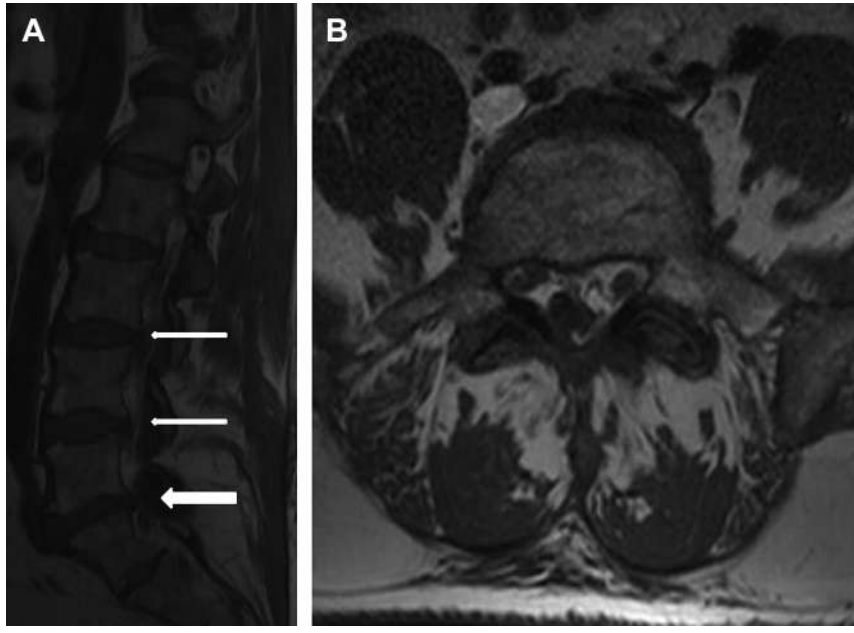


Fig. 4. Disc bulge. (*A*) T1-weighted sagittal and (*B*) T2-weighted axial MRI showing diffuse disc bulges at levels L3-4 and L4-5 (*thin arrows*) and posterior central disc extrusion at L5-S1 (*thick arrow*) resulting in narrowing of the left lateral recess that contacts the traversing left S1 nerve root.

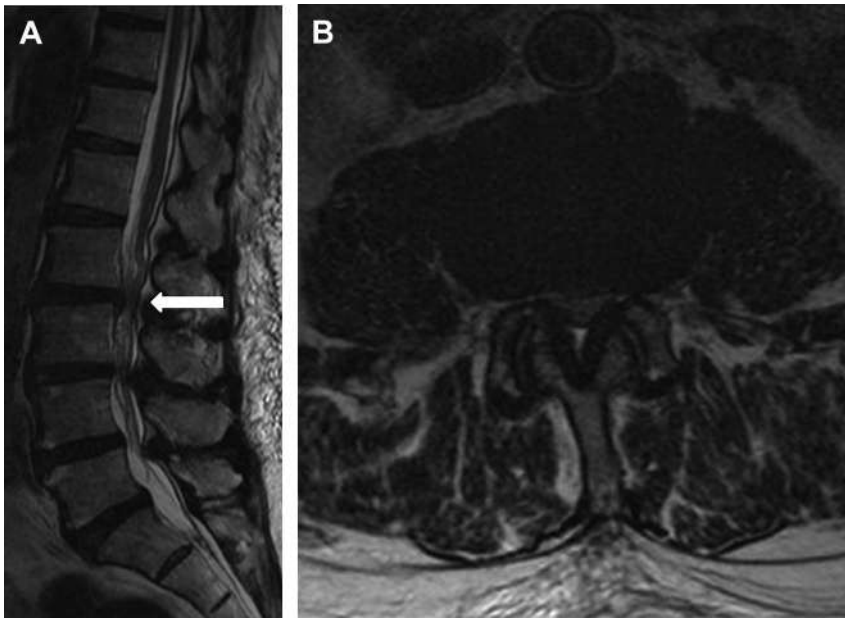


Fig. 5. Degenerative spinal stenosis. (*A*) T1-weighted sagittal and (*B*) T2-weighted axial MRI showing severe dural compression at L2-3 (*arrow*) secondary to severe facet and ligamentum flavum hypertrophy and circumferential disc bulge with caudal extension of the central disc extrusion. Severe dural compression at L3-4 and moderate dural compression at L4-5.

including L4-5 and L5-S1 are the most frequent sites of spondylolisthesis. If there are no neurologic signs or symptoms, and the grade of slippage is I or II, spondylolisthesis is treated conservatively, much like other causes of chronic mechanical low back pain. If there is neurologic compromise or grades III or IV slippage, the patient should be referred for surgical evaluation.

Spondylolysis refers to a defect in the pars interarticularis without vertebral slippage. It is common, is found in more than 5% of people older than age 7, and typically is asymptomatic. It is thought to result from a congenital defect in the pars with or without a stress fracture related to childhood activity (**Figs. 6** and **7**).[18]

Differential Diagnosis

One approach to organizing the differential diagnosis of low back pain is to consider it in terms of nonspecific "mechanical" low back pain versus back pain with lower extremity symptoms versus systemic and visceral diseases, as shown in **Table 4**.

By far the most common causes of low back pain are mechanical, representing about 97% of patients. In clinical practice, it is often difficult to determine the precise source of a patient's mechanical back pain. In fact, Deyo and Weinstein[17] have reported that a definitive diagnosis cannot be made in up to 85% of patients due to the weak association between symptoms, pathologic changes, and findings on imaging. The inability to make precise diagnoses results in the frequent use of nonspecific diagnostic terms, such as sprain, strain, spasm, and degenerative changes.

