

JAMA | Review

Low Back Pain

A Review

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IMPORTANCE Low back pain is defined as pain localized below the costal margin and above the inferior gluteal fold, with or without leg pain. Low back pain affects approximately 619 million people worldwide and is the leading cause of years lived with disability worldwide.

OBSERVATIONS Approximately 90% of patients presenting for care with low back pain have *nonspecific low back pain*, which is defined as low back pain that is not associated with specific spinal disorders (such as lumbar radiculopathy, lumbar spinal stenosis, vertebral fracture, axial spondyloarthritis, infection, or malignancy). Low back pain is classified as acute if the duration is shorter than 6 weeks, subacute if the duration is 6 to 12 weeks, and chronic when the duration is longer than 12 weeks. The age-standardized prevalence of low back pain is higher in females (9330 per 100 000) than in males (5520 per 100 000). The prevalence of low back pain increases with age, peaking at approximately 85 years. Risk factors for low back pain include obesity, depressive symptoms, occupational exposures (eg, heavy lifting), tobacco use, chronic disease (eg, diabetes), and previous low back pain. Acute nonspecific low back pain is usually self-limited, and approximately 72% of individuals recover by 12 months. Prognosis is less favorable for chronic nonspecific low back pain, but 42% of patients recover within 12 months. Initial management of patients with low back pain of any duration includes reassurance that serious underlying disease is unlikely, discussion about the expected time course of recovery, and the recommendation to remain physically active. Patients should be encouraged to continue their usual activities (including work), avoid prolonged rest, and be advised to self-manage their condition, which consists of symptom-relief strategies (such as heat application) and activity pacing (maintaining or gradually increasing usual activities and work). For patients with acute nonspecific low back pain, first-line therapies include heat application, spinal manipulation, massage, and acupuncture (typically provided by physical therapists, chiropractors, acupuncturists, and massage therapists) as well as nonsteroidal anti-inflammatory drugs (NSAIDs; such as ibuprofen) and skeletal muscle relaxants (such as cyclobenzaprine). For chronic nonspecific low back pain, first-line therapies include exercise of any type, psychological therapies (eg, cognitive behavioral therapy), or combined multidisciplinary approaches (such as pain management programs and integrated exercise and psychological care) along with spinal manipulation, massage, and acupuncture. NSAIDs should be considered as second-line therapy for chronic nonspecific low back pain.

CONCLUSIONS AND RELEVANCE Low back pain is a leading cause of disability worldwide. Acute nonspecific low back pain is often self-limited, whereas chronic nonspecific low back pain has a less favorable prognosis. For patients with acute nonspecific low back pain, first-line treatments include selected nonpharmacological therapies and medications (such as NSAIDs and skeletal muscle relaxants). For patients with chronic nonspecific low back pain, first-line treatment consists of exercise, psychological therapies (such as cognitive behavioral therapy), and combined multidisciplinary care.

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Low back pain is defined as pain located below the costal margin and above the inferior gluteal folds, with or without leg pain and is the leading cause of years lived with disability worldwide, affecting people of all ages.¹ Approximately 1 in 4 US workers report low back pain,² with mean lifetime prevalence of approximately 40% in adults.³ In primary care, fewer than 10% of low back pain cases are due to specific spinal causes (such as lumbar radiculopathy due to disk herniation or spinal stenosis), other diagnoses (such as vertebral fracture), inflammatory disease (such as axial spondyloarthritis),⁴ vertebral malignancy (such as metastatic disease), infection (such as vertebral osteomyelitis), or cauda equina syndrome.⁵ When no specific cause is identified, low back pain is termed *nonspecific low back pain*. Nonspecific low back pain is usually classified as acute when the duration is shorter than 6 weeks, subacute when the duration is 6 to 12 weeks, and chronic when the duration is longer than 12 weeks. This review summarizes current evidence on the epidemiology, pathophysiology, clinical evaluation, prognosis, and treatment of nonspecific low back pain in the outpatient setting (Box 1).

Methods

We searched PubMed from January 1, 2005, through February 10, 2026, for English-language randomized clinical trials, cohort studies, and systematic reviews and/or meta-analyses using the term *nonspecific low back pain*. We reviewed the most recent clinical practice guidelines published by the World Health Organization (WHO),⁶ the American College of Physicians (ACP),⁷ and the UK National Institute for Health and Care Excellence (NICE),⁸ and examined the reference lists of relevant articles. Of 1428 records identified, 108 were included, consisting of 50 meta-analyses, 18 randomized clinical trials, 15 systematic reviews, 13 prospective cohort studies, 8 international guidelines, 3 Delphi consensus studies, and 1 cross-sectional study. We prioritized randomized clinical trials that tested efficacy of clinically practical interventions.

Epidemiology and Risk Factors

The Global Burden of Disease study,⁹ an international program that estimates disease prevalence and disability worldwide, estimated that 619 million individuals worldwide had low back pain in 2020. The age-standardized prevalence was higher in females compared with males (9330 per 100 000 vs 5520 per 100 000).⁹ The prevalence of low back pain increases with age, peaking around 85 years.⁹ Other factors associated with higher prevalence include obesity (odds ratio [OR], 1.5 [95% CI, 1.2-1.9]),¹⁰ depressive symptoms (OR, 1.6 [95% CI, 1.3-2.0]),¹¹ and occupational exposures (such as heavy lifting) (OR, 1.10 [95% CI, 1.01-1.20] per 10 kg lifted) (absolute rates not available).¹² Additional risk factors include tobacco use,¹³ chronic diseases (such as diabetes or asthma),¹⁴ and prior low back pain.¹⁵

Pathophysiology

The process by which nonspecific low back pain develops and persists remains unclear. The term *nonspecific low back pain* implies no

Box 1. Frequently Asked Questions About Nonspecific Low Back Pain

What is nonspecific low back pain?

Nonspecific low back pain is defined as pain below the costal margin and above the inferior gluteal folds. Nonspecific low back pain does not have clinical features suggestive of a specific underlying condition (such as radiculopathy, spinal stenosis, axial spondyloarthritis, fracture, infection, or malignancy) or referred pain from nonspinal structures (such as kidney, gastrointestinal, pelvic, or vascular conditions).

When should diagnostic imaging be performed for patients with nonspecific low back pain?

Routine imaging with radiography, computed tomography, or magnetic resonance imaging is not recommended for patients with nonspecific low back pain of any duration because these tests do not improve clinical outcomes and may identify incidental findings that lead to additional testing and unnecessary interventions. Imaging should be reserved for patients with severe or progressive neurological deficits (such as motor weakness, saddle anesthesia, or urinary retention) or signs or symptoms of serious conditions (such as cancer, infection, fracture, or cauda equina syndrome).

What are first-line treatments for patients with acute and chronic nonspecific low back pain?

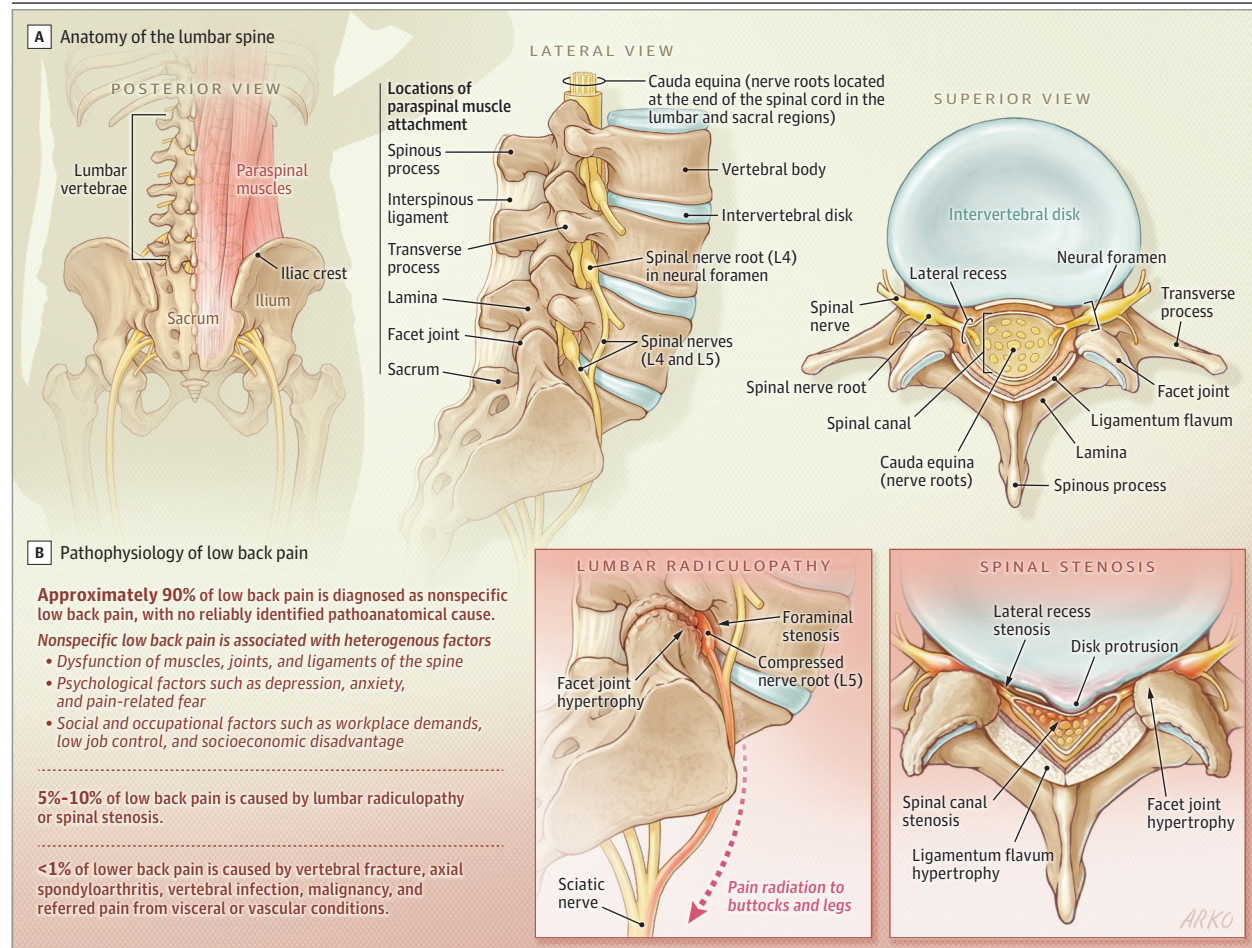
Initial management for acute and chronic nonspecific low back pain includes advice and education to support self-management, reassurance, and encouragement to remain active. For acute low back pain, when additional treatment is needed, nonpharmacological therapies (such as superficial heat, spinal manipulation, acupuncture, or massage) may provide short-term pain relief. Nonsteroidal anti-inflammatory drugs and skeletal muscle relaxants may be considered when pain limits function. For chronic low back pain, first-line treatment is self-management, exercise, and psychological therapies delivered alone or as part of multidisciplinary biopsychosocial care. Other nonpharmacological therapies (such as spinal manipulation, massage, or acupuncture) may provide modest short-term benefit.

known pathoanatomical cause¹⁶ and the pain is not associated with specific spinal disorders (such as lumbar radiculopathy, lumbar spinal stenosis, vertebral fracture, axial spondyloarthritis, infection, or malignancy). Approximately 90% of patients presenting for care with low back pain have nonspecific low back pain that is conceptualized as a heterogeneous condition related to biological factors (such as dysfunction of muscles, joints, or ligaments of the back; **Figure 1**) in addition to psychological factors (such as depression, anxiety, and negative beliefs about low back pain),¹⁷ social and occupational factors (such as workplace demands and low job control), and socioeconomic disadvantage.¹⁷ The interaction of these factors and their contribution to low back pain varies among individuals, reflecting the personal, context-dependent nature of low back pain and the heterogeneity in symptoms and response to treatment.¹⁸

Clinical Presentation

Patients with low back pain typically present with pain and activity-related functional limitation, including difficulty bending, lifting, walking, or tolerating prolonged sitting or standing, and a reduced ability

Figure 1. Illustrations of Lumbar Spine Anatomy



In most patients with low back pain, no pathoanatomical cause can be reliably identified. Although multiple lumbar spine structures, including muscles, fascia, ligaments, tendons, facet joints, neurovascular elements, vertebrae, and

intervertebral disks may generate pain, reliable methods to identify the specific pain-generating structure are lacking. Rarely, damage to these structures (such as through trauma or infection) results in specific low back pain.

to perform tasks at work and at home.¹⁹⁻²¹ Pain, localized between the costal margin and inferior gluteal folds, may radiate into the buttock or thigh without a dermatomal distribution. Approximately 30% of patients do not recall a precipitating event, and 76% report a prior episode of low back pain.^{19,22}

Symptoms often worsen with movement or sustained postures and improve with rest or position change. Coexisting cardiovascular, metabolic (eg, diabetes), and mental health conditions (eg, depression) are estimated to be present in 72% of patients with nonspecific low back pain.^{23,24}

Clinical Evaluation and Diagnosis

A diagnosis of nonspecific low back pain is made after excluding specific spinal and nonspinal disorders as causes of the pain.^{25,26} This evaluation includes a focused history and physical examination to determine the likelihood of specific underlying disorders and to assess for the presence and degree of neurological involvement. Lumbar radiculopathy or spinal stenosis accounts for 5% to 10% of initial presentations of low back pain in primary care. Vertebral fracture,

axial spondyloarthritis, vertebral infection, and malignancy as well as referred pain from visceral or vascular conditions each occur in fewer than 1% of patients with low back pain.⁵

Patient History

Clinicians should ask patients with low back pain about symptom duration, distribution, frequency, exacerbating and relieving factors, and the associated effects on daily activities.²⁷ The history should include questions about previous low back pain episodes and prior treatment (including treatment response) as well as factors associated with persistent pain and disability, including psychosocial factors such as pain-related fear (ie, fear of movement due to anticipated pain), depressive symptoms, and occupational factors (such as workplace compensation issues and workplace dissatisfaction),^{28,29} to guide diagnosis, prognosis, and treatment selection. Screening tools that include these factors can be used during initial evaluation to assist with prognostic risk stratification and therapy selection, but their accuracy varies and should complement clinical judgment and contextual factors (Table 1).³⁰⁻³⁵

The possibility of low back pain due to problems outside the spine (such as hip pathology), pain referred from other areas, visceral

Table 1. Prognostic Screening Instruments for Estimating Risk of Poor Recovery at Initial Primary Care Presentation for Acute Nonspecific Low Back Pain

Test ^a	Description	Outcome definition ^b	Discrimination ^c	Score range	Clinical use
STarT Back Tool ³⁰	9-item self-reported questionnaire to screen for prognostic indicators of low back pain that are relevant to initial decision-making in primary care.	Poor outcome defined as pain scale score ≥ 3 at follow-up (length of pain: 3–6 mo).	Pooled analysis ³¹ of 5 studies including 1153 people with acute low back pain treated in primary care: C statistic, 0.59 (95% CI, 0.55–0.63).	Score range, 0–9; higher scores indicate increased risk	Stratifies patients by psychosocial risk (low, medium, or high) to guide treatment selection and intensity. Designed for primary care to support targeted use of psychologically informed care.
Predicting the Inception of Chronic Pain Model ³²	5-item self-reported screening questionnaire to predict the transition from acute to chronic low back pain.	Chronic low back pain defined as pain scale score ≥ 2 at 3 mo without any period of recovery.	External validation in 1652 people with acute low back pain treated in primary care: C statistic, 0.66 (95% CI, 0.63–0.69).	Score range, 0%–100%; higher scores indicate increased risk	Estimates individual probability (0%–100%) of developing chronic pain. Most useful to identify patients at low risk for transitioning to chronic pain, supporting reassurance and limiting unnecessary referral.
Örebro Musculoskeletal Pain Questionnaire ³³	25-item self-reported screening questionnaire to identify psychological and functioning-related risk factors among individuals with acute musculoskeletal pain.	Poor outcome defined as pain scale score ≥ 3 at follow-up (length of pain: 3–6 mo).	Pooled analysis ³¹ of 4 studies including 360 people with acute low back pain treated in primary care: C statistic, 0.69 (95% CI, 0.62–0.76).	Score range, 0–210; higher scores indicate increased risk	Identifies patients at low, moderate, or high risk of persistent pain-related work disability. May help guide early referral and workplace-focused interventions, particularly in occupational settings.
da Silva Clinical Prediction Model ³⁴	5-item self-reported questionnaire to predict recovery from low back pain at 1 wk, 1 mo, and 3 mo.	Recovery from pain defined as pain scale score ≤ 1 during the past week (assessed at 3 mo).	External validation in 737 people with acute low back pain treated in primary care: C statistic, 0.71 (95% CI, 0.63–0.78).	Score range, 0%–100%; higher scores indicate decreased risk	Uses early pain intensity and change over time to estimate individual likelihood (0%–100%) of rapid recovery. May assist short-term prognosis discussions and reassurance.

^a The instruments were developed for prognostic risk estimation and should be used at the initial patient presentation. However, use of these instruments for longitudinal monitoring or for detecting clinically meaningful change is limited.

^b Scores range from 0 to 10. A score of 10 indicates the worst pain.

^c Indicates how well the model predictions differentiate between individuals with the outcome and those without the outcome. The C statistic is a measure of the area under a receiver operating characteristic curve.

origin (such as pancreatitis), and vascular diseases (such as peripheral artery disease) or viral syndromes should be considered.

Clinicians should also ask about clinical features (known as red flags) that increase the likelihood of cancer, vertebral fracture, and infection; for axial spondyloarthritis, additional diagnostic investigation and specific treatment may be required (Figure 2).²⁶ Individual signs and symptoms often have limited diagnostic value for identifying serious underlying pathology, but several characteristics meaningfully increase the probability of vertebral fracture, including older age (≥ 70 years; posttest probability, 9% [95% CI, 3%–25%]), severe trauma (such as a fall from a height or a motor vehicle crash; posttest probability, 11% [95% CI, 8%–16%]), and corticosteroid use for 6 months or longer (posttest probability, 33% [95% CI, 10%–67%]), and osteoporosis.³⁶ A history of cancer increases the likelihood of spinal malignancy (posttest probability, 33% [95% CI, 22%–46%]).³⁶

Clinicians should also evaluate risk factors for vertebral infection including presence of fever, intravenous drug use, recent infection or spinal procedure, and for axial spondyloarthritis in those with insidious onset at a younger age (<45 years), morning stiffness lasting at least 30 minutes, improvement with exercise but not at rest, pain alternating between the left and right buttock, and awakening due to back pain at night. Although data on combinations of red flags are limited, the presence of multiple features should further increase concern for an underlying diagnosis of serious spinal pathology and consideration of further diagnostic evaluation (such as magnetic resonance imaging [MRI]).

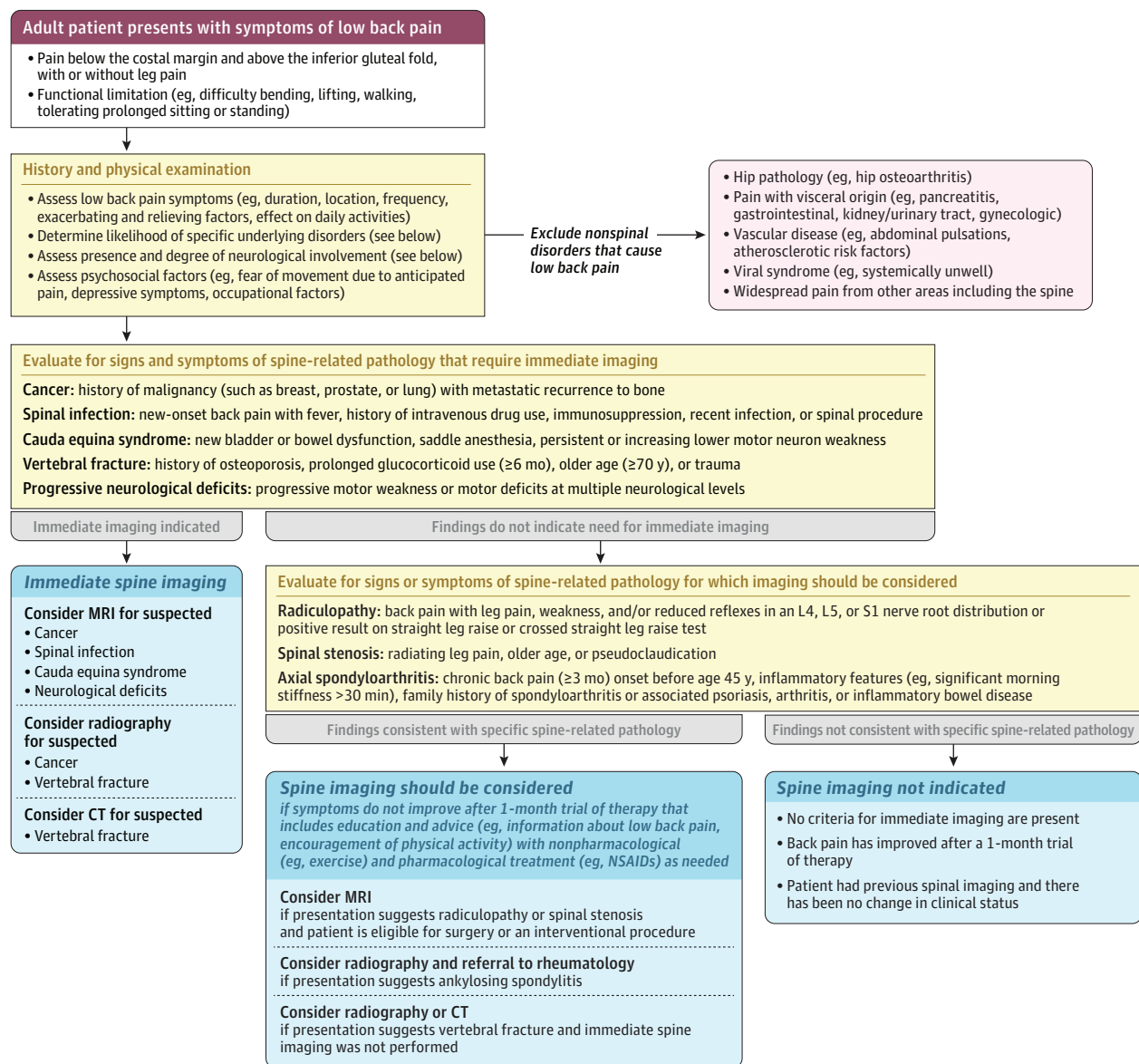
Although affecting only 0.1% of patients presenting to primary care,⁵ all with low back pain should be evaluated for rapidly progressive or severe neurological deficits, including cauda equina syndrome (compression of the lumbosacral nerve roots), which typically presents as new urinary retention or incontinence, saddle anesthesia, and progressive motor weakness of the lower extremities. Presence of these findings requires immediate imaging with MRI and urgent evaluation by neurosurgery.³⁷

Physical Assessment

Important elements of the physical examination include evaluation of spine symmetry, posture, and range of motion, palpation to differentiate spinal (bone) from paraspinal (soft tissue) tenderness, and a general mobility assessment (such as the sit-to-stand test and assessment of gait) to assess functional limitations.

In patients with back and leg symptoms, the presence of leg pain following L4, L5, or S1 dermatomal distributions together with weakness, dermatomal sensory loss, or reduced reflexes is consistent with lumbar radiculopathy.³⁸ Physical examination for possible lumbar radiculopathy includes straight leg testing and evaluation of knee extension (quadriceps) strength and patellar reflexes (L4 nerve root), great toe and foot dorsiflexion strength (L5 nerve root), foot plantar flexion and ankle reflexes (S1 nerve root), and distribution of sensory symptoms to identify dermatomal and myotomal deficits consistent with nerve root compression. The straight leg raise test is positive when passive elevation of the extended leg places tension on the sciatic nerve and lumbosacral nerve roots reproduces the patient's leg symptoms when the leg is elevated at an angle of 30° to 70° while the patient lies supine.³⁸ In a Cochrane meta-analysis³⁹ of cohort and case-control diagnostic accuracy studies that included a median of 126 participants (IQR, 71–2504 participants), the

Figure 2. Algorithm of the Diagnostic Approach for Nonspecific Low Back Pain



CT indicates computed tomography; MRI, magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs. This diagnostic algorithm has not been validated in clinical trials.

straight leg raise test was associated with high sensitivity for lumbar disk herniation (pooled sensitivity, 0.92 [95% CI, 0.87-0.95]) but low specificity (pooled specificity, 0.28 [95% CI, 0.18-0.40]). The crossed straight leg raise test (leg pain reproduced when the unaffected leg is elevated at an angle of 30° to 70° while the patient lies supine) was less sensitive (pooled sensitivity, 0.28 [95% CI, 0.22-0.35]) but more specific (pooled specificity, 0.90 [95% CI, 0.85-0.94]) for diagnosis of lumbar disk herniation.

