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Review Article

A Review of Lumbar Spinal Stenosis with Intermittent Neurogenic Claudication: Disease and Diagnosis

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Abstract

Objective. Lumbar spinal stenosis (LSS) is a degenerative spinal condition affecting nearly 50% of patients presenting with lower back pain. The goal of this review is to present and summarize the current data on how LSS presents in various populations, how it is diagnosed, and current therapeutic strategies. Properly understanding the prevalence, presentation, and treatment options for individuals suffering from LSS is critical to providing patients the best possible care. **Results.** The occurrence of LSS is associated with advanced age. In elderly patients, LSS can be challenging to identify due to the wide variety of presentation subtleties and common comorbidities such as degenerative disc disease. Recent developments in imaging techniques can be useful in accurately identifying the precise location of the spinal compression. Treatment options can range from conservative to surgical, with the latter being reserved for when patients have neurological compromise or conservative measures have failed. Once warranted, there are several surgical techniques at the physician's disposal to best treat each individual case.

Key Words: Spinal Stenosis; Claudication; Interspinous Spacer

Introduction

Lumbar spinal stenosis (LSS) was first described by Sachs and Frankel more than a century ago in work that was published in 1900 [1]; however, the clinical description of LSS was not defined until 1954 by Dutch neurosurgeon Henk Verbiest [2]. Soon thereafter, LSS was recognized as a clinical entity contributing to physical impairment. Later on, Porter and colleagues correlated back pain and weakness to narrowing of the spinal canal. Currently, the US Social Security Act recognizes spinal stenosis as a disabling condition. The regulation is written specifically for LSS and states that it results "in pseudoclaudication, established by findings on appropriate medically acceptable imaging, manifested by chronic nonradicular pain and weakness, and resulting in inability to ambulate effectively" [3].

Degenerative LSS is a progressive condition, and it may persist for decades without any symptoms. Case reports from the 1970s and 1980s demonstrated successful surgical treatments based on subjective assessment by surgeons. In the early 1990s, Johnsson, Rosén, and Udén followed 32 untreated patients with spinal stenosis with

mean patient age of 60 years for a mean period of 49 months and described the natural course of LSS, prognosis, and treatment [4]. Based on the study, no significant change in symptoms was reported in the majority of patients (70%), and 15% of patients exhibited some deterioration. Concluding that observation is a reasonable treatment for LSS, the investigators found that significant neurologic deterioration is rare [4,5].

The first successful spinal fusion surgery was performed in 1911 for prevention of progressive deformity secondary to Pott's disease, that is, tuberculosis of the spine. That was the beginning of understanding of spinal biomechanics and fixation devices for the spinal surgery armamentarium. Despite the sophisticated developments, the incidence of residual or recurrent postoperative back pain remains high [6]. Surgery targets the symptom of pain rather than its root cause and does not retard disc degeneration or stimulate disc regeneration. In contrast, it increases the disc degeneration at adjacent segments [7]. The literature to date has focused primarily on various surgical treatments, although nonsurgical treatments such as epidural steroid injections are commonly considered options based on the clinical presentation of symptomatic LSS. The efficacy of the surgical intervention in the long term has been challenged, and the procedures are linked to increased risks of morbidity, especially in an elderly population. Also, surgical procedures are found to have a larger financial burden, where the costs are estimated to exceed $100 billion per year due to reduced productivity [8].

The effectiveness of the treatment for LSS depends on the accuracy of the diagnosis, which can be challenging. Combining patient history and physical examination with imaging test results and symptoms is at the basis of a proper diagnosis. No universal gold standard for LSS diagnosis has been established, and therefore the impressions of expert clinicians often lie at the basis of a clinical diagnosis [9].

The chronic symptoms of LSS are often missed or misdiagnosed. An inadequate or normal physical examination that may show no abnormalities coupled with inability to make a proper clinical judgment can contribute to difficulty in making the correct diagnosis. In addition, the appropriate interpretation of patients' symptoms, obtaining the patients' history, and combining this with specific attributes from the physical examination are critical. To prevent misdiagnoses at the level of primary care physicians, a clinical support tool may be of assistance to correctly identify LSS patients [10]. Such a diagnostic tool was reported by Konno et al., where a clinical prediction was made by assigning a risk score to a patient's history, physical examination scores, and imaging results, and then comparing that with the diagnosis of an experienced specialist [11]. In this multicenter prospective study, 468 patients exhibiting pain and numbness of the lower legs were seen by 104 experienced orthopedic physicians in 22 clinics and 50 hospitals. Based on this study, the sensitivity of the tool was estimated at 93%, the specificity at 72%, and the overall prevalence of LSS was 47% in a population consisting of patients with a mean age (range) of 64 (20–96) years, and where 46% were male [11].

The overall goal of this review is to present a thorough overview of LSS, including prevalence, clinical presentation, accurate diagnosis, and current treatment options.

Prevalence of Lumbar Spinal Stenosis

LSS is one of the most commonly diagnosed spinal disorders and is found to severely affect quality of life. It often leads to a surgical intervention in older patients. In the United States, 135.5–137.5 persons per 100,000 Medicare beneficiaries underwent lumbar stenosis surgery between 2002 and 2007. The costs of these surgical interventions led to a socioeconomic burden of $1.65 billion in hospital bills alone [12]. Early studies by De Villiers and Booysen reported a prevalence of LSS of 6% of 850 lumbar myelograms [13], and later Fanuele et al. described a prevalence of 13.1% in 17,744 patients under evaluation at multiple spine centers across the United States [14]. The Framingham Study, a cross-sectional observational study, was set up to determine the prevalence of congenital and acquired LSS in 3,529 patients [15]. In this study, a distinction was made between relative (i.e., a threshold measurement of the cross-sectional diameter of the canal of $\leq$12 mm) and absolute (i.e., a threshold measurement of $\leq$10 mm) LSS. Prevalence rates of 4.7% relative LSS and 2.6% absolute LSS were found in the congenital group. These percentages were 22.5% and 7.3% in the acquired LSS group, respectively. It was found in this study that the prevalence increased with age, as in the group with patients in the age range 60–69, the relative and absolute prevalence were 47.2% and 19.4%, respectively [15]. In Japan, a population-based study with 2,666 patients also found that the prevalence of LSS increased with age [16]. The prevalence was estimated at 1.7% in female patients and 2.2% in male patients aged 40–49. In the age group 70–79, the prevalence was estimated at 11.2% and 10.3% for female and male patients, respectively [16]. Another study in Japan with 1,009 patients in a cohort resembling the general population estimated the prevalence of symptomatic LSS at 10% [17]. In the LAIDBack Study, the prevalence of LSS using imaging results in 148 patients without current low back pain or sciatica was studied [18]. The occurrence of mild stenosis was defined as a narrowing of the central canal cross-sectional area by one-third or less, moderate at a narrowing between one-third and two-thirds, and severe was defined as a stenosis larger than two-thirds. This study reported a mild stenosis in 21–30%, moderate stenosis in 6%, and severe stenosis at 7% in a population aged 55 years and older [18]. The occurrence of radiographic LSS in patients without any lower back pain or other symptoms has been reported

before in the literature. For instance, Jensen et al. reported 29% radiographic abnormalities in a population of 98 asymptomatic patients [19]. Thus, these findings stress the importance of the patients' medical history and physical examinations in accordance with the radiographical results in correctly diagnosing LSS [15,18–20].

Clinical Presentation

To describe the diagnosis and treatment options for LSS, description of the clinical presentation is warranted. LSS is defined by any type of narrowing of the spinal canal, nerve root canals, or intervertebral foramina [21]. LSS can be classified based on the cause of the stenosis, (i.e., congenital or acquired) or based on the anatomic location (i.e., central, foraminal, or lateral) [9,21]. Central stenosis occurs when the spinal canal and dural sac are affected, foraminal stenosis occurs when the spinal foramina is affected, and lateral stenosis occurs when the lateral recess is affected [22,23]. It is thought that the changes due to central stenosis can lead to some degree of lateral stenosis, whereas lateral stenosis can occur autonomously [9]. Acquired LSS is commonly associated with aging and progressive degenerative processes of the spine [15]. Intermittent neurogenic claudication (INC), also referred to as pseudoclaudication, is the most common clinical presentation of LSS, manifested as back pain, leg pain, and weakness that significantly compromises the ability to ambulate [24]. Patients with INC have normal peripheral pulses and vascular studies, whereas patients with peripheral vascular claudication present the following: abnormal peripheral pulses and vascular studies, leg pain that is more severe than back pain after walking some distance, and pain relief quickly with rest.

The clinical presentation of LSS is the most frequent indication for spinal surgery in patients older than age 65 years. The clinical symptoms, history, and physical examination may help to make a diagnosis of the clinical syndrome of LSS. The typical clinical presentation of LSS entails the absence of pain when seated, improvement of symptoms on bending forward, and a wide-based gait. This clinical presentation is the most useful finding for a clinical diagnosis. The flexion of the lumbar spine (e.g., when using a bicycle or shopping cart) improving symptoms occurs because extension closes the spinal canal, thereby worsening the symptoms. This can be prevented directly by decompression laminectomy surgery or indirectly by interspinous spacers.

As discussed in the *Prevalence of Lumbar Spinal Stenosis* section, the likelihood of the clinical syndrome of LSS increases with age. Usually, persons aged 60 years or younger are less likely to have the clinical syndrome of LSS. Concurrent spinal conditions such as facet osteoarthritis and degenerative disc disease increase the likelihood of the clinical syndrome of LSS. In addition, congenital short pedicles can result in stenosis at an earlier age. A patient with a mechanically unstable spine typically presents with mechanical low back pain either with or without radiculopathy. In the treatment of lumbar stenosis with spondylolisthesis, it is pivotal to determine whether the spondylolisthesis is stable or unstable and whether a surgical intervention would render a stable spondylolisthesis unstable [25].

Patients with LSS often experience chronic back pain due to degenerative disease of the spinal structures. Degenerative disc disease (DDD) can be the initial process in the cascade of events. As the diffusion of nutrition decreases due to degeneration of the endplates, it promotes the degeneration of discs, contributing to decreased disc heights, altering the biomechanics of the spine, and leading to facet arthropathy, osteophytes, spondylosis, and degenerative spinal stenosis. As the condition progresses, the instability of the spine due to spondylolisthesis and degenerative scoliosis becomes inevitable [26].

Diagnosis and Evaluation of Lumbar Spinal Stenosis

There are no widely accepted gold standard diagnosis criteria for LSS, but the presence of imaging showing narrowing of the spinal neural structure is a necessary finding. Confirmatory diagnosis typically consists of assessing history, physical examination, and radiographic images. History should be consistent with pain while standing or walking that is relieved by forward flexion or sitting. Typical clinical features include lower extremity pain with equivocal straight leg raise testing and softs signs of radiculopathy. In many cases, the physical examination is normal while sitting, including reflexes, strength, and sensation. Imaging showing disc bulging, loss of disc height, facet joint arthropathy, osteophyte formation, and ligamentum flavum hypertrophy can lead to narrowing of the spinal canal. With the extension of the spine, the laminar edges of adjacent vertebral bodies overlap with relaxation and inward buckling of the ligamentum flavum, along with the movement of the superior facets in a rostral-anterior direction. Neurogenic claudication occurs as a result of ischemia (due to venous congestion and diminished arterial blood flow) or mechanical compression of nerve roots and is defined by pain or radiculopathy in the buttocks and/or lower extremities that worsens with walking and improves with forward bending or sitting. Walking may additionally exacerbate symptoms, as the demand for increase in oxygen in the spinal nerve roots may exceed the available blood flow [27].

Spinal canal narrowing might also be exacerbated by the shifting of one vertebral body anteriorly or posteriorly, relative to the adjacent vertebral body. This condition is known as spondylolisthesis. The lumbosacral joint is exposed to extensive anterior-directed shear forces. The paired facet joints, pars interarticularis, and

intervertebral discs are the main anatomical structures that are subjected to these forces. Degenerative spondylolisthesis occurs due to the aging process. It is associated with the occurrence of marked facet joint arthritis with rotatory vertebral slip and occurs most frequently at L4-5, although it can be seen at other levels. Isthmic spondylolisthesis occurs due to a defect in the pars interarticularis (whether congenital or acquired) and occurs most often at L5-S1 [28]. In isthmic spondylolisthesis, the intervertebral disc preserves the stability of the spinal segment. Once disc degeneration occurs, the main source of stability is lost and the vertebral slip increases. This is an important differentiating factor when determining treatment options for lumbar spinal stenosis.

Classification of spondylolisthesis is based on the degree of shifting of one vertebral body anteriorly or posteriorly relative to an adjacent vertebral body in the spine. Grade 1 is shifting of <25%, Grade 2 is 25–50%, Grade 3 is 50–75%, Grade 4 is 75–100%, and Grade 5 (spondyloptosis) is >100% [29].

Generally, the initial study utilized for the evaluation of back pain is the x-ray. X-rays are inexpensive and readily available. Moreover, the vertebrae can be assessed for changes in disc height loss, the vacuum disc phenomenon, vertebral alignment, and osteophyte formation. Assessment of sagittal and coronal vertebral alignment is important, as imbalances may rule out certain treatment options. Dynamic flexion and extension radiographs may be used to assess for spinal instability. Implementation of "Scotty Dog" views can help to evaluate for pars interarticularis defects in order to distinguish between degenerative and isthmic spondylolisthesis. However, radiographs are limited in their ability to evaluate soft tissue, discs, and nerves, making the identification of spinal stenosis difficult utilizing x-ray alone.

To image bony anatomy, computed tomography (CT) is the best choice [30]. It can be used to diagnose disc herniation and spinal stenosis. However, it does not reliably depict nerve root impingement and has the downside of radiation exposure. Therefore, it is not the first choice for the imaging of spinal stenosis. Another alternative can be CT myelogram. Compared with normal CT, the contrast in the subarachnoid space increases the visibility of neural structures. Therefore, it is comparable with magnetic resonance imaging (MRI) for neural impingement and stenosis. However, the patient is still exposed to radiation, it involves a lumbar puncture, and contrast medium is required.

MRI does not require ionizing radiation and is noninvasive. It has a high sensitivity in diagnosing stenosis and has a high soft tissue contrast. It is the best modality to evaluate disc pathology and stenosis, as it depicts spinal cord, nerve roots, and bone marrow abnormalities [31]. Before obtaining an MRI, a history should be taken regarding implantable hardware or devices, and an evaluation of the safety of the metal in place should be undertaken.

A spinal midsagittal diameter of the dural sac that is <10 mm as measured on MRI is considered spinal stenosis. The neuroforamen is considered stenotic when the anterior-posterior diameter is <3 mm on sagittal imaging [32].

When there is a normal diameter of the bony canal, separate evaluation of the dural sac is needed, such as in examples of epidural lipmatosis. A central canal spinal stenosis can occur in the area under the facet joints or in the neural foraminal. The most common type of lumbar spinal stenosis is acquired degenerative spinal stenosis. It is often due to a combination of factors: disc bulging or herniation, hypertrophy or in-folding of the ligamentum flavum, and hypertrophy of osteoarthritic facet joints. The altered biomechanics between the affected spinal structures are important factors to the development of stenosis over time [33]. Descriptive signs for stenosis may be the obliterated CSF space and/or deformity of the spinal cord [30].

Schizas et al. describes a grading system for spinal stenosis based on dural sac morphology. The dural sac was imaged on T2 axial MRI based on the rootlet:cerebrospinal fluid ratio. The grading was applied to MRI images of 95 subjects divided into three groups: 37 patients with symptomatic lumbar spinal stenosis who were surgically treated, 31 patients with symptomatic lumbar spinal stenosis who were treated with conservative therapies, and 27 low back pain patients. Patients with grades that did not show cerebrospinal fluid were more likely to fail conservative therapies [34].

Physical Exam Findings in Spinal Stenosis

The physical examination, according to Thomas et al. from 2003, should start with simple observation, beginning with your differential diagnosis in mind [35]. A detailed history and examination are typically distinct from that of a herniated disc. Patients with LSS will most typically have a kyphotic standing posture to minimize symptoms, which is not seen in patients with a herniated disc. This kyphotic standing posture is a result of straightening or reversal of the normal lumbar lordotic curvatures. It occurs when lumbar flexion increases the cross-sectional area of the intervertebral foramen, vertebral canal, and lateral recess.

One of the most important disease states to differentiate is between neurogenic and vascular claudication. Patients with spinal stenosis typically will report pain that is improved with flexion, sitting, or leaning forward. The pain is triggered by standing or extension of the spine and walking. Typically, the pain is above the knees, and walking down stairs is worse than walking up. A patient with vascular claudication will typically have symptoms that are exacerbated by walking but are relieved by standing. Patient symptoms are typically in the calf and lower legs. Evaluation of pedal pulses should be evaluated, as the symptoms of neurogenic vs vascular

claudication can be similar, and both disease processes may be present in the same patient. Therefore, a vascular-focused physical examination including peripheral pulses should be performed on each patient with a suspected presentation of spinal stenosis.

Another important differentiation should be made between typical spinal stenosis vs disc herniation. This includes age of onset, which is typically >50 years in the stenosis patient, as opposed to <50 in disc herniation patients. The onset is usually insidious in the stenosis patient, as opposed to sudden in the disc herniation population. Sitting is typically better with stenosis, whereas sitting often exacerbates pain in a patient with a disc herniation. Patients suffering from a disc herniation often have focal motor weakness, dural tension signs, and focal muscle strength deficits. These findings are less common in stenosis patients.

Range of motion of the lumbar spine should be assessed in the sagittal, transverse, and coronal planes. In patients with degenerative changes, stiffness and rigidity may be expected. Additional points of physical examination should be to exclude other factors that may be contributing to or complicating the stenosis picture. A detailed examination of the hips and sacroiliac joints with Patrick's maneuver and internal and external rotations of the hip should be assessed to ensure that that there are no other signs of other conditions that may contribute to the patient's presentation. Neurological examination may not be conclusive in the early stage of the disease. Several investigations should be conducted. The sensory examination should include light touch, pinprick, and vibration. As idiopathic peripheral neuropathy presents with similar distal symptoms, the dermatomal vs stocking pattern sensory changes should be assessed. In one-third of patients with LSS, motor weakness is present due to the L5 myotome being affected. Hall et al. found a decrease in or absence of Achilles reflexes in 43% of patients and a decrease in or absence of patellar reflexes in 18% of patients [36]. Hyperactivity in the reflexes may indicate stenosis in the cervical spine and should also be evaluated. Along with the supine straight leg raise and slump test, the femoral stretch test should also be evaluated and performed in patients lying on their side.

The Modified Romberg Test

The modified Romberg Test of Standing Balance on Firm. The Romberg test or Romberg sign is used to assess balance in patients. Patients with spinal stenosis may demonstrate difficulties with balance, especially if patients have peripheral neuropathy and other comorbidities that may impact their overall clinical picture [37,38].

Oswestry Disability Index

The Oswestry Disability Index Questionnaire is utilized to evaluate, as a self-assessment, the degree of disability the patient feels due to their issues. Evaluations of the degree of spinal canal stenosis and the relationship between the patient's perceived disability level have been studied as well [22]. Sirvanci et al. concluded that lumbar spinal stenosis remains a clinical radiologic syndrome. The clinical picture and MRI findings should be evaluated before making a decision to perform surgery. The patient's MRI should be used to determine the levels of decompression. However, Sirvanci et al. showed no correlation of central lateral recess stenosis vs Oswestry Disability Index [22].

Zurich Claudication Questionnaire

The Zurich Claudication Questionnaire was developed by Stucki et al. [39]. The claudication questionnaire is a disease-specific self-reported outcome instrument that was utilized for further treatment, which quantifies severity of symptoms, physical functioning characteristics, and the patient's satisfaction after treatment. It was designed to complement existing generic measures of lumbar spinal disability and health status specifically for patients with lumbar spinal stenosis. An increased score shows worsening disability.

The Japanese Orthopedic Association Back Pain Evaluation Questionnaire

The Japanese Orthopedic Association (JOA) updated the previous JOA score from 1986 and provided new outcome measures in 2007 [40–43]. The evaluation criteria for the evaluation were based on psychological, biological, and anatomical outcome measures resulting from the original Japanese Orthopedic Association score for low back pain. It was based on assessing the limitations of the original JOA scoring system developed in 1986. The new JOA scoring system contains a self-administered questionnaire and is a more accurate outcome measure for evaluating patients with low back pain.

Anterior Spine vs Posterior Elements

The biomechanics of the spine depend on the pattern of stress on the vertebral bodies, intervertebral discs, and ligamentous structures during flexion, extension, rotations, and bending. Each vertebra consists of three columns: the anterior column, middle column, and posterior column. The anterior column carries the axial load and resists extension. It consists of the anterior longitudinal ligament, anterior two-thirds of the vertebral body, and annulus fibrosus. The middle column ensures resistance to flexion and carries a part of the axial load. It consists of the posterior longitudinal ligament, posterior third of the vertebral bodies, annulus fibrosis, and nucleus pulposus. The posterior column ensures resistance to flexion and provides stability during rotation and lateral bending. It consists of the pedicles, facet joints, ligamentum flavum, interspinous ligaments, and supraspinous ligaments [26].

The anterior spine consists of the vertebral bodies and intervertebral discs protected by strong annulus fibrosus and cartilaginous endplates above and below. The intervertebral discs (IVDs) are the largest avascular structures in the body, and the nutrition of the discs is by diffusion from endplates. Progressive degenerative process may involve endplates, further decreasing nutrition and enhancing dehydrogenation of discs, and eventually triggering the most common progressive disability, degenerative disc disease. Even though the etiology of DDD is poorly understood, it is widely accepted that the aging process promotes dehydration by decreasing the water content of the IVDs. Genetic factors based on twin studies and mice knockout studies, along with environmental factors including overuse injuries, falls, smoking, and obesity, have been identified as contributing factors to DDD [44].

The presence of a stenotic spinal canal on MRI is not diagnostic of LSS due to poor correlation to symptomatology. There is no significant correlation between the area of the dural sac and clinical symptoms of spinal stenosis. Clinical diagnosis becomes more challenging due to overlapping symptomatology in degenerative LSS, peripheral arterial disease (PAD), and peripheral neuropathy. Leg pain and back pain seem to be the primary symptoms of LSS; however, pain and numbness in the leg are also reported as primary symptoms of LSS [45,46]. Weakness of the lower extremities, sensory changes, areflexia, and bladder or bowel incontinence are symptoