ORIGINAL ARTICLE

The MIST Guidelines: The Lumbar Spinal Stenosis Consensus Group Guidelines for Minimally Invasive Spine Treatment

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■ **Abstract**

Background: Lumbar spinal stenosis (LSS) can lead to compression of neural elements and manifest as low back and leg pain. LSS has traditionally been treated with a variety of conservative (pain medications, physical therapy, epidural spinal injections) and invasive (surgical decompression) options. Recently, several minimally invasive procedures have expanded the treatment options.

Methods: The Lumbar Spinal Stenosis Consensus Group convened to evaluate the peer-reviewed literature as the basis for making minimally invasive spine treatment (MIST) recommendations. Eleven consensus points were clearly defined with evidence strength, recommendation grade, and consensus level using U.S. Preventive Services Task Force criteria. The Consensus Group also created a treatment algorithm. Literature searches yielded 9 studies (2 randomized controlled trials [RCTs]; 7 observational studies, 4 prospective and 3 retrospective) of minimally invasive spine treatments, and 1 RCT for spacers.

Results: The LSS treatment choice is dependent on the degree of stenosis; spinal or anatomic level; architecture of the stenosis; severity of the symptoms; failed, past, less invasive treatments; previous fusions or other open surgical approaches; and patient comorbidities. There is Level I evidence for percutaneous image-guided lumbar decompression as superior to lumbar epidural steroid injection, and 1 RCT supported spacer use in a noninferiority study comparing 2 spacer products currently available.

Conclusions: MISTs should be used in a judicious and algorithmic fashion to treat LSS, based on the evidence of efficacy and safety in the peer-reviewed literature. The MIST Consensus Group recommend that these procedures be used in a multimodal fashion as part of an evidence-based decision algorithm. ■

Key Words: lumbar spinal stenosis, minimally invasive spine treatment, percutaneous image-guided lumbar decompression, systematic literature review, epidural injection, interspinous spacer

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INTRODUCTION

Creation of the Guideline Development Group

Open surgical treatment of lumbar spinal stenosis (LSS) has been an established practice for decades, and recently several minimally invasive treatment options have expanded the available clinical treatment options. Most significantly, these minimally invasive options are supported by prospective, randomized trials. However, proper patient selection for these new treatment options is essential to success, as it is with other surgical and pain care treatments. Recognizing these issues, several cross-disciplinary leaders in the interventional spine community, representing many surgical and pain societies, have formed a consensus group to evaluate the current state of LSS diagnosis and treatment, and to make recommendations to guide clinical practice in this emerging area.

Using the Institute of Medicine (IOM) clinical practice guidelines for 2011, a group of nationally recognized spine experts was convened and charged with creating clinical practice guidelines for minimally invasive spinal treatment (MIST).[1] Within the IOM framework, the workflow included these steps: identify the workgroup and establish the charge; identify and reconcile conflicts of interest; identify and evaluate the evidence; and make recommendations based on that evidence. For topics and practice areas where the evidence base was still emerging, clinical consensus, based on the available best practice experience, was created. Each consensus point was clearly defined, with evidence strength, recommendation grade, and consensus level provided. Consensus areas were clearly delineated as recommendations based on consensus, whereas recommendations based on the literature cite supporting studies.

Defining Workflow, Evidence Ranking, Recommendations, and Consensus

The development of consensus guidance has been performed previously, with recent works published by the Polyanalgesic Consensus Conference and the Neuromodulation Appropriateness Consensus Committee. Those committees used the U.S. Preventive Services Task

Force (USPSTF) criteria for evidence level and degree of recommendation, along with the strength of consensus.[2] Given the early state of the literature regarding clinical use of minimally invasive LSS treatment, the goal of this article was to fill gaps in knowledge with expert consensus for this rapidly expanding clinical practice. The literature base for noninjection treatments is relatively small, and the USPSTF criteria provide a basic and straightforward method of communicating the state of the literature to the reader (ie, based on randomized controlled trial [RCT] evidence, case control evidence, or consensus opinion). The USPSTF evaluation of proper study design in this context uses Jadad criteria[3] for evaluating randomization, drop-out rate, withdrawal rate, and reasoning and blinding methods. As the literature base grows and matures, more robust grading criteria can be applied. USPSTF criteria for evidence levels (Table 1), meaning of recommendation degrees (Table 2), and strength of consensus (Table 3) appear with the consensus points in this publication. The MIST working group also conducted systematic literature searches, which prioritized RCTs, and served as the evidence base for the recommendations and discussions that follow.

Table 1. Hierarchy of Studies by the Type of Design (U.S. Preventive Services Task Force)[2]

Evidence Level	Study Type
I	At least 1 controlled and randomized clinical trial, properly designed
II-1	Well-designed, controlled, nonrandomized clinical trials
II-2	Cohort or case studies and well-designed controls, preferably multicenter
II-3	Multiple series compared over time, with or without intervention, and surprising results in noncontrolled experiences
III	Clinical experience-based opinions, descriptive studies, clinical observations, or reports of expert committees

Table 2. Meaning of Recommendation Degrees (U.S. Preventive Services Task Force)[2]

Degree of Recommendation	Meaning
A	Extremely recommendable (good evidence that the measure is effective and that benefits outweigh the harms)
B	Recommendable (at least moderate evidence that the measure is effective and that benefits exceed harms)
C	Neither recommendable nor inadvisable (at least moderate evidence that the measure is effective, but benefits are similar to harms and a general recommendation cannot be justified)
D	Inadvisable (at least moderate evidence that the measure is ineffective or that the harms exceed the benefits)
I	Insufficient, low-quality, or contradictory evidence; the balance between benefit and harms cannot be determined

Table 3. Strength of Consensus

Strength of Consensus	Definition*
Strong	>80% consensus
Moderate	50% to 79% consensus
Weak	<49% consensus

*Quorum defined as 80% of participants available for vote.

LUMBAR SPINAL STENOSIS

The North American Spine Society (NASS) clinical guideline development group has defined LSS as condition and symptom constellations that arise from decreased canal space within the lumbar spinal column (NASS consensus).[4] Although LSS may be congenital in nature, as well as degenerative, most guidelines focus on developmental LSS, and the NASS guidelines are no exception. When LSS becomes symptomatic, it causes a spectrum of clinical syndromes characterized by neurogenic claudication, and ranging from buttock and leg pain (frequent), to fatigue or "heaviness in the legs," to significant neurologic compromise (rare). The NASS guidelines suggest that significant neurologic compromise, while rare, is not necessarily correlated with severity of stenosis based on radiographic imaging. Obviously, the time course of the development of the stenosis is critical: typically, the faster the onset, the more pronounced the clinical presentation. These current MIST guidelines are not inclusive of subjects with significant neurologic compromise requiring urgent or emergent evaluation for surgical decompression, and are intended for the segment of patients who are seeking elective LSS treatment.

History and Physical Findings

The most consistent findings of symptomatic spinal stenosis occur with older individuals who have little to no pain at rest while sitting or when lying recumbent. However, upon standing and/or soon after ambulating, the individual experiences back, buttock, and/or leg pain that is progressive in nature, and possibly has a neuropathic component (tingling and numbness) as well

as an aching component (mechanical and ischemic but nonvascular). Patients may also describe a sense of heaviness in the back and/or lower extremities. These symptoms appear at various times but typically present within a few minutes to 15 minutes of ambulating. Symptoms may limit activities of daily living (ADLs) and are referred to as neurogenic claudication. Symptoms typically resolve immediately or within a short time after sitting or lying down. If patients' pain is not worsened by ambulation, they have a low likelihood of spinal stenosis as their primary cause of discomfort.[5–7] Likewise, there are subjects who have neurogenic claudication as a component of their pain but also have superimposed radicular pain or mechanical back pain from other anatomical sources. Although spinal stenosis may contribute to these clinical presentations, this would represent a mixed clinical picture and would not be classic spinal stenosis based on history. A history of extension-based pain relieved by flexion seems to be consistent throughout the literature. In many evaluations, the physical examination more often than not reveals normal reflexes, sensation, and motor strength while sitting and lying. The history is usually far more sensitive in determining the presence of LSS than the physical examination and imaging studies, although both are necessary to ensure that comorbidities are evaluated and diagnosed.

The use of questionnaires for identification and evaluation of LSS has previously been graded as having insufficient evidence in support of or against the practice, and physical examination tests have also been graded as having low specificity for identifying LSS.[5–7]

Current Nonsurgical Treatment Options: Treatment Continuum

For decades the mainstays of nonsurgical treatment for LSS included physical therapy, spinal manipulation, exercise, and stretching (Figure 1). Medical management consisted of nonsteroidal anti-inflammatory drugs (NSAIDs), anticonvulsants, antidepressants for neuropathic pain, and opioids. Traditional interventional treatment consisted of interlaminar lumbar epidural steroid injections (ESIs) and transforaminal ESIs. Although these strategies can reduce pain significantly, they often have the limitation of short-term duration of benefit (1 to 6 months). Historically, the next step is to consider open decompressive surgery, often with spinal fusion, when the simple interventions fail. A large gap existed in the treatment algorithm with regard to duration of effectiveness and degree of invasiveness between conservative nonsurgical treatment and surgical treatment options. In the past decade, several image-guided percutaneous interventions were introduced that have now potentially expanded the algorithm of conservative, minimally invasive surgical treatment options.[8,9]

CONSENSUS POINT 1

The LSS treatment choice is dependent on the degree, level, and architecture of the stenosis; severity of the symptoms; failed, past, less invasive treatments; and patient comorbidities (Level I-I, Grade A, Consensus strong).

Diagnosis of Lumbar Spinal Stenosis by Radiologic Imaging

LSS is regarded as narrowing of the central canal, foramen, or lateral recess of the lumbar spine[10] (Figure 2). Figure 2 demonstrates the anatomic positioning of the various categories of LSS relative to the bony structures of the spinal vertebrae, suggesting a visual framework of central and lateral recess, and foraminal and extraforaminal stenosis as outlined in Figure 3. Symptoms consistent with LSS include pain in the buttocks and numbness and weakness in the lower extremities exacerbated by prolonged standing or walking. This presentation must then be correlated with radiographic evidence of spinal narrowing and neural compression (Figure 4).[11] Spinal narrowing that is not

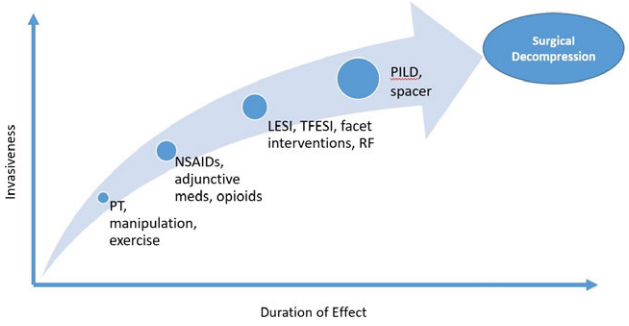

Figure 1. Graphic representation of level of invasiveness vs. length of efficacy of nonsurgical treatment options. LESI, lateral epidural spinal injection; NSAIDs, nonsteroidal anti-inflammatory drugs; PILD, percutaneous image-guided lumbar decompression; PT, physical therapy; RF, radiofrequency; TFESI, transforaminal epidural spinal injection.

congenital can arise from ligamentum flavum (LF) thickening, disc disease, osteoarthritic facet hypertrophy, or a combination of all 3[12] (see Figure 4). In settings when the diagnosis of LSS appears to correlate with patient symptoms and objective imaging, diagnosis of LSS would seem to be straightforward. Unfortunately, despite the fact that stenosis is defined as narrowing of the spinal space with resulting compression, the exact radiographic definition of LSS remains unclear and, in fact, there is no exact radiologic definition of LSS.[11]

There are many reasons for uncertainty in the radiographic diagnosis of LSS. There are abundant proposed grading systems, but unfortunately, no single system has proven superior, creating inconsistencies in the literature.[13,14] There are measurable, quantitative criteria, but more commonly, qualitative criteria are used, leading to inconsistent inter-reader agreement.[15] Another complication to reaching the elusive diagnosis of LSS is that multiple imaging modalities are employed. Currently, MRI, computed tomography (CT), and CT myelography are all used in the diagnosis.[13] There is broad consensus that MRI is the best study to yield soft-tissue contrast and is the most commonly used modality.[16] CT and CT myelography can be utilized when there are contraindications to MRI or accurate bony anatomy is indicated.[13] Unfortunately, the values among imaging modalities and measurement type will vary. Plain radiographs are of limited value except to demonstrate alignment of the vertebral bodies.[17]

The NASS defines LSS as "a condition in which there is diminished space available for the neural and vascular elements in the lumbar spine secondary to degenerative changes in the spinal canal."[4] The question then becomes which radiographic findings and measurements translate into meaningful clinical information. In the 2012 Delphi survey sponsored by the Lumbar Spinal Stenosis Outcome Study Working Group Zurich, a group of radiologists considered to be musculoskeletal experts developed criteria felt to be the most important in describing LSS.[11] Five of the 6 criteria felt to be most relevant to describing LSS were qualitative in nature. These included disc protrusion/extrusion/sequestration, perineural intraforaminal fat, hypertrophic facet joint degeneration, absent fluid around the cauda equina, and hypertrophic LF.[11] Although there was broad agreement among the experts on which qualitative criteria were important, there was no consensus on the most important parameter of the 5.

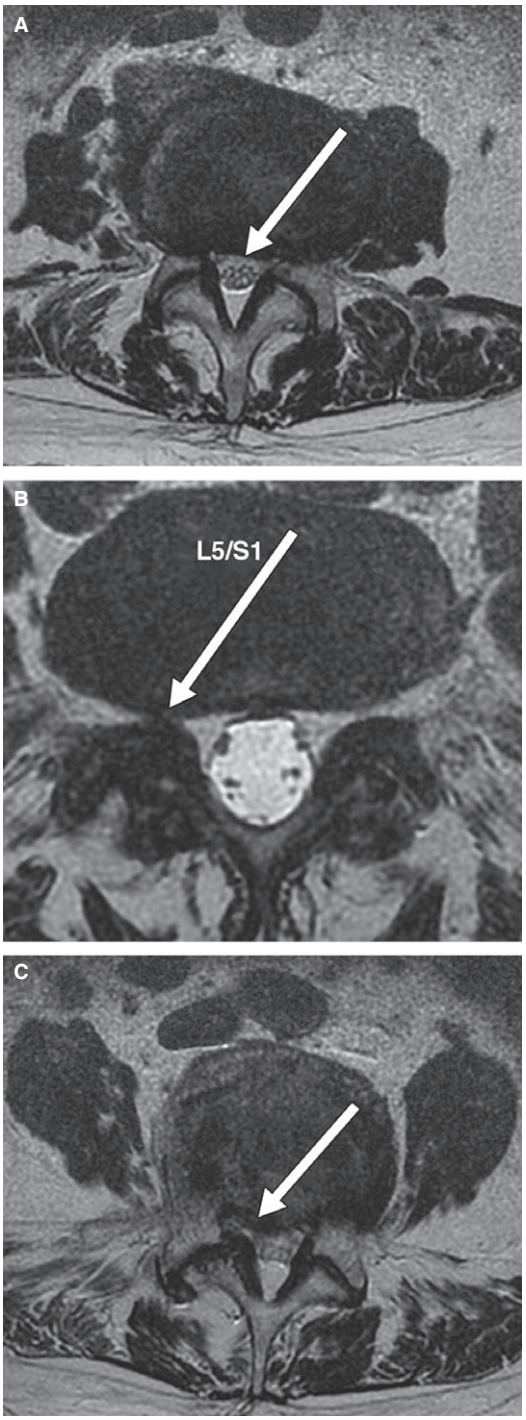

Figure 2. Radiologic criteria for the diagnosis of lumbar spinal stenosis. Axial T2-weighted magnetic resonance images show lumbar spinal stenosis at different locations in different patients. A, Central spinal canal stenosis (arrow). B, Neuroforaminal stenosis (arrow). C, Right lateral recess stenosis (arrow). Reprinted from Andreisek G, Imhof M, Wertli M, et al. A systematic review of semiquantitative and qualitative radiologic criteria for the diagnosis of lumbar spinal stenosis. *Am J Roentgenol.* 2013;201: W735–W746.[12] Reprinted with permission of the *American Journal of Roentgenology.*

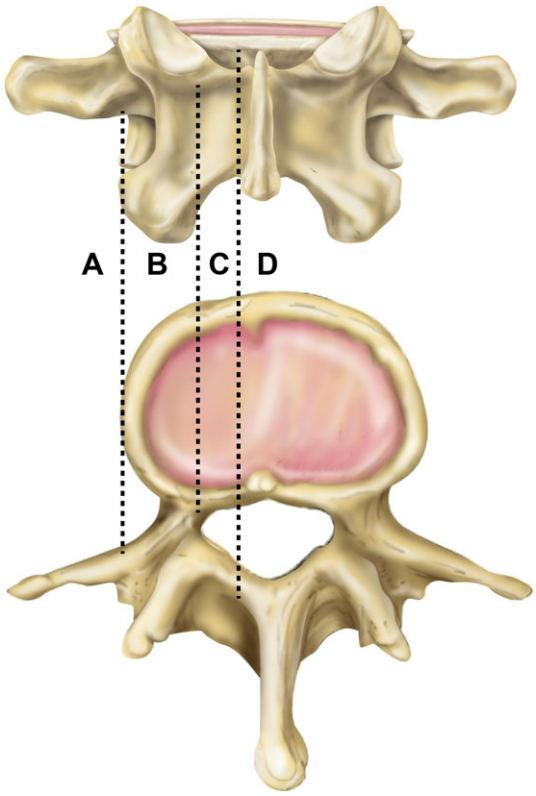

Figure 3. Lumbar vertebrae: illustration of anatomical spaces. A, The extraforaminal space is lateral to the neuroforamen. B, The foramen is created by the roof and floor of the adjacent pedicles. C, The lateral recess begins laterally at the pedicle and covers the area medially to the start of the central canal. D, The central canal encompasses the area between the lateral recess and is bound anteriorly by the vertebral body or disc and posteriorly by the vertebral arch. Courtesy of T. Dolan, Department of Academic Multimedia, University of Kentucky, Lexington, Kentucky, U.S.A.

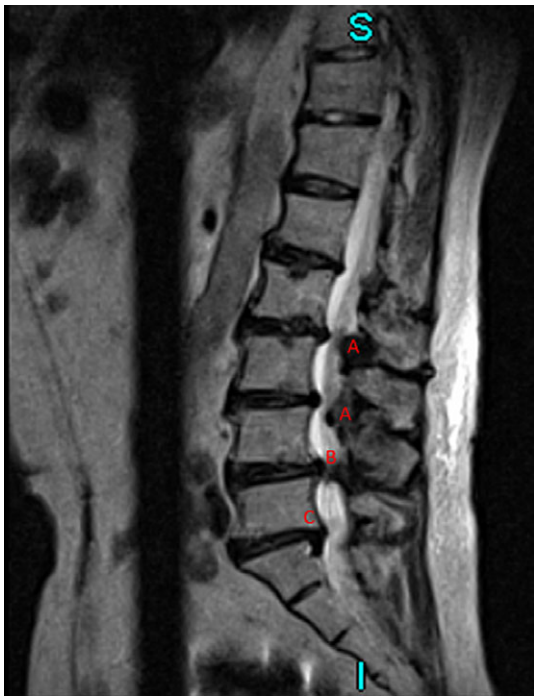

Figure 4. MRI with multiple causes of spinal stenosis. A, Ligamentum flavum hypertrophy. B, Disc herniation and ligamentum flavum hypertrophy. C, Retrolisthesis with disc extrusion.

In this same study, only 1 quantitative measurement, anteroposterior (AP) diameter of the osseous spinal canal, was consistently rated as important by the panel experts.[11] In this particular study, a cutoff value of less than 11 mm in AP diameter at the L3–4 disc level resulted in the diagnosis of central canal stenosis. Three additional quantitative parameters are worth mentioning, given the paucity of quantitative measurements, but did not reach the statistical median score of 9 needed to be included in panel recommendations: cross-section of the dural sac of <100 mm^2, midsagittal diameter of the dural sac of <12 mm, and diameter of the foramen of <3 mm.

As discussed previously, hypertrophy of the LF is considered to be important, both as a cause of LSS and in the reporting of radiographic finding. LF thickening occurs more frequently at the L3–4 and L4–5 lumbar segments, more so than at L5–S1. The LF is typically measured perpendicular to the border of the lamina corresponding to the intervertebral disc (Figure 5).[18,19] It has been proposed that the upper limit of normal for LF thickness is <4 mm; however, in a recent study by Abbas et al.[18] comparing patients known to have LSS to patients without LSS symptoms, there was wide variation in LF thickness. They noted that even in patients who did not have symptoms of LSS, LF thickness could exceed 4 mm. Of interest, in patients with spondylolisthesis, thinner LF was noted compared with patients without spondylolisthesis.[18] This was felt to be related to the forces applied to the LF, creating stretch, and therefore thinning the ligament as the vertebral body moves anterior relative to the adjacent vertebra.

A large systematic review attempted to address the lack of quantitative criteria for the diagnosis of LSS.[20] The investigators evaluated the 10 most common quantitative parameters applied to LSS, regardless of reference to central, lateral, or foraminal stenosis. The 2 most commonly reported quantitative values were the AP diameter of the osseous spinal canal (Figure 6) and the cross-sectional area of the dural sac (Figure 7). The most common cutoff point for central canal stenosis in the AP diameter was 10 mm; however, some authors

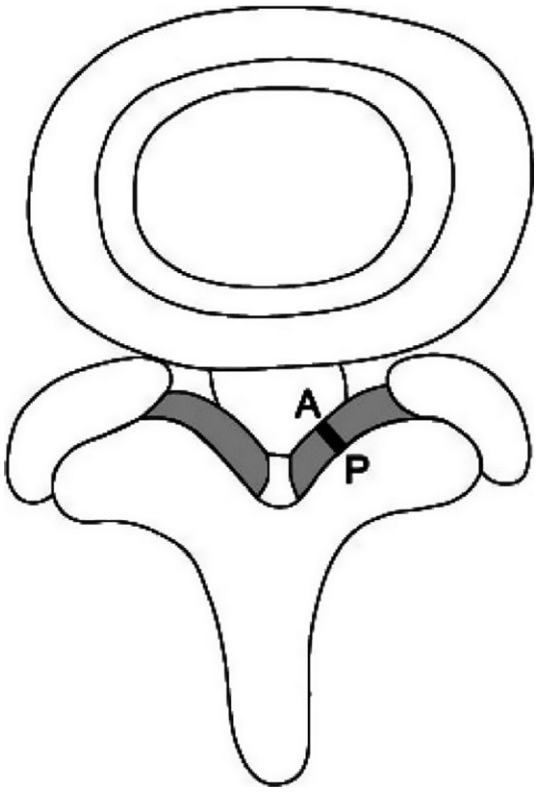

Figure 5. Ligamentum flavum thickness measurement (AP). The thickness of the ligamentum flavum is measured perpendicular to the border of the lamina corresponding to the intervertebral disc. Reprinted from Abbas et al. (2010)[18] under Open Access, Creative Commons terms and conditions.

went as low as 7 mm and some as high 13 mm. The cross-sectional cutoff was fairly consistent in defining stenosis at <100 mm^2. [20]

In 2013, Andreisek et al. performed a systematic review evaluating semiquantitative and qualitative criteria for LSS.[12] Semiquantitative criteria differ from the quantitative criteria set out in Steurer et al.[20] in that the semiquantitative criteria are inherently subjective. Despite this subjectivity, as noted previously, radiologists use these types of criteria more frequently than quantitative radiologic parameters.[12] The panel set defining criteria for central and lateral LSS with standardization that was both reproducible and usable in clinical practice (Table 4).

CONSENSUS POINT 2

There are poor correlations between severity of spinal stenosis radiographically and clinical presentation (Grade I, Level II, Consensus strong).

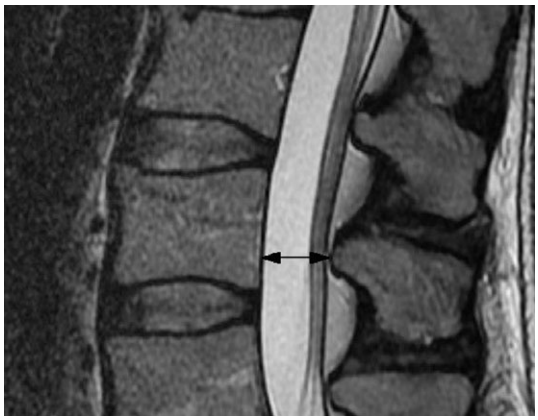

Figure 6. Middle lumbar spine. The black arrow indicates the anteroposterior diameter of the osseous spinal canal. Reprinted from Steurer et al. (2011)[20] under Open Access, Creative Commons terms and conditions.

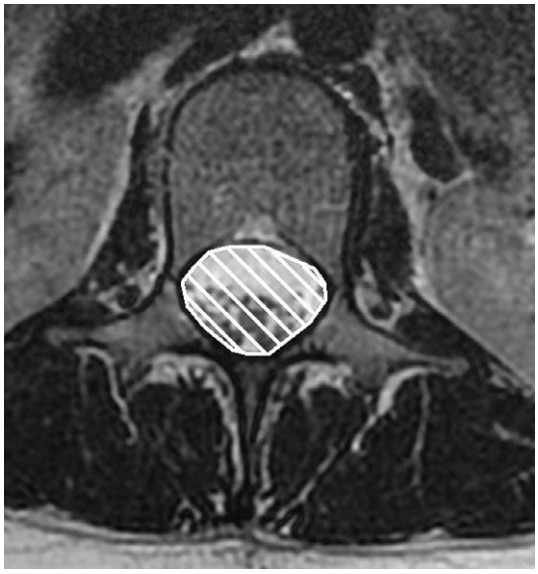

Figure 7. Lumbar spine at the level of L1. Cross-sectional area of the spinal canal is indicated by the white hatched area. Reprinted from Steurer et al. (2011)[20] under Open Access, Creative Commons terms and conditions.

CONSENSUS POINT 3

When possible, an interpretation of the MRIs by the treating physician performing either a direct or indirect surgical decompressive procedure is critical to improve success, avoid failure, and improve safety (Grade C, Level II, Consensus strong).

Radiology Reports

Physicians treating patients whose complaints are compatible with symptomatic LSS and present with neurogenic claudication initially rely on information from the radiologic imaging report. First, is stenosis present? Second, if present where is the location? Finally, which anatomic structure—bone, ligament, or disc—is causing the stenosis?[11] In 2014, a consensus conference of 15 international experts convened to define minimum standards of what should be included in a radiologic report for patients with suspected LSS.[21] A total of 27 radiologic parameters and criteria were reviewed by the panel. Five key radiologic criteria were chosen as a minimum standard in clinical reporting. In choosing these specific measures, the experts felt there was reproducibility, findings present in a majority of patients, report comprehension by the referring physicians, criteria that accounted for anatomic variability, and a relationship between symptoms and outcome. Interestingly, all 5 radiologic criteria were qualitative in nature, with the panel citing poor evidence correlating quantitative criteria and patient symptoms or outcomes (Table 5).

The panelists did agree upon 5 quantitative parameters that, while too time consuming to obtain or too difficult to measure in clinical practice, should be used in research studies to help standardize findings. These conclusions were based on measures that were reproducible, would create a discriminating threshold to discern LSS from not LSS, facilitate measurement, account for variation in anatomy, and correlate clinical presentation and outcomes[21] (Table 6).

Table 4. Stenosis Classification Criteria: Compression Ratio in Relation to Normal Size[12]

	Central (Spinal Compression)	Lateral (Compression of Lateral Recess)
Mild	<1/3	<1/3
Moderate	1/3 to 2/3	1/3 to 2/3
Severe	>2/3	>2/3

Table 5. Qualitative Radiologic Criteria for Stenosis

Central Stenosis	Lateral Stenosis	Foraminal Stenosis
Central zone compromise Relationship between fluid around the cauda equina	Lateral recess nerve compression	Foraminal nerve root impingement* Foraminal zone compromise

*As there are many descriptions of foraminal nerve root impingement, the grading system by Pfirrmann et al. was recommended.[22]

Table 6. Quantitative Radiologic Criteria for Stenosis

Central Stenosis	Lateral Stenosis	Foraminal Stenosis
Anteroposterior diameter of dural/thecal sac (<10 mm) Thecal sac area compression in % of normal midsagittal diameter Cross-sectional area of dural tube/sac*	Lateral recess height (<2 mm) Lateral recess depth (<2 to 3 mm)	None found

*The authors suggest that the cross-sectional area of the dural sac should be considered the most important parameter to document.

Like many other modalities in medicine, neuroimaging is a tool for the physician who must correlate the patient's subjective symptoms with objective information to arrive at a clinical diagnosis and develop a treatment plan, with the intent to achieve treatment success. With such a wide-ranging set of radiographic criteria, both quantitative and qualitative, and significant variability within those parameters, imaging, while important, must still be correlated with the patient's clinical presentation.

CONSENSUS POINT 4

Symptomatic lumbar spinal stenosis is a diagnosis that requires both radiographic evidence and presence of neurogenic claudication symptoms (Grade B, Level I, Consensus strong).

Diagnosis of Spinal Stenosis by Physical Examination

The diagnosis of LSS presents an interesting challenge for physicians. As previously noted, LSS can be defined by radiologic findings, but these are often not specific and cannot be used alone to guide treatment.[23] LSS can be entirely asymptomatic.[23,24] Alternatively, it can present with neurogenic claudication in the case of central canal stenosis, radicular pain in the case of foraminal or lateral recess stenosis, or as a combination.[25,26] Physical examination findings may differ among these groups. As the accurate diagnosis of clinically significant LSS has implications for treatment decisions, the history and physical examination are of critical importance.

The initial examination of the patient with suspected spinal stenosis begins with a visual examination of the lumbar spine.[27] The curvature of the spine should be noted, along with any scoliotic deformity or aberration of the normal thoracic kyphosis or lumbar lordosis.

Patients with LSS are often noted to have loss of normal lumbar lordosis, and they may sit and walk in a forward-flexed position.[28] Superficial examination of the skin with any evidence of infection or skin abnormalities should be noted, as should evidence of scarring indicative of previous surgery.

Neurologic examination of the LSS patient while at rest may appear normal. A straight leg raising test, the results of which are characteristically positive in patients with disc herniation and radiculopathy, is typically absent in patients with LSS.[29] Weakness in an L5 distribution (extensor hallucis longus) is the most common motor finding. Asymmetrical reflexes at the knee or ankle may also be seen. Symmetrically diminished or absent reflexes, particularly at the ankle, are more likely age-related.[30] Other abnormalities on examination may be secondary to lateral recess or foraminal stenosis.[31]

Vascular examination, to help distinguish between vascular and neurogenic claudication, should include palpation of distal pulses and assessment of distal skin temperature and appearance. If symptoms refer to the buttock, hip, or groin, a brief assessment of the hips is warranted to rule out intrinsic hip pathology. Testing hip range of motion with flexion, abduction, and external rotation; a hip scour test; as well as palpating the gluteal tendon insertion onto the greater trochanter can be helpful. Assessment for possible confounding diagnoses of sacroiliac and/or facet joint pathology should also be done by the clinician.

In LSS, the hallmark finding is the presence of neurogenic claudication. It is useful to recreate this finding during the physical examination. Prolonged extension may recreate and exacerbate the patient's symptoms. The stoop test, during which a patient is asked to walk with exaggerated lumbar extension until symptoms of neurogenic claudication are noted, can be performed.[32] If leaning forward or sitting relieves the symptoms, neurogenic claudication secondary to LSS is suspected. Similarly, having patients stand during the examination will very likely recreate their symptoms, and the standing intolerance time should be noted.

Clearly, surgical indications for spinal stenosis are similar for other spinal pathologies requiring surgery, with a mindful eye on cauda equina syndrome: weakness, numbness around the groin representing saddle paresthesia, or bladder or bowel dysfunction. Advanced imaging is warranted in these circumstances.

The differential diagnosis of LSS is made difficult by a number of factors. The symptom of pain with walking can be caused by a number of different disease processes, particularly in the elderly population with degenerative disease and multiple other comorbidities.[26] It is critically important to differentiate neurogenic claudication from vascular claudication.[29] Therefore, popliteal and pedal pulses should be checked by palpation and ankle-brachial index if necessary. If co-existent disease is suspected, further angiographic imaging is warranted. As in all patients presenting with lumbar pain, evaluation should rule out tumor, infection, or compression fracture.[30]

Comorbidities and Disease Recognition

Lumbar spinal stenosis refers to the potential compression of the neural structures.[33] Compression of nerve roots may be due to congenital (developmental) or acquired factors such as spondylolisthesis, degenerative disc, LF hypertrophy, or osteoarthritis.[34] Associated symptoms may include motor weakness, heaviness of the limbs, numbness, or paresthesia.

Typically, patients with LSS are more than 50 years old and often have chronic back pain with a recent onset of radicular symptoms (buttock, thigh, or calves).[7] This patient group has difficulty with prolonged standing and walking that results from increased lumbar lordosis, a narrowing spinal canal, and foraminal narrowing and nerve compression. Patients experience relief of symptoms with a flexed posture that reduces lumbar lordosis and decreases canal and foraminal narrowing. A helpful historic symptom is a positive "shopping cart sign."[35] Patients with symptomatic neurogenic claudication will lean over a shopping cart while grocery shopping to minimize lumbar lordosis and reduce compression on the cauda equina and nerve roots. Depending on the level of neural compression, there may be motor and sensory deficits, including paresthesia that may present as "heavy legs" and numbness. The diagnosis of neurogenic claudication may be distinguished from vascular claudication in that vascular claudication occurs with walking only and is not dependent on position changes to increase lumbar stenosis (Table 7).[33] Dyck and Doyle described the use of the Van Gelderen bicycle test to distinguish between vascogenic and neurogenic claudication.[36] In this test, vascular claudication is reproduced, whereas neurogenic claudication is not. Vascular claudication is usually related to atherosclerotic occlusive disease with narrowed arterial vessels. Early symptoms occur in the calves followed by the thighs and buttocks. The cramping in the legs is relieved with rest or by hanging the legs over

the side of the bed, which is thought to improve gravitational blood flow to the legs.[37] Vascular claudication presents with absent or diminished peripheral pulses. Ankle to brachial blood pressure measures may present a ratio of <1, suggestive of atherosclerotic occlusive disease and vascular claudication. A complete vascular workup is suggested in this patient group.

The resting physical examination is usually unremarkable, but in advanced cases neurologic examination may reveal sensory or motor deficits. There may be signs of forward flexion of the spine, loss of lumbar lordosis, and wide-based gait. The wide-based gait is a reaction to symptomatic loss of sensation and motor weakness.[7] Leg weakness may be elicited by lumbar extension. Weakness in legs is often characterized by reduced strength of the extensor hallucis longus, patchy areas of hypoesthesia, and absent deep-tendon reflexes. Results of the straight leg raising test are negative and distal pulses are present.

The differential diagnoses in patients with spinal stenosis includes disc disease, spondylolisthesis, sacroiliitis, and facet syndrome.[33] Many times, these conditions co-exist and complicate the definitive diagnosis. Discogenic disease may present variously if related to a herniated disc, annular tear, or internal disc derangement. Internal disc derangement or herniation may follow significant trauma or inciting event, such as a sudden lifting event, fall, or awkward twisting motion.[38] Most patients complain of deep-seated axial back pain that is aggravated with bending, lifting, and axial loading. There may be associated weakness and numbness down the leg. A heaviness or cramping sensation is present in the buttocks and legs. Patients describe difficulty arising or going to a sitting position. On examination, there may be point tenderness of the lumbar spine and paraspinal muscles. Flexion-extension, rotation, and lateral bending of the spine is usually painful. If herniation is present, especially with foraminal extension, pain is reproduced with straight leg raise and femoral stretch testing. In internal disc disruption and annular tear, no nerve root signs may be present. Mechanical compression with herniated disc may present with motor weakness, sensory loss, and reflex changes.

Table 7. Neurogenic vs. Vascular Claudication

	Neurogenic	Vascular
Pulses	Present	Absent or diminished peripheral pulses
Palliative maneuvers	Bending over or sitting	Stop walking
Provocative maneuvers	Going downhill (increased lumbar lordosis)	Going uphill (increased metabolic demand)
Van Gelderen bicycle test	No leg pain	Leg pain
Wide-based gait	Present	Absent
Romberg sign	Present	Absent
Shopping cart sign	Present	Absent
Neurologic examinations	Diminished S1 and L4 reflexes	Absent
	Weakness on muscles (extensor hallucis longus)	Absent
	Sensory changes in vibration or touch	Absent

Spondylolisthesis is the displacement of a vertebral body in relation to adjacent vertebral segments, which may lead to nerve root compression.[35] This can be suggestive of an unstable spine, and flexion and extension films are critical to determine the degree of instability. When the spondylolisthesis is greater than grade 2, surgical decompression and instrumentation may be needed to stabilize the spine, and indirect decompression by spacer or percutaneous image-guided lumbar decompression (PILD) is not indicated. Facet cysts or edema in the facet joints, as demonstrated on a T2-weighted MRI, may also suggest instability.

Other structures such as sacroiliac and facet joints may present with referred pain patterns. These patterns are nondermatomal and follow sclerotomes.[33] The pain is described as dull, deep, and poorly localized in comparison to dermatomal pain. The pathogenesis of referred pain is associated with central sensitization phenomenon.[39] An example of this is seen when hypertonic saline is injected into facet joints, resulting in the classic referred pain pattern in the back, buttocks, or legs.[40] Facet joint pain is typically in the back with referral to the groin, lateral thigh, and/or posterior proximal legs to the knees. It is important to note that most patients with LSS may also have hypertrophy of the facet joints or disc bulging and can have pain related to comorbid pathologies. The pain of facet joint disease is aggravated with extension and rotation. Diagnostic facet or medial branch blocks can be done to determine if facet joints are involved with the patient's pain.

In similar fashion, pain referred from the sacroiliac joint may be in the back, buttocks, or groin. Patients complain of pain on palpation and when recumbent over the symptomatic sacroiliac joint.[41] On examination, a positive Patrick's or Gaenslen's test result is noted. Provocative and diagnostic sacroiliac joint injection can be done to determine if the sacroiliac joint is involved with a patient's pain. Patients with hip joint pain frequently present with pain radiating to the groin,

and from the anterior thigh to the knee, which can mimic an L4 radiculopathy. In many cases, patients present with both hip and spine disorders, confounding the diagnosis. Hip-spine syndrome exists when pathologic changes in the hip lead to flexion contracture and compensatory lumbar hyperlordosis, resulting in nerve root compression and sciatica. Diagnostic hip joint injections may help to determine the painful structure.

CONSENSUS POINT 5

Neurogenic claudication needs to be differentiated from other claudication sources (Grade A, Level II-2, Consensus strong).

Stenosis Characterization

For the purpose of this recommendation, it is important to characterize LSS as symptomatic or asymptomatic, the architecture and the degree of spinal stenosis, and the number of levels involved (Table 8). This allows for the development of an algorithm supportive of patient characteristics.

ALGORITHM FOR CANDIDACY OF INTERVENTIONAL TREATMENTS

The proposed algorithm serves as a guide to help clinicians improve treatment selection for patients diagnosed with symptomatic LSS (Figure 8). It is imperative to consider some important aspects of the algorithm.

- First, assessment for instability is critical when determining candidacy for minimally invasive surgical options (beyond injection-based treatment). This is accomplished by examination of flexion and extension films when instability is suspected, along with the presence of significant spondylolisthesis. If it is unclear if instability exists, flexion/extension films represent a low-cost/low-risk means of evaluation for any spondylolisthesis. Facet joint hypertrophy with fluid collection or facet cyst formation may also suggest instability. Patients deemed unstable with a grade 2 spondylolisthesis or greater are not candidates for minimally invasive indirect decompression with an interspinous spacer (ISS).
- Second, direct decompression (PILD) should be considered if instability exists and patients are not candidates for open surgery and/or fusion, with the presence of LF hypertrophy. These are represented as the dashed green lines within the algorithm.
- Third, for those patients with central stenosis and multifactorial causes for the development of spinal stenosis (facet hypertrophy contributing to central stenosis, disc bulge, without significant ligament flavum hypertrophy), indirect decompression methods are preferred. For patients with predominant lateral recess stenosis, indirect decompression methods are preferred.

THERAPIES TO CONSIDER FOR THE TREATMENT ALGORITHM

Pharmacologic Options

There is limited peer-reviewed literature describing the treatment of symptomatic LSS with NSAIDs. Neuropathic pain medications can be helpful in improving the standing and walking intolerance for patients with symptomatic LSS. A study of the use of pregabalin in 104 patients with intermittent neurogenic claudication unresponsive to NSAIDs for at least a month demonstrated an improvement in VAS and Japanese Orthopedic Association Back Pain Evaluation Questionnaire scores at 6 weeks' follow-up.[42] It should be noted that the side effect profile of central nervous system (CNS) active drugs can be difficult to tolerate in many patients in the LSS age group.

Table 8. Characterization of Lumbar Spinal Stenosis

Clinically Relevant History	Symptomatic	Asymptomatic		
Stenosis type	Central	Lateral stenosis	Foraminal stenosis	
Stenosis level	L1–2	L2–3	L3–4	L4–5 L5–S1
Number of levels	<2	>2		
Degree of instability	Spondylolisthesis > grade 1	Flexion/extension > 3 mm translation/and or 5 degree angulation in flexion/extension	Cobb angle > 10	Osteopenia or osteoporosis
Architecture of stenosis	Ligamentum flavum hypertrophy	Facet hypertrophy	Disc bulge/protrusion	

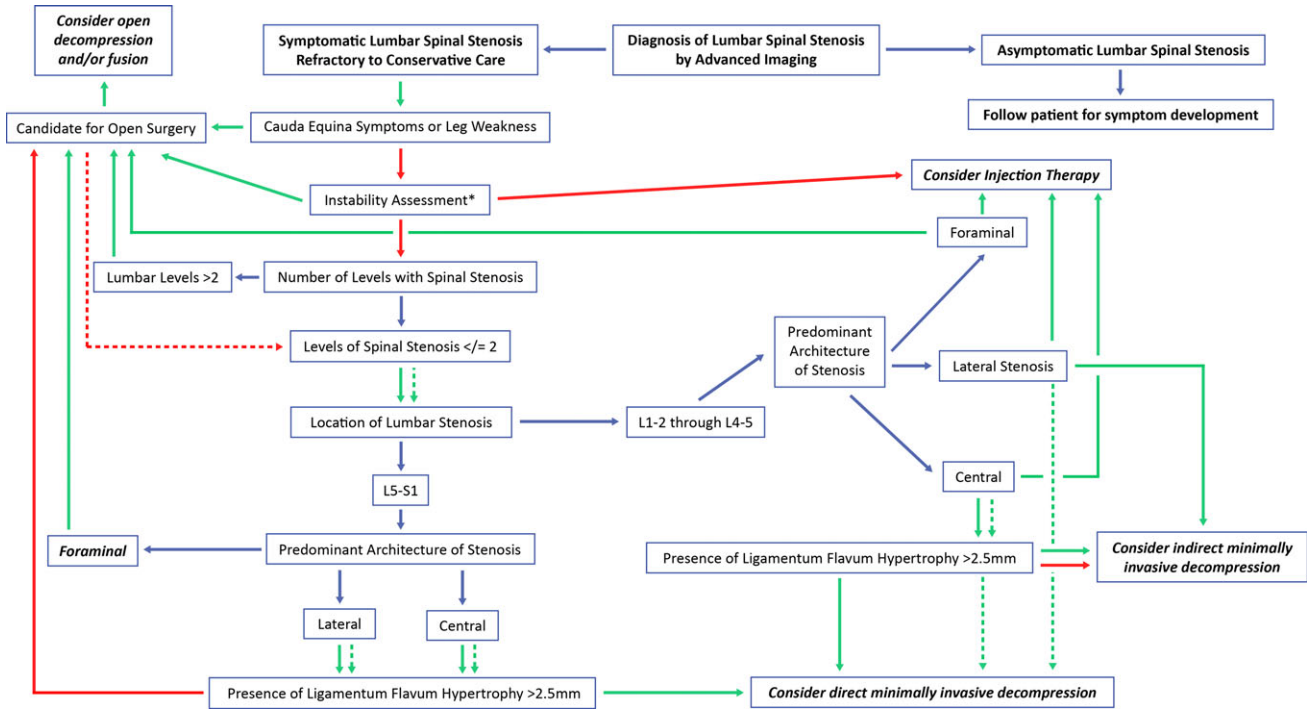

Figure 8. Algorithm for interventional treatments of lumbar spinal stenosis. Blue lines, specific components to larger diagnosis; green lines, affirmative; dashed green lines, instability exists and patients are not candidates for open surgery and/or fusion, with the presence of ligamentum flavum hypertrophy; red lines, negative; dashed red lines, instability exists and patients are not candidates for open surgery and/or fusion, without the presence of ligamentum flavum hypertrophy. *Instability in algorithms defined as spondylolisthesis greater than grade 2.

Lubelski et al. [43] demonstrated, in a retrospective review of over 1,300 patients, that the prediction of quality-of-life (QOL) improvements in patients with LSS treated with membrane-stabilizing agents (MSAs) is associated with 4 categories: need for surgery within 1 year of initiation, time of surgery after initiation, improvement in QOL based on the EuroQol 5 dimensions questionnaire (EQ-5D) QOL index, and improvement in EQ-5D score. They concluded that MSAs improved QOL for those with LSS, and the greatest improvement would be for patients who had worse QOL before treatment, were less depressed, had greater median income, and were married.

Nonoperative treatments for spinal stenosis with neurogenic claudication were recently evaluated in a Cochrane Database review.[44] From the 8,635 citations aggregated and evaluated, 56 full-text articles were evaluated and 21 trials were included, encompassing 1,851 individuals. There was low-quality evidence for opioids, no better than placebo or paracetamol. Reviewers also highlighted the low quality of evidence for prostaglandin inhibitors, and the very low quality of evidence for treatment with gabapentin and methylcobalamin for improving walking distance.

CONSENSUS POINT 6

There is low-quality evidence for using NSAIDs, neuropathic pain medications (MSAs), and opioids as monotherapy in the treatment of spinal stenosis. These therapies should be employed judiciously, balancing the patient's individual risks and benefits (Grade D, Level I, Consensus strong).

Nonpharmacologic Options

The Utility of Back Braces for the Treatment of Spinal Stenosis. Conservative measures for the treatment of LSS include rehabilitation with a focus on a flexion-based or neutral-positioned program with spinal stabilization. Back braces, particularly lumbar or lumbosacral corsets, have proven to be efficacious in providing adequate relief of symptoms and lumbar support throughout the rehabilitation process.

In 2001, a comparative study was done to evaluate the effectiveness of wearing a lumbosacral corset in symptomatic degenerative LSS.[45] The patients (n = 21) who participated in the study were evaluated for walking distance and pain score (VAS) with and without wearing the corsets. The study showed statistically significant functional improvement in walking distance and a reduction in VAS score with the use of lumbosacral corsets.

In 2009, Levendoglu et al. investigated the quantitative effects of lumbar corsets used in LSS on walking time.[46] Patients with LSS walked on a treadmill while wearing or not wearing various lumbar corsets. Symptom initiation time (SIT) and total walking time (TWT) were recorded. SIT and TWT were significantly longer for the patients with the corsets compared to those without.

Spinal orthoses provide stability, pain relief, normal spinal alignment, and balance.[47] The aim of physical therapy for patients with LSS is to strengthen abdominal and back muscles, preserve motion in the spine, and improve overall fitness. Corsets or braces can help ease this pain, thereby optimizing rehabilitation outcomes. Previous research indicates orthoses may weaken postural muscles, recommending that they be worn for a few hours per day.[48] However, a meta-analysis published in 2017 demonstrated no negative effect by the continuous use of lumbosacral orthoses for 1 to 6 months.[49]

CONSENSUS POINT 7

There is little evidence supporting the use of axial bracing for the treatment of neurogenic claudication and spinal stenosis. If instability is suspected, bracing may be helpful in the treatment of neurogenic claudication related to spinal segmental motion (Grade C, Level II, Consensus moderate).

Injection Therapies. Following noninvasive treatments in the care continuum, it is common practice for a variety of image-guided injective therapies to be prescribed. There are several decades of experience with the application of injective treatments for spinal stenosis; however, the practice has recently become a subject of some debate.[50,51] Several leading interventional pain societies have pointed out limitations of these studies with regard to the positive outcomes of several RCTs.[52] Despite this controversy, several RCTs using injective therapies for LSS exist to guide expert opinion.

Manchikanti et al. evaluated the efficacy of caudal epidural injections in 100 subjects with LSS.[53] The outcome measures were a numerical rating scale (NRS), Oswestry Disability Index (ODI), and opioid intake. Response was determined as 50% pain relief at 3 weeks postprocedure. The treatment groups evaluated lidocaine vs. lidocaine with steroid in a double-blinded clinical setting without placebo control. In this study, 54% of subjects receiving lidocaine and 62% of subjects receiving lidocaine and steroid met endpoint criteria at 3 weeks following the injections. The investigators suggested that local anesthetic injections could be of benefit while avoiding the detrimental effects of repeat steroid application.

Likewise, Manchikanti and colleagues found similar results when the same study design was used to evaluate the effects of interlaminar epidural injections for LSS comparing local anesthetic to local anesthetic plus steroid.[54] In this double-blind clinical-setting RCT of 120 subjects, 72% of local anesthetic and 74% of local anesthetic plus steroid subjects met criteria of 50% pain relief. Lee et al.[55] compared interlaminar epidural injections to bilateral transforaminal injections in 99 subjects randomly assigned to the route of delivery receiving local anesthetic and steroid. The outcome measures of NRS, Patient Satisfaction Index, and Roland 5-point pain score suggested both techniques provided significant relief in the 2-week to 4-month study period (1 to 3 injections), with bilateral transforaminal injections resulting in a significantly greater decrease in Roland 5-point pain scores. There was no placebo control group.

Smaller scale studies evaluating ESIs include the study by Koc et al.,[56] who assigned subjects to either physical therapy, interlaminar ESI, or control. At 6 months the ESI group demonstrated pain scores that were improved vs. control and equivalent vs. physical therapy. Likewise, Wilson-MacDonald et al.[57] compared epidural injection of bupivacaine and methylprednisolone to sham procedure defined as intramuscular injection of bupivacaine and methylprednisolone. There was a small positive difference in outcomes favoring ESI. In one of the first studies to address the question, Fukusaki et al.[58] compared epidural saline to mepivacaine alone and mepivacaine and steroid in 53 subjects receiving 1 to 3 injections during the study period. The steroid group demonstrated superior results to saline or mepivacaine alone, and all treatment effects had waned by the end of

the 3-month study period. Keeping in mind that the Fukusaki et al. study was performed in 1998 and was among the first to evaluate injective therapy, much less LSS, the length of effect of injective therapy was not well established at the time, and the finding that the treatment effect may be 3 months or less was relatively new information. The investigators concluded that epidural injections were not a long-term treatment.

Two RCTs have evaluated transforaminal approaches applied to LSS: the aforementioned Lee et al. study[55] comparing interlaminar ESI to transforaminal ESI (TFESI) and a 2011 study by Nam and Park.[59] Nam and Park evaluated the effect of transforaminal injection of local anesthetic alone to local anesthetic and steroid (0.5% lidocaine and 20 mg of triamcinolone). Outcome measures were the VAS and ODI. The local anesthetic and steroid group had significantly greater improvement in the outcome measures, though both groups demonstrated improvement. There was no placebo group in the study.

Friedly et al. compared lidocaine to lidocaine and glucocorticoid injection (interlaminar and transforaminal were both allowed in the study design per physician preference, as was the selection of the steroid).[51] In this 400-subject RCT, addition of glucocorticoid offered minimal benefit over local anesthetic alone in the treatment of LSS. There was no placebo group in the study.

It is clearly acknowledged that facet hypertrophy contributes to the pathology of LSS. Given that there is clearly a component of extension-based mechanical pain in patients with LSS, and given the similarities on physical examination with axial low back pain of facet joint origin, relatively little work has been done evaluating the responsiveness of LSS symptomatology with regard to the potential role of facet interventions in LSS. A recent feasibility study by Hwang et al. evaluated lumbar facet interventions as an alternative to epidural application of medication for LSS.[60] This preliminary study reported a 50% reduction of symptoms in a population with documented central canal stenosis. Building on this feasibility study, Shim and colleagues reported in a cross-over design study that facet interventions appear to be similar to ESI in reduction of symptoms of LSS.[61] This study should also be considered as preliminary data, however, due to significant design flaws, such as poor documentation of length of relief and lack of differentiation between radicular pain and neurogenic claudication.

Although the literature suggests that facet interventions for LSS may have limited utility, 1 recent study suggested that radiofrequency (RF) ablation may have utility in treating pain in patients with radiographic evidence of central and lateral spinal stenosis.[62] In this study of 127 subjects, preselected by a radiologist with musculoskeletal expertise and having confirmed evidence of central and lateral stenosis, there was a positive correlation of treatment success with RF ablation, but interestingly not with lumbar medial branch blocks. This outcome, while interesting, creates a diagnostic dilemma: How do you screen candidates for a treatment that may have success when the diagnostic tool does not identify possible candidates accurately? The investigators pointed out that although facet hypertrophy certainly has a role in the creation of LSS from an anatomic standpoint, many facet interventions have been marginally successful in treating this component of the syndrome. Given these seemingly paradoxical findings, further clarification is clearly warranted. To our knowledge, there are no studies addressing lumbar RF ablation in subjects with LSS as the primary focus of the study.

In addition to the studies comparing medications (local anesthetic with/without steroid) or type of injection (ESI vs. TFESI) with regard to efficacy for LSS, there are at least 2 studies comparing lumbar ESIs to PILD.[63,64] In both studies it is the premise of the study design that lumbar epidural spinal injection (LESI) is the de facto gold standard of treatment of LSS prior to surgical intervention for patients who have exhausted more conservative therapies. In these RCTs, LESI did demonstrate efficacy consistent with the outcomes of other RCTs mentioned earlier. Although the Brown[63] and mild® Decompression Alternative to Open Surgery (MiDAS)[64] studies were not designed to directly evaluate the effectiveness of LESI for LSS, they do independently confirm the outcomes of short-to-intermediate improvement in pain control and function consistent with previous studies. Since the primary outcome was not to evaluate ESI as a treatment, these findings theoretically confirm the efficacy of ESI without bias, as the design of the trial was to evaluate the effectiveness of PILD.

There have been several systematic reviews evaluating the efficacy of injection therapy for symptomatic LSS.[65–68] These all suggest a short- to intermediate-term benefit for the symptomatic treatment of LSS. A recent editorial suggested that the role of ESIs should be reconsidered based on glucocorticoid risk profiles noted from several sources.[68] In the editorial accompanying the Friedly et al. paper, the author suggested

that the role of ESI treatment in general should be reevaluated, while certainly suggesting that the role of glucocorticoid injection should be seriously reevaluated in the treatment algorithm.[69] While caution with transforaminal injections is certainly warranted, the statement by Andersson does ignore the data in the Friedly et al. paper in which both treatment groups saw improvement, albeit with more side effects in the steroid group.[51,69] In keeping with the data presented previously, the other systematic reviews demonstrated consistent short- to intermediate-term improvement of symptomatic LSS treated with ESI. These systematic reviews support the benefit of caudal and interlaminar injections (local anesthetic only and local anesthetic with steroid) as well as transforaminal injections of local anesthetic with or without steroid.[65–68] In the most recent systematic review, caudal/interlaminar injections received a Level 2 recommendation and transformational injection received a Level 3 recommendation for LSS symptomatology.[52]

Although it is reasonable to repeat ESI when patients have sustained pain relief and then develop recurrent pain, it is important to understand that some payer guidelines (including Medicare) now stipulate that patients should have a minimum of 3 months of pain relief and then develop recurrent pain of a similar nature before it is reasonable to proceed with additional injection therapy (Appendix S1). For patients exhibiting shorter-term relief of less than 3 months, one should not proceed with further injection therapy but rather continue down the treatment algorithm to one of the treatment options directed at decompression.

Lumbar facet interventions as treatment for LSS likely have not been vigorously investigated, as these treatments have been considered theoretically to have little benefit in the treatment of neurogenic claudication associated with central canal stenosis and/or lateral recess stenosis. Recent preliminary publications suggest this may be an area of increased study.[60,61]

CONSENSUS POINT 8

There is ample evidence to support the use of minimally invasive treatment strategies for the management of symptomatic lumbar spinal stenosis. Depending on the duration and extent of relief, these minimally invasive options can be repeated or continued to more surgical treatment solutions (Grade B, Level II-2, Consensus strong).

CONSENSUS POINT 9

When performing spinal interventional treatments, it is imperative to follow the described anticoagulation recommendations and to ensure that detection of injury can occur by either maintaining a reactive patient or the use of appropriate neurological monitoring (Grade A, Level II-2, Consensus strong).

Percutaneous Image-Guided Lumbar Decompression

PILD, as defined by the Centers for Medicare and Medicaid Services (CMS), involves noninvasive techniques to debulk the posterior elements of the spine (lamina and LF) using instrumentation in an image-guided (CT or fluoroscopy) fashion, with the assistance of contrast media to evaluate the effects of treatment on the compressed area via an epidurogram.[70] Currently, there are 2 percutaneous disposable devices in the marketplace for lumbar decompression: Totalis (Vertiflex Spine, Caralsbad, CA, U.S.A.) and mild® (Minimally Invasive Lumbar Decompression, Vertos Medical, Aliso Viejo, CA, U.S.A.). mild® is the only image-guided technique meeting the CMS definition of PILD, and as such PILD will refer to mild® for the purposes of this section. Totalis is not currently commercially available and is not included in this algorithmic discussion of available patient options.

PILD by definition treats LSS secondary to LF hypertrophy. LF hypertrophy in the studies performed to date has been defined as ligamentum thickness of >2.5 mm on MRI evaluation. While other anatomic elements beyond LF hypertrophy (facet hypertrophy, disc encroachment into the spinal canal) can clearly contribute to LSS, PILD is not designed to address these pathologies, although in clinical settings the overall reduction of spinal canal pressure from debulking the ligament has been shown to treat multifactorial etiologies. Although PILD is indicated for patients with central stenosis due to LF hypertrophy and neurogenic claudication as the presenting complaint, it is not intended to debulk lateral foramen or primary bony abnormalities. Interestingly, a majority of patients treated in the MiDAS Evidence-based Neurogenic Claudication Outcomes Research (ENCORE) study[8,64] did have comorbid foraminal stenosis, facet hypertrophy, or disc bulging, and these were actually a positive

predictor of success with a percutaneous decompression. Thus, these comorbid findings should not be considered as a contraindication to using this procedure. Levels L3–4, L4–5, and L2–3 are most commonly associated with LSS, and a recent study evaluating the incidence of LSS secondary to LF hypertrophy suggested that the L3–4 and L4–5 spinal levels most commonly developed ligamentous-based stenosis.[71]

There are 7 prospective studies and 4 retrospective studies evaluating the safety and effectiveness of PILD. The earliest work by Deer and Kapural first described the technique of PILD and evaluated the safety of the procedure involving 90 subjects in 2010.[72] Publication of this prospective trial was quickly followed by a second 2010 prospective publication by Chopko and Caraway[73] demonstrating clinical improvement as measured by the VAS, ODI, Zurich Claudication Questionnaire (ZCQ), and 12-Item Short Form (SF-12) Health Survey. In this study, 78 subjects were prospectively enrolled to undergo PILD by 1 of 14 American spine specialists in a multicenter study. Inclusion criteria were MRI evidence of LF thickness of >2.5 mm, canal sectional area of ≤100 mm^2, anterior spondylolisthesis of ≤5.0 mm, and ability to ambulate at least 10 feet before being limited by pain. In keeping with the Deer and Kapural initial study, no device-related complications were noted, and there was statistically significant improvement across all outcome measures at 6 weeks. Later in 2010, Lingreen and Grider published a 2-site retrospective evaluation of 42 consecutive patients,[74] also without any device- or procedure-related complications noted. In this study, VAS scores at 6 weeks were reduced by 40%, with 86% of patients reporting satisfaction with the results. Interestingly, even those subjects not experiencing efficacy from the procedure felt that the minimally invasive nature of PILD made it a viable option for the treatment of LSS prior to considering open surgical decompression.

Subsequently, between 2011 and 2013, several studies were published continuing to demonstrate the safety and efficacy of PILD. Chopko published a prospective single-site study with 14 subjects followed over 23 weeks that demonstrated 53% improvement in pain scores.[75] This was followed by a case series by Wong following 17 subjects over 1 year and demonstrating improvements in VAS and ODI similar to those in previous reports.[76] The Wong study also served as a detailed procedure description. Similar to the study by Wong, Mekhail and colleagues followed 58 subjects at 11 clinical sites retrospectively for 1 year, demonstrating VAS, ODI, ZCQ, and SF-12 score improvement.[77] Finally, Basu reported similar results in 27 subjects in a prospective study evaluating ODI, VAS, and ZCQ scores and patient satisfaction.[78] Taken together, these results established a track record of safety and began to demonstrate the likely effectiveness of PILD at least in the first year following decompression. Limitations of the body of literature to that point are as follows: (1) only 1 study that was not industry sponsored, and (2) lack of a comparator group.

In 2012, Brown published the first RCT involving PILD.[63] In this double-blinded study, 38 subjects were randomized to either PILD or LESI and followed for 12 weeks. Outcome measures were the ODI, ZCQ, and VAS. Similar inclusion criteria were utilized to those described for the initial Deer and Kapural studies. The ZCQ results demonstrated higher patient satisfaction with PILD vs. LESI and sustained improvement for PILD through the 12-week duration of the study.

In 2016, the 6-month and 12-month results of the MiDAS ENCORE study were published.[8,64] In this study, 302 subjects were randomized to PILD or LESI and followed for 1 year. Medication management for the 2 groups was similar, as were the other patient demographics. Subjects in the PILD group were treated initially with PILD and followed for 1 year, while subjects in the LESI group could receive up to 4 treatments per year with image-guided LESI using 80 mg of depo-methylprednisolone acetate or triamcinolone acetonide (49 mg for diabetic subjects). Patients could not receive transforaminal injections, participated in neuromodulation trials (spinal cord stimulation or intrathecal drug delivery), or undergo surgery. Outcome measures were the ODI, numeric pain rating scale, and ZCQ and were evaluated using validated minimally important change measures. The PILD group had a 58% responder rate compared to 27% for the LESI group ($P < 0.001$), with a primary safety endpoint demonstrating no difference between PILD and LESI. These results suggested that PILD was superior to LESI with no difference in safety, while subjects also experienced a durable outcome with PILD over 1 year compared with LESI. Limitations of this study and conclusions reached by the investigators included criticisms that the study was industry sponsored, that LESI and PILD were not comparable treatments, as the former is accepted as having a relatively short-term effect while the latter is designed

to be a longer lasting treatment, and lack of patient blinding. Defense of the study acknowledges the contemporary reality that LESI represents the only widely accepted nonmedical/surgical treatment for symptomatic LSS and may be performed as many as 4 times a year, and that the study was specifically requested by CMS to satisfy reimbursement requirements. Subsequently, 2-year data have been presented that demonstrate the durability of relief in patients treated with the minimally invasive lumbar decompression (MILD) procedure (results currently in press).

A single-site prospective study was performed to determine the safety and efficacy of the MILD procedure at 6 months.[78] Twenty-seven consecutive patients were identified and enrolled in the prospective study, with outcomes measures of the VAS, ODI, and ZCQ. All patients had LF > 2.5 mm, with previous failure of conservative therapy. Success was defined as: a reduction in VAS score of 2 or more points, improvement of 15 or more points on the ODI, no procedure-related complications, and did not require reoperation. A total of 44 levels were decompressed in 27 patients. Ten patients underwent a 1-level decompression, 17 underwent decompression at 2 levels. Most procedures were at L3–4, followed by L4–5. Mean LF thickness was 4.5 mm. Mean VAS score at baseline was 9.1 and improved to 3.9 at 6 months postprocedure (2-tailed *t*-test, $P < 0.0001$). This represents a 57.1% improvement in pain on average. Eighty-eight percent of the patients fulfilled the criteria of improvement of at least 2 points in the VAS score (or >30% improvement compared to baseline pain). Without the complete data set, the number of patients with at least 50% improvement (the standard for other pain care therapies) could not be determined. The ODI baseline mean score was 55.1, with improvement to a mean value of 31.1, an improvement of 24 points (2-tailed *t*-test, $P < 0.0004$), while the ZCQ score was 1.86 at 6 months (2-tailed *t*-test, $P < 0.001$). This single-center, prospective, nonrandomized, noncontrolled observational study showed both efficacy and safety.

Taken *in toto* the adverse events in the RCTs and the observational studies that comment on safety note 1 procedural hemorrhage that abated with application of Gelfoam (Pfizer, New York, NY, U.S.A.). No other incidents of hemorrhage, dural tear, or neurologic deficit were noted. Mekhail et al. compared rates of complication with PILD to those of open decompressive surgery.[77] Their 0% incidence of complications in the PILD group stands in contrast to the surgical complication rate. The interested reader is directed to that article.

CONSENSUS POINT 10

Based on the systematic review of the available literature for PILD (Table 9), the consensus committee has determined that there is sufficient support to warrant Level I evidence using the USPSTF criteria. The 2 comparative prospective studies that led to reimbursement approval by the CMS are both Level I (USPSTF criteria). All RCT evidence compares PILD to lumbar ESI and not to open decompression (Grade A, Level I, Consensus strong).

Interspinous Spacers for Indirect Lumbar Decompression

Interspinous spacers were developed as a less invasive strategy to avoid many of the risks of traditional laminectomy and eliminate the complication of postlaminectomy syndrome. The basic premise of these devices is to limit, or even block, extension at specific levels of the spine, thus minimizing the physiologic effects of acquired spinal degeneration. This "extension blocking" effect results in tightening of the hypertrophic LF and prevents it from buckling into the spinal canal. This helps to maintain a bigger central spinal and neuroforaminal canal. The concept of interspinous process devices for the treatment of LSS began in the 1950s, at which time metal plugs were inserted between the spinous processes.[80,81] The therapeutic goal of the current generation of ISS devices is to produce slight lumbar flexion at the treated level(s), thus maximizing the potential space in the spinal canal, while allowing the untreated levels to move freely.

An inherent advantage of ISS over other minimally invasive treatments discussed in this review is its versatility to potentially improve stenosis at both the central and neuroforaminal canals. Moreover, ISS use is reversible—in the event the procedure provides insufficient relief, the device can be removed with little consequence and no bearing on the patient's ability to proceed with a surgical decompression.

Although a variety of spacer-platforms have been utilized (past and present), this section will focus on the primary, stand-alone ISS on the market, the Superion® Indirect Decompression System (S-IDS) by Vertiflex, Inc.

Table 9. Systematic Review of PILD Literature

Study	Study Type	Details	U.S. Preventative Services Task Force Rating[2]
MiDAS (Benyamin et al., 2016; Staats & Benyamin, 2016)[8,64]	RCT	MILD vs. LESI with follow-up at 6 months, 1 and 2 years (in press) for the MILD arm Outcome measures: VAS, ODI, ZCQ, SF-12	Level I
Brown (2012)[63]	RCT	21 subjects randomized to MILD and 17 to LESI with VAS, ODI, and ZCQ and followed at 6 and 12 weeks. Improved satisfaction at 6 and 12 weeks for PILD vs. LESI; PILD also demonstrated improved pain and function scores vs. LESI in the 12-week period.	Level I
Deer et al. (2012)[79]	Observational, prospective	46 subjects with LSS followed prospectively at 12 weeks, 6 months, and 1 year following PILD Outcome measures: VAS, ODI, ZCQ	
Chopko & Caraway (2010)[73]	Observational, prospective	78 patients followed prospectively Outcome measures: VAS, ODI, ZCQ, SF-12	
Mekhail et al. (2012)[77]	Observational, retrospective	58 subjects with LSS followed retrospectively at 11 U.S. sites Outcome measures: VAS, ODI, ZCQ, SF-12 Results: Significant decrease in pain; physical function significantly improved by all measures	
Basu (2012)[78]	Observational, prospective	27 subjects with LSS enrolled in single site Outcome measures: ODI, ZCQ, VAS at baseline and 6 months	
Chopko (2011)[75]	Observational, prospective	14 subjects with LSS receiving MILD Outcome measures: VAS, ODI Results: Significantly improved VAS while ODI failed to improve	
Lingreen & Grider (2010)[74]	Observational, retrospective	42 subjects with LSS at 2 U.S. centers Outcome measures: VAS, patient self-reported improvement to stand and ambulate for >15 minutes pre- and post-procedure. 40% reduction in pain with 86% subjects suggesting they would recommend the PILD procedure.	
Wong (2012)[76]	Observational, retrospective	17 subjects with LSS receiving PILD Outcome measures: ODI, VAS followed 1 year Results: 70% reduction in VAS and significant improvement in ODI at 1 year	

LESI, lumbar epidural steroid injection; LSS, lumbar spinal stenosis; MiDAS, mild® Decompression Alternative to Open Surgery; MILD, minimally invasive lumbar decompression; ODI, Oswestry Disability Index; PILD, percutaneous image-guided lumbar decompression; RCT, randomized controlled trial; SF-12, Short Form 12 Health Survey; ZCQ, Zurich Claudication Questionnaire.

(Carlsbad, CA, U.S.A.). Prior to the S-IDS, the X-STOP® interspinous spacer (X-ISS) decompression system by Medtronic (Minneapolis, MN, U.S.A.) was the most commonly utilized ISS in the United States. The device was approved for use by the U.S. Food and Drug Administration (FDA) in 2005; however, Medtronic ultimately discontinued distribution in 2015 citing minimal long-term benefit and a relatively high rate of complications, which included dislodgement of the device.[82] Later that year, the S-IDS was approved by the FDA. The device was intended to rectify the deficiencies of the X-ISS (ie, device movement) and introduce a percutaneous implantation technique that could be utilized by interventional spine specialists.

The S-IDS is an H-shaped, 1-piece implant composed of titanium alloy as opposed to the X-ISS, which was a 2-piece implant composed of polyetheretherketone (PEEK) polymer. The X-ISS required an open implantation through an incision approximately 1-inch in length (per level), whereby the 2 components would be assembled at the level of the spine. In contrast, the S-IDS is delivered percutaneously, as a single piece, through a cannula, using a series of dilators to open tissues leading to the intralaminar opening. The S-IDS has superior and inferior cam lobes that rotate during deployment, so as to capture the superior and inferior spinous processes, respectively (Figure 9). The S-IDS is indicated to treat skeletally mature patients with intermittent neurogenic claudication secondary to a diagnosis of moderate degenerative LSS, with or without Grade 1 spondylolisthesis, confirmed by imaging, with evidence of thickened LF, narrowed lateral recess, and/or central canal or foraminal narrowing. The S-IDS may be implanted at 1 or 2 adjacent lumbar levels in patients in whom treatment is indicated at no more than 2 levels, from L1 to L5.[83,84]

Literature Review of Interspinous Spacers. The sentinel article establishing the efficacy of ISS for the treatment of intermittent neurogenic claudication secondary to moderate LSS was published by Zucherman et al. in *Spine* in 2005.[85] This was a multicenter, prospective, randomized trial comparing the X-ISS ($n = 100$) to nonoperative therapy ($n = 91$). At 2 years, the X-ISS

cohort improved by 45% from baseline in symptom severity score compared to 7.4% in the control group. The mean improvement in the physical function domain was 44.3% in the X-ISS cohort compared to -0.4% in the control group. Most importantly, the subjects in the X-ISS cohort had significantly better outcomes in each domain of the ZCQ. The utility of X-ISS was further supported by the publication of an observational study on 175 patients treated with this particular ISS.[86] The researchers reported clinically significant decreases in back and leg VAS scores as well as an overall reduction in VAS score. Although these publications support the utility of ISS, they failed to establish its place in the treatment algorithm compared to traditional, decompressive surgery.

In 2013, Strömqvist et al. authored perhaps the strongest publication supporting the comparability of ISS to traditional surgery.[87] The 2-year study compared the X-ISS (n = 50) to surgical decompression (n = 50). In addition to establishing noninferiority, the primary endpoint in this study was the ZCQ; the secondary endpoints were VAS scores, Short Form 36 (SF-36) scores, complications, and reoperations. The primary and secondary outcomes for both groups were significantly improved; with the exception of reoperation rate (laminectomy: 6%; X-ISS: 26%), the results were similar at all time points, with no statistically significant differences noted between the 2 treatments. The evidence contained in this publication is regarded as Level 1.