There are also nonmechanical causes of low back pain, including neoplasms, infections, and inflammatory conditions, as listed in **Table 4**. Nonmechanical causes of back pain are usually accompanied by systemic signs and symptoms or a severe,

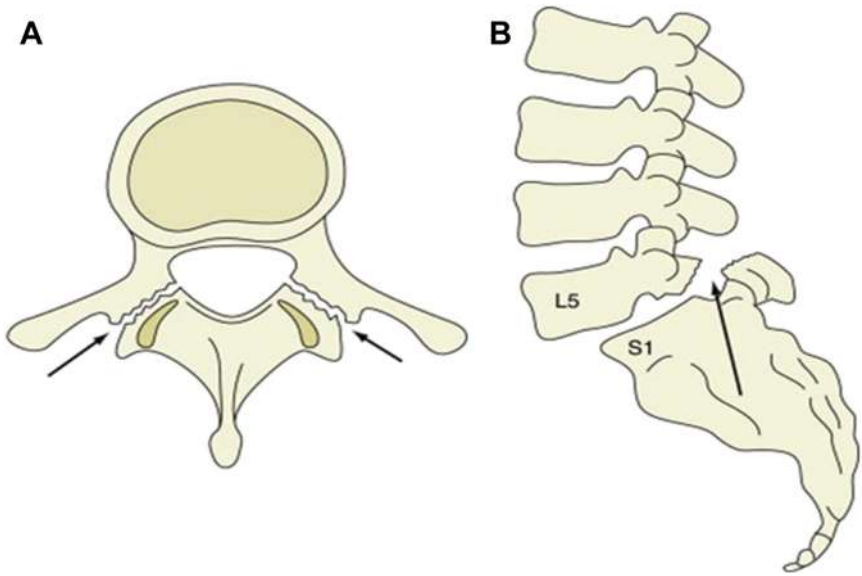


Fig. 6. (*A*) Spondylolysis with bilateral defects in the pars interarticularis (*arrows*). (*B*) Spondylolysis of the L5 vertebra (*arrow*) resulting in isthmic spondylolisthesis at L5-S1. (*From* Firestein GS, Budd RC, Gabriel SE, et al. Kelley's textbook of rheumatology. Philadelphia: Saunders; 2013. p. 672; with permission.)

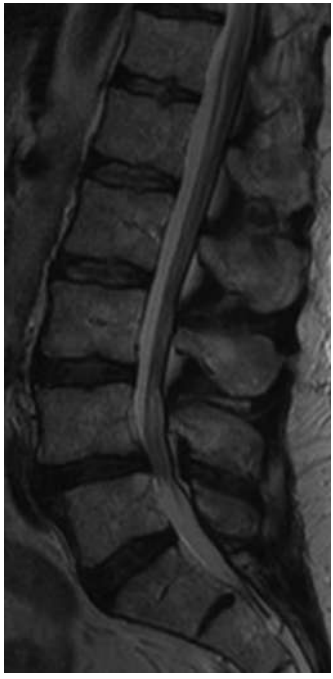


Fig. 7. Spondylolisthesis. T1-weighted sagittal MRI showing grade 1 anterolisthesis of L4 on L5, likely degenerative.

Table 4
Differential diagnosis of low back pain and estimated prevalence of each condition in primary care practice

Nonspecific "Mechanical" Low Back Pain (97%)	Back Pain with Lower Extremity Symptoms	Systemic and Visceral Diseases
Idiopathic musculoligamentous strain/ sprain (70%)	Disc herniation (4%)	Neoplasia (0.7%) • Multiple myeloma • Metastatic carcinoma • Lymphoma/leukemia • Spinal cord tumors • Retroperitoneal tumors
Disc/facet degeneration (10%)	Spinal stenosis (3%)	Infection (0.01%) • Osteomyelitis • Septic discitis • Paraspinous abscess • Epidural abscess • Shingles
Osteoporotic compression fracture (4%)	—	Inflammatory disease (0.03%) • Anklyosing spondylitis • Psoriatic spondylitis • Reactive arthritis • Inflammatory bowel disease
Spondylolisthesis (2%)	—	Visceral disease (0.05%)
Severe scoliosis, kyphosis, asymmetric transitional vertebrae (<1%)	—	• Prostatitis • Endometriosis • Chronic pelvic inflammatory disease • Nephrolithiasis • Pyelonephritis • Perinephric abscess • Aortic aneurysm • Pancreatitis • Cholecystitis • Penetrating ulcer
Traumatic fracture (<1%)	—	Other • Osteochondrosis • Paget's disease

Adapted from Wipf JE, Deyo RA. Low back pain. In: Branch WT, editor. The office practice of medicine. 3rd edition. Philadelphia: Saunders; 1994. p. 646.

rapidly progressing course. Visceral organ pain, including bowel, kidney, and pelvic organ pain, can also be referred to the spine. Overall, nonmechanical spine conditions and referred visceral organ pain are much less common causes of low back pain than mechanical causes. In fact, fewer than 5% of all primary care patients with low back pain will have a serious systemic pathologic condition.

DIAGNOSTIC EVALUATION

Given that a precise anatomic cause for low back pain usually cannot be found, the primary objectives in the diagnostic evaluation of the patient with low back pain are to evaluate for evidence of systemic disease or neurologic compromise that may require further workup or surgical evaluation, and to probe for factors that may predispose the patient to a prolonged course or chronic pain syndrome. These objectives can usually be met by taking a thorough history and physical examination.

