A Pathway for the Diagnosis and Treatment of Lumbar Spinal Stenosis

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KEYWORDS

• Lumbar • Stenosis • DDD • Decompression • Laminectomy • Neurogenic claudication

KEY POINTS

- Lumbar spinal stenosis most commonly occurs due to degenerative spinal changes in the elderly population, and presentation can range from asymptomatic to severely disabled.
- Imaging findings, in conjunction with symptoms, can help guide treatment and inform surgical versus nonsurgical shared decision making.
- Recent data have shown that surgical intervention can lead to improved functional outcomes and decreased pain compared with nonsurgical management.

INTRODUCTION

Lumbar spinal stenosis (LSS) is defined as the narrowing of the lumbar spinal canal with subsequent compression of neural elements. The cause of the disease and its associated pathology is varied. Although some symptoms may be dismissed as part of the normal aging process by patients and physicians alike, LSS can be debilitating and take a significant toll on the patients and their families.[1] The exact prevalence of LSS is unknown, and furthermore, the proportion of those who are asymptomatic versus symptomatic is similarly imprecise. The Framingham study, a cross-sectional observation study, attempted to quantify this information. Of their 191 study participants, who had a mean age of 52.6 years, the prevalence of LSS was 23.6%. Not surprisingly, as age increased, the percentage of patients with LSS increased.[2] Other research estimates that LSS affects more than 200,000 patients in the United States[3] and that approximately 6 of 100 patients diagnosed with degenerative lumbar conditions will require lumbar fusion within 1 year of diagnosis.[4] In another study, the estimated prevalence of symptomatic LSS was around 10%.[5] Although the exact magnitude of LSS is uncertain, what is clear is the staggering toll LSS exacts on the health care system. A retrospective cohort analysis of Medicare claims looking at surgical intervention for LSS from 2002 to 2007 demonstrated that mean hospital charges for decompression compared with complex fusion procedures were US $23,724 and $80,888, respectively.[6]

Anatomy

To fully understand the pathology of LSS, functional knowledge of the normal anatomy of the lumbar spine and its contribution to pathology is necessary. The anterior border of the spinal canal is composed of the vertebral body, the intervertebral disk, and the posterior longitudinal ligament. The lateral border is formed by the pedicles, the lateral ligamentum flavum, and the neural foramen. The posterior border is formed by the facet joints, lamina, and ligamentum flavum (**Fig. 1**). The paired nerve roots

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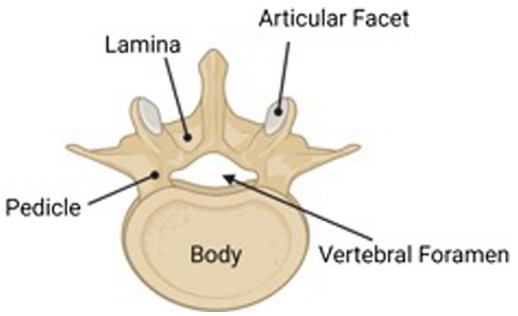


Fig. 1. Anatomy of lumbar vertebrae.

travel through the spinal canal before exiting at each respective neural foramen below the pedicle (right L4 nerve root exits below the right L4 pedicle above the L4-5 disk).

Pathophysiology

As individuals age, the intervertebral disk undergoes a process of degeneration. Within the disk, the annulus undergoes a transformation in which the level of type I collagen increases, subsequently leading to decreased hydration of the gelatinous nucleus pulposus. This decreased hydration causes the disk to desiccate, hindering its ability to handle a mechanical load. As the disk further degenerates, disk height is lost and the disk may begin to bulge and impinge on the spinal canal as well as causing the ligamentum flavum to buckle and the facet joints to settle. The facet joints, which help produce the smooth gliding motion necessary for movement in the lumbar spine, begin to see increased stress across the joint, which leads to further joint degeneration, hypertrophy, and osteophyte formation. These osteophytes can impinge on the thecal sac as well as the nerve root in the neural foramen. The intervertebral foramen, the space in which the nerve root exists, becomes tighter secondary to these degenerative changes, further compressing neural elements. This cascade, known as degenerative spondylosis, is one of the most common causes of LSS[7] (Figs. 2, 3, and 4).

The degenerative changes thus far described can be worsened by dynamic factors such as segmental instability. Instability can be in the form of translational or rotational abnormality. A translational abnormality is most often seen as an anterior slippage of one vertebral body on top of the next vertebral segment (typically L4-on-L5) resulting in substantially decreased room for the neural elements; this is known as degenerative spondylolisthesis. In scoliosis, a rotational instability is seen that leads to altered spine biomechanics and further narrowing of the central canal, lateral recess, and the intervertebral foramen, in addition to potential coronal or sagittal imbalance.

Classification

Stenosis can be anatomically classified as central, lateral recess, or foraminal based on the location of neural compression. Central stenosis is usually caused by a combination of disk bulging, hypertrophied ligamentum flavum, and facet joint overgrowth. Lateral recess stenosis is due to facet joint osteophytes as well as disk protrusion. Foraminal stenosis causes compression of the exiting nerve root and dorsal root ganglion due to loss of disk height, foraminal disk protrusion, or osteophyte formation. Finally, extraforaminal stenosis is usually due to far lateral disk herniation with resultant exiting nerve root compression.

Other acquired conditions, although less common, should be considered when attempting to determine the cause of LSS. These conditions can include space-occupying masses, postsurgical fibrosis, rheumatologic conditions such as ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis, or congenital conditions such as achondroplasia or congenital stenosis.[8]

ASSESSMENT AND EVALUATION

Clinical Presentation

The diagnosis of LSS is becoming increasingly common, which may be due in part to increased access to advanced imaging and an aging patient population. A distinguishing feature of LSS is the association of pain with postural changes. Lumbar extension decreases cross-sectional spinal canal volume, which corresponds with increased pain associated with lumbar stenosis, compared with lumbar flexion, which results in the opposite effect. These symptoms are referred to as neurogenic claudication or pseudoclaudication, which consists of pain in the buttock, groin, and thigh regions. However, patients often present with additional symptoms such as heaviness, fatigue, burning, aching, dysesthesia, and rarely weakness in the buttock, groin, and thigh regions. Symptoms are typically bilateral but can affect one extremity more than the other.[9] Pain is thus exacerbated by walking, going up stairs, or standing and relieved by sitting down or leaning forward, sometimes referred to as the "shopping cart sign." Patients should be followed subjectively based on their walking tolerance in terms of time or distance.

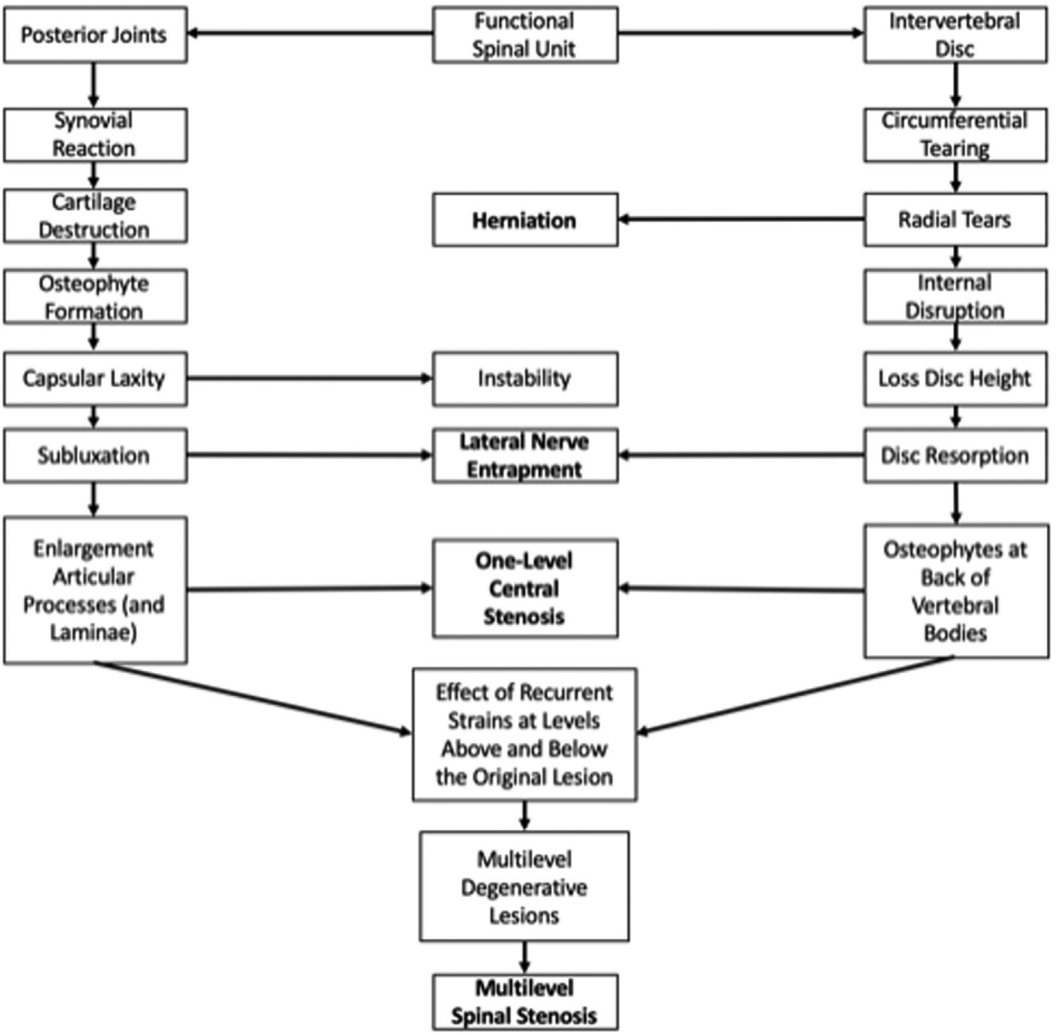


Fig. 2. The degenerative cascade.

Neurogenic claudication should not be confused with vascular claudication or other forms of nonspecific lower back pain. Vascular claudication is pain caused by peripheral arterial disease leading to insufficient blood flow to the extremity. The symptoms of vascular claudication occur more distally in the extremity, most commonly calf pain, contrary to neurogenic claudication that primarily occurs more proximal in the buttocks and thigh regions. Vascular claudication is commonly aggravated by increased movement and activity in a distance-related fashion, meaning that a patient typically can walk a certain distance until pain arises and requires rest to alleviate the pain[10]; this is in stark contrast to neurogenic claudication in that it is not the amount of activity that causes the pain but the type of activity, typically involving lumbar extension.

Patients with LSS may also present with radicular leg pain in addition to neurogenic claudication. Neurogenic claudication results from compression of the thecal sac, whereas radicular pain or radiculopathy is due to compression of a nerve root or dorsal root ganglion in the lateral recess or the neural foramen, respectively. This leg pain occurs in a dermatomal distribution. Most commonly, a patient may present with numbness and weakness in the extensor hallucis longus and tibialis anterior group due to compression of the L4 or L5 nerve root within the lateral recess.

Low back pain involvement with spinal stenosis varies, likely due to variable presentation of degenerative disk disease and facetogenic disease resulting in neural compression. Neurogenic claudication differs from other forms of nonspecific low back pain in that sitting typically

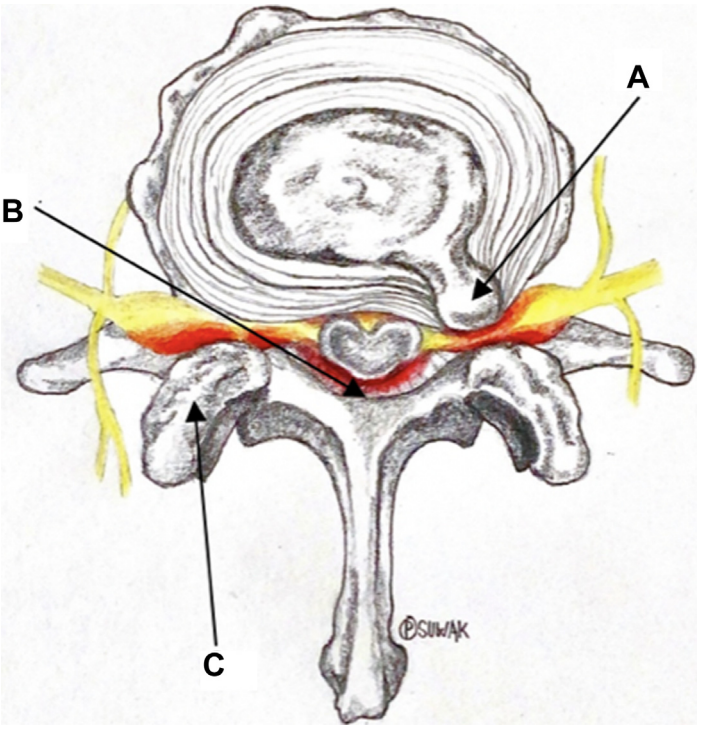


Fig. 3. Axial cross section demonstrating areas of stenosis secondary to intervertebral disk desiccation (A), thickened ligamentum flavum (B), and hypertrophied facet capsules (C).

brings relief for patients with LSS, whereas nonspecific low back pain is typically worsened from prolonged periods of sitting. However, back pain is much less of a reliable finding in terms of both diagnosis and outcomes compared with the presence of neurogenic claudication.

In addition, other common maladies plaguing older populations that can lead to back and leg pain include osteoarthritis of the spine or hip, peripheral arterial disease, and greater trochanteric pain syndrome. These conditions may lead to symptoms that mimic the pseudoclaudication of LSS and may warrant further evaluation and management. Symptoms of bowel or bladder incontinence are atypical for spinal stenosis, and a careful history must be elucidated because various causes of genitourinary dysfunction are common in older patient populations.

Physical Examination

The physical examination of patients with LSS is often equivocal. It is important to note a patient's body position during standing and ambulation because forward flexion of the trunk is how a patient with LSS will decrease their symptoms. In addition, the neurologic examination is commonly normal with patients not demonstrating any evidence of weakness or sensory deficit. Decreased deep tendon reflexes are typical in older patients, and provocative maneuvers, such as the straight leg raise test, may not elicit a clear response. Hyperreflexia in the lower extremities or proximal thigh weakness should prompt an evaluation for the remainder of the neural axis to evaluate for myelopathy. The hip is frequently confused for spinal pathology, and care should be taken to examine the hip in addition to obtaining a lower extremity vascular examination to avoid misdiagnosis of the patient's condition.

Radiology

Diagnostic testing of LSS typically starts with plain radiographs, including anteroposterior and lateral views, with consideration of flexion and extension lateral views. Because most patients with LSS are elderly, there will likely be a variety of spondylotic or degenerative changes. In fact, even severe degenerative spine changes can be seen in asymptomatic patients. In addition, particular attention should be paid to evaluate for coronal and sagittal deformity, which is best evaluated with full-length standing films. The gold standard of radiologic diagnosis of LSS remains MRI. Dural sac cross-sectional area is used to measure the degree of stenosis. An area between 76 and 100 mm indicates relative

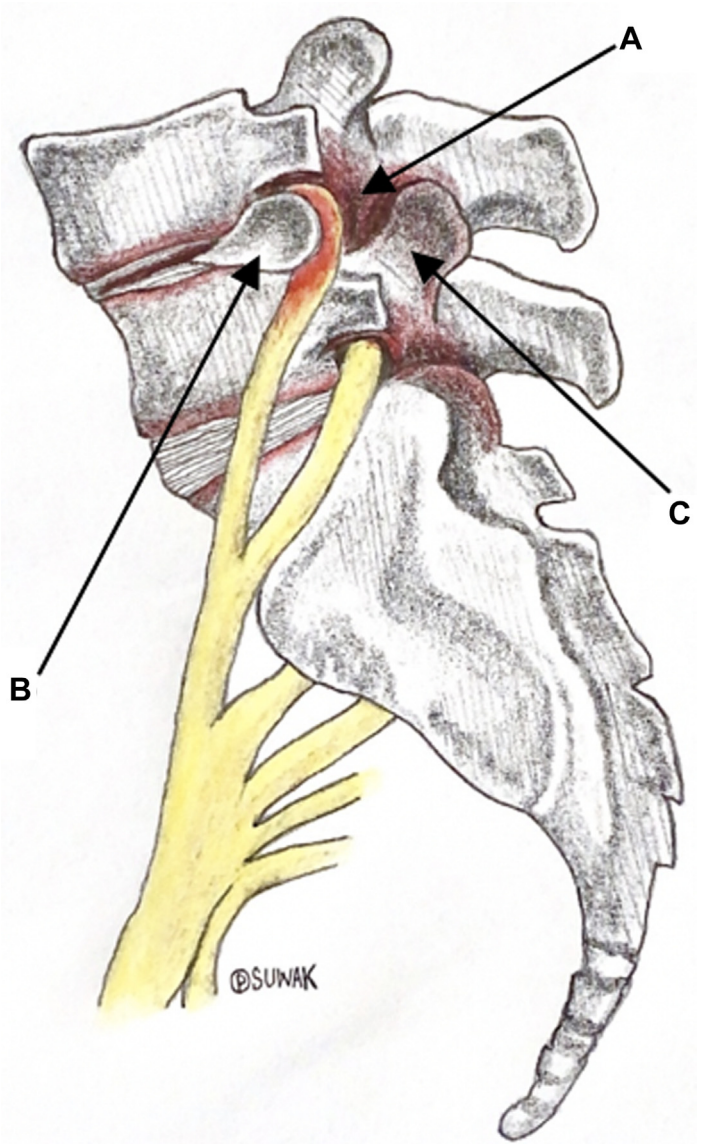


Fig. 4. Sagittal view demonstrating foraminal stenosis (A) secondary to disk degeneration and collapse (B) as well as facet osteophytes (C).

stenosis, whereas less than 75 mm indicates substantial stenosis.[11] Another morphologic classification system exists that grades stenosis, A (no or minor stenosis) through D (extreme stenosis), based on the nerve rootlet/cerebrospinal fluid (CSF) ratio on axial T2 images[12] (Table 1). In patients who are unable to tolerate an MRI or have previous spinal instrumentation, a computed tomographic (CT) myelogram, a study in which dye is injected into the CSF, allows for evaluation of the thecal sac and the surrounding soft tissue and bony pathology. Typically, workup for lumbar spinal stenosis does not require electromyography or nerve conduction velocity studies, but may be helpful when diagnostic imaging is equivocal or for evaluation of demyelinating disease, peripheral neuropathy, or peripheral nerve compression.

DISCUSSION

Once clinical diagnosis of LSS has been reached, and both the severity of symptoms and the degree of compression on imaging has been assessed, a meaningful discussion regarding the management of LSS between patient and physician should be undertaken. The goal in managing LSS is to restore function and reduce

Table 1
Classification of spinal stenosis based on morphology of the dural sac on MRI[12]

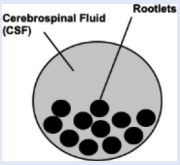
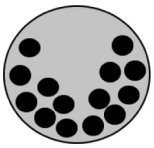
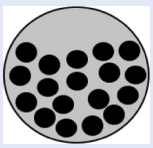
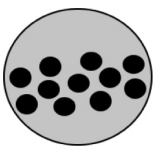
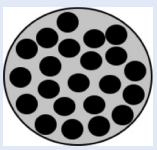
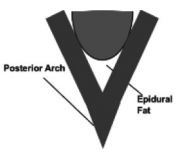
Grade	Description	Image
A	CSF present in dural sac with heterogeneous distribution. This is true for A1-A4. If not true, then it is grade B, C, or D.	
A1	Nerve rootlets are dorsal and CSF occupies >50% of the dural sac.	
A2	Rootlets remain dorsal and in contact with the dura, but they are in a horseshoe pattern.	
A3	Rootlets are dorsal and occupy >50% of the dural sac area.	
A4	Rootlets are central and occupy >50% of the dural sac area.	
B	Rootlets can be individually identified, but they occupy the entire dural sac area.	
C	Rootlets cannot be individually identified; entire dural sac area is a homogeneous gray signal	

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Table 1
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Grade	Description	Image
D	Rootlets cannot be individually identified, and there is no posterior epidural fat	

From Schizas C, Theumann N, Burn A, et al. Qualitative Grading of Severity of Lumbar Spinal Stenosis Based on the Morphology of the Dural Sac on Magnetic Resonance Images. *Spine*. 2010;35(21):1919-1924.

pain; this can be achieved through both medical treatments and surgical interventions. Typically, conservative management is the first-line treatment; this includes lifestyle changes, oral antiinflammatory medications, physical therapy, and epidural corticosteroid injections.

Nonoperative Management

Although widely used and often efficacious, there are no formal studies evaluating the use of analgesics or nonsteroidal anti-inflammatory drugs in patients with LSS. Studies looking into the use of acetaminophen in the treatment of spinal pain have shown the drug to be ineffective.[13] In addition, because the patient population with LSS is typically older and as a result, often has multiple comorbidities including hypertension, cardiovascular disease, and diabetes, nonsteroidal anti-inflammatory drugs may actually do more harm than good by negatively impacting a patient's cardiovascular, renal, and gastrointestinal systems.[14,15] Opioids, although pure analgesics, have not been shown to improve functional outcomes when combined with therapy, and in fact, the adverse effects including cognitive impairment and sedation can be dangerous and lead to increased risk of falls in older populations.[16] Gabapentin, which is effective in the treatment of different neuropathic pain syndromes, has been shown in small studies to improve pain scores, lead to increased walking distances, and improve sleep.[17] However, careful attention must be paid to the side effects of gabapentin including lethargy and dizziness, which could be detrimental to older populations.

There is no standardized physical therapy regimen to treat LSS. Traditional exercise programs focus on decreasing lumbar lordosis and extension forces while improving core strength. It is not uncommon for patients with LSS to be

deconditioned as a result of their symptoms and other age-related comorbidities. Aerobic training including stationary biking, ellipticals, treadmills, or aquatic programs can lead to improved walking tolerance and pain scores.[18] The efficacy of other modalities including ultrasonography, transcutaneous electrical nerve stimulation, and heat packs in addition to physical therapy are inconclusive.[19] Lumbar corsets, which maintain a small degree of lumbar flexion, may offer some benefit and have been shown to decrease pain and increase walking distance.[20] Ultimately, a physical therapy program must be tailored to the individual.

The purpose of epidural steroid injections (ESIs) is to reduce inflammation and edema at the stenotic segment. Fluoroscopy is typically used for caudal, interlaminar, or transforaminal ESIs. It is unclear whether ESIs result in long-term improvement in patients with LSS. Several studies have shown no differences in functional outcomes at either 6 or 12 weeks.[21] However, some case series using multiple injection protocols have led to long-term improvements up to 2 years in some patients.[22] A transient headache is a common adverse effect but more rare complications including epidural abscess, meningitis, and spinal hematomas have been noted.