Symptoms affecting the buttocks, thigh, or leg pain or weakness precipitated by standing and walking that are relieved by sitting or lumbar flexion suggests lumbar spinal stenosis.⁴⁰

When Imaging Is Indicated

Routine spinal imaging (ie, radiography, computed tomography, or MRI) is not recommended for patients with acute and chronic

nonspecific low back pain in the absence of certain clinical features (such as history of cancer, fever, trauma, and new bladder or bowel dysfunction) that suggest serious pathology (Figure 2).^{26,37}

Degenerative changes of the lumbar spine on plain radiograph (eg, disk degeneration, facet arthropathy, and end plate changes) are common imaging findings and may not be associated with low back pain.⁴¹ A population-based prospective cohort study of 3369 adults (mean age, 53 years; 51% women) reported degenerative changes on MRI in 77.8% of participants with low back pain and in 74.4% without low back pain; the proportion of people with these findings increased with age (50% of persons <41 years and $>90\%$ of persons >69 years), but was common among people without low back pain symptoms.⁴¹ Disk degeneration at any lumbar level among people without low back pain increased from 38.1% in those aged 41 to 49 years to 58.1% in

those aged 50 to 59 years and to 81.9% in those aged 60 to 68 years.⁴¹

Lumbar imaging findings of degenerative changes may result in inappropriate attribution of symptoms to these findings (eg, degenerative disk disease or disk bulge), potentially resulting in increased patient anxiety, inappropriate diagnosis, and unnecessary testing and treatments that do not improve clinical outcomes.^{37,42,43}

Prognosis and Treatment

Acute nonspecific low back pain is usually self-limited, and approximately 72% of individuals recover by 12 months.⁴⁴ For chronic nonspecific low back pain, 42% of patients recover within 12 months.⁴⁵ A systematic review⁴⁶ of 60 prospective inception cohort studies that included 17 974 participants with nonspecific low back pain reported that among those with acute low back pain, the mean pain severity score improved from 56 (95% CI, 49-62) at inception to 26 (95% CI, 21-31) at 6 weeks on a pain scale from 0 to 100, where 100 indicates worst pain (minimal clinically important difference is a change by 30% from baseline).^{46,47} Improvement was also reported among patients with subacute nonspecific low back pain; the mean pain severity score went from 63 (95% CI, 55-71) at inception to 29 (95% CI, 22-37) at 6 weeks on a pain scale from 0 to 100. The prognosis for chronic nonspecific low back pain was less favorable with only small improvements over time; the mean pain severity score went from 56 (95% CI, 37-74) at inception to 48 (95% CI, 32-64) at 6 weeks, and many patients continued to experience persistent low back pain symptoms at 52 weeks.⁴⁶

Guidelines recommend that all patients with nonspecific low back pain of any duration should receive education and advice as initial management, consisting of reassurance that serious pathology is unlikely, advice to remain physically active, and education to support self-management (Tables 2 and 3, Figure 3, and Box 2).^{26,68,69} Information on the outcome measures for pain severity and disability, including interpretation of effect size, appears in the eTable in the Supplement.⁷⁰

Acute Nonspecific Low Back Pain

With or without treatment, patients with acute nonspecific low back pain typically experience substantial improvement in pain and function within approximately 6 weeks after symptom onset.⁴⁶ Initial management includes patient education, treatment advice, and self-management strategies (such as activity pacing in which patients are encouraged to maintain or gradually increase their usual activities and work). Prolonged rest should be discouraged and can delay recovery.^{56,57,71}

Nonpharmacological Treatments

Spinal manipulation,⁵⁹ acupuncture,^{61,62} superficial heat,⁵⁸ and massage⁶⁰ (typically provided by physical therapists, chiropractors, acupuncturists, and massage therapists) are recommended⁷ and may provide small, short-term reductions in pain severity. A meta-analysis⁵⁹ of 15 clinical trials, including 1699 people with acute nonspecific low back pain, reported that spinal manipulation was associated with greater improvement in pain severity (mean difference [MD], -10.0 [95% CI, -15.6 to -4.3] on scale from 0-100) and disability (12 trials and 1381 adults; standardized MD [SMD], -0.4

[consistent with a small effect size; 95% CI, -0.7 to -0.07]) compared with sham or other treatments. A meta-analysis⁶¹ of 2 clinical trials including 100 people with acute nonspecific low back pain reported that a single session of acupuncture was associated with greater improvement in pain severity (MD, -9.4 [95% CI, -17 to -1.8] on scale from 0-100) but there was no significant change in disability compared with sham acupuncture. In a meta-analysis⁵⁸ of 2 clinical trials including 258 people with acute nonspecific low back pain and compared with oral placebo, heat wrap was associated with a moderate improvement in pain severity (MD, 1.1 [95% CI, 0.7 to 1.5] on a scale from 0-5 when 5 indicates the worst pain) and disability (MD, -2.1 [95% CI, -3.2 to -1.0] on a scale from 0-24 when 24 indicates the worst disability). Although recommended by the ACP guideline,⁷ evidence supporting massage for acute nonspecific low back pain is limited to 1 small trial with 61 participants that showed statistically significant large improvements in pain severity but no effects on disability compared with inactive control.^{60,72}

Structured or supervised exercise is not routinely indicated for acute nonspecific low back pain because it has limited effectiveness.⁷³ A meta-analysis⁷³ of 11 trials that included 1443 patients with acute nonspecific low back pain reported that exercise (including aerobic, flexibility, back-specific resistance exercise, or combined exercise programs) was not associated with significantly greater improvement in pain severity (SMD, 0 [95% CI, -0.1 to 0.1]) or disability (including 1494 adults; SMD, 0.1 [95% CI, 0 to 0.2]) compared with conservative treatment including spinal manipulation, advice, medication, or usual care.

Early referral for physical therapy may benefit patients with risk factors for chronic low back pain,⁷⁴ but feasibility is limited by relatively rapid recovery even without therapy, treatment access, and costs. In a randomized clinical trial⁷⁴ including 220 adults with acute nonspecific low back pain, early physical therapy (spinal manipulation plus exercise) provided small improvements in disability at 3 months (MD, -3.2 [95% CI, -5.9 to -0.5] on a scale from 0-100 when 100 indicates worse disability) but not pain severity (MD, -0.4 [95% CI, -0.8 to 0.1] on a scale from 0-10 when 10 indicates the greatest pain) and showed no significant differences in disability and pain severity at 1 year compared with usual care.

In a randomized clinical trial³⁰ including 1000 adults with acute or subacute low back pain who were at medium or high risk of persistent disability (assessed using the STarT Back Tool; Table 1) compared with medical care, more intensive clinician-supported biopsychosocial self-management consisting of individualized pain education, exercise, and cognitive behavioral therapy (CBT) strategies produced a small statistically significant improvement in disability compared with other guideline-concordant care (MD, -1.2 [95% CI, -1.9 to -0.5] on a scale from 0-24), but had no significant effect on pain severity at 1-year follow-up.⁷⁵ A risk-stratified care pathway in which treatment was matched to patient risk of poor prognosis (ie, using the STarT Back Tool³⁰) showed small, statistically significant reductions in disability among patients with low back pain in 1 UK randomized clinical trial including 851 participants (MD, -1.8 [95% CI, -2.6 to -1.1] on a scale from 0-24).⁷⁶ However, these results have not been replicated in other settings,^{77,78} including in a US randomized clinical trial⁷⁸ with 2300 adults who had acute nonspecific low back pain and showed no statistically significant difference in disability (MD, -2.1 [95% CI, -4.9 to 0.6] on a scale from 0-100) or in use of opioids or imaging compared with usual care.

Table 2. Pharmacological Interventions for Acute and Chronic Nonspecific Low Back Pain