Patient History

When assessing a patient with low back pain, providers should ask about time course, precipitating factors (trauma), location, character, severity, radiation, and exacerbating and alleviating factors. Most patients presenting with acute low back pain have a prior history of low back pain to which the current episode can be compared. Many, but not all, patients will recall an inciting activity that may have exacerbated the current flare. Most mechanical back pain is relieved by lying down and is not bothersome at night. Pain that is not relieved by lying down is more likely to be caused by malignancy or infection, but this is not a specific finding for these conditions. The likelihood of spinal infection is increased in patients with a history of injected drug use, skin or soft tissue infections, urinary tract infections, or fever.

Mechanical pain typically localizes to the paraspinal regions, occasionally spreading to the flanks or buttocks, but does not radiate into the legs. Radicular or sciatic pain radiates into the lower extremities and may be associated with paresthesias, sensory loss, motor weakness, or decreased reflexes. The distribution of pain and associated symptoms can help identify the nerve root involved. **Table 5** lists the signs and symptoms of the lumbar radiculopathies by nerve root. Radiculopathy syndromes caused by disc herniation often worsen with cough, sneeze, or Valsalva maneuvers.

Back pain that radiates to the lower extremities, occurs episodically with walking or standing erect, and is relieved by sitting or forward spine flexion is typical of neurogenic claudication and suggests central spinal stenosis (must also consider vascular claudication).

The presence of radicular symptoms or neurogenic claudication suggests neurologic involvement, from either disc herniation or spinal stenosis, but can often be managed conservatively. However, the presence of bowel or bladder dysfunction may signal severe compression of the cauda equina, as do saddle anesthesia, bilateral leg numbness, and back pain. The cauda equina syndrome is usually caused by massive midline disc herniation, but can also be caused by tumor or abscess

Table 5
Signs and symptoms of lumbar radiculopathies by nerve root

Root	Pain Distribution	Dermatomal Sensory Distribution	Motor Weakness	Affected Reflex
L1	Inguinal region	Inguinal region	Hip flexion	Cremasteric
L2	Inguinal region Anterior thigh	Anterior thigh	Hip flexion Hip adduction	Cremasteric Thigh adductor
L3	Anterior thigh 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 Great toe	Foot/toe dorsiflexion Knee flexion Hip adduction	—
S1	Posterior thigh Posterior leg Lateral foot	Posterolateral leg Lateral aspect of foot	Foot/toe plantar flexion 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.

compressing the cauda equina. Of note, progressive neurologic deficits or suspected cauda equina syndrome or cord compression requires emergent surgical evaluation.

Historical red flags that may signal systemic disease include a personal history of cancer, advanced age, unexplained fever or weight loss, duration of pain greater than 4 weeks, pain occurring at night, or pain that has not responded to previous therapies. A list of these red flags is summarized in **Table 6**.

Even in the absence of neurologic compromise or systemic disease, some patients are more likely than others to have a prolonged pain and disability course, including patients with comorbid depression or anxiety, somatization disorder, substance abuse, job dissatisfaction, pursuit of disability compensation, and involvement in litigation.[19,20] When evaluating a patient with back pain, it is important to assess for the above psychosocial factors and emotional distress level as these factors are stronger predictors of outcomes than pain characteristics and physical examination findings.[21] Some authors now advocate using a prognostic tool to help determine which patients would benefit from earlier, structured treatments to decrease the development of prolonged pain and disability (see Treatment of Acute Back Pain section).[22]

Physical Examination

A general physical examination should be performed in all patients presenting with back pain, including careful examination of the abdomen given the possibility of visceral organ pain radiating to the spine, and special attention to potential malignant sources (breast, prostate, lymph nodes) or infectious sources (flank or suprapubic pain, skin or soft tissue infection, track marks, heart murmur) if the patient history raises concern for systemic disease.

The examination of the back should include inspection of the spine and patient posture, range of motion, and palpation of the spine and paraspinous structures. Spinal inspection may reveal scoliosis, kyphosis, or lordosis. Lumbar spine mobility is often reduced in patients presenting with low back pain. It is not useful as a tool to differentiate causes of low back pain because it varies widely between individuals, but may be useful to establish a baseline for the individual from which to compare response to therapies. Spinal pain that is reproduced by palpation or percussion may indicate spinal infection, but this is a sensitive, not specific, test, and interexaminer reproducibility is poor.[23]

For patients with lower extremity symptoms, a straight leg raising test and full neurologic assessment, as well as palpation of the pedal pulses to help distinguish neurologic from vascular claudication, should be performed.

Table 6
Red flags for serious or systemic cause of low back pain

Patient Factors	Pain Characteristics	Associated Signs/Symptoms
History of trauma	Nighttime pain	Unexplained weight loss
History of cancer	Duration greater than 4–6 wk	Unexplained fevers
Age >50 y	Unresponsive to conservative therapies	Comorbid infection such as urinary tract infection
History of osteoporosis or prolonged corticosteroid use		Focal neurologic deficits with progressive or disabling symptoms
Injection drug use		Cauda equina syndrome
Immunosuppression		
Diabetes		

The straight leg raising test helps to confirm radiculopathy. It is performed with the patient in a supine position. The examiner slowly raises the affected leg off the table with the foot dorsiflexed. The test is positive when radicular pain is reproduced between 30° and 70° of hip flexion (**Fig. 8**). The crossed straight leg raising test is performed by elevating the unaffected leg and is deemed positive when lifting the unaffected leg reproduces symptoms in the affected leg. The straight leg test is sensitive (73%–98% sensitivity), but not specific (11%–61% specificity), for herniated discs. The crossed straight leg test is less sensitive for herniated discs, but 90% specific.[24,25]

Other neuromechanical tests that may be performed in patients with pain radiating into the lower extremities are summarized in **Table 7**.