Patel et al. reported the 2-year data of the prospective, multicenter (29 sites), randomized controlled FDA Investigational Device Exemption (IDE) pivotal trial comparing the S-IDS (n = 190) to the X-ISS (n = 201), which served as the control.[84] Leg pain was the predominant complaint among the entire cohort, which decreased by 70% in both groups at 2 years. Additionally, 77% of subjects with leg pain and 68% of those with back pain reported clinically significant improvements (≥ 20 mm on the VAS) at 2 years. The study established noninferiority of the S-IDS over X-ISS (primary endpoint). Complications and/or reoperations were not statistically different between the 2 groups.

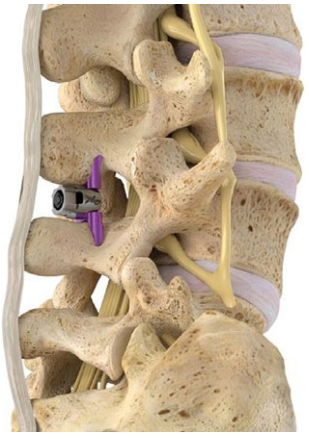

Figure 9. Fully deployed indirect decompression system at the L3 to L4 spinal level.

Lauryssen et al. published a review article in 2015 comparing the results of the IDE trial to a compilation of 19 published studies on the use of decompressive laminectomy for the treatment of LSS.[88] The article compared back and leg pain, ODI score, and ZCQ score between those in the IDE study treated with ISS and the published results for patients treated with laminectomy. The percentage improvements at 24 months uniformly favored those treated with ISS compared to baseline scores.

The 3-year data of the aforementioned IDE trial, published in 2015, favored the S-IDS over the X-ISS.[9] The primary endpoints of this study compared individual patient success based on the ZCQ, no reoperations at the index level, no implant/procedure-related complications, and no clinically significant confounding treatments. At 3 years, the proportion of S-IDS patients (52.5%) achieving the primary endpoints was significantly greater than that of X-ISS patients (38%; P = 0.023). In 2017, the 4-year data were published by Nunley et al.[89] At 4 years, 84.3% of patients (n = 89) treated with the S-IDS showed clinically significant success on at least 2 of the 3 domains of the ZCQ. Additionally, 73% of patients reported improvements in leg VAS scores over baseline and 69% in back VAS scores. At 5 years, 84% (n = 88) of patients treated with the S-IDS demonstrated clinically significant success on at least 2 of the 3 domains of the ZCQ, 80% had improvements over baseline in leg pain and 65% in back pain.[90]

In the 2005 *Spine* publication, improvements (≥15% improvement) in the ODI were noted in 65% of the subjects treated with the S-IDS.[85] Improvements in ODI score were sustained at 4 and 5 years ; 62% of subjects at 4 years and 65% at 5 years showed clinically significant improvements over baseline.[89,90] It should be noted that although the literature has focused on comparing spacer devices with similar features, there has not been a direct comparison of the S-IDS to decompression methods (percutaneous or open), merely noninferiority studies of the S-IDS compared to the X-ISS.

Potential Complications. The 2005 study by Zucherman et al.[85] resulted in an intraoperative or procedure-related complication rate of 7% for the X-ISS. These included respiratory distress, coronary ischemia, pulmonary edema, wound dehiscence, hematoma, and incisional pain. The device-related complication rate was 4%. These complications included malpositioned implant, implant migration, spinous process fracture, and increased pain at the implant level. In the Patel et al.[84] pivotal trial comparing the S-IDS to the X-ISS, the X-ISS was associated with a serious adverse event rate, classified as device or procedure related, of 9.5%; the rate of neurological complications was 2.5%.

In the Patel et al. IDE study,[83,84] the incidence of serious adverse events classified as device or procedure related was 8.4% with the S-IDS; a neurological complication rate of 3.5% was reported. At 2 years, the incidence of nonhealed spinous process fractures was 11.1% with the S-IDS and 5.0% with the X-ISS; healed spinous process fracture incidence was 5.3% with the S-IDS and 3.5% with the X-ISS. Approximately 80% of spinous process fractures were identified by the 6-week follow-up visit in each group. Of note, the investigators concluded that spinous process fractures were largely asymptomatic and had no influence on the clinical effectiveness of either device. The reoperation rate with the X-ISS ranged from 4.5% to 26% at 2 years.[86,87] At 3 years, the reoperation rate for the X-ISS was reportedly 20.3% compared to 18.8% for those treated with the S-IDS ($P = 0.77$).[9]

Cost Effectiveness. The cost associated with ISS is comparable to that of traditional surgery ($13,950)[91]; however, when one considers the percutaneous approach of spacers vs. an open decompressive laminectomy, the minimally invasive nature of the former is understandably more attractive (to physicians and patients alike) due to the lower complication rate. Obviously, conservative care carries the lowest average cost ($10,540) and the least risk; however, evidence suggests an ISS is superior to conservative care.[85]

Parker et al. compared conservative care, ISS, and laminectomy in an effort to elucidate which was more cost effective and used quality adjusted life years (QALY).[91] A Markov model simulated cost, health outcomes, and incremental cost effectiveness of the 3 treatment modalities. Although conservative care carried the lowest overall cost, it also imparted the lowest QALY (0.06 compared to 0.26 for ISS/surgery). Despite the larger up-front cost of ISS and laminectomy, they both were found to provide superior value (cost and effectiveness) compared to conservative care. These findings suggest not only that ISS is superior to conservative care from an efficacy standpoint, but also that it is more cost effective for payers. Moreover, if ISS and laminectomy are considered equally cost effective, one must choose the less invasive therapy with the lower complication rate, which is ISS.[34]

Systematic Review of Spacer Literature.

CONSENSUS POINT 11

Based on the independent systematic review of the available literature for spacers (Table 10) placed by interventional pain physicians and interventional radiologists, the consensus committee has determined that there is sufficient support to warrant Level I evidence using the USPSTF criteria. The recommendation is based on an RCT noninferiority study of 2 spacers and not comparing spacers to open decompression (Grade B, Level I, Consensus strong).

Surgical Decompression by Open Surgical Methods

When considering the open surgical options for LSS, the surgeon considers that spondylosis is the degenerative process that most often contributes to LSS. Clinical features that are commonly attributed to this include lower back pain, radicular leg pain, and neurogenic claudication. The treatment algorithm has been variable and relies heavily on physician preference, but as seen in this review there is the implementation of conservative measures first, which may lead to improvement in the symptoms. Surgical intervention is indicated in patients whose symptoms persist despite conservative measures and demonstrate surgically correctable pathologies.

For those patients having a primary complaint of radiculopathy, it is important to determine if an intervertebral disc herniation exists, as this is approached differently from LSS.[92] The pathology lies in a soft disc without other abnormality. Therefore, you would expect an underlying biomechanically stable spine without abnormality in the adjacent facet joints, LF. or bone. These patients are usually younger with a more acute disease course. For those who do not have resolution with time and conservative measures, a

Table 10. Systematic Review of Spacer Literature

Study	Study Type	Details	U.S. Preventative Services Task Force Rating[2]
Patel et al. (2015)[84]	RCT noninferiority study, Superion vs. X-STOP	391 subjects with LSS randomized to receive S-IDS spacer (experimental group) or X-ISS (control group) followed for 2 years. Outcome measures: VAS, ODI, ZCQ, back and leg pain Results: 70% decrease in leg pain in each group, 2.0 mm or greater improvement in back and leg pain (68% and 77%, respectively), and ODI improvement all similar in both groups demonstrating noninferiority	Level I

LSS, lumbar spinal stenosis; ODI, Oswestry disability index; RCT, randomized controlled trial; ZCQ, Zurich Claudication Questionnaire.

decompression surgery alone using a less invasive technique may be sufficient.

More definitive surgical procedures for LSS vary in their indication, adoption, and support by the evidence in peer-reviewed literature. The most well-known and accepted indication for surgical decompression is for the urgent treatment of cauda equina syndrome.[93] Beyond this indication, conservative measures are recommended first. It is generally accepted by surgeons that patients should undergo at least a 3-month period of consistent conservative measures. Beyond this, the literature supports surgical intervention with improvement in symptoms.[94,95] Surgeons also generally feel that early decompression of nerve compromise is recommended, as longer term compression may lead to chronic changes.[94]

Surgical decompression of compromised neural elements is meant to treat radicular leg pain and neurogenic claudication, and is not supported as a treatment of primary low back pain.[96,97] Biomechanical stability is an entity separate from compressed neural elements. In general, intervention with an instrumented fusion is geared toward the treatment of back pain, correction of a deformity, and improvement in fusion rates.[98] Those patients with dynamic instability,[99] degenerative scoliosis/kyphosis,[100] and spondylolisthesis[101] have indications for instrumented fusion. Intraoperatively, an extensive and wide decompression may warrant a fusion.[102] Revision surgery for patients with failed back surgery syndrome is generally performed in those with severe adjacent level disease, as well as in those with instability, and therefore may necessitate an instrumented fusion.[103]

In summary, surgical decompression is generally reserved for decompression of neural elements in those patients failing conservative measures, but who are sustaining neural compromise. Assessment of biomechanical stability is necessary, with instrumented fusion being performed for dynamic instability, degenerative scoliosis/kyphosis, spondylolisthesis, extensive and wide decompression, and revision surgery. The goal of surgery is to achieve stability and release of nerve compromise. Therefore, although instrumentation may improve the fusion rate, it does not necessarily improve recovery rate and pain control.[104]

CONCLUSIONS

Minimally invasive spine treatments should be used in a judicious and algorithmic fashion to treat lumbar spinal stenosis, based on the evidence of efficacy and safety in the peer-reviewed literature. An obvious next step for minimally invasive spine research will be studies with head-to-head comparison of direct and indirect modalities as well as direct/indirect modalities to open decompression; this should not be misconstrued to suggest that safety data or clinical effectiveness have not been established, merely to point out the next steps for further study. The MIST Consensus Group recommend that these procedures be used in a multimodal fashion as part of an evidence-based decision algorithm.

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CONFLICTS OF INTEREST

The MIST project was organized by the West Virginia Society for Interventional Pain Physicians in association with the American Society of Pain and Neuroscience. The board of directors accepted nominations from members and chose members based on publication, research, clinical experience, and diversity. The members were asked to disclose any conflicts of interest. A

senior editor who had no relevant conflicts of interest reviewed the paper to reconcile any perceived bias before submission.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Appendix S1. Local Coverage Determination (LCD): Lumbar Epidural Injections (L35937).

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