Treatment	Drugs and dosage	Efficacy/effectiveness ^a	Adverse events	Considerations
Acute nonspecific low back pain (length of pain: <6 wk)				
NSAIDs	Ibuprofen: 400 to 800 mg every 6 to 8 h (maximum: 2400 mg/d); naproxen: 250 to 500 mg every 8 to 12 h (maximum: 1250 mg/d); celecoxib: 200 mg every 12 h (maximum: 400 mg/d).	Compared with placebo at ≤3-wk follow-up, ⁴⁸ use of NSAIDs was associated with significantly reduced pain (MD, -7.3 [95% CI, -11.0 to -3.6]) on a scale from 0 to 100 and disability (MD, -2.0 [95% CI, -2.9 to -1.2]) on a scale from 0 to 24.	Compared with placebo, ⁴⁸ there was no clear difference in risk of adverse events at ≤3 wk with use of NSAIDs (RR, 0.9 [95% CI, 0.6 to 1.2]; 95 per 1000 with use of NSAIDs vs 111 per 1000 with placebo).	Use with caution in patients with asthma, kidney disease, history of stomach ulcers, and coprescription of blood pressure-lowering medication or blood clotting inhibitors. Contraindications include chronic kidney disease or active gastrointestinal bleeding.
Skeletal muscle relaxant drugs	Cyclobenzaprine: 5 to 10 mg 3 times/d; tizanidine: 4 to 12 mg 3 times/d.	Compared with placebo at ≤2-wk follow-up, ⁴⁹ use of nonbenzodiazepine antispasmodics was associated with reduced pain (MD, -7.7 [95% CI, -12.1 to -3.3]) on a scale from 0 to 100 but not disability (MD, -3.3 [95% CI, -7.3 to 0.7]) on a scale from 0 to 100.	Compared with placebo, ⁴⁹ there was an increased risk of adverse events with use of nonbenzodiazepine antispasmodics (RR, 1.6 [95% CI, 1.2 to 2.0]). Common adverse events include dizziness, drowsiness, headache, and nausea; increased risk of misuse and dependency in some instances (eg, carisoprodol).	Use with caution in patients with older age, history of substance abuse, concurrent use of sedating medications, and heart, liver, or kidney impairment. Contraindications include high risk for falls in older adults, glaucoma, and significant heart, liver, or kidney disease.
Acetaminophen	Acetaminophen: 500 to 1000 mg every 4 to 6 h (maximum: 4000 mg/d).	Compared with placebo at 1-wk follow-up, ⁵⁰ use of acetaminophen was not associated with decreased pain (MD, 1.5 [95% CI, -1.3 to 4.3]) on a scale from 0 to 100 or disability (MD, -0.5 [95% CI, -1.2 to 0.3]) on a scale from 0 to 24.	Compared with placebo, ⁵⁰ there was no clear difference in risk of adverse events with use of acetaminophen (RR, 1.1 [95% CI, 0.9 to 1.3]; 115 per 1000 vs 107 per 1000 with placebo).	No longer considered first-line therapy due to ineffectiveness. Use with caution in patients with liver disease, alcohol use disorder, and risk of cumulative overdose from combination products. Contraindications include severe hepatic impairment.
Chronic nonspecific low back pain (length of pain: >12 wk)				
NSAIDs	Same as described for acute nonspecific low back pain.	Compared with placebo at <2-wk follow-up, ⁵¹ use of NSAIDs was associated with reduced pain (MD, -11.1 [95% CI, -13.8 to -8.4]) on a scale from 0 to 100 and disability (MD, -8.4 [95% CI, -10.6 to -6.3]) on a scale from 0 to 100.	Compared with placebo, ⁵¹ there was no clear difference in the risk of experiencing an adverse event with use of NSAIDs (RR, 1.1 [95% CI, 1.0 to 1.2]) but there was an increased risk of gastrointestinal adverse events (RR, 2.5 [95% CI, 1.2 to 5.2]).	Use with caution in patients with asthma, kidney disease, history of stomach ulcers, and coprescription of blood pressure-lowering medication or blood clotting inhibitors. Contraindications include chronic kidney disease or active gastrointestinal bleeding.
Antidepressants	Duloxetine: start at 30 mg once/d and increase to 60 mg once/d after 1 wk if tolerated.	Compared with placebo at 4- to 16-wk follow-up, ⁵² use of duloxetine was associated with reduced pain (MD, -5.3 [95% CI, -7.2 to -3.3]) on a scale from 0 to 100 and disability (MD, -0.9 [95% CI, -1.3 to -0.5]) on a scale from 0 to 24.	Compared with placebo, ⁵² there was an increased risk of adverse events with use of duloxetine (RR, 1.2 [95% CI, 1.1 to 1.3]; 652 per 1000 vs 558 per 1000 with placebo) but no increased risk of serious adverse events (OR, 1.8 [95% CI, 0.8 to 3.9]; 21 per 1000 vs 12 per 1000, respectively). Common adverse events include nausea, somnolence, constipation, dry mouth, and fatigue.	Use with caution in patients with glaucoma, high blood pressure, and heart or kidney impairment. Contraindications include liver disease and use of monoamine oxidase inhibitors.
Tramadol	Immediate-release tablet: 25 mg/d taken in morning ^b ; extended-release tablet: 100 mg/d. ^c	Compared with placebo, ⁵³ use of tramadol was associated with reduced pain (SMD, -0.6 [95% CI, -0.7 to -0.4]) and disability (SMD, -0.2 [95% CI, -0.3 to -0.1]).	Compared with placebo, there was an increased risk of serious adverse events with use of tramadol (RR, 18 [95% CI, 6 to 37] more events per 1000) and nonserious adverse events (162 [95% CI, 61 to 284] more events per 1000). Common adverse events ⁵⁴ include nausea (NNH, 7), dizziness (NNH, 8), and constipation (NNH, 9).	Use with caution in patients with risk of seizure, physiological opioid dependence, opioid use disorder, serotonin syndrome, and concurrent use of sedating medications. Contraindications include uncontrolled epilepsy, severe respiratory depression, and use of monoamine oxidase inhibitors.
Opioids	Codine alone or combined with acetaminophen: 30 to 60 mg every 4 h; hydrocodone alone or combined with acetaminophen, aspirin, or ibuprofen: 5 to 10 mg every 4 h.	Compared with placebo at ≤3-mo follow-up, ⁵⁵ use of opioids was associated with reduced pain (MD, -9.0 [95% CI, -11.7 to -6.3]) on a scale from 0 to 100.	Compared with placebo, there was an increased risk of adverse events with use of opioids (RR, 1.4 [95% CI, 1.2 to 1.6]) and serious adverse events (RR, 2.2 [95% CI, 1.2 to 4.1]). Common adverse events ⁵⁵ include headaches, somnolence, and nausea.	Use with caution in patients with risk of physiological opioid dependence, opioid use disorder, concurrent use of sedating medications, and older age. Contraindications include severe respiratory depression and acute asthma.

Abbreviations: MD, mean difference; NNH, number needed to harm; NSAIDs, nonsteroidal anti-inflammatory drugs; OR, odds ratio; RR, risk ratio or relative risk; SMD, standardized mean difference.

^a The minimum clinically important difference for pain severity and disability is commonly defined as a 30% reduction from baseline. Absolute thresholds for pain intensity range from 1 to 2 points (on a scale from 0 to 10) or range from 10 to 20 points (on a scale from 0 to 100). Absolute thresholds for disability range from 2 to 5 points on the Roland Morris Disability Questionnaire (on a scale from 0 to 24) and by approximately 10 points on the Oswestry Disability Index (on a scale from 0 to 100).

^b May titrate in 25-mg increments every 3 days as tolerated to reach 25 mg 4 times daily; after this regimen is reached, may increase by 50 mg every 3 days as tolerated to reach 50 mg 4 times daily.

^c Titrate in 100-mg increments every 5 days if needed (maximum: 300 mg/d).

Table 3. Nonpharmacological Interventions for Acute and Chronic Nonspecific Low Back Pain

