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Algorithmic approach to the management of the patient with lumbar spinal stenosis

Terence P. Doorly, MD
Associate Chief of Neurosurgery
The North Shore Medical Center
Salem, Massachusetts
Medical Director
Neurosurgery and Spine
The Musculoskeletal Center
Peabody, Massachusetts

Cheryl L. Lambing, MD
Assistant Clinical Professor
Department of Family Medicine
University of California, Los Angeles
Ventura County Medical Center
Family Medicine Residency
Director, Family Care Center
Director, Osteoporosis Center, Ventura County
Ventura, California

Gerard A. Malanga, MD
Clinical Professor
UMDNJ–New Jersey Medical School
Newark, New Jersey
Director, Pain Management
Overlook Pain Center
Summit, New Jersey

Philip M. Maurer, MD
Director, Interventional Spine Program
Booth Bartolozzi Balderston Orthopaedics/
PENN Orthopaedics
Department of Orthopaedics
Pennsylvania Hospital
Philadelphia, Pennsylvania

Ralph F. Rashbaum, MD
Instructor
Department of Anesthesiology & Pain Therapy
Texas Tech University Health Science Center
Co-founder, Texas Back Institute
Lubbock, Texas

CASE STUDY. A 71-year-old generally healthy woman presents for her first visit in 3 years. She ambulates slowly from the waiting room, with a more stooped posture than previously. She reports a 2-year history of slowly worsening buttock and leg pain when she walks any distance. She has noticed that her symptoms are much less when she leans on a shopping cart in the grocery store. Her buttock/leg pain resolves within a few minutes when she sits down. The patient exhibits signs and symptoms suggestive of lumbar spinal stenosis (LSS).

Natural history of lumbar spinal stenosis

Lumbar spinal stenosis (LSS) is described as a clinical syndrome of buttock or lower extremity pain, which may occur with or without back pain that is associated with diminished space available for the neural and vascular elements in the lumbar spine.[1] There are several categories of LSS, of which degenerative LSS is the most common and the focus of this article.

The normal process of aging leads to degenerative changes in the lumbar spine. These changes include the formation of osteophytes (bone spurs), hypertrophy of the facet joints and ligamentum flavum, bulging of the intervertebral discs, and deformities such as spondylolisthesis and scoliosis.

The result is gradual narrowing (stenosis) of the central spinal canal, the area under the facet joints (subarticular recess), and the neural foramina. Significant stenosis produces compression of the underlying neural and vascular elements that results in the typical painful symptoms (**FIGURE 1**).

Lumbar spinal stenosis is a slowly progressive disorder that typically does not present until age 50 or older. However, not all patients with LSS develop significant or disabling symptoms. In one report of 50 patients with mild disease, nearly half of the patients had either no pain or mild pain (on a 10-point visual analog scale) 10 years after diagnosis.[2] Approximately 30% to 50% of patients with clinically mild or moder-

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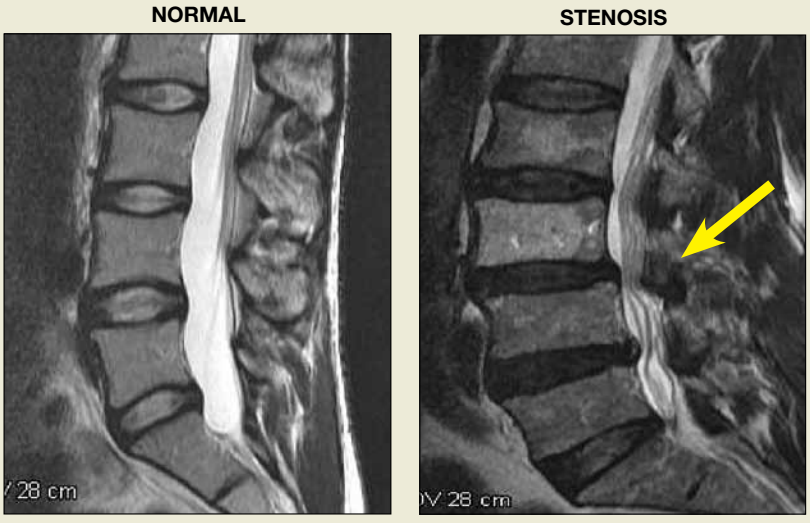
FIGURE 1 **MRI of lumbar spinal stenosis**



Sagittal T2 MRI showing normal lumbar lordosis with spacious spinal canal

Sagittal L3-4 T2 MRI showing degenerative changes, including disc bulging, loss of disc height, facet and ligament hypertrophy, and grade 1 spondylolisthesis at L3-4, producing spinal stenosis.

Images reprinted with permission from Terence P. Doorly, MD

ate degenerative LSS have a favorable natural history and rarely suffer rapid or catastrophic neurologic deterioration.[1] However, functional limitations that typically occur with moderate or severe LSS can be life-altering.

Assessment and differential diagnosis

A focus of the assessment is to rule out other causes of buttock/leg pain and to exclude any significant "red flag" pathologies. The patient history is usually notable for leg and/or buttock pain (neurogenic claudication) that is gradual in onset and is sometimes accompanied by low back pain (**FIGURE 2, BOX 2**). LSS typically progresses slowly; rapid progression suggests another etiology. As with the patient described earlier, symptoms are provoked by standing upright and walking, and relieved with flexion (eg, leaning forward on a shopping cart or sitting).[1] Radicular pain symptoms can be unilateral or bilateral and range from dull and aching to dysesthetic or sharp.

Assessing the impact of symptoms on function and daily activity is important, as this guides treatment planning. The functional assessment may be facilitated by asking patients to write down those activities they want or need to do and still can do on 1 sheet of paper, and those activities they want or need to do but can't do on another sheet of paper ("can do/can't do assessment"). The impact of other conditions such as arthritis, peripheral vascular disease, diabetes, peripheral neuropathy, and motor neuron disease should be investigated. In addition, the use of medications that can cause myopathy, such as statins, cimetidine, or cyclosporine, should be explored.

There are no universally accepted findings on physical examination, although a stooped forward posture is common (**FIGURE 2, BOX 2**). Generally, the range of motion is forward flexion without pain, but restricted, often with pain, in extension. The patient typically has normal strength and normal sensory examination results, but often has decreased or absent ankle jerk reflexes bilaterally. There is usually no tenderness over the spine on palpation. Physical findings that are most strongly linked to LSS include a wide-based gait, thigh pain that worsens with 30 seconds of lumbar extension, progressive leg weakness with continued walking, and neuromuscular deficits.[1]

Signs or symptoms suggestive of red flag pathology include cauda equina syndrome (lower extremity pain, weakness, and numbness that may involve the perineum and buttocks, associated with bladder and bowel dysfunction), fever, nocturnal pain, steroid use, gait disturbance, structural deformity, unexplained weight loss, previous carcinoma, severe pain upon lying down, recent trauma with suspicious fracture, or the presence of severe or progressive neurologic deficit. If any of these conditions is present, further diagnostic work-up in a timely fashion is indicated.[3]

It is particularly important to differentiate the neurogenic claudication of LSS from vascular claudication, as the treatments are vastly different (**TABLE**).[3] Since vascular claudication results from an impaired blood supply caused by atherosclerosis, measuring the ankle brachial index is helpful to distinguish between the 2 types of claudication. In addition, patients with neurogenic claudication tolerate the bicycle test well, whereas patients with vascular claudication become symptomatic as tissue hypoxia occurs. Similarly, limited evidence suggests that the 2-stage treadmill test, which capitalizes on the postural dependency of stenotic symptoms, also may be useful to differentiate patients with neurogenic vs vascular claudication.[4] Findings significantly associated with neurogenic claudication include an earlier onset of symptoms with level walking (P=.0009), increased total walking time on an inclined treadmill (P=.014), and prolonged recovery time after level walking (P=.001).[4]

Investigation

The history and physical examination are usually sufficient to make a presumptive diagnosis of LSS. It is important to realize, however, that if stenosis is identified on radiologic studies but is not correlated with symptoms, it is of little clinical significance. There is, in fact, no clear relationship between symptoms and the degree of stenosis.[5] Indeed, investigation suggests that approximately one-third to two-thirds of asymptomatic adults have a substantial spinal abnormality as shown by magnetic resonance imaging (MRI).[6,7] Imaging is not indicated for LSS unless there are moderate functional loss, neurologic deficit, or red flags suggestive of other causes of spinal disease (**FIGURE 2**). An MRI should be ordered prior to referral if clinical suspicion is high and the symptoms are increasing, but not suggestive of vascular claudication or peripheral neuropathy.