Neurologic testing for patients with lower extremity symptoms should focus on the L5 and S1 nerve roots, because more than 95% of disc herniations occur at these levels. Testing should include evaluation of muscle strength, sensation, and reflexes at each level (**Fig. 9** summarizes the signs and symptoms associated with compression of each lumbar nerve root).

The L5 nerve root motor function can be tested by evaluating the strength of foot and great toe dorsiflexion. The L5 nerve root sensory function can be tested by evaluating sensation of the medial foot and the space between the first and second toe. There is no reflex associated with the L5 nerve root.

The S1 nerve root function is tested by evaluating sensation at the posterior calf and lateral foot and by eliciting the Achilles reflex. Of note, loss of Achilles (ankle) reflexes often occurs with advancing age even in the absence of nerve root compression. In one study, bilateral ankle reflexes were found to be absent in 30% of individuals between the ages of 61 and 70, and in more than 50% of those aged 81 to 90.[26] Therefore, absent ankle reflex is more likely to be clinically meaningful if it is unilateral and affects the symptomatic leg. The S1 nerve root motor function is tested by evaluating strength of foot plantar flexion; however, weakness of plantar flexion is a late finding.

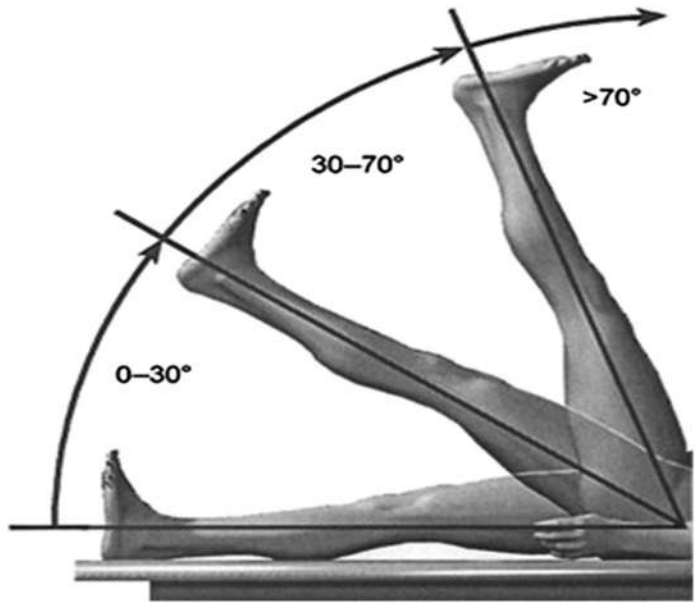


Fig. 8. Straight leg raising test. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and back pain. Continuum: Lifelong Learning Neurol 2001;7:20; with permission.)

Table 7
Neuromechanical tests useful in evaluating the patient with back pain radiating into the lower extremities

Test	Description
Straight leg raising test	With the patient in the supine position, the examiner raises the symptomatic extremity slowly off the examining table. The test is positive when the radicular symptoms are reproduced when the extremity is elevated between 30° and 70°.
Lasegue test	With the patient in the supine position, the symptomatic lower extremity is flexed to 90° at the hip and knee. The knee is then extended slowly, which produces radiating pain as a result of L5 and S1 nerve root compression.
Bragard sign	A follow-up to a positive straight leg test. If pain is generated by straight leg raising, the symptomatic extremity is lowered until the pain recedes. At that point the foot is dorsiflexed. If this maneuver reproduces radicular pain, the test is positive.
Contralateral (crossed) straight leg raising test	With the patient in supine position, the examiner raises the unaffected extremity. The test is positive if this maneuver causes pain in the affected extremity.
Prone straight leg raising test	With the patient in prone position, the symptomatic extremity is slowly extended at the hip by the examiner. If this exacerbates pain in the anterior thigh, a high lumbar radiculopathy (L2-3) is suggested.
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 exacerbates radicular pain.
Patrick (Faber) test	The lateral malleolus of the symptomatic extremity is placed on the patella of the opposite extremity, and the symptomatic extremity is slowly rotated externally. Accentuation of pain suggests that pain is caused by a hip or sacroiliac joint lesion 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. 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.

Adapted from Devereaux M. Low back pain. Med Clin North Am 2009;93(2):488–489; with permission.

Imaging and Additional Testing

A judicious approach to imaging in patients with low back pain is recommended for many reasons. First, most patients with nonspecific mechanical low back pain or radiculopathy will recover spontaneously within 4 to 6 weeks. Second, abnormalities on imaging have been shown to correlate poorly with clinical symptoms. In fact, imaging abnormalities have been found in about 20% of people in the absence of low back

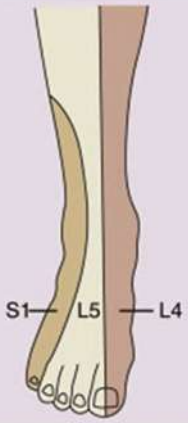
Lower extremity dermatome	Disc	Nerve root	Motor loss	Sensory loss	Reflex loss
	L3-4	L4	Dorsiflexion of foot	Medial foot	Knee
	L4-5	L5	Dorsiflexion of great toe	Dorsal foot	None
	L5-S1	S1	Plantarflexion of foot	Lateral foot	Ankle

Fig. 9. Neurologic features of lumbosacral radiculopathy. (*From* Firestein GS, Budd RC, Gabriel SE, et al. Kelley's textbook of rheumatology. Philadelphia: Saunders; 2013. p. 668; with permission.)

pain.[13] Given these findings, abnormalities detected on imaging may or may not be clinically relevant to the patient's current symptoms. Furthermore, they typically do not alter treatment strategy, may cause patient distress, and may lead to further unnecessary tests and procedures. In addition, obtaining unnecessary radiographs and computed tomography (CT) scans exposes patients to potentially harmful radiation and contributes to the economical burden of low back pain.