Treatment	Description	Efficacy/effectiveness ^a	Common adverse events
Acute nonspecific low back pain (length of pain: <6 wk)			
Advice to avoid bed rest	Continue normal daily activities within limits permitted by back pain.	Compared with following advice to rest in bed for 2 to 4 d, ⁵⁶ those who followed advice to stay active reported reduced pain at 4 wk (SMD, 0.2 [95% CI, 0.02 to 0.4]) and improved functional status at 4 wk (SMD, 0.3 [95% CI, 0.1 to 0.5]).	Considered safe.
Education	Information to improve an individual's understanding of their low back pain and appropriate management.	Compared with not receiving information or receiving placebo advice for 2 to 12 wk, ⁵⁷ the receipt of pain management information was associated with reduced pain (MD, -3.3 [95% CI, -5.9 to -0.8]) on a scale from 0 to 100 but not reduced disability (MD, -1.3 [95% CI, -3.3 to 0.6]) on a scale from 0 to 100.	Considered safe. No difference in adverse events for education group vs control group (RR, 1.3 [95% CI, 0.3 to 4.9]). ⁵⁷
Superficial heat	Application of heat to the lumbar spine; common modalities include hot water bottles, hot towels, heat wraps, and heating pads.	Compared with receiving an oral placebo, ⁵⁸ the use of a heat wrap was associated with reduced pain after 5 d (MD, 1.1 [95% CI, 0.7 to 1.5]) on a scale from 0 to 5 and reduced disability after 4 d (MD, -2.1 [95% CI, -3.2 to -1.0]) on a scale from 0 to 24.	Considered safe. No major adverse events. Minor events, including skin pinkness, resolved quickly. ⁵⁸
Spinal manipulation	Manual therapy involving controlled, high-velocity, low-amplitude thrusts to spinal joints, typically using short- or long-lever techniques to move the joint beyond its current restriction but within anatomical limits.	Compared with receiving sham or alternative treatments for ≤6 wk, ⁵⁹ receipt of spinal manipulation was associated with reduced pain (MD, -10 [95% CI, -15.6 to -4.3]) on a scale from 0 to 100 and reduced disability (SMD, -0.4 [95% CI, -0.7 to -0.07]).	Considered safe. No major adverse events. Minor events included transient local discomfort or increased pain. ⁵⁹
Massage	Manipulation of soft tissue (eg, muscle, tendon, ligament, fascia) through specific methods.	Compared with receiving an inactive control for 4 wk, ⁶⁰ receipt of massage was associated with reduced pain (SMD, -1.2 [95% CI, -1.9 to -0.6]) but not reduced disability (SMD, -0.5 [95% CI, -1.1 to 0.1]).	Considered safe. No major adverse events. Minor events included transient increased pain or skin discomfort.
Acupuncture	Insertion of fine needles into anatomical points on the body.	Compared with receiving sham acupuncture, ⁶¹ receipt of acupuncture was associated with reduced pain (MD, -9.4 [95% CI, -17.0 to -1.8]) on a scale from 0 to 100 but not reduced disability.	Considered safe. No major adverse events. Minor events included transient increased pain, bleeding, bruising, or fainting. ⁶²
Chronic nonspecific low back pain (length of pain: >12 wk)			
Advice and education	Same as described for acute nonspecific low back pain.	Compared with not receiving advice or receiving placebo advice for 2 to 12 wk, ⁵⁷ receipt of advice was associated with reduced pain (MD, -11.2 [95% CI, -17.0 to -5.3]) on a scale from 0 to 100 and reduced disability (MD, -7.9 [95% CI, -13.1 to -1.5]) on a scale from 0 to 100.	Considered safe.
Exercise	Supervised or home-based program aimed at improving aerobic fitness, muscle strength, spinal stability, and/or flexibility.	Compared with not receiving treatment for ≤12 wk, ⁶³ participating in an exercise program (all types combined) reduced pain (MD, -15.2 [95% CI, -18.3 to -12.2]) on a scale from 0 to 100 and reduced disability (MD, -6.8 [95% CI, -8.3 to -5.3]) on a scale from 0 to 100.	Considered safe. No major adverse events. Minor events included transient increased low back pain and muscle soreness. ⁶³
Psychological therapy	Interventions such as cognitive behavioral therapy provided by a psychologist to modify behavior, physiological reactivity, and cognition.	Compared with being on a waiting list, ⁶⁴ those who received cognitive behavioral therapy reported reduced pain (SMD, -0.5 [95% CI, -0.8 to -0.2]) and reduced disability (SMD, -0.5 [95% CI, -0.8 to -0.2]).	Considered safe.
Spinal manipulation	Same as described for acute nonspecific low back pain.	Compared with receiving other conservative interventions for 1 mo, ⁶⁵ those who received spinal manipulation reported reduced pain (MD, -4.7 [95% CI, -8.3 to -1.2]) on a scale from 0 to 100 and reduced disability (SMD, -0.3 [95% CI, -0.4 to -0.1]).	Considered safe. No major adverse events. Minor events included muscle soreness, stiffness, and/or a transient increase in pain. ⁶⁵
Massage	Same as described for acute nonspecific low back pain.	Compared with receiving an inactive control for 0 to 6 mo, ⁶⁶ those who received massages reported reduced pain (SMD, -0.8 [95% CI, -0.9 to -0.6]) and reduced disability (SMD, -0.7 [95% CI, -1.1 to -0.4]).	Considered safe. No difference in adverse events (RD, 0.06 [95% CI, 0 to 0.1]; 60 per 1000 for massage vs 4 per 1000 for control). ⁶⁶
Acupuncture	Same as described for acute nonspecific low back pain.	Compared with receiving a sham treatment for ≤7 d, ⁶⁶ those who received acupuncture reported reduced pain (MD, -9.2 [95% CI, -13.8 to -4.6]) on a scale from 0 to 100 but not reduced disability (SMD, -0.2 [95% CI, -0.4 to 0.1]).	Considered safe. No major adverse events. Minor events included pain at insertion points, hematoma, bleeding, and worsening of low back pain. ⁶⁶
Multidisciplinary or multicomponent	Combines physical, vocational, and behavioral components provided by multiple health care professionals from different clinical backgrounds.	Compared with receiving usual care for 12 mo, ⁶⁷ those who received multidisciplinary care reported reduced pain (SMD, -0.2 [95% CI, -0.4 to -0.04]) and disability (SMD, -0.2 [95% CI, -0.4 to -0.1]).	Considered safe. No additional adverse events beyond those relevant for interventions included in the program. ⁶⁷
Abbreviations: MD, mean difference; RD, risk ratio or relative risk; SMD, standardized mean difference.		or range from 10 to 20 points (on a scale from 0 to 100). Absolute thresholds for disability range from 2 to 5 points on the Roland Morris Disability Questionnaire (scale from 0 to 24) and by approximately 10 points on the Oswestry Disability Index (on a scale from 0 to 100).	

^a The minimum clinically important difference for pain severity and disability is commonly defined as a 30% reduction from baseline. Absolute thresholds for pain intensity range from 1 to 2 points (on a scale from 0 to 10)

Figure 3. Summary of Low Back Pain Treatment Guidelines From the US, UK, and the World Health Organization

		ACUTE NONSPECIFIC LOW BACK PAIN		CHRONIC NONSPECIFIC LOW BACK PAIN		
		US	UK	US	UK	World Health Organization
Recommended treatments		American College of Physicians (ACP)	National Institute for Health and Care Excellence (NICE)	ACP	NICE	World Health Organization
NONPHARMACOLOGICAL	Advice and education (eg, information about low back pain, encourage physical activity, self-management)					
	Superficial heat (eg, heat wrap)					
	Exercise (eg, aerobic, strength training, flexibility, stretching)					
	Spinal manipulation ^a					
	Massage ^a					
	Acupuncture					
	Psychological therapy (eg, cognitive behavioral therapy, mindfulness-based stress reduction) ^b					
	Multidisciplinary and/or multicomponent therapy					
	Electrotherapy (eg, TENS)					
PHARMACOLOGICAL	Nonsteroidal anti-inflammatory drugs (NSAIDs) (ie, ibuprofen, diclofenac, naproxen, celecoxib)					
	Acetaminophen					
	Skeletal muscle relaxants (eg, cyclobenzaprine, tizanidine, orphenadrine)					
	Opioids (eg, oxycodone, tapentadol, tramadol, buprenorphine, morphine) ^c					
	Antidepressants (ie, SSRIs, TCAs)					
	Antidepressants (ie, SNRIs)					
	Antiepileptics (eg, gabapentin, pregabalin)					
	Systemic steroids (eg, corticosteroids)					

Recommended
 Conditional recommendation
 Inconclusive/insufficient evidence
 Recommended against
 No mention

The recommendations were taken from the US, UK, and the World Health Organization clinical practice guidelines; the criteria required for the recommendations are detailed fully in the published guidelines.⁶⁻⁸ The US guideline provides recommendations for noninvasive treatments for acute and chronic low back pain.⁷ The UK guideline provides recommendations for invasive and noninvasive treatments for acute and chronic low back pain.⁸ The World Health Organization guideline provides recommendations for noninvasive treatments for chronic low back pain.⁶ SNRIs indicate serotonin and norepinephrine reuptake inhibitors; SSRIs, selective serotonin reuptake inhibitors; TCAs, tricyclic antidepressants; TENS, transcutaneous electrical nerve stimulation.

^aFor conditional recommendations (yellow boxes), treatment must be provided as part of a treatment package that includes exercise with or without psychological therapy.

^bFor conditional recommendations (yellow boxes), treatment must be provided as part of a treatment package that includes exercise with or without spinal manipulation and/or massage.

^cFor conditional recommendations (yellow boxes), treatment only provided if other treatments are contraindicated, not tolerated, or have been ineffective after discussion of known risks and realistic benefits.

Pharmacological Treatments

For patients with low back pain that substantially limits function or activity, or when nonpharmacological care is not feasible due to limited access, high cost, or the need for timely symptom relief, first-line pharmacological options include nonsteroidal anti-inflammatory drugs (NSAIDs; such as ibuprofen or naproxen) and skeletal muscle relaxants (such as cyclobenzaprine or tizanidine).^{7,8} In a meta-analysis⁴⁸ of 4 clinical trials that included 815 patients with acute nonspecific low back pain, NSAIDs were associated with a small statistically significant reduction in pain severity (MD, -7.3 [95% CI, -11.0 to -3.6] on a scale from 0-100) compared with placebo. There was no clear statistically significant difference between selective cyclooxygenase-2 inhibitor NSAIDs and nonselective NSAIDs in reducing pain severity.⁴⁸ NSAIDs should be used with caution in older adults and in patients with kidney disease, peptic ulcer disease, or

cardiovascular disease. In a meta-analysis⁵¹ of 3 randomized clinical trials that included 1167 patients with acute and chronic low back pain, there was a statistically significant higher number of participants with gastrointestinal adverse events reported in the NSAIDs group compared with placebo (28/702 [3.9%] vs 9/465 [1.9%]; relative risk, 2.5 [95% CI, 1.2 to 5.2]).