MRI and computed tomography (CT) are equally capable of confirming the diagnosis of LSS. MRI provides superior soft tissue contrast with excellent visualization of soft tissue pathology and neural elements; by comparison, CT is more sensitive for calcified structures and provides better visualization of both structural integrity and bridging bone. MRI has the advantage of being a nonionizing technique.[1] In 2007, after an evidence-based review, the North American Spine Society recommended MRI as the most appropriate, noninvasive test for evaluating degenerative LSS.[1] Exceptions, and situations in which CT may be used (possibly with myelography), include patients who are claustrophobic or those who have metallic implants, for whom MRI findings are inconclusive; or cases in which a poor correlation exists between symptoms and MRI findings.[1] MRI or CT is usually reserved for selected patients being considered for surgery after medical/interventional management has failed.

Treatment

The generally slow, progressive nature of LSS cannot be stopped, but it can be managed. With this in mind, the goals of treatment are to relieve the patient's pain and to improve functioning. Shared decision making with the patient is therefore critical to determine the optimal treatment and its timing. Using the 2-page "can do/can't do" assessment described earlier, this process can be aided by having a thorough discussion with the patient regarding the extent to which LSS symptoms affect his or her functioning and activities of daily living. The impact of the disorder on mood and sleep should also be investigated and managed appropriately. If symptoms do not significantly affect function or activities of daily living, however, watchful waiting may be a reasonable treatment approach.

When watchful waiting is no longer appropriate, the treatment options for LSS can broadly be categorized as nonsurgical or surgical. The North American Spine Society has concluded that in patients with:

- mild to moderate symptoms of LSS, medical/interventional treatment is effective approximately 70% of the time at 6 months and 57% at 4 years.[2]
- moderate to severe symptoms of LSS, surgery is more effective than medical/interventional treatment.[2,8]
- severe symptoms of LSS, decompression surgery alone is effective approximately 80% of the time, and medical/interventional treatment alone is effective approximately 33% of the time.[1]

