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Diagnosis and Management of Lumbar Spinal Stenosis
A Review

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IMPORTANCE Lumbar spinal stenosis is a prevalent and disabling cause of low back and leg pain in older persons, affecting an estimated 103 million persons worldwide. Most are treated nonoperatively. Approximately 600 000 surgical procedures are performed in the US each year for lumbar spinal stenosis.

OBSERVATIONS The prevalence of the clinical syndrome of lumbar spinal stenosis in US adults is approximately 11% and increases with age. The diagnosis can generally be made based on a clinical history of back and lower extremity pain that is provoked by lumbar extension, relieved by lumbar flexion, and confirmed with cross-sectional imaging, such as computed tomography or magnetic resonance imaging (MRI). Nonoperative treatment includes activity modification such as reducing periods of standing or walking, oral medications to diminish pain such as nonsteroidal anti-inflammatory drugs (NSAIDs), and physical therapy. In a series of patients with lumbar spinal stenosis followed up for up to 3 years without operative intervention, approximately one-third of patients reported improvement, approximately 50% reported no change in symptoms, and approximately 10% to 20% of patients reported that their back pain, leg pain, and walking were worse. Long-term benefits of epidural steroid injections for lumbar spinal stenosis have not been demonstrated. Surgery appears effective in carefully selected patients with back, buttock, and lower extremity pain who do not improve with conservative management. For example, in a randomized trial of 94 participants with symptomatic and radiographic degenerative lumbar spinal stenosis, decompressive laminectomy improved symptoms more than nonoperative therapy (difference, 7.8 points; 95% CI, 0.8-14.9; minimum clinically important difference, 10-12.8) on the Oswestry Disability Index (score range, 0-100). Among persons with lumbar spinal stenosis and concomitant spondylolisthesis, lumbar fusion increased symptom resolution in 1 trial (difference, 5.7 points; 95% CI, 0.1 to 11.3) on the 36-Item Short Form Health Survey physical dimension score (range, 0-100), but 2 other trials showed either no important differences between the 2 therapies or noninferiority of lumbar decompression alone compared with lumbar decompression plus spinal fusion (MCID, 2-4.9 points). In a noninferiority trial, 71.4% treated with lumbar decompression alone vs 72.9% of those receiving decompression plus fusion achieved a 30% or more reduction in Oswestry Disability Index score, consistent with the prespecified noninferiority hypothesis. Fusion is associated with greater risk of complications such as blood loss, infection, longer hospital stays, and higher costs. Thus, the precise indications for concomitant lumbar fusion in persons with lumbar spinal stenosis and spondylolisthesis remain unclear.

CONCLUSIONS AND RELEVANCE Lumbar spinal stenosis affects approximately 103 million people worldwide and 11% of older adults in the US. First-line therapy is activity modification, analgesia, and physical therapy. Long-term benefits from epidural steroid injections have not been established. Selected patients with continued pain and activity limitation may be candidates for decompressive surgery.

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Multimedia

Supplemental content

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Lumbar spinal stenosis is a common cause of pain and disability in older persons. Although there are no rigorous epidemiological studies of the prevalence of lumbar spinal stenosis, one estimate suggests that approximately 103 million individuals have symptomatic lumbar spinal stenosis worldwide.[1] More than 350 000 persons in the US 45 years or older underwent decompressive laminectomy and another 370 000 had lumbar fusion in 2014,[2] with the great majority of these procedures done for lumbar spinal stenosis.

Symptomatic lumbar spinal stenosis is characterized by low back and leg pain in the setting of compression of the central canal and/or exiting nerve roots by disk, osteophyte, ligamentum flavum, or other structures (**Figure 1**). This review summarizes current evidence regarding diagnosis and treatment of acquired, degenerative, lumbar spinal stenosis.

Symptomatic lumbar spinal stenosis is costly. In one study,[3] participants with lumbar spinal stenosis accrued $9892 over 2 years for nonoperative management costs, including outpatient visits, diagnostic tests, medications, medical devices, injections, and rehabilitation. US Surgical costs in 2018 exceeded $15 billion.[2]

Methods

We searched the PubMed, Ovid, and PEDro (Physical Therapy Evidence Database) research databases between January 2000 and December 2021 for articles about diagnosis and treatment of lumbar spinal stenosis. We crossed the key words *lumbar spinal stenosis* or *spinal stenosis* with *burden*, *incidence*, *risk factors*, *prevalence*, *surgery*, *medication*, *acetaminophen*, *gabapentin*, *NSAIDs*, *opioids*, *epidural injections*, *physical therapy*, *exercise*, *physical examination*, *sensitivity*, *specificity*, *diagnosis*, *diagnostic test*, *history*, and *clinical guidelines*. We searched references from identified publications for additional relevant articles. We excluded animal experiments, case reports, and articles not available in English or that did not provide quantitative data. We selected articles on diagnosis that reported sensitivity and specificity, and articles on treatment that reported either mean between-group differences in change in outcome from baseline, or sufficient detail for us to calculate these differences, or the proportion of participants in each study group improving by a specified amount. We required that clinical trials include at least 25 participants per treatment group. Of the 68 articles included in this review, 23 were randomized clinical trials; 9 were evaluations of patient reported outcome measures; 8 were treatment guidelines; 6 were systematic reviews; 9 were narrative reviews; 6 were observational studies; 6 were cross-sectional studies; and 1 was a cost-effectiveness analysis.

Pathophysiology

Lumbar spinal stenosis is characterized by narrowing of the lumbar spinal canal and/or the neural foramina, with compression of the spinal nerve roots (Figure 1). The spinal cord typically ends at about L1, transitioning to a bundle of nerve roots resembling a horse's tail (termed *cauda equina*). The nerve roots exit the central canal through neural foramina to innervate the lower extremities. Spondylolisthesis (forward displacement of one vertebra with respect to an adjacent vertebra) introduces an additional source of compression (**Figure 2**). Lumbar spinal stenosis typically develops with older age and is a degenerative condition. Less common etiologies include congenital stenosis (due to a congenitally small central canal); metabolic syndromes such as Paget disease, in which overgrowth of bone affected by Paget disease can compress spinal nerve roots; and epidural lipomatosis, in which excess fat deposited in the epidural space (typically from endogenous or exogenous corticosteroid excess) can compress traversing spinal nerve roots.

Acquired degenerative lumbar spinal stenosis arises from degenerative changes, including combinations of disk protrusion, facet joint hypertrophy, ligamentum flavum hypertrophy, and spondylolisthesis (Figure 1). As disk height is lost, load (weight) is transferred to the facet and intervertebral joints, which form osteophytes. The posterior longitudinal ligament folds and hypertrophies. The combination of disk protrusion, facet joint osteophytes, ligamentum flavum buckling and hypertrophy, and spondylolisthesis all contribute to displacement of nerve roots in the central canal and/or neural foramina and lateral recesses, through which nerves exit to the lower extremities[4] (Figure 1).

The mechanism whereby compression of nerve roots gives rise to back pain, lower extremity pain, paresthesia, and weakness is not fully understood. One theory is that the osteophytes and ligament hypertrophy that occur with spinal stenosis compress small arterioles, causing ischemia of the nerve. Alternatively, spinal canal stenosis may prevent normal venous drainage, leading to increased venous pressure, accumulation of toxic metabolites, and nerve root damage.[4]

Clinical Presentation

Patients with degenerative lumbar spinal stenosis generally present for care when symptoms affect their ability to work, shop, perform housework, walk distances, or participate in other valued activities. Patients typically present[5] with discomfort in the lower lumbar spine, buttocks, and thighs, sometimes extending to the lower legs and feet.[6,7] The lower extremity pain may be unilateral or bilateral and may be accompanied by numbness and paresthesia of the feet and lower legs, reflecting compression of sensory fibers. Later in the course, patients may experience worsening of balance and a wide-based gait reflecting compression of the posterior column fibers that provide awareness of position.[6,7]

Approximately 20% of people older than 60 years have imaging evidence of lumbar spinal stenosis,[8-10] more than 80% of whom are asymptomatic. Treatment is not indicated for asymptomatic stenosis.

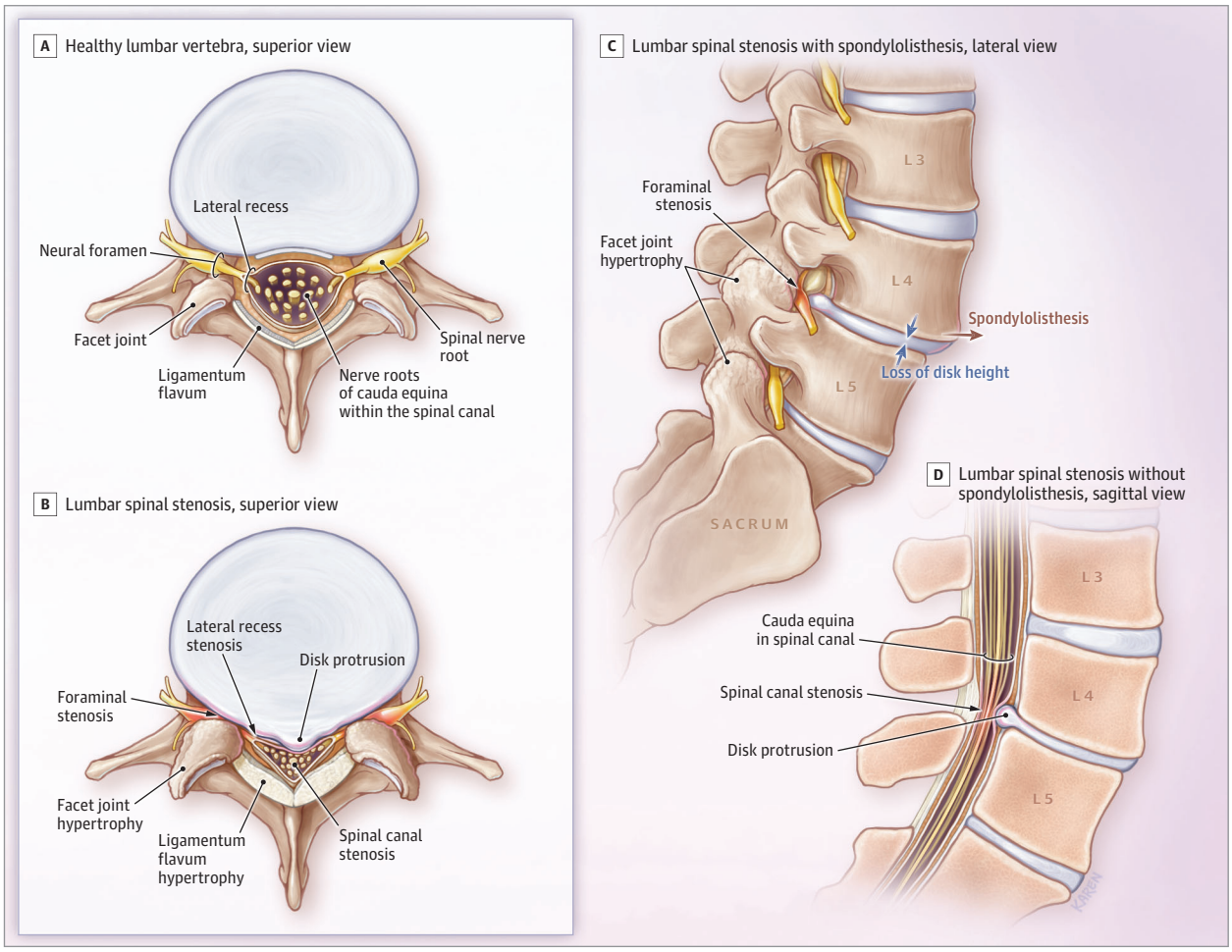
Assessment and Diagnosis

The diagnosis of lumbar spinal stenosis can generally be made with a careful history and physical examination (**Box 1**). Imaging can confirm the structural diagnosis and clarify the anatomy if therapeutic injections or surgery are contemplated. There are no history or physical examination findings that are both highly sensitive and specific for lumbar spinal stenosis. Few studies have examined the diagnostic value of combinations of symptoms and signs. When imaging is indicated, cross-sectional imaging with magnetic resonance imaging (MRI) or computed tomography (CT) can confirm the diagnosis.

History

Discomfort from lumbar spinal stenosis is typically exacerbated by standing and walking and relieved with sitting and bending

Figure 1. Pathoanatomy of Degenerative Lumbar Spinal Stenosis



Central and foraminal stenosis arise from facet joint space loss, osteophyte formation, ligamentum flavum hypertrophy, and disk protrusion.

forward. Pain in the buttock or lower extremity that is exacerbated by lumbar extension (eg, walking) is referred to as neurogenic claudication. Absence of low back pain while sitting is approximately 52% to 70% sensitive and approximately 55% to 83% specific for lumbar spinal stenosis.[6,7] Some patients with spinal stenosis report that walking is more comfortable when leaning on a shopping cart, when the spine is flexed. Worsening of spinal stenosis symptoms when the back is extended and improvement of symptoms when the lower back is flexed occurs because spinal extension reduces the cross-sectional area of the central spinal canal and neural foramina. Patients often report poor balance and unsteadiness, especially later in the clinical course.[7] A report of poor balance in a person with back pain is associated with a 70% sensitivity and a 53% specificity for spinal stenosis.[7] Spinal stenosis can compress the posterior column fibers, reducing proprioception and impairing balance.

On physical examination, a wide-based gait and positive Romberg sign are associated with a specificity of more than 90%, but a sensitivity of only approximately 40% for lumbar spinal stenosis.[7] The spine is generally not tender. Lumbar extension often elicits pain in the lumbosacral junction, buttocks, and thighs. Thigh pain on lumbar extension is associated with a sensitivity of 51% and a specificity of 69% for the diagnosis of spinal stenosis.[7] Flexion of the lumbar spine typically relieves these symptoms.

The most frequently involved interspace in lumbar spinal stenosis is L4-5. Of the 289 participants in the SPORT (Spine Patient Outcomes Trial),[11] 92% had stenosis at L4-5, 66% at L3-4, 28% at L2-3, and 26% at L5-S1. Sensory deficits (to pinprick or vibration) in the L3 to S1 distributions, weakness of ankle or great toe flexion or extension, and loss of ankle reflexes have sensitivities of approximately 50% and specificities of approximately 80% for lumbar spinal stenosis among persons with back pain.[7]

Persons with lumbar spinal stenosis often have concomitant painful conditions, including leg claudication due to peripheral artery disease (PAD), hip osteoarthritis, and greater trochanteric pain syndrome. Neurogenic claudication from spinal stenosis is distinguished from these conditions in part because thigh and occasionally lower leg pain are worse with standing and relieved with sitting. In contrast, the pain of vascular claudication is typically not exacerbated by standing.[12] History of tobacco use and an abnormal ankle-brachial index are also clues to recognition of vascular claudication.[13]

In contrast to lumbar spinal stenosis, greater trochanteric pain syndrome (sometimes referred to as trochanteric bursitis)

is characterized by tenderness directly over the trochanteric bursa, and hip osteoarthritis is characterized by groin, anterior thigh, and/or buttock pain that is exacerbated by internal rotation of the flexed hip. Relief of symptoms after injection of local anesthetic (with or without corticosteroids) into the trochanteric bursa or hip can help discern whether greater trochanteric pain syndrome or hip osteoarthritis, respectively, is contributing to the patient's symptoms.

Clinicians evaluating patients for possible lumbar spinal stenosis should obtain a plain radiograph to evaluate for concomitant degenerative scoliosis and spondylolisthesis (Figure 2), which may influence treatment. In a consecutive series[14] of 272 patients who underwent surgery for lumbar spinal stenosis, 34% had spondylolisthesis of 5 mm or more and 7% had scoliosis greater than 15°. Cross-sectional imaging is indicated when treatment with epidural injections or surgery are under consideration. Cauda equina syndrome, in which sacral and lower lumbar nerve roots are compressed, may result in rapidly progressive lower-extremity muscle weakness, urinary retention, fecal incontinence, or numbness of the genital, rectal, and perineal regions. Patients with these findings should undergo urgent imaging with MRI or CT and surgical referral.

Because of its superior delineation of soft tissues such as disk, muscle, cartilage, nerve root, and ligament, MRI is the modality of choice (**Figure 3**), unless contraindicated. CT can be used for persons with contraindications to MRI. Some authors have proposed formal quantitative criteria for lumbar spinal stenosis on MRI. A spinal canal cross-sectional area less than 191 mm^2 has sensitivity of 93% and specificity of 45% for lumbar spinal stenosis.[15] A spinal cross-sectional area of less than 147 mm^2 has sensitivity of 75% and specificity of 79%.[15]

Treatment

Randomized clinical trials of treatments for lumbar spinal stenosis are summarized in **Table 1**. Further information on the outcomes measures for each trial, including the minimal clinically important difference (MCID) is provided in eTable 1 in the Supplement. **Table 2** summarizes these effectiveness data.

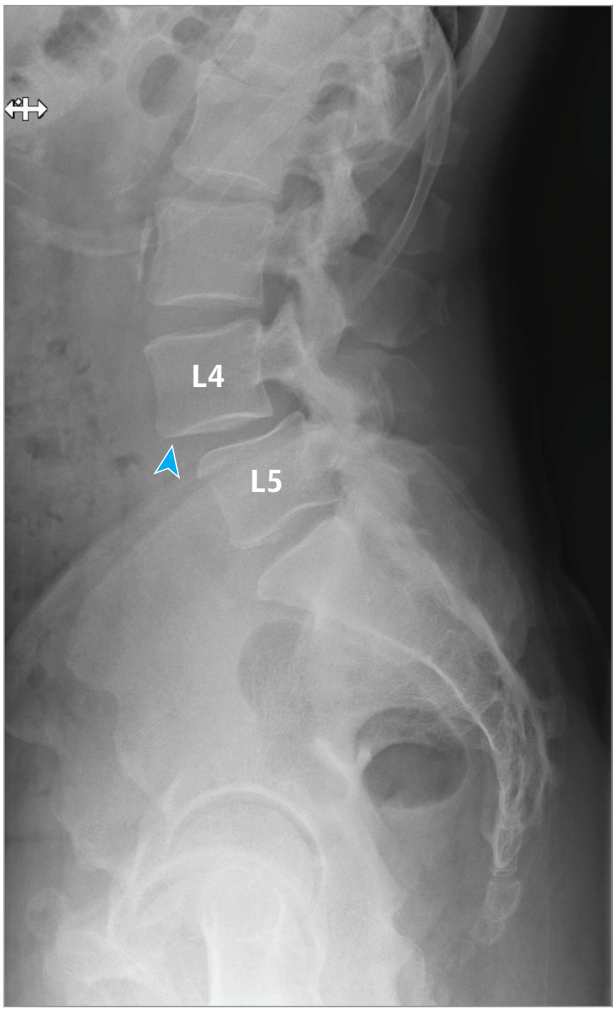
Patients with lumbar spinal stenosis generally benefit from an explanation of the relationship between posture and symptoms. To avoid exacerbating symptoms, clinicians may suggest exercises (such as biking or swimming side stroke) that are typically carried out in a lumbar flexion position.[26]

Although many studies have assessed the effectiveness of nonsteroidal anti-inflammatory medications (NSAIDs), acetaminophen, and other medications in patients with low back pain,[40,45] there is little research on the effectiveness of these medications specifically in patients with lumbar spinal stenosis. Findings from studies of other spinal disorders, such as nonspecific back pain and disk protrusion, should be applied cautiously to patients with lumbar spinal stenosis.

A meta-analysis of 2 clinical trials[41] of acetaminophen to treat patients with low back pain, including 1096 participants in the acetaminophen group and 547 in the placebo group, suggested that 4 g/d of acetaminophen was not associated with greater benefit than placebo for reducing pain and disability at the 12 week follow-up (mean difference, −0.50; 95% CI, −2.92 to 1.92) on a 0 to 10 pain scale and a mean difference of 0.10 (95% CI, −0.39 to 0.59) on the 0 to 24 Roland-Morris Disability Questionnaire. NSAIDs are effective in reducing pain and disability due to chronic low back pain of various etiologies,[45] but many older patients cannot take NSAIDs because of chronic kidney disease or epigastric pain. A meta-analysis[45] of 6 clinical trials that compared NSAIDs with placebo in patients with chronic low back pain (735 assigned to NSAIDs; 619 to placebo) reported that compared with placebo, NSAIDs were associated with greater improvement in pain intensity on the 100-mm visual analog scale after 12 or 16 weeks (12- and 16-week follow-up data from various studies were combined for the meta-analysis; mean difference, −6.97; 95% CI, −10.74 to −3.19).

Gabapentin may be effective for pain relief due to lumbar spinal stenosis (Table 1). In a randomized trial[16] of 55 participants, gabapentin plus a standard regimen of physical therapy with exercises, corset, and NSAIDs was associated with greater pain reduction (difference of 2.1 points on visual analog scale; range, 0-10; MCID, 1.5 to 2.8) than the standard regimen without gabapentin. Dizziness and drowsiness are the most common adverse effects of gabapentin,

Figure 2. Plain Radiograph Showing L4-5 Spondylolisthesis in a 71-Year-Old Man



The blue arrow indicates forward slippage of L4 with respect to L5.

Box 1. Typical Symptoms and Physical Examination Findings in Lumbar Spinal Stenosis

Symptoms
- Pain (unilateral or bilateral) radiating from the low back to below buttocks
- Pain relieved with sitting
- Pain reduced when leaning on shopping cart
- Poor balance

Physical Examination
- Wide-based gait, abnormal Romberg
- Pain exacerbated by lumbar extension
- Deficit to vibration sense in medial (L4) and lateral (S1) malleolus or great toe (L5)

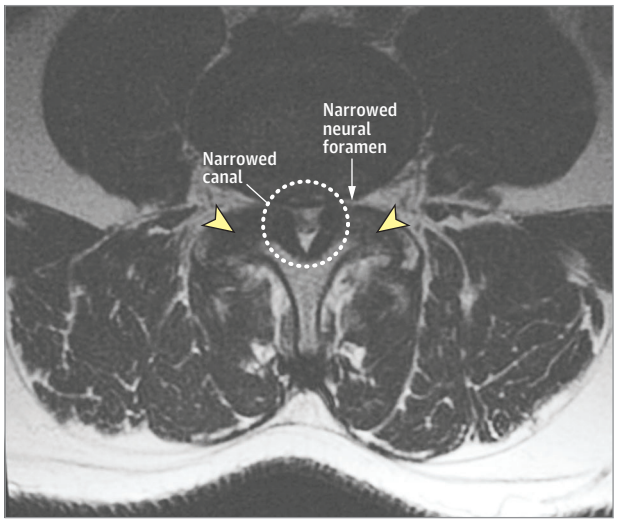
affecting up to 40% of treated patients compared with approximately 20% in placebo control patients.[49] Daily nasal calcitonin was more effective than daily gabapentin and placebo in a study of 90 participants,[18] but a systematic review and meta-analysis and Cochrane review of 232 and 213 participants, respectively, reported that calcitonin was not associated with reduced pain or improved walking distance among people with lumbar spinal stenosis compared with placebo or acetaminophen.[54]

Duloxetine, a serotonin norepinephrine reuptake inhibitor, improved pain more than placebo in randomized clinical trials of patients with chronic low back pain[50,55] but has not been studied in patients with lumbar spinal stenosis. However, adverse effects are common. In a clinical trial[50] of 458 patients with chronic low back pain, constipation occurred in 10.7% of the duloxetine group vs 2.2% of controls. Similarly, compared with placebo, duloxetine was associated with a higher frequency of nausea (9.0% vs 2.7%), dry mouth (6.0% vs 0%), somnolence (19.2% vs 7.1%), and dizziness (6.4% vs 0.9%). However, because the clinical trial of gabapentin included only 55 participants with lumbar spinal stenosis and duloxetine has not been studied specifically in patients with lumbar spinal stenosis, recommendations for these therapies must be viewed cautiously.

Clinical trials of opiates for people with lumbar spinal stenosis have not been reported. One randomized clinical trial[56] of 240 participants with chronic back pain or hip or knee osteoarthritis found that after 12 months, participants randomized to receive opioids did not report greater pain relief than those receiving nonopioid medications (NSAIDs, acetaminophen, others; mean difference in Brief Pain Inventory interference scale, 0.1; 95% CI, −0.5 to 0.7). A 2017 systematic review[40] of pharmacological therapies for low back pain found that treatment with opioids was associated with small improvements in pain and function with no meaningful difference between NSAIDs and opioids.

Physical therapy can be effective for patients with lumbar spinal stenosis. In a clinical trial[24] of 86 participants with lumbar spinal stenosis randomized to physical therapy or home exercises, physical therapy improved the Zurich Claudication Questionnaire symptom severity scale by the MCID in 62.8% of patients at 6 weeks, compared with 32.6% in patients randomized to home exercise. In a clinical trial[25] of 104 patients with lumbar spinal stenosis, a 6-week comprehensive training program, consisting of patient education, prescribed exercises, and manual therapy, improved performance on the self-paced walk test by at least the MCID among 82% of patients, compared with 63% of those randomized to a 6-week self-directed training program.

Figure 3. Magnetic Resonance Imaging Showing Central and Foraminal Lumbar Spinal Stenosis at L4-5 in a 67-Year-Old Man



The narrowed canal is denoted with a dashed circle, and the facet joints are denoted with yellow arrows.

Multiple clinical trials have studied manual therapy for spinal stenosis, including lumbar distraction mobilization, hip and sacroiliac joint mobilization, manual stretching, and muscle strengthening. In a clinical trial[33] of 58 participants with lumbar spinal stenosis, 79% reported being at least somewhat better following a 6-week program that included manual therapy, treadmill walking, and strengthening and stretching exercises compared with 41% of patients randomized to the flexion exercise group. The results were similar at 1 year. Schneider et al[31] randomized 259 patients with lumbar spinal stenosis to 1 of 3 treatment groups: medications with or without epidural injections, manual therapy with individualized exercise, and group exercises. Participants randomized to manual therapy combined with individual exercises had improved their Zurich Claudication Questionnaire scores significantly more at the 2-month follow-up (mean difference, 2.0; 95% CI, 0.4 to 3.6) than did those randomized to medications with or without injections. Participants randomized to group exercises had similar improvement to those receiving medications and/or epidural injections (mean difference, −0.4; 95% CI, −2.1 to 1.3). The differences between groups were negligible at 6 months.

Additional exercise treatments that have been studied for lumbar spinal stenosis include cycling, treadmill walking, and aquatic therapy. A trial[26] of 68 participants randomized to an exercise program (consisting of heat therapy, mechanical lumbar traction, and a home exercise program with flexion and neural mobilization exercises) with either cycling or treadmill walking reported that at 6 weeks the Oswestry Disability Index significantly improved in each intervention group, from 31.8 to 23 in the cycling group and from 33 to 25.9 in the treadmill walking group (mean difference, 2.1 points; 95% CI, −3.1 to 7.7). Another randomized clinical trial that compared aquatic therapy (consisting of ambulation, walking, stretching, minisquat, pelvic curl, pelvic tilt, knee to chest, double knee lift, and deep water exercise) with conventional therapy (consisting of

Table 1. Randomized Trials of Treatments for Lumbar Spinal Stenosis

Source	Treatment	Comparator	No. of participants	Primary outcome[a]	Follow-up time	Mean between-group difference (95% CI)[b]
Yaksi et al,[16] 2007	Usual care + gabapentin (started at 900 mg/d, increased to maximum dose of 2400 mg/d)	Usual care (therapeutic exercises, corset, NSAIDs)	55	Pain (VAS) (0-10, 0: best, MCID, 1.5-2.8)[17]	4 mo	2.1[c,d]
Haddadi et al,[18] 2016	Gabapentin (900 mg/d)	Placebo	90	ODI (0-100; 0, best; MCID, 10-12.8)[17,19,20]	12 wk	5.46[e,c]
	Calcitonin (200 IU delivered nasally)	Placebo	90	ODI	12 wk	16.35[c,f]
Friedly et al,[21] 2017	Corticosteroid injections[g]	Lidocaine (1-3 mL of 0.25%-1%)	400	RMDQ score (0-24, 0: best, MCID, 3-5)[22]	3 mo	−0.1 (−1.3 to 1.0)
				Leg pain intensity (0-10; 0, best)	3 mo	−0.1 (−0.7 to 0.5)
				RMDQ score	12 mo	0.4 (−0.9 to 1.6)
				Leg pain intensity	12 mo	−0.1 (−0.7 to 0.5)
Manchikanti et al,[23] 2012	Corticosteroid injections[h]	Lidocaine (0.5%)	100	≥50% Reduction in NRS (0-10) or ODI (0-50) scores	2 y	6% (51% improved in lidocaine group, 57% in steroid group)[e]
Minetama et al,[24] 2019	Supervised physical therapy 2/wk for 6 wk (manual therapy, stretching, exercises, cycling, treadmill walking)	Home exercises	86	Percentage achieving MCID (0.36[19]) on the ZCQ symptom severity (1-5)	6 wk	30.2% (9.1% to 48.6%); 62.8% in PT group and 32.6% in home exercise group[f]
Ammendolia et al,[25] 2018	6-wk Comprehensive training program (2 sessions/wk), for 6 wk with single booster session 4 wk later	6-wk Self-directed training program	104	Proportion of participants achieving at least 30% (MCID) improvement in the SPWT at 6 mo	8 wk	24% (6% to 40%); 84% in comprehensive, 60% in self-directed[f]
	Intervention included education, set of 18 exercises, manual therapy				3 mo	21% (4% to 38%); 88% in comprehensive, 67% in self-directed[f]
					6 mo	19% (2% to 35%); 82% in comprehensive, 63% in self-directed[f]
					12 mo	22% (4% to 39%); 81% in comprehensive, 59% in self-directed[f]
Pua et al,[26] 2007	Cycling (2/wk for 6 wk)	Treadmill with body weight support (2/wk for 6 wk)	68	ODI	3 wk	3.2 (−1.7 to 8.2)
					6 wk	2.1 (−3.1 to 7.7)
Hammerich et al,[27] 2019	1-3 Epidural steroid injections and education (10-wk intervention period)	1-3 Epidural injections, education, and 8-10 sessions of multimodal PT (10-wk intervention period)	54	ODI	10 wk	1.08 (−5.94 to 8.10)
					6 mo	4.70 (−2.32 to 11.72)
					1 y	2.72 (−4.30 to 9.74)
Homayouni et al,[28] 2015	Aquatic therapy (every other day for 24 sessions)	Conventional PT (10 sessions and home exercises for subsequent wk)	50	Pain (VAS)	3 mo	1.52
				6-min walk test (MCID, 50-105.9 m)[29,30]	3 mo	24.9 m
Schneider et al,[31] 2019	Group exercise (classes supervised by fitness instructors in senior community centers)	Medical care (medications and/or epidural injections)	259	ZCQ (12-55, 12; best, MCID, 4.2-5.3[32])	2 mo	−0.4 (−2.1 to 1.3)
	Manual therapy/individual exercise (spinal mobilization, stretches, strength training)	Medical care (medications and/or epidural injections)	259	ZCQ	2 mo	2.0 (0.4 to 3.6)[f]
	Group exercise (supervised by fitness instructors)	Medical care (medications and/or epidural injections)	259	ZCQ	6 mo	0.5 (−1.3 to 2.3)
	Manual therapy or individual exercise (spinal mobilization, stretches, strength training)	Medical care (medications and/or epidural injections)	259	ZCQ	6 mo	1.1 (−0.6 to 2.8)

(continued)

Table 1. Randomized Trials of Treatments for Lumbar Spinal Stenosis (continued)

Source	Treatment	Comparator	No. of participants	Primary outcome[a]	Follow-up time	Mean between-group difference (95% CI)[b]
Whitman et al,[33] 2006	Manual PT, exercise, and walking (2/wk for 6 wk)	Flexion exercise, walking, and subtherapeutic ultrasound (2/wk for 6 wk)	58	Percentage reporting "somewhat better" or greater improvement	6 wk	79% in treatment group met threshold, 41% in comparators (difference, 38%)
				Percentage reporting "somewhat better" or greater improvement[i]	1 y	62% in treatment group met threshold, 41% in comparators (difference, 21%)
Delitto et al,[34] 2015	Decompression surgery	PT (2/wk for 6 wk)	169	SF-36 physical function score (0-100; 100, best; MCID, 3.3[35])	2 y	0.9 (−7.9 to 9.6)
Weinstein et al,[11] 2008	Decompression[j]	Usual nonoperative treatment (PT, education with home exercise, NSAIDs if tolerated)	289	SF-36 bodily pain (0-100, 100: best, MCID, 7.8[35])	2 y	7.8 (1.5 to 14.1)
				SF-36 physical function	2 y	0.1 (−6.4 to 6.5)
				ODI	2 y	−3.5 (−8.7 to 1.7)
Malmivaara et al,[36] 2007	Decompression[k]	Nonoperative treatment (assessment by physiatrist, 1-3 PT, NSAIDs if tolerated)	94	ODI	2 y	7.8 (0.8 to 14.9)
Försth et al,[37] 2016	Decompression + fusion	Decompression alone	247	ODI	2 y	−2 (−7 to 3)
Ghogawala et al,[38] 2016	Decompression + fusion	Decompression alone	66	SF-36 physical-component summary score (0-100; 100, best; MCID, 7.8[20,35])	2 y	5.7 (0.1 to 11.3)
Austevoll,[39] et al 2021	Decompression + fusion	Decompression alone	267	Percentage with reduction in ODI ≥30% (noninferiority margin, 15%)	2 y	−1.4% (−12.2% to 9.4%)[l]

Abbreviations: MCID, minimal clinically important difference; NRS, numeric rating scale; NSAIDS, nonsteroidal anti-inflammatory drugs; ODI, Oswestry Disability Index; PT, physical therapy; RMDQ, Roland-Morris Disability Questionnaire; SF-36, 36-Item Short Form Health Survey; SPWT, self-paced walk test; VAS, visual analog scale; ZCQ, the Zurich Claudication Questionnaire.

[a] Positive mean between group difference values indicate greater improvement in the treatment group compared to control group.

[b] For detailed information about each primary outcome measure, see eTable 1 in the Supplement.

[c] The authors did not provide between-group differences, so this value is calculated using within-group differences.

[d] $P < .05$ for comparison between groups.

[e] Comparison between groups is nonsignificant.

[f] $P < .01$ for comparison between groups.

[g] One to 3 mL of 0.25% to 1% lidocaine followed by 1 to 3 mL triamcinolone (60-120 mg), betamethasone (6-12 mg), dexamethasone (8-10 mg), or methylprednisolone (60-120 mg).

[h] Lidocaine of 0.5%; nonparticulate betamethasone, 6 mg.

[i] Measured on the Global Rating of Change scale, which has range −7 to 7 with −7 meaning "a very great deal worse" and 7 "a very great deal better"; 3 refers to "somewhat better" and defined improvement.

[j] Surgeons were given the option to perform fusion and did so in 6% of cases.

[k] Surgeons were given the option to perform fusion and did so in 20% of cases.

[l] Difference between groups in percent of patients achieving a reduction in ODI score of 30% or greater.

ultrasound, hot pack and transelectrical nerve stimulation, trunk muscle endurance, and stretching exercises), reported that each therapy significantly improved pain at 3 months, measured by an improvement on a visual analog scale of 2.57 for aquatic therapy and 1.05 points for conventional therapy (MCID, 1.5-2.8),[17] but the 2 therapies did not significantly differ in the extent of improvement in the visual analog scale (difference, 1.52 on 0-10 scale) or in 6-minute walk time (difference, 24.9 m; MCID, 50-105.9 m).[28,29] In a randomized clinical trial,[27] in which all 54 participants received 1 to 3 epidural steroid injections, 31 were randomized to education only and 23 to education and 8 to 10 sessions of physical therapy over a 10-week period. No significant differences were found in the Oswestry Disability Index (range, 0-100) between the groups at 10 weeks (1.08; 95% CI, −5.94 to 8.10), at 6 months (4.70; 95% CI, −2.32 to 11.72), or at 1 year (2.72; 95% CI, −4.30 to 9.74).

Overall, these randomized clinical trials typically demonstrated benefits of structured, supervised exercise programs and manual therapy for improving pain and functional status in persons with lumbar spinal stenosis.

Randomized clinical trials that assessed the efficacy of epidural steroid injections in lumbar spinal stenosis (Table 1) showed that injections of a local anesthetic were associated with marked pain relief for up to 2 years. The sustained improvement in those receiving a short-acting anesthetic suggests a strong placebo effect and underscores the need for controlled trials.[23]

A high-quality randomized trial[21,51] of 400 participants with lumbar spinal stenosis showed no clinically meaningful or statistically significant differences between epidural steroid injections and local anesthetic injections for the primary outcome of Roland-Morris Disability Questionnaire scores (range, 0-24) at 6 weeks' follow-up (point difference, 1.0; 95% CI, −0.1 to 2.1). At the 3-week follow-up, those receiving epidural steroid injections had a 1.8-point greater mean improvement in the Roland-Morris Disability Questionnaire score (95% CI, 0.9 to 2.8) than those randomized to anesthetic

Table 2. Summary Table of Treatments for Lumbar Spinal Stenosis

Therapy	Efficacy (vs control)	Common adverse effects	Comments
Exercise, PT			
Supervised PT	63% Improve with PT vs 33% with home exercise at 6 wk[24] 82% improve with comprehensive training program vs 63% in self-directed program at 6 mo[25]	Generally safe	
Manual therapy	62% Improved with manual therapy, walking vs 41% with flexion exercises, walking[33]	Generally safe	Manual therapy especially effective in LSS[31,33]
Medications			
Acetaminophen (paracetamol)	No studies in LSS; generally ineffective in LBP[40,41]	Cardiovascular events (0.005 cases per person-year if frequency of use exceeds 22 d/mo; 0.003 cases per person-year for those who do not take acetaminophen[42]), kidney, hepatic risks with chronic use (1%-10%[43]), especially if underlying hepatic, kidney problems[44]	Little harm but minimal efficacy
NSAIDs	No studies in LSS; in LBP, 0.7 points on 0-10 VAS[45]	Symptomatic ulcers, GI tract bleeding, ulcer perforation, hypertension, fluid retention, kidney dysfunction occur (1%-5% of patients,[43,46-48] widely recognized though a Cochrane review of LBP showed no increased risk of NSAID vs placebo[45]	NSAIDs are effective in LBP and osteoarthritis but not studied in a definitive randomized trial of LSS; low risk of adverse effects in trials likely due to selection criteria and short duration LSS population older with higher risk of kidney, cardiac, and GI toxic effects
Gabapentin	Improved pain, 2.1 points on 0-10 VAS[16]	Dizziness, drowsiness in ≈ 40% vs 20% with placebo[49]	Efficacy based on small study
Duloxetine	No studies in LSS; in chronic LBP 61% sustained pain response vs 46% in placebo[50]	In LBP trial duloxetine had greater frequency of several adverse effects than placebo (somnolence, 19.2% vs 7.1%; constipation, 100.7% vs 2.2%; nausea, 9% vs 2.7%; dry mouth, 6% vs 0%)[50]	Modestly effective in LBP though adverse effects common
Epidural corticosteroid injections	No meaningful benefit vs lidocaine injection at 3 mo or 12 mo (0.1 point on leg pain VAS, 0-10); slight benefit at 3 weeks[21,51]	Severe infection (0.01%-0.1%), epidural hematoma (<1 in 150 000), permanent neurologic injury (rare; reported in case reports)[52]	May be useful in short-term (3 wk) but no sustained benefit ≥6 wk
Surgery			
Decompressive surgery	2 Studies showed 3.5- to 7.8-point greater improvement on ODI (0-100) than usual care[11,36]; 1 Study with rigorous PT group showed 0.9-point greater improvement on SF-36 physical function (0-100)[34]	At 30-d follow-up, mortality was 0.3% for decompression and 0.6% for decompression with fusion; readmission was 6.6% for decompression and 9.4% for decompression with fusion; and wound complications was 1.8% for decompression and 3.3% for decompression with fusion[53]	In a randomized clinical trial with rigorous PT group, surgery less efficacious[34]
Decompressive surgery with lumbar fusion	2 of 3 Studies of fusion vs decompression without fusion showed no clinically important difference[37-39]		Fusion is indicated for patients with spondylolisthesis or scoliosis; in cases with these deformities, indications remain unclear

Abbreviation: GI, gastrointestinal; LBP, low back pain; LSS, lumbar spinal stenosis; NSAID, nonsteroidal anti-inflammatory drug; ODI, Oswestry Disability Index; PT, physical therapy; SF-36; 36-Item Health Survey; VAS, visual analog scale.

injections alone, but these results should be interpreted with caution because the primary outcome for the trial was negative. In summary, epidural steroid injections may offer modest short-term pain relief but do not appear to last more than 3 weeks.

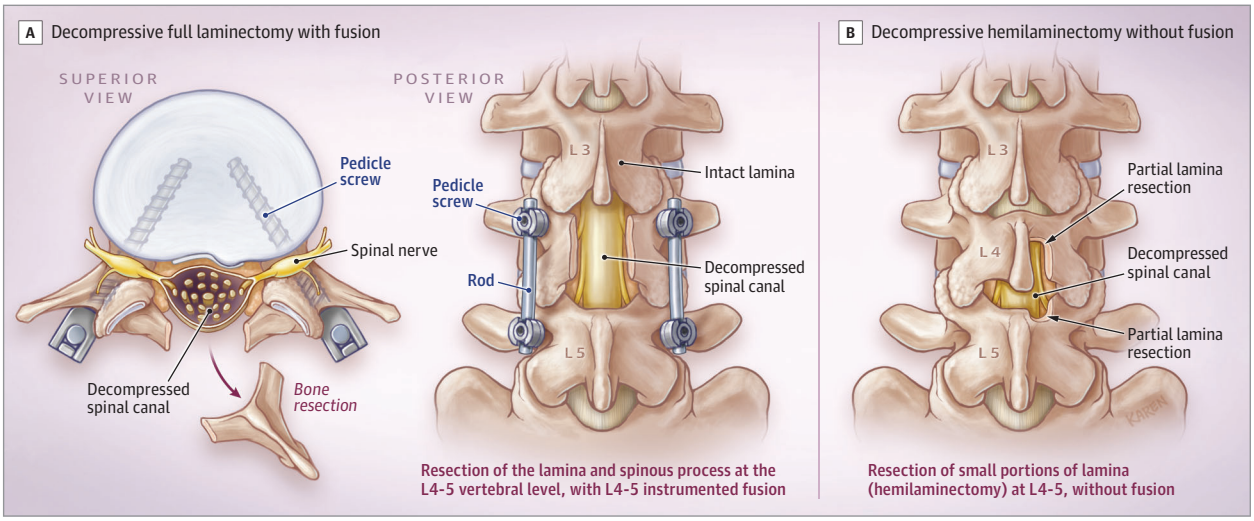
Epidural injections are associated with small risks of important adverse events.[57] Severe infection (eg, epidural abscess, discitis, osteomyelitis, meningitis) may occur in 0.01% to 0.1% of spinal injections,[52] and epidural hematomas in fewer than 1 in 150 000. Permanent neurological injury, such as foot drop is rare, reported in case reports.[52] Up to 10% of persons who receive epidural steroid injections may have suppression of morning cortisol levels,[51] though there is a paucity of controlled data on the risks of hyperglycemia, weight gain, hypertension, or facial swelling.

A relatively small proportion of patients who have persistent symptoms and functional limitations despite nonoperative treatment are referred for spinal decompression surgery. Direct surgical decompression, in which bone and/or disk are moved away from the affected nerve root(s), can be performed through an open or minimally invasive approach for lumbar spinal stenosis[37,58] (**Figure 4**A).

Three randomized clinical trials have compared nonoperative regimens with decompressive surgery (Table 1). The results are mixed but suggest a benefit of decompressive surgery compared with nonoperative care. Malmivaara et al[36] randomized 94 participants with lumbar spinal stenosis severe enough to merit surgical intervention to either decompression or nonsurgical treatment (physical therapy, education, home exercises, and NSAIDs if tolerated). Compared with nonoperative therapy, surgery improved the Oswestry Disability Index score by 7.8 points on a scale of 0 to 100 (95% CI, 0.8-14.9) and back pain by 2.1 points on a scale of 0 to 10 (95% CI, 1.0-3.3) at 2-year follow-up (MCID, 11-12.8 points).

The SPORT Trial[11,59] randomized 289 participants with lumbar spinal stenosis judged to be surgical candidates either to

Figure 4. Lumbar Decompression and Fusion for Lumbar Spondylolisthesis



decompression or to a nonoperative regimen consisting of physiatrist assessment, 1 to 3 visits with a physical therapist, and NSAIDs, if tolerated. In the intention-to-treat analysis, surgery improved the mean 36-Item Health Survey Short Form (SF-36) bodily pain score (range, 0-100) by 7.8 points (95% CI, 1.4-14.1) at the 2-year follow-up, even though 40% of participants randomized to medical therapy received surgery and 33% of those assigned to the surgical group did not receive surgery. However, in another study[34] of 169 participants with lumbar spinal stenosis randomized either to operative decompression or to a standardized physical therapy program, surgery did not improve physical function, disability, or pain compared with physical therapy (adjusted mean difference in SF-36 score, 0.90; 95% CI, −7.9 to 9.6) at the 2-year follow-up. These trials of decompression have important limitations including the substantial crossover in SPORT. In addition, heterogeneity in nonoperative regimens makes it more difficult to reach uniform conclusions about the benefits of operative therapy.

In patients with concomitant degenerative spondylolisthesis and/or scoliosis, decompression for lumbar spinal stenosis is often performed in combination with lumbar arthrodesis (fusion), in which adjacent vertebrae are fused to prevent motion (Figure 4B). The rationale for fusion is that pain may arise in part from spine instability, defined as abnormal movement or load transfer in the spine, which may worsen following decompression unless the construct is fused. Fusion is generally accomplished with autologous bone graft (eg, from the iliac crest) and often supplemented with instrumentation, in which screws, rods, and or interbody cages are inserted using anterior, anterolateral, lateral, posterolateral, or posterior approaches. Biologic products such as bone morphogenetic protein are used by some surgeons to help promote bony healing.

The precise indications for fusion in the setting of concomitant spondylolisthesis are debated, given the greater risk of complications and higher cost of fusion, balanced with the potential for improved outcomes with fusion. In a randomized clinical trial of 66 participants with lumbar spinal stenosis and spondylolisthesis, Ghogawala et al[38] reported that fusion significantly improved the SF-36 physical component summary score (0-100 scale, 100 best) compared with decompression alone (mean difference, 5.7; 95% CI, 0.1-11.3) at the 2-year follow-up. There was no significant effect of fusion on improvement in the Oswestry Disability Index score (0-100 scale, 100 best) (8.5 points; 95% CI, 17.5-0.5). In a noninferiority clinical trial[39] of 267 persons with lumbar spinal stenosis and concomitant spondylolisthesis, decompression alone was noninferior to decompression plus fusion for the primary outcome of a 30% reduction in the Oswestry Disability Index score (noninferiority margin of a 15% difference in attaining the primary outcome). In this trial, 71.4% of participants randomized to decompression alone and 72.9% randomized to decompression plus fusion achieved the outcome (between-group difference, 1.4%; 95% CI, 12.2%-9.4%). These results indicated that decompression alone was noninferior to decompression plus fusion.