As a result, joint guidelines from the American College of Physicians (ACP) and the American Pain Society explicitly state: "Clinicians should not routinely obtain imaging or other diagnostic tests in patients with nonspecific low back pain."[27] The guidelines advise that diagnostic imaging is only indicated for patients with signs or symptoms of severe neurologic deficit or serious underlying disease (summarized in **Table 8**). Other patients may be imaged if they do not have improvement in their back pain after 4 to 6 weeks or if they develop any red flags.[27,28]

Table 8
Indications for diagnostic imaging in patients with low back pain and recommended initial imaging modality

Characteristic	Initial Imaging Modality
Progressive neurologic findings	Magnetic resonance imaging
Constitutional symptoms (fever, chills, weight loss)	Plain radiographs
History of traumatic onset	Plain radiographs
History of malignancy with new onset pain	Magnetic resonance imaging
Age >50 y	Plain radiographs
Infectious risk, such as injection drug use, immunosuppression, indwelling urinary catheter, prolonged steroid use, skin or urinary tract infection	Magnetic resonance imaging
Osteoporosis	Plain radiographs
Radiculopathy or pseudoclaudication persisting for more than 4–6 wk	Magnetic resonance imaging

If there is a concern for serious underlying pathologic condition or pain has not improved after 4 to 6 weeks, plain anteroposterior and lateral radiographs of the lumbosacral spine may be useful in evaluating for tumor, infection, spinal instability, spondylosis, and spondylolisthesis.

CT and magnetic resonance imaging (MRI) are more sensitive than plain radiographs in the early detection of malignancy and infection. Both modalities can also show herniated discs and stenosis; however, MRI is more sensitive for infections, metastatic cancer, and rare neural tumors and is preferred when available because of better visualization of soft tissues and avoidance of radiation. CT or MRI should be obtained when a patient has progressive neurologic deficits, findings highly concerning for malignancy or infection, or unexplained pain persisting for 12 weeks or longer. For patients with a typical radiculopathy syndrome persisting beyond 6 weeks, MRI should only be obtained if the patient is a candidate for a procedure such as corticosteroid injection or surgery.

For patients in whom an underlying serious or systemic cause for low back pain is suspected, it is also advisable to obtain specific blood and urine tests to aid in the diagnosis, which may include a complete blood count, erythrocyte sedimentation rate, antinuclear antibody with reflexive testing, prostate-specific antigen, a metabolic panel, blood cultures, urinalysis, and/or urine cultures.

For patients in whom there is a need to distinguish spinal stenosis or radiculopathy from a peripheral neuropathy syndrome, it may be helpful to obtain electromyography and nerve conduction testing. Ankle-brachial indices and arterial duplex studies may help differentiate vascular from neurogenic claudication.

Fig. 10 shows a diagnostic algorithm regarding the evidence-based evaluation and initial treatment of low back pain.

TREATMENT FOR ACUTE LOW BACK PAIN

It is important for providers to reassure their patients with acute nonspecific low back pain with or without radiculopathy that most people have significant improvement of their symptoms within 4 to 6 weeks without any specific treatment.[29] In fact, up to 90% of patients seen within 3 days of onset will recover after 2 weeks.[30] For patients with radiculopathy, prognosis is also generally favorable, although speed of recovery is usually slower: about one-third of patients are improved at 2 weeks, and about 75% by 3 months.[31] Patients with spinal stenosis are more likely to have chronic symptoms: in one small study of 32 patients with spinal stenosis followed for a mean of 49 months without surgical intervention, 15% had symptom improvement, 15% symptom worsening, and 70% unchanged symptoms.[32]

Although most patients have favorable outcomes without intervention, some are at higher risk for prolonged disability, including those with comorbid depression or anxiety, poor coping skills, job dissatisfaction, and higher initial disability levels. Recent studies have shown evidence for improvement in patient outcomes and resource utilization when initial treatment recommendations are stratified according to patient prognosis based on the above risk factors.[22] Therefore, it may be advisable for clinicians to use a prognostic tool to help identify patients who would benefit from earlier targeted interventions in addition to self-care advice. One validated prognostic tool is the Keele STarT Back Screening Tool,[33] shown in **Figs. 11** and **12**.

Hill and colleagues[22] found that patients randomized to targeted interventions based on the Keele prognostic score (low-risk patients received self-care advice, medium-risk patients were referred to physical therapy, and high-risk patients were referred to cognitive behavioral therapy-enhanced physical therapy) had statistically significant