Skeletal muscle relaxants (eg, cyclobenzaprine, tizanidine) were associated with statistically significant but small pain severity reductions compared with placebo in a meta-analysis⁴⁹ of 16 trials that included 4546 patients with acute nonspecific low back pain (MD, -7.7 [95% CI, -12.1 to -3.3] on a scale from 0-100), but the skeletal muscle relaxants were associated with an increased risk of adverse events, particularly sedation, compared with placebo. Benzodiazepines (eg, diazepam) are not recommended. In addition to the potential for misuse, physiological dependence, and benzodiazepine use disorder,

Box 2. Education and Self-Management Messages for Patients With Nonspecific Low Back Pain

All patients with nonspecific low back pain should receive advice and education that are tailored to their needs and capabilities to support self-management. *Self-management* refers to the everyday actions patients can take to manage their low back pain and maintain their usual activities, including symptom-relief strategies (such as superficial heat) and pacing activity levels toward meaningful functional goals. Clinicians should provide guidance on these strategies, which can be reinforced with evidence-based resources (such as brief written materials).^{46,47}

Understanding low back pain

Patients should be reassured that most low back pain arises from minor strains of muscles, joints, or ligaments of the back and does not indicate serious pathology or lasting harm. Fluctuations in pain intensity are common and do not imply further tissue damage. For acute nonspecific low back pain, substantial improvement typically occurs within 6 weeks. For patients with chronic nonspecific low back pain, clinicians should emphasize that functional improvement is achievable even if pain does not fully resolve, and that remaining engaged in valued activities is an important goal.

Imaging

Clinicians should explain that diagnostic imaging is rarely indicated for nonspecific low back pain and that imaging frequently identifies incidental findings such as disk bulges, protrusions, degeneration, and facet arthropathy that are also common in people without pain and usually reflect age-related changes rather than the source of symptoms.

Validating the pain and addressing patient misconceptions

Clinicians should acknowledge that the patient's pain is real and distressing, and should elicit beliefs about the cause of symptoms, correct misconceptions (eg, pain does not equal harm; movement is safe), and reinforce the importance of maintaining or returning to usual activities, including a graded return to work or other meaningful roles. Patients should be reassured that temporary increases in pain with activity are common and not harmful.

diazepam did not significantly improve disability at 1 week (MD, 0.3 [95% CI, -2.8 to 3.5] on a scale from 0-24) compared with placebo in a randomized clinical trial⁷⁹ including 114 adults with acute nonspecific low back pain.

Acetaminophen (paracetamol) is not recommended as a single therapy for low back pain based on a single randomized trial⁸ including 1652 adults and reporting that the time to recovery did not significantly differ between use of scheduled acetaminophen (1330 mg administered 3 times per day; median recovery of 17 days [95% CI, 14-19]), use of as-needed acetaminophen (maximum dose of 4000 mg/d; median recovery of 17 days [95% CI, 15-20]), or use of placebo (median recovery of 16 days [95% CI, 14-20]).⁸⁰ In a meta-analysis⁸¹ including 2 trials, use of systemic corticosteroids (eg, prednisone, methylprednisolone) was not significantly associated with improved pain severity (MD, 0.6 [95% CI, -0.4 to 1.7] on a scale from 0-10) for nonspecific low back pain or improved function (SMD, -0.1 [95% CI, -0.5 to 0.3]); systemic corticosteroids are not recommended.

Use of opioids should be reserved for patients with severe, disabling acute nonspecific low back pain that does not respond to recommended pharmacological treatments (such as NSAIDs and skeletal

muscle relaxants) with prescriptions for short courses (eg, a few days or less) of immediate-release formulations (such as codeine or hydrocodone as needed).⁸² Recommendations vary across guidelines; NICE⁸ recommends use of weak opioids such as codeine (with or without paracetamol) for acute nonspecific low back pain only if an NSAID is contraindicated, not tolerated, or has been ineffective, whereas the ACP guideline⁷ for acute nonspecific low back pain makes no recommendation because of insufficient evidence, and the guideline from the ACP and the American Academy of Family Physicians for acute musculoskeletal injuries⁸³ recommends against opioids including tramadol. More recent evidence also questions the benefit of opioids; in a randomized trial⁸⁴ including 347 adults with acute low back and neck pain, modified-release oxycodone/naloxone did not significantly improve pain severity at 6 weeks compared with placebo (MD, 0.5 [95% CI, 0 to 1.1] on a scale from 0-10).

Chronic Nonspecific Low Back Pain

Nonpharmacological treatments are recommended as first-line therapy for chronic nonspecific low back pain to reduce disability, improve function, and manage common comorbidities (such as depression) that are associated with poorer prognosis and functional outcomes.^{6,7} Pharmacological treatments are recommended as second-line therapies when nonpharmacological care does not achieve sufficient pain relief.⁷ Given their limited benefits and risk of adverse effects, invasive interventions (such as radiofrequency denervation or spinal surgery) should be reserved for patients who have not responded to first- and second-line treatments.^{8,85}

Nonpharmacological Treatments

Advice on staying active and education about low back pain are recommended treatments for patients with chronic nonspecific low back pain. Education should focus on helping patients understand why pain can persist in the absence of ongoing tissue injury, address unhelpful beliefs (eg, that persistent pain indicates ongoing damage or movement is harmful), and support self-management strategies, including maintaining physical activity, managing acute worsening of symptoms, and reducing avoidance of movement and activities due to concerns that they may worsen pain.⁸⁶ Clinicians can explain^{86,87} that persistent pain does not necessarily indicate ongoing tissue damage, and may reflect increased sensitivity of the nervous system that can develop over time.

Exercise and psychological interventions, alone or in combination, are recommended as first-line treatments for chronic nonspecific low back pain in guidelines from the ACP,⁷ NICE,⁸ and the WHO.⁶ In a meta-analysis⁶³ of 35 trials that included 2746 people with chronic nonspecific low back pain, supervised exercise programs (including strength training, aerobic exercise, Pilates, flexibility, and combined exercise programs) vs no treatment were associated with moderate and small statistically significant reductions in pain severity (MD, -15.2 [95% CI, -18.3 to -12.2]) and disability (MD, -6.8 [95% CI, -8.3 to -5.3]) on a scale from 0 to 100. No specific exercise modality has demonstrated superior benefit compared with alternative exercise modalities,⁸⁸⁻⁹¹ and patients should be encouraged to engage in exercise programs that align with their preferences, capabilities, out-of-pocket costs, and accessibility to promote adherence and adequate cumulative dose (≥ 20 hours over 8-12 weeks).⁸⁸

Psychological treatments, including CBT, were associated with small to moderate statistically significant reductions in pain severity

compared with those on the waiting list (SMD, -0.5 [95% CI, -0.8 to -0.2]) in a meta-analysis⁶⁴ of 22 trials that included 2340 adults with chronic nonspecific low back pain. Brief formats of CBT may retain effectiveness. In a randomized clinical trial including 263 adults,⁹² a single 2-hour session class (included pain education, mindfulness principles, and CBT skills) designed to help participants manage pain was noninferior at 3 months to an 8-session CBT program for pain severity (MD, 0.1 [97.5% CI, $-\infty$ to 0.6] on a scale from 0-10).

Interventions that integrate exercise with psychological approaches may reduce pain severity and disability for 1 year or longer.^{93,94} In a randomized clinical trial⁹⁵ including 276 adults with chronic nonspecific low back pain, graded sensorimotor retraining (consisting of individualized education to improve understanding of pain and its contributing factors, exercises to improve sensory perception and body awareness, and a gradual progression of movements and activities that are painful, feared, or avoided) significantly reduced disability up to 1 year (MD, -1.8 [95% CI, -3.1 to -0.5] on a scale from 0-24) compared with a sham and attention control. Similarly, in a randomized clinical trial⁹⁶ including 492 adults with chronic nonspecific low back pain, compared with usual care, cognitive functional therapy consisting of individualized pain education, graded movement exposure, and lifestyle change reduced pain severity up to 1 year (MD, -1.5 [95% CI, -2.0 to -0.9] on a scale from 0-10) and reduced disability (MD, -4.8 [95% CI, -6.0 to -3.5] on a scale from 0-24).