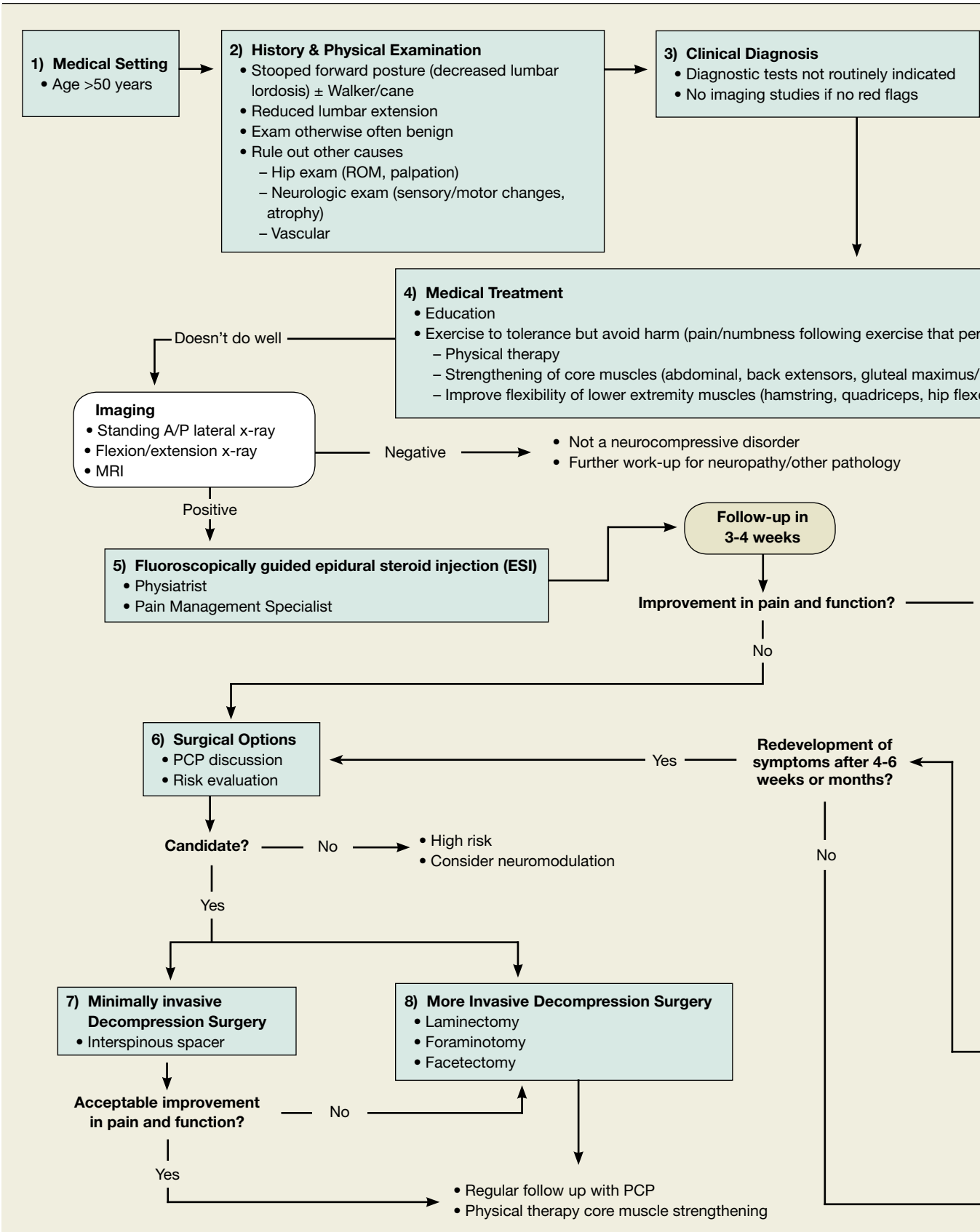
Patient Factors Affecting Outcomes—In addition, results of surgical treatment are good to excellent in 53% to 82% of patients at >4 years of follow-up.[8,9] Reoperation is necessary within 10 years in 23% of patients.[8]

In considering nonsurgical vs surgical treatment, 2 important questions arise: (1) Are there patient factors that are likely to negatively or positively affect outcomes from surgical treatment? and (2) What are the consequences of delaying surgery?