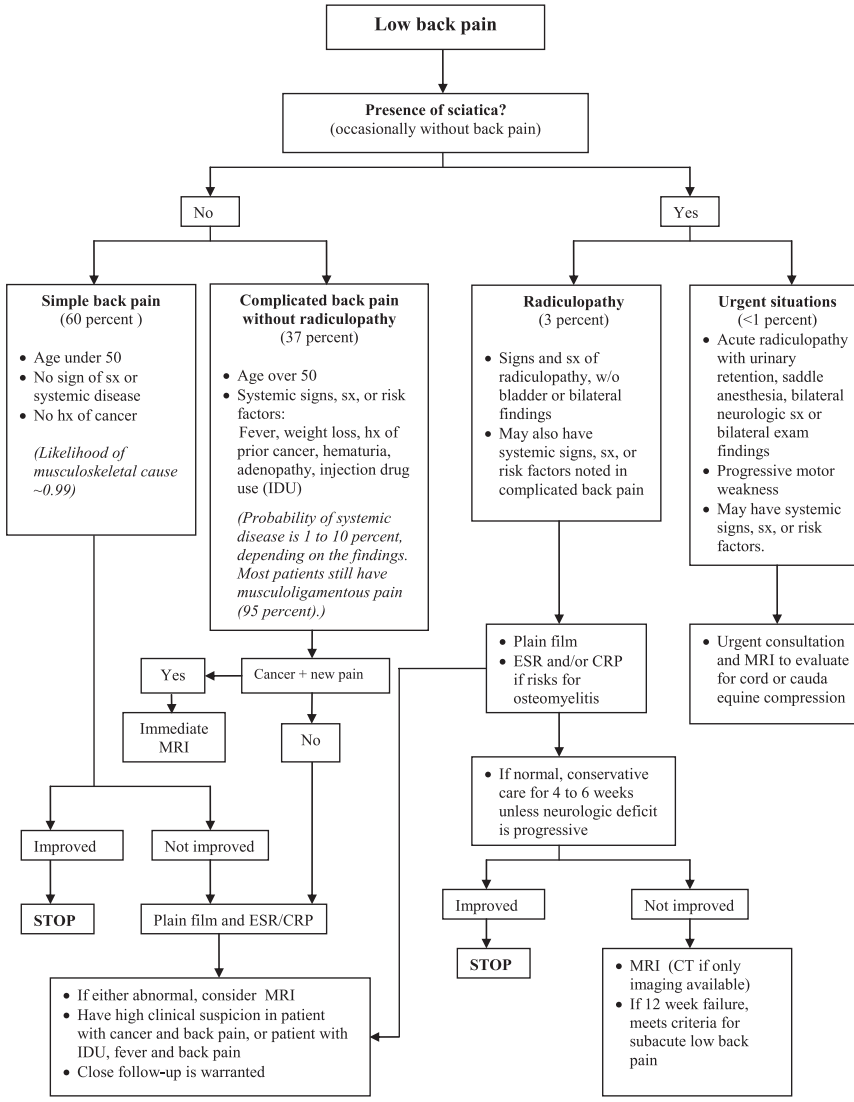


Fig. 10. Algorithm for the evaluation of low back pain. CRP, C-reactive protein; ESR, Erythrocyte sedimentation rate. (*Adapted from* Wipf JE, Deyo RA. Low back pain. Common medical problems in ambulatory care. Med Clin North Am 1995;79:239; with permission.)

improvements on a 1-year disability assessment compared with patients in the usual care group. In addition, care for the targeted intervention group was more cost-effective.

Activity Recommendations and Self-Care

All patients with acute nonspecific low back pain, including those with lower extremity symptoms, should be given general self-care advice including return to usual activity and the avoidance of prolonged bed rest. Studies indicate that bed rest does not increase the speed of recovery and in fact may delay it.[34] Self-care advice may also include heat application and self-education with evidence-based materials.

Keele STarT Back Screening Tool	No	Yes
Has your back pain spread down your leg(s) at some time in the last 2 weeks	○	○
Have you had pain in the shoulder or neck at some time in the last 2 weeks	○	○
Have you only walked short distances because of your back pain	○	○
In the last 2 weeks, have you dressed more slowly than usual because of back pain	○	○
Do you think it's not really safe for a person with a condition like yours to be physically active	○	○
Have worrying thoughts been going through your mind a lot of the time	○	○
Do you feel that your back pain is terrible and it's never going to get any better	○	○
In general have you stopped enjoying all the things you usually enjoy?	○	○

Overall, how bothersome has your back pain been in the last 2 weeks?

Not at all	Slightly	Moderately	Very much	Extremely
○	○	○	○	○

Fig. 11. Keele STarT back screening tool. Keele STarT back tool. (*Courtesy of* Keel University, Keele, Staffordshire, UK; with permission. The copyright (©2007) of the STarT Back Tool and associated materials is owned by Keele University, the development of which was part funded by Arthritis Research UK: i) the tool is designed for use by health care practitioners, with appropriate treatment packages for each of the stratified groups;ii) the tool is not intended to recommend the use of any particular product. No license is required for non-commercial use. If you would like to incorporate the tool in any way into commercial product materials, please contact Keele University for further advice.)

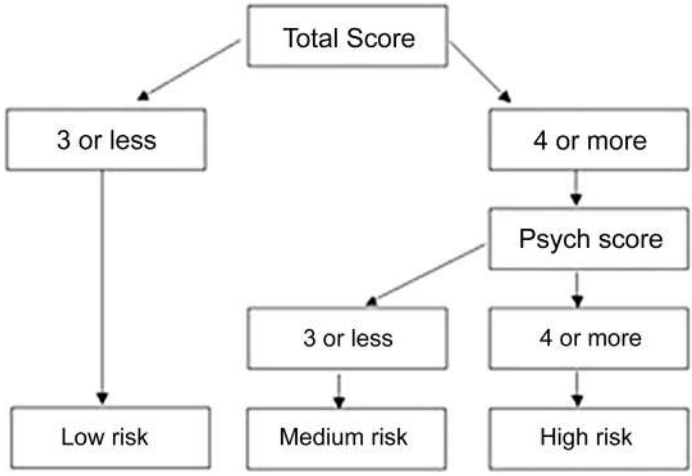


Fig. 12. Scoring the Keele STarT back screening tool. "Psych score" refers to score on questions 5 to 9. (*Courtesy of* Keel University, Keele, Staffordshire, UK; with permission. The copyright (2007) of the STarT Back Tool and associated materials is owned by Keele University, the development of which was part funded by Arthritis Research UK: i) the tool is designed for use by health care practitioners, with appropriate treatment packages for each of the stratified groups; ii) the tool is not intended to recommend the use of any particular product. No license is required for non-commercial use. If you would like to incorporate the tool in any way into commercial product materials, please contact Keele University for further advice.)

Analgesics

In addition to self-care advice, clinicians may recommend or prescribe analgesic medications to help alleviate pain in the short term. Several classes of medications have been shown to provide some pain relief when used for short time intervals for low back pain, including nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, skeletal muscle relaxants, tramadol, and opioids. When choosing a medication, clinicians should be mindful of effectiveness, tolerability, and side-effect profiles. The 2007 joint guidelines from the ACP and American Pain Society recommend either NSAIDs or acetaminophen as first-line analgesic agents for the treatment of low back pain.[27] **Table 9** lists the medication comparisons.

Of note, there is no good evidence supporting the use of systemic glucocorticoids,[37,38] lidocaine patches, anticonvulsants, or antidepressants in the treatment of acute low back pain, and therefore, their use is not recommended.

Nonpharmacologic Noninvasive Treatments

There is no high-quality evidence that nonpharmacologic therapies are superior to self-care advice in the treatment of acute low back pain, including spinal manipulation[39] and exercise therapy,[40] as well as massage, acupuncture, and yoga. However, these modalities may be of benefit in patients found to be at higher risk for prolonged pain and disability as discussed above.

For patients with acute low back pain who do not improve with self-care and short-term analgesics after 4 to 6 weeks, clinicians should first re-evaluate for an underlying serious condition (cancer or fracture) or systemic disease as per the algorithm in **Fig. 10**. If no serious cause is found, providers may begin to implement the treatments outlined in later discussion for subacute and chronic low back pain.

TREATMENT OF CHRONIC LOW BACK PAIN

If low back pain persists for more than 12 weeks and serious conditions have been ruled out, the focus of care should shift from pain-resolution to pain-management strategies that control pain while maximizing function and quality of life and preventing disability.

Treatment of chronic low back pain is often multidisciplinary, involving a combination of self-care, analgesics, spinal manipulation, physical therapy with or without cognitive behavioral therapy, massage, acupuncture, yoga, and in some cases, invasive interventions such as glucocorticoid injections and surgical procedures.

Analgesics

Regarding analgesics, most of the evidence for their benefit comes from short-term trials; therefore, the efficacy and safety for long-term use is unproven. Short-term courses of acetaminophen or NSAIDs are typically recommended for acute exacerbations of chronic low back pain if the side-effect profiles are acceptable for the patient. The long-term use of NSAIDs is limited by their potential gastric, renal, and cardiac toxicity.

Opioids have been increasingly used for chronic low back pain; however, evidence to support their use is minimal. A 2013 *Cochrane Review* found low- to moderate-quality evidence for short-term efficacy for pain and function when opioids were compared with placebo, but none of the trials persisted beyond 12 weeks.[41,42] In addition, the meta-analysis found that there is no high-quality evidence that long-term use of opiates is superior to other medications (NSAIDs, antidepressants) for pain relief and function. Furthermore, patients who use chronic opiates, especially in high doses,

Table 9
Pharmacotherapy for treatment of acute low back pain

Drug Class	Drug Names/Dose Regimens	Benefits/Evidence	Adverse Effects/Contraindications
NSAIDs	• Ibuprofen 400–600 mg po q6-8 h • Naproxen 250–500 mg po q12 h • Meloxicam 7.5–15 mg po daily • Diclofenac 50–75 mg po q12 h • Etodoloc 200–400 mg po q6-8 h • Ketorolac 30–60 mg im × 1	• 2008 *Cochrane Review* showed greater symptom improvement compared with placebo after 1 week: RR 1.19 (95% CI 1.07–1.35)[35] • Recommended as first-line therapy, along with acetaminophen, for acute LBP in the 2007 ACP/APS guidelines[27]	• Nephrotoxicity (avoid in patients with kidney disease or at high risk for renal injury) • Gastrointestinal toxicity (avoid in patients with a history of gastritis, upper GI bleed, or peptic ulcer disease; consider coadministration of a proton pump inhibitor in higher risk patients) • Increased risk of cardiovascular events (avoid in patients with known CAD and those at very high risk) • Higher risk in elderly patients • Use lowest dose for shortest duration
Acetaminophen	Acetaminophen 325–650 mg po q4-6 h (Not to exceed 4 g per 24 h or 2 g per 24 h in patients with underlying liver disease or heavy alcohol use)	• Similar to slightly less efficacy compared with NSAIDs • Less side effects than NSAIDs • Recommended as first-line therapy, along with NSAIDs, for acute LBP in the 2007 ACP/APS guidelines[27]	• Hepatotoxicity: risk varies by dose and patient; higher risk with concurrent alcohol use, underlying liver disease, or higher dose • May cause asymptomatic transaminase elevations at therapeutic doses
Centrally acting skeletal muscle relaxants	• Benzodiazepines • Cyclobenzaprine 5–10 mg po tid • Methocarbamol 1000 mg po qid • Carisoprodol 350 mg po tid and qhs • Baclofen 5–10 mg po tid • Tizanadine 4–8 mg po q6-8 h	• 2003 systematic review found that non-benzodiazepine muscle relaxants were more effective than placebo for short-term relief of LBP: RR 0.8, 95% CI 0.71–0.89[36]	• Sedation • Dizziness • Dependence/abuse potential (benzodiazepines and carisoprodol) • Hepatotoxicity and multiple drug interactions (tizanidine) • Use of muscle relaxants should generally be limited to 1–3 wk
Opioid agonists	• Tramadol (nonopiate that acts at opiate receptor) • Opioids (codeine, hydrocodone, oxycodone, hydromorphone, morphine, methadone, fentanyl)	• Data are limited for efficacy and safety in treatment of acute low back pain (most studies focus on chronic low back pain) • Avoid first line; if used, limit duration and consider scheduled rather than as needed administration	• Sedation • Confusion • Nausea • Constipation • Respiratory depression (at higher doses) • Dependence and abuse potential (higher risk with longer term use)