Fusion is associated with adverse events. In a Medicare claims study,[53] the 30-day all-cause rehospitalization rate was 6.6% for decompression alone compared with 9.4% for decompression combined with fusion. The 30-day mortality rate for decompression alone was 0.3% compared with 0.6% for decompression with fusion, and the 30-day rate of wound complications for decompression alone was 1.8% compared with 3.3% for decompression with fusion. Three trials[37-39] comparing decompression alone to decompression plus fusion for lumbar spinal stenosis reported that fusion lengthens the time of surgery, from an average of 80 to 124.4 minutes to 150 to 289.6 minutes. In these 3 studies, fusion also increased blood loss from 83.4 to 288 mL to 429 to 648 mL.

Given the inconsistent clinical trial findings and substantial adverse effects and costs, the decision to perform instrumented fusion in a patient with spondylolisthesis and spinal stenosis should involve shared decision-making. Patients must balance the greater risk of complications and longer period of rehabilitation associated with fusion, with the likelihood that fusion may improve outcomes, especially in those with instability demonstrated on flexion and extension films.

Although lumbar decompression is the most frequently performed surgical treatment for spinal stenosis, other surgical options include percutaneous decompression (through the minimally invasive lumbar decompression procedure) or indirect decompression through an interspinous spacer device inserted

between a vertebrae. A review and meta-analysis[60] showed that interspinous devices were associated with improved symptoms for patients with mild to moderate stenosis but had higher rates of reoperation than open decompression. Reoperations ranged from 0% to 17% over the 2-year follow-up for decompression and from 17% to 33% for interspinous devices (odds ratio, 3.96; 95% CI, 1.88-8.35). These approaches require further investigation.

Guidelines

The North American Spine Society (NASS) guidelines[61] state that there is insufficient evidence to recommend either for or against pharmacological treatments with intramuscular calcitonin, intranasal salmon calcitonin, methylcobalamin, intravenous lipoprostaglandin, prostaglandin, and gabapentin. The Danish Health Authority[62] recommends against NSAIDs, paracetamol (acetaminophen), muscle relaxants, and opioids. The Lumbar Spinal Stenosis Consensus Group[63] cites low-quality evidence for the use of NSAIDs and opioids to treat symptoms from lumbar spinal stenosis.

NASS[61] indicates that evidence is insufficient to recommend physical therapy for spinal stenosis, whereas the Danish Health Authority[62] makes a weak recommendation in favor of physical therapy and the World Federation of Neurosurgical Societies (WFNS) spine committee[64] reached a strong positive consensus in support of the statement: "In nonsevere clinical conditions, a conservative approach based on at least 3 weeks of therapeutic exercise may be the first therapeutic choice." NASS[61] cites fair evidence recommending for epidural injections in the short-term and poor-quality evidence recommending for epidural steroid injections in the longer-term. The WFNS spine committee[64] similarly reports short-term to intermediate-term benefits of epidural injections but states that the inclusion of steroids does not seem to confer a benefit compared with local anesthetic alone. The Danish Health Authority[62] indicates a weak and conditional recommendation in favor of decompressive surgery, whereas NASS[61] cites fair evidence recommending the use of decompressive surgery. The WFNS spine committee[64,65] agreed on the effectiveness of surgical decompression for patients with moderate to severe symptoms. Additionally, the Danish Health Authority,[62] NASS,[61] and WFNS spine committee[64,66] recommend consideration of fusion surgery for patients with spine instability and spinal stenosis (**Box 2**).

Prognosis

Current understanding of the prognosis and natural history of lumbar spinal stenosis is based on studies that followed up patients with lumbar spinal stenosis who did not undergo an operation. One study[67] of 146 patients with lumbar spinal stenosis (mean age, 68 years, 42% women) who did not undergo an operation and were followed up for 3 years reported that approximately one-third of participants indicated improvement; approximately 50% reported no change in symptoms; and approximately 10% to 20% of patients said that their back pain, leg pain, and walking were worse. Abrupt worsening in muscle strength is uncommon,[68] and there has been limited research on the extent to which motor or sensory deficits improve with surgical decompression. These observations suggest that, except for patients with cauda equina syndrome, treatment decisions should be guided by pain and interference with daily activities rather than by concern for rapid neurological deterioration. However, patients with imaging studies that show severe stenosis should be counseled on the nature of cauda equina symptoms and urgency of seeking care if these symptoms occur.

Box 2. Questions Commonly Asked by Primary Care Physicians

Question 1
Should a patient with symptoms of back pain radiating to the thighs, tingling in the feet, and reduced light touch sensation, who is suspected of having spinal stenosis, undergo an MRI or be referred to surgery?

Answer
First-line therapy can be prescribed without imaging tests or referral and consists of education, nonsteroidal anti-inflammatory drugs (NSAIDs), if tolerated, and exercise supervised by a physical therapist. It would be reasonable to obtain a plain radiograph and to refer the patient to a specialist experienced in seeing spinal stenosis if they do not improve.

Question 2
In a patient with spinal stenosis, are sensory loss in the dorsal feet and ankle dorsiflexion weakness indications for immediate surgery?

Answer
Neurological deficits tend to progress slowly and are not indications for prompt surgery unless they progress quickly or include cauda equina syndrome (bowel or bladder incontinence, saddle anesthesia).

Question 3
Are epidural injections typically recommended for patients with spinal stenosis?

Answer
Epidural injections may help for a few weeks, but relief is unlikely to last 6 weeks or more. If they have a short-term goal (eg, a wedding to attend), the injection may be reasonable; otherwise, it may not be worth the risk of infection and nerve injury.

Question 4
When is fusion appropriate for patients undergoing laminectomy?

Answer
Fusion may be indicated for patients with concomitant spondylolisthesis who have instability on flexion and extension views on radiography.

Limitations

This review has several limitations. Our search may have missed some relevant references. Second, a formal quality assessment of included studies was not performed. Third, papers written in languages other than English were not included.

Conclusions

Lumbar spinal stenosis affects approximately 103 million people worldwide and 11% of older adults in the US. First-line therapy is activity modification, analgesia, and physical therapy. Long-term benefits from epidural steroid injections have not been established. Selected patients with continued pain and activity limitation may be candidates for decompressive surgery.

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Submissions: We encourage authors to submit papers for consideration as a Review. Please contact Mary McGrae McDermott, MD, at mdm608@northwestern.edu.

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