If symptoms persist or progressively worsen after using conservative treatment modalities, surgical management can be considered. Ultimately, it is the patient's desire combined with failure of nonoperative management that will drive the decision for surgical intervention. Proper patient selection is critical to achieving successful outcomes with spinal stenosis surgery. Ideally, the patient will exhibit symptoms of neurogenic claudication including pain, numbness, and paresthesias in the posterolateral legs and thighs associated with prolonged walking, standing, or extension-type activities and relieved with forward flexion. In fact, one of the most common reasons for early failure after LSS surgery was absence of neurogenic symptoms coupled with nonsevere stenosis on imaging.[23]

Data have shown that certain patient variables are associated with better outcomes after surgical management compared with conservative management including male sex, shorter duration of systems, higher income levels, higher levels of education, better overall mental health, no diabetes mellitus, and few medical comorbidites.[24] In addition, it appears that baseline Oswestry Disability Index, a validated measurement of a patient's permanent functional disability, and smoking had the greatest effect on LSS surgical outcomes.[24] Typically, surgical decompression is performed on an elective basis unless there is a rapidly progressive neurologic decline or bowel/bladder dysfunction. It is imperative that before proceeding with any surgical management, the patient is medically optimized and understands the risks and associated complications of any surgical procedure.

Surgical Management

There are several surgical techniques that can be used in the treatment of LSS surgery. The most common is a decompressive laminectomy. The purpose of decompressive laminectomy is to relieve the pressure at a specific level of the spinal cord and its respective nerve roots. Other possible surgical techniques include laminotomy, minimally invasive decompression, and indirect decompression through the use of an interbody device. Among the available surgical options, there is currently no evidence to support superiority of one technique over the others.[25] Nonetheless, preferences among these surgical techniques exist.

Decompressive laminectomy is the most common technique used and serves as the gold standard for LSS surgical treatment.[26] The procedure involves removal of the lamina in its near entirety and any thickened ligaments to allow for sufficient decompression (Fig. 5). It is essential during this procedure to maintain and preserve most of the facet joints and the pars interarticularis. Loss of anatomic landmarks and overzealous resection can lead to pars fracture and iatrogenic spondylolisthesis. Several studies have demonstrated that patients undergoing surgical management have had significant improvement in primary outcomes compared with nonsurgical management.[27]

One of the largest studies focusing on the topic is the Spine Patient Outcomes Research Trial (SPORT), a multicenter prospective study that evaluated patients undergoing operative compared with nonoperative treatment. More than 650 patients with at least 12 weeks of symptoms were separated into 2 cohorts—a prospective observational cohort and a randomized control cohort—and both groups were further split into an operative and a nonoperative group. Patients with lumbar instability (defined by greater than 4 mm translation or 10° of angular motion) were excluded. Notably, nonoperative treatment was not standardized. In addition, the study was impacted by a high rate of crossover (approximately 40%) between nonoperative and operative groups. Nonetheless, the data from the SPORT study generally represents

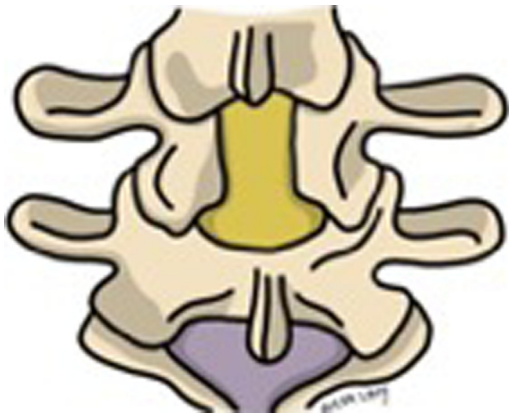

Fig. 5. In a laminectomy, the lamina is removed to decompress the spinal cord.

the highest level of evidence to date. In an as-treated analysis, decompressive laminectomy for symptomatic degenerative spinal stenosis provided significant improvements in function, pain, and disability compared with nonoperative treatment for both short term (3 month) and 4-year follow-up.[28]

The SPORT trial also investigated long-term outcomes at 8 years follow-up. The as-treated analysis demonstrated that the surgical group in the randomized control cohort had diminished benefits in surgical treatment after 4 years, specifically showing no significant effect of surgery between 6 and 8 years follow-up. It should be noted that 52% of the patients randomized to the nonoperative treatment eventually underwent surgery during the 8-year period. However, in the observational study group, surgical intervention demonstrated a consistent improvement in primary outcomes maintained across the 8-year follow-up period.[29]

Additional studies have shown the significant benefit of surgical management for LSS in both short- and long-term outcomes. At 1-year follow-up, patients with LSS treated surgically demonstrated significantly better and improved patient-reported outcomes than patients treated nonsurgically.[30] The same Maine Lumbar Spine Study group showed in their prospective observational cohort similar improved patient-reported outcomes in long-term follow-up of 8 to 10 years. Specifically, patients who underwent surgical management were more active and had significantly less severe leg pain than patients who underwent nonsurgical management at 8 to 10 years' follow-up.[24] Other randomized controlled trials have investigated patient-reported outcomes of decompressive surgical management versus nonsurgical management in patients with LSS and demonstrated similar favorable results for surgical management. Patients who underwent decompressive surgery experienced significantly greater improvement in overall disability, leg pain, and back pain at all follow-up examinations during the 2-year study period.[31] Taken into consideration, the various published findings indicate that surgical management of LSS provides a reliable and effective benefit for patients up to 4 years follow-up compared with nonsurgical management.

A laminotomy procedure is an alternative approach to a decompressive laminectomy, especially in the circumstances of primarily lateral recess stenosis. In a laminotomy, only a portion of the vertebral lamina is removed, which allows for decompression of the nerve root (Fig. 6). Some propose that by maintaining the midline structures, namely, the spinous process and interspinous and supraspinous ligaments, as opposed to removing them during a laminectomy, there is a decreased possibility of iatrogenic instability and postoperative lumbar back pain.[32] Studies comparing unilateral or bilateral laminotomy to laminectomy in patients with lateral recess LSS without significant central stenosis demonstrated that laminotomy resulted in better perceived recovery at final follow-up visits as well as lower rates of iatrogenic instability and less postoperative back pain.[33–35]

Minimally invasive surgeries have been increasing since the introduction of laparoscopic instruments. Microendoscopic decompression of LSS provides small incisions that preserve the surrounding soft tissue structures while providing equivalent resection to that of open laminectomy. Compared with open decompressive laminectomy, studies have shown that microendoscopic decompressive laminectomy has been proved to yield significantly less operative blood loss, shorter time to mobilization and length of hospital stay, less muscle destruction, less low back and leg pain at final follow-up, as well as less probability of requiring opioids for postoperative pain.[36,37]

Interbody fusion, a surgical technique in which the intervertebral disk is removed and replaced with a metal, plastic, or bone spacer, is also used in the management of lumbar degenerative conditions. Although more commonly used for the treatment of spondylolisthesis, the placement of an interbody device can provide indirect decompression of the lumbar spine by increasing disk height, foraminal height, foraminal area, and spinal canal diameter[38,39] (Fig. 7). By decompressing the neural elements

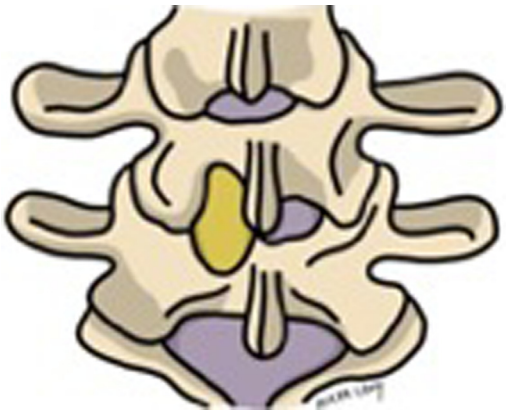

Fig. 6. In a laminotomy, as opposed to a laminectomy, only a portion of the lamina is removed.

in a minimally disruptive way, the surgeon avoids direct resection of posterior structures and its associated morbidities. However, the addition of an interbody increases both the cost and time of surgery and may increase the complication rate.

Postoperatively, patients are encouraged to get out of bed with physical therapy and ambulate as soon as possible. Patients are advised to avoid bending, lifting, and twisting for 6 to 12 weeks. Typically, patients are seen in the office 2 to 3 weeks after surgery for an assessment of the surgical incision and radiographs and are given a prescription for outpatient physical therapy. In the event of a fusion procedure, flexion-extension radiographs are obtained at each subsequent clinic visit to ensure no further instability has occurred. Long-term follow-up is necessary to confirm that a solid fusion has occurred and there are no hardware-related complications. Absence of bridging bone on radiographs or continued symptoms should warrant a CT scan to assess for pseudoarthrosis of the fusion mass. Follow-up at regular intervals can proceed as per patient needs.

Complications

As with all surgical procedures, potential complications are ever present, including infection, CSF leaks, nerve root injuries, vascular sequelae, nonunion or hardware failure, instability, and adjacent segment disease. CSF leaks are one of the most common complications following LSS surgery with rates ranging from 1.8% to 17.4%.[40,41] Management of these tears varies by surgeon but are generally managed by repair ± spinal sealant ± closed-suction drainage. Vascular complications following LSS surgery including deep vein thrombosis, pulmonary embolism, postoperative hematoma, and rarely, vascular catastrophe lead to unexplained hypotension. Typical postoperative prophylaxis includes compression socks and pneumatic sequential compression devices, thromboembolic prophylaxis, and early ambulation. When postsurgical infections occur, they usually occur in the early postoperative period (<3 months). It is important to diagnose infections early and obtain C-reactive protein measurements as an adjunct to the physical examination. If infection is suspected, irrigation and debridement with retention of intact hardware, if present, as well

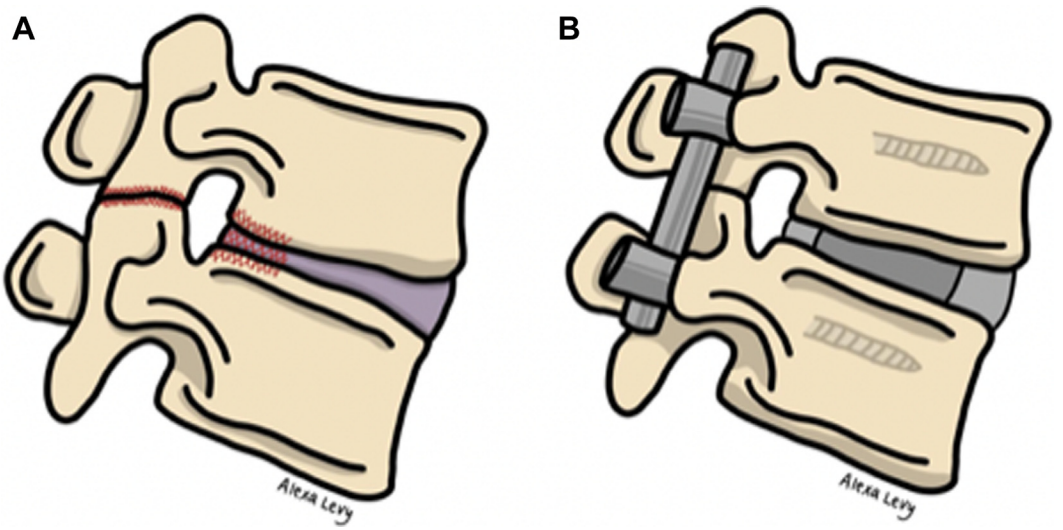

Fig. 7. By replacing the degenerative disk (*A*) with a spacer (*B*), the interbody fusion technique indirectly decompresses the lumbar spine by restoring spine biomechanics.

as long-term intravenous antibiotics are required. During LSS surgery, despite adequate decompression, overresection of the lumbar facets can lead to iatrogenic spondylolisthesis and subsequent long-term instability requiring further surgeries. Adjacent-segment disease, a condition in which adjacent levels further degenerate causing debilitating symptoms, can be a potential issue after lumbar fusion. However, although the rate of radiographic adjacent-level degeneration is high, clinically symptomatic patients only make up a small subset of the overall group. Although LSS is typically a condition of older patients with greater comorbidities, studies have shown that morbidity rates associated with decompression and/or fusion are comparable to that in young populations undergoing similar procedures.[42] In addition, in patients older than 80 years with LSS and degenerative spondylolisthesis, operative treatment produced significant benefit over nonoperative management and they experienced no significant increases in complications or mortality rates after surgery compared with younger patients.[43] However, other studies have shown that although older patients may actually experience significantly greater relief of pain, there were more postoperative adverse complications in the older group including cardiac and respiratory events and infections.

SUMMARY

LSS is typically an age-associated degenerative condition characterized by a narrowing of the spinal canal, resulting in nerve root compression. Although asymptomatic disease is common, symptoms can often be debilitating and patients may present with unilateral or bilateral pain in the buttock, groin, and thigh regions with associated heaviness, fatigue, weakness, and paresthesias. Initial workup includes radiographic images of the lumbar spine, including lateral standing films to assess for instability. Although the diagnosis of LSS is based on clinical examination, advanced imaging either through MRI or CT myelogram is essential for accurate assessment of LSS. Initial nonoperative treatment of LSS includes medication, such as gabapentin, physical therapy, and, if necessary, ESIs. If conservative treatment fails and the patient is an appropriate candidate for surgery, operative intervention can be initiated. The type of lumbar decompressive surgery is based on symptoms, levels involved, and whether there is evidence of instability. A decompressive laminectomy is the most common procedure. Recent literature supports operative intervention over nonoperative management for LSS in appropriately selected patients. Postoperatively, the patient will quickly be mobilized and regularly monitored by their operating surgeon to assess for any possible complications including infection, adjacent segment disease, instability, and nonunion or hardware failure.

CLINICS CARE POINTS

- Lumbar disk degeneration is nearly ubiquitous in elderly patients.
- The diagnosis of LSS is based on the clinical presentation. Many patients with radiographic stenosis do not exhibit symptoms of neurogenic claudication. In addition, one should be wary of mimickers of LSS such as hip disease and peripheral arterial disease.
- Initial radiographic images are necessary to obtain to evaluate for instability. MRI is the preferred radiologic study in assessing spinal stenosis.
- Conservative management with medications and physical therapy are first line for the treatment of LSS. ESIs can be considered as a potential option. Most patients will do well with nonoperative care.
- Patients with neurogenic claudication with correlating radiographic findings of LSS who fail nonoperative care are the ideal surgical candidates.
- Open decompressive laminectomy is the gold standard of treatment of LSS without instability.
- An as-treated analysis performed in the SPORT study, a large multicenter level 1 prospective randomized controlled trial, demonstrates the efficacy of operative intervention over nonoperative management for LSS.

DISCLOSURE

The authors have nothing to disclose.

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