have a significant risk of adverse effects, including dependence, misuse, and overdose.[43] Therefore, the long-term use of opioids for chronic low back pain should be restricted to patients who demonstrate a functional improvement with opioid use, are at low risk for misuse, and can be monitored closely for adverse effects.

Antiepileptics and tricyclic antidepressants (TCAs) are frequently used to treat patients with radicular low back pain or spinal stenosis. However, a 2008 systematic review concluded there is not compelling evidence that antidepressants are superior to placebo in the treatment of nonspecific low back pain.[44] Similarly, a 2013 systematic

Table 10
Evidence-based nonsurgical treatments for chronic low back pain

Treatment	Benefit	Recommendation with Evidence Grade	Comments
NSAIDs	Moderate	Suggested as first-line therapy (2B)	Use limited by gastric and renal toxicity
Acetaminophen	Small	Suggested as first-line therapy (2B)	May cause asymptomatic liver enzyme elevation
Opioids	Small	Suggest not using as first-line therapy (2B)	Use limited by risk of side effects, dependency, misuse
Antidepressants	None to small	May be used to treat comorbid depression but not as sole back pain analgesic (2B)	—
Nonpharmacologic noninvasive therapies • Acupuncture[1] • Physical therapy • Massage therapy • Cognitive behavioral therapy • Spinal manipulation • Yoga[2] (viniyoga)	Moderate	Suggested (2B)	[1]Efficacy of sham acupuncture vs acupuncture inconsistent [2]Evidence insufficient to judge nonviniyoga
Nonsurgical invasive therapies • Epidural steroid injection	Moderate (short term only)	Suggested (2B)	Evidence for use in patients with disc herniations causing radiculopathy

Evidence Grade Explanation:

1A: Strong recommendation, high-quality evidence. Strong recommendation, can apply to most patients in most circumstances without reservation.

1B: Strong recommendation, moderate-quality evidence. Strong recommendation, likely to apply to most patients.

1C: Strong recommendation, low-quality evidence. Relatively strong recommendation; might change when higher-quality evidence becomes available.

2A: Weak recommendation, high-quality evidence. Weak recommendation, best action may differ depending on circumstances or patients or societal values.

2B: Weak recommendation, moderate-quality evidence. Weak recommendation, alternative approaches likely to be better for some patients under some circumstances.

2C: Weak recommendation, low-quality evidence. Very weak recommendation; other alternatives may be equally reasonable.

Data from Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007;147:478.

review concluded there is only low-quality evidence for the use of antiepileptics given scarcity and poor methodology of existing trials.[45] Furthermore, the use of these medications is often limited by side effects, including somnolence, dizziness (antiepileptics and TCAs), and anticholinergic effects (TCAs).

Nonpharmacologic Noninvasive Treatments

Nonpharmacologic noninvasive evidence-based treatments for chronic low back pain include physical therapy, spinal manipulation, acupuncture, massage, yoga, and cognitive behavioral therapy. These treatments have B-grade evidence, meaning there is fair-quality evidence of moderate benefit, or small benefit but no significant harms, costs, or burdens.[27,46] All patients with chronic low back pain should be advised to remain active. Beyond that, use of the other nonpharmacologic treatments can be pursued based on provider and patient preferences and treatment availability.

Invasive Nonsurgical Treatments

Invasive nonsurgical treatments for chronic low back pain include epidural steroid injections, intradisc steroid injections, facet joint injections, medial branch blocks, and radiofrequency denervation. Of these, there is moderate-quality evidence only for epidural steroid injections in patients with sciatica or radiculopathy, and the benefit is short term (less than 6 weeks).[47] **Table 10** summarizes the evidence-based nonsurgical treatments for chronic low back pain.

Surgical Referrals

Urgent surgical evaluation is recommended for patients with severe or progressive motor weakness or evidence of cauda equina syndrome. In the absence of severe progressive neurologic deficits, surgery may be considered an elective treatment of patients with radiculopathy and spinal stenosis who have chronic disabling symptoms and have not responded to appropriate trials of nonsurgical treatments.[48] In general, surgical outcomes may be superior to nonsurgical management in the short term, but the difference does not persist after longer-term follow-up.

SUMMARY

Low back pain is a common, frequently recurring condition that often has a nonspecific cause. Most nonspecific acute low back pain will improve within several weeks with or without treatment. The diagnostic workup should focus on evaluation for evidence of systemic or pathologic causes. Psychosocial distress, poor coping skills, and high initial disability increase the risk for a prolonged disability course. All patients with acute or chronic low back pain should be advised to remain active. The treatment of chronic nonspecific low back pain involves a multidisciplinary approach targeted at preserving function and preventing disability. Surgical referral is indicated in the presence of severe or progressive neurologic deficits or signs and symptoms of cauda equina syndrome.

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