For patients with substantial functional disability or in those who do not improve with first-line management, multidisciplinary rehabilitation programs (integrating physical, vocational, and behavioral components delivered by multiple disciplines) provide moderate reductions in pain severity and disability.⁶⁷ In a meta-analysis⁶⁷ including 7 clinical trials and 821 adults with chronic nonspecific low back pain, multidisciplinary rehabilitation, compared with usual care, was associated with small improvements in pain severity (SMD, -0.2 [95% CI, -0.4 to -0.04]) and disability (including 6 trials and 722 adults; SMD, -0.2 [95% CI, -0.4 to -0.1]) and was more effective than exercise or psychological therapies alone. However, limited availability of multidisciplinary rehabilitation programs, high costs, and high treatment intensity may limit access for many patients.

Other nonpharmacological therapies for chronic nonspecific low back pain include spinal manipulation⁶⁵ and acupuncture,⁶⁶ which provide short-term reductions in pain severity. Guideline recommendations for these treatments vary. Guidelines from the ACP⁷ and the WHO⁶ recommend these therapies, whereas the NICE guideline⁸ recommends spinal manipulation only as part of multimodal care and does not recommend acupuncture. Affordable mobility assistive products (eg, walking aids) may be offered to support function and participation.⁶ The therapies for chronic nonspecific low back pain that are not recommended by guidelines due to a lack of demonstrated effectiveness include spinal traction, therapeutic ultrasonography, electrotherapies (eg, transcutaneous electrical nerve stimulation), and lumbar supports.^{6,8}

Pharmacological Treatments

Pharmacological treatments should be considered only when patients with chronic nonspecific low back pain do not improve or worsen with recommended nonpharmacological treatments.⁷ Compared with placebo, the benefits of most medicines are no better

than nonpharmacological treatments and most are associated with an increased risk of adverse events.⁹⁷

NSAIDs (such as ibuprofen and naproxen) are the first-line pharmacological treatment for nonspecific low back pain.⁶⁻⁸ In a meta-analysis⁵¹ of 9 clinical trials that included 2537 adults with chronic nonspecific low back pain, NSAIDs were associated with moderate statistically significant reductions in pain severity (MD, -11.1 [95% CI, -13.8 to -8.4] on a scale from 0-100) compared with placebo. However, NSAIDs were associated with significantly increased gastrointestinal adverse events (28 of 702 [4.0%] vs 9 of 465 participants [1.9%] who received placebo; relative risk, 2.5 [95% CI, 1.2 to 5.2]) and require careful patient selection and monitoring.⁵¹ Second-line pharmacological treatments recommended by the ACP guideline⁷ include the serotonin and norepinephrine reuptake inhibitor duloxetine (an antidepressant). In a meta-analysis⁵² of 4 trials that included 1415 adults with chronic nonspecific low back pain, duloxetine was associated with small reductions in pain severity (MD, -5.3 [95% CI, -7.2 to -3.3] on a scale from 0-100) compared with placebo. However, the NICE⁸ and WHO⁶ guidelines recommend against antidepressants because the small benefits are outweighed by an increased risk of adverse events (such as nausea and somnolence).

Use of acetaminophen,⁵⁰ corticosteroids,⁸¹ skeletal muscle relaxants (such as tizanidine or cyclobenzaprine),⁴⁹ and antiepileptics (such as pregabalin or gabapentin)⁹⁸ are not recommended for treatment of chronic nonspecific low back pain due to lack of benefit and increased risk of medication-related adverse events (such as dizziness, sedation, and gastrointestinal events).

The ACP guideline⁷ recommends opioids only for patients with severe, chronic low back pain unresponsive to other treatments,^{7,55,82} but the NICE⁸ and WHO⁶ guidelines recommend against opioids for chronic nonspecific low back pain. In a meta-analysis⁵³ including 5 clinical trials and 1378 patients with chronic nonspecific low back pain, tramadol (a synthetic dual mechanism opioid analgesic) was associated with moderate statistically significant reductions in back pain severity (SMD, -0.6 [95% CI, -0.7 to -0.4]) compared with placebo but tramadol significantly increased the risk of serious and nonserious adverse events, including nausea and dizziness.⁵⁴ When initiating opioids, clinicians should prescribe them for a trial period (eg, ≤ 3 -7 days) at the lowest effective dose and shortest possible duration (eg, 25 to 30 mg of tramadol every 6 to 8 hours), with regular reassessment.⁸² For patients already receiving opioids long-term, the lack of meaningful benefit should prompt consideration of gradual tapering, which may improve pain severity, function, and quality of life.⁹⁹

There is limited evidence for cannabis-based treatments. In a randomized clinical trial¹⁰⁰ including 820 adults with chronic low back pain, a cannabis oral extract containing tetrahydrocannabinol with other naturally occurring cannabis constituents, provided a small statistically significant reduction in pain severity (MD, -0.6 [95% CI, -0.9 to -0.3] on a scale from 0-10) but increased adverse events (such as dizziness and fatigue) compared with placebo (83.3% vs 67.3%). Cannabis-based treatments are not currently approved by the US Food and Drug Administration for chronic noncancer pain.¹⁰¹

Invasive Treatments

Spinal injections, radiofrequency denervation, spinal cord stimulation, and spinal surgery are rarely indicated for chronic nonspecific

low back pain. These interventions should be considered only for patients in whom a plausible source of low back pain has been identified and noninvasive treatments have been unsuccessful.^{8,85}

Spinal injections, including epidural and facet joint injections, have not demonstrated benefits for patients with chronic nonspecific low back pain. In a network meta-analysis¹⁰² including 47 trials and 5290 adults with chronic low back pain, spinal injections with local anesthetic and/or steroids were not associated with significantly more benefit than sham procedures, with effect estimates ranging from 0.2 to 0.8 points worse on a pain severity scale ranging from 0 to 10. Therefore, clinical practice guidelines do not recommend spinal injections for nonspecific low back pain.^{8,85}

The effectiveness of radiofrequency denervation (involving thermal ablation of spinal nerves) remains uncertain. The NICE guideline⁸ recommends denervation only for patients with suspected facet-mediated pain who have a positive response (eg, >50% pain reduction) to diagnostic medial branch blocks by local anesthetic injection. However, a meta-analysis^{103,104} including 6 trials and 348 adults with chronic low back pain reported no significant benefit compared with sham or no treatment (MD, -0.6 [95% CI, -1.4 to 0.1] on a scale from 0-10).

Few high-quality, sham-controlled trials not sponsored by the pharmaceutical and device industries are available on spinal cord stimulation for chronic nonspecific low back pain. Evidence suggests that spinal cord stimulation does not provide sustained benefits that outweigh the costs and risks (such as infection, neurological damage, and migration of the device leads requiring repeated surgery).¹⁰⁵

Spinal fusion surgery and disk replacement surgery are not recommended for nonspecific low back pain.⁸ In a randomized trial¹⁰⁶ including 349 adults with chronic low back pain, lumbar fusion did not improve walking capacity compared with intensive rehabilita-

tion (MD, 34 m [95% CI, -8 to 77 m]) and produced only a slight statistically significant reduction in disability (MD, -4.1 [95% CI, -8.1 to -0.1] on a scale from 0-100) at 24 months. Two smaller randomized trials including 64 adults¹⁰⁷ and 60 adults¹⁰⁸ with chronic low back pain reported consistent findings that spinal fusion was not significantly better than intensive rehabilitation (pain education, graded activity exposure, and exercise) for improvement in pain severity and function. Based on shared decision-making with explicit discussion of limited benefit, potential harms, and uncertainty of long-term outcomes, referral for specialist surgical assessment may be considered for patients with refractory chronic low back pain symptoms unresponsive to nonsurgical therapies for at least 1 year that substantially limit daily activities and work.⁸⁵

Limitations

This review has limitations. First, the quality of the included literature was not formally reviewed. Second, some relevant articles may have been missed. Third, the search was limited to English-language articles.

Conclusions

Low back pain is a leading cause of disability worldwide. Acute nonspecific low back pain is often self-limited, whereas chronic nonspecific low back pain has a less favorable prognosis. For patients with acute nonspecific low back pain, first-line treatments include selected nonpharmacological therapies and medications (such as NSAIDs and skeletal muscle relaxants). For patients with chronic nonspecific low back pain, first-line treatment consists of exercise, psychological therapies (such as CBT), and combined multidisciplinary care.

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Submissions: We encourage authors to submit papers for consideration as a Review. Please contact Kristin Walter, MD, at kristin.walter@jamanetwork.org.

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