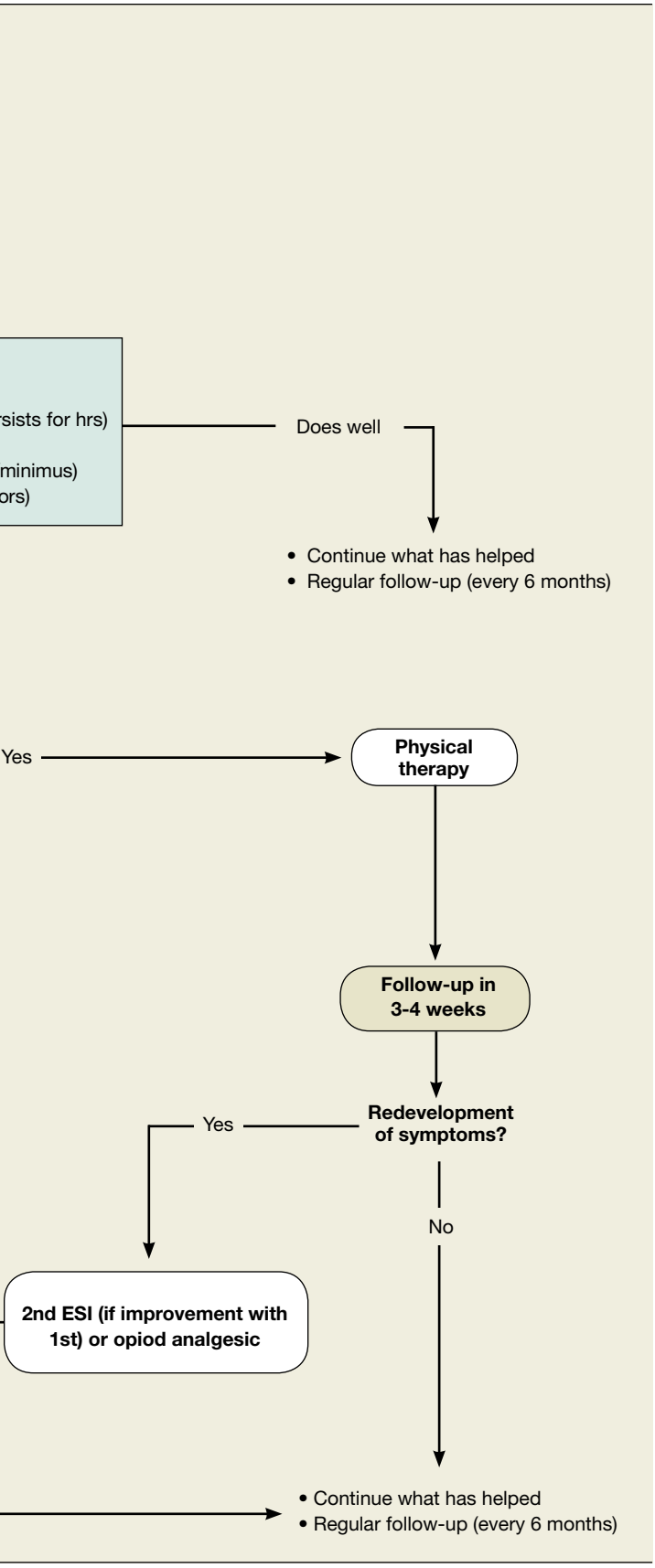
The answer to the first question comes from a systematic review of 21 trials involving patients who underwent surgical treatment of LSS.[10] Depression, cardiovascular comorbidity, a disorder influencing walking ability, and scoliosis were predictive of poorer subjective outcomes postoperatively. Conversely, better walking ability, patient-rated health, higher income, less overall comorbidity, and pronounced central stenosis were predictive of a better subjective outcome postoperatively. Furthermore, surgery for lumbar spinal stenosis in patients older than 75 can be conducted safely and with similar outcomes to those in younger patients.[11]

Delaying Surgery—With respect to the second question about the consequences of delaying surgery, some insight comes from a prospective evaluation of 100 patients with symptomatic LSS: 68 patients with generally moderate symptoms were initially managed conservatively with orthosis, and 32 patients with moderate/severe symptoms were initially managed with more invasive decompression surgery without fusion.[2] All patients participated in physical therapy in the form of ambulation and stabilizing exercises. At 6-month follow-up, 62% (42/68) of patients managed conservatively experienced a good result (defined as full to partial restitution of function with at least clear improvement), compared with 84% (27/32) of those managed surgically. Twenty of the 26 patients who did not achieve a good result in the conservative treatment group were subsequently treated surgically after 3 to 27 months (median 3.5 months). At 6-month postoperative follow-up, 90% (18/20) of these patients experienced a good result. At 4-year follow-up, 58% (11/19; 1 patient had died) of those initially managed conservatively and subsequently surgically had a good result, compared with 87% (27/31; 1 patient had

FIGURE 2 **LSS Management Algorithm**



This algorithm represents the consensus of the authors who provide care for patients with lumbar spinal stenosis across the spectrum of clinical care.



died) of those initially managed surgically (1 underwent reoperation). The authors concluded, "In principle, surgery for LSS seems to be equally beneficial whether it is given early or late (up to 3 years) after severe symptoms."[2]

Nonsurgical treatment

Nonsurgical treatment options include physical therapy, analgesic medications, epidural injections, lumbosacral braces, and lifestyle interventions (eg, weight loss) (**FIGURE 2, BOXES 4, 5**). Other treatments that have been employed are spinal manipulation, traction, and electrical stimulation.

Physical therapy—While there is limited evidence of long-term benefits with physical therapy alone,[1,12] physical therapy, including exercise, may be effective in controlling symptoms, but should be part of a comprehensive treatment plan.[1] Strengthening of core muscles (abdominal, back extensors, gluteus maximus/minimus) is important for dynamic support of the spine. Improving flexibility of lower extremity muscles (hamstring, quadriceps, hip flexors) can be helpful as well. One study of 68 patients with LSS revealed that walking on a treadmill with body weight support was comparable to cycling, when both were combined with exercise for 6 weeks, in reducing disability and pain.[13] Similarly, in a study of 52 patients with LSS, use of a wheeled walker to induce lumbosacral flexion significantly improved ambulation; 71% of patients increased their walking distance by at least 250%.[14] In addition, 71% reported excellent or good pain relief (≥50% reduction in pain on a 10-point visual analog scale) after using the wheeled walker for 3 to 5 days.[14] The use of a lumbosacral brace during the daytime also can increase walking distance to a mean of 393 m, compared with 315 m without a lumbosacral brace ($P<.05$), and can decrease pain to 4.7 on a 10-point visual analog scale, compared with a pain rating of 5.9 for those not wearing a lumbosacral brace ($P<.05$).[15] Physical therapy and use of a lumbosacral brace must be continued however, to maintain the benefits.

Medical and interventional management—The use of pharmacologic agents for the management of LSS alone has been limited in clinical trials. Instead, patients with LSS generally have been included in trials involving patients with various types of back pain.[16-18] Consequently, there is insufficient evidence to determine the role of nonsteroidal anti-inflammatory drugs (NSAIDs), adjuvant analgesics, muscle relaxants, intranasal or intramuscular calcitonin, methylcobalamin, or intravenous lipoprostaglandin E(1) in the management of LSS.[1] Despite this lack of evidence, NSAIDs and analgesics are commonly used and often provide short-term improvement in pain. Similarly, while limited data suggest a modest benefit associated with spinal manipulation,[19] there is insufficient

TABLE Features distinguishing neurogenic claudication from vascular claudication[3]

Description	Neurogenic claudication	Vascular claudication
Quality of pain	Cramping	Burning, cramping
Low back pain	Frequently present	Absent
Sensory symptoms	Frequently present	Absent
Muscle weakness	Frequently present	Absent
Reflex changes	Frequently present	Absent
Arterial pulses	Normal	Decreased or absent
Arterial bruits	Absent	Frequently present
Skin/dystrophic changes (eg cyanosis, hair loss)	Absent	Frequently present
Aggravating factors	Erect posture, ambulation, extension of spine	Any leg exercise
Relieving factors	Sitting, bending forward, squatting	Rest
Walking uphill	Symptoms produced later	Symptoms produced earlier
Walking downhill	Symptoms produced earlier	Symptoms produced later
Bicycle test	No symptoms provoked unless erect	Provokes symptoms

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evidence from controlled clinical trials to establish a benefit from traction or electrical stimulation.[1]

Gabapentin—It is worth noting that Yaksi et al[20] investigated the addition of gabapentin in a randomized trial involving 55 patients with LSS treated with therapeutic exercises, lumbosacral corset with steel bracing, and NSAIDs. The addition of gabapentin resulted in an increase in walking distance (P=.001), improved pain scores (P=.006), and recovery of sensory deficit (P=.04), compared with those who did not receive gabapentin.

Epidural injections—The use of epidural injections, primarily with a corticosteroid, for the treatment of LSS has been far more extensively studied (**FIGURE 2, BOX 5**).[21] Fluoroscopic x-ray–guided interlaminar epidural injections are preferred over nonfluoroscopically guided injections because of improved success by documenting the injection of the affected spinal level, resulting in short-term pain relief.[1]

The use of sequential radiographically guided transformational epidural steroid injections or caudal injections can produce significant, long-term relief of pain in patients with radiculopathy or neurogenic intermittent claudication from LSS.[1,22] For example, in 1 prospective cohort study, 34 patients with LSS received an average of 2.2 injections of lidocaine and triamcinolone within a 6-week period and experienced significant improvement over 12 months. Walking tolerance was improved in 59% of the patients at 6 weeks (P<.0001), 56% at 6 months (P<.0001), and 51% at 12 months (P=.0005). Similar benefits were observed with standing tolerance. Measures of pain relief, patient satisfaction, and outcomes also demonstrated significant improvement over the 12-month study period.[23] In a second study, long-term benefits were observed in 140 patients with LSS who received a mean of 2.2 triamcinolone/local anesthetic injections, with a mean follow-up period of 17 months.[24] One-third of the patients achieved pain relief lasting longer than 2 months after their injection(s), while 53% reported sustained improvement in their functional status.[24]

In summary, of patients with mild to moderate LSS who initially receive medical/interventional treatment and are followed for 2 to 10 years, approximately 20% to 40% will ultimately require surgical intervention.[1]

Surgical treatment

In addition to implementing and evaluating the patient's response to medical and interventional therapies, the primary care physician plays an important role when nonsurgical management fails and the patient develops progressive symptoms. (**FIGURE 2, BOX 6**) In this role, the primary care physician should continue to work in close collaboration with the patient to understand any change in the patient's needs and concerns, as well as goals. Revisiting the list of activities the patient can and can't do may be helpful in assessing the degree of functional impairment. A more detailed discussion of the role of surgery, the types of procedures, and the potential risks and

benefits should be undertaken. The patient should be reassured about surgery and encouraged to see a spine specialist to discuss these options further. In addition, patient education should be continued. Educational resources for patients with LSS include the:

- American Association of Neurological Surgeons (www.neurosurgerytoday.org)
- North American Spine Society (www.spine.org)
- American Academy of Orthopaedic Surgeons (www.aaos.org)

The goals of surgical treatment are 3-fold: first, to provide symptom relief by relieving nerve compression; second, to prevent or slow further structural deterioration that may lead to a more involved treatment solution; and third, to treat with the most effective and least aggressive approach. It is worth noting that patients 75 or older with LSS show similar significant improvement in activities of daily living, as well as in pain relief, in the 10 years following lumbar decompression as do patients ages 65 to 74 who undergo the procedure.[25]

Surgical options range from minimally invasive decompression surgery such as an interspinous spacer (**FIGURE 2, BOX 7**) to more conventional, invasive decompression surgery (eg, laminectomy, foraminotomy, facetectomy, or micro/laminectomy—with or without fusion) (**FIGURE 2, BOX 8**).

Conventional decompression surgery— Laminectomy has been the standard surgical treatment for LSS for many years, with an established ability to provide significant improvement in symptoms and functioning.[26,27] Treatment with decompression surgery alone (without fusion) is effective about 80% of the time in patients with severe symptoms of LSS.[1] The advantage of conventional decompressive laminectomy is that it provides good visibility and working space by removing the spinous processes, interspinous and supraspinous ligaments, in addition to large portions of the laminae and facet joints, thereby enabling direct access to the spinal canal. The benefits of laminectomy over medical/interventional management have been well documented.[28] However, concern remains about the increased risk associated with invasive surgical procedures in the elderly, particularly the length of the procedure.[11] In addition, the resection of the osteoligamentous posterior tension band may produce secondary spinal instability and mechanical back pain.[29] These concerns have resulted in a number of less-invasive procedures being described in recent years. These less-invasive procedures focus on various types of microsurgical decompression, sometimes facilitated by tubular retractors, in which the osteoligamentous tension band is largely preserved.[29]

Minimally invasive decompression: Interspinous spacer— More recently, new procedures, such as insertion of an interspinous spacer, aim to provide decompression of the spinal canal and its contents without removing any part of the osteoligamentous tension band. In addition, decompression is achieved without violating the spinal canal. The interspinous spacer, which is placed between the spinous processes of the stenotic levels, is designed to limit extension of the stenotic spinal segments. The spacer maintains the stenotic segment in a neutral or slightly flexed position when the individual is upright, thereby reducing the neural compression that produces symptoms. Other ranges of motion, flexion, lateral bending, and rotation, are preserved. Levels of the spine adjacent to the implant are unaffected. There is usually minimal or no tissue or bone resection, and the supraspinous ligament and other key structures are maintained.

The interspinous spacer has been investigated in patients with mild or moderate symptoms of LSS. Typically, these patients do not warrant a highly invasive procedure. A 2-year, randomized, controlled, multicenter, prospective clinical trial compared patients implanted with the interspinous spacer (n=100) to those treated with nonsurgical care consisting of medical/physical therapies and epidural injections (n=91).[30] At 2 years, patients implanted with the interspinous spacer experienced a 45% improvement in the symptom severity score, compared with a 7% improvement in the control group (P<.001).[30] Improvements in the mean physical function scores were 44% in the interspinous spacer group and 0% in the control group (P<.001).[30]

The proportion of patients who satisfied specific thresholds for all 3 criteria (symptom severity, physical function, and patient satisfaction) was 48% in the interspinous spacer group, compared with 5% in the control group. During the 2-year follow-up period, 6 patients in the interspinous spacer group and 24 in the control group underwent major decompression surgery for unresolved symptoms of LSS.[30] There were no device-related intraoperative complications.[30] Four-year follow-up data were observed for a subset of patients in the trial.[31] Using a 15-point improvement from baseline in the Oswestry Disability Index as the criterion for a successful surgical outcome, 14 of 18 patients (78%) had successful outcomes (mean improvement in disability index score from baseline, 29 points) at an average follow-up of 4.2 years.[31]

In another in vivo study, 24 consecutive patients with stenosis at 1 or 2 levels underwent placement of an interspinous spacer device.[32] These patients had previously received caudal epidural injections for symptomatic relief that lasted from a few weeks to a few months. Maximal improvement in symptom severity occurred at 3 months, with a mean decrease of 0.95 from a preoperative baseline of 3.37 on a 5-point scale.[32] Although symptom severity scores gradually increased over the subsequent follow-up visits, the overall improvement remained clinically significant for up to 12 months, with a decrease of 0.54 from baseline values. Changes in physical function from baseline were not clinically significant at 3, 6, or 12 months, al-

though this may have been due to symptom scores in some patients at baseline that were below the minimum number required to show significant improvement. At 1 year postoperatively, 7 patients had symptom recurrence severe enough to require caudal epidural injection treatment. Two of these patients, who had slippage of the interspinous device, remained symptomatic and underwent removal of the device followed by decompression and fusion surgery.[32]

Follow-up— Following minimally invasive or more invasive decompression surgery, the primary care physician plays an important role in the recovery process, working in close collaboration with the spine specialist and physiatrist. Since full recovery may take several months, the primary care physician should encourage the patient to "take it easy" and perform only light activities, and to follow the recovery protocol and recommendations of the spine specialist. During this period of relative inactivity, it is important that comorbidities be closely managed. Should symptoms recur months or years later, referral to a physiatrist or spine specialist may be needed for further management.

Primary care treatment plan

In summary, the primary care physician plays a critical role in the ongoing management of patients with LSS. Correctly establishing the diagnosis of LSS by differentiating neurogenic from vascular claudication, as well as other causes, is essential. This requires determining when further work-up is needed, including MRI. Selecting among the many nonsurgical options is important and often challenging, as many treatments have been poorly investigated in clinical trials. Recognition and management of depression, sleep disorders, and other possible consequences of LSS are necessary. Establishing a collaborative relationship with patients and providing patient education about the degenerative process are critical. Information about accommodation to symptoms and treatment options, along with their benefits and risks, is a core component of comprehensive management. Consultation with and/or referral to a spine specialist must occur when symptoms compromise the patient's functioning. Finally, involvement of the primary care physician in providing effective postoperative management is essential for optimal long-term outcomes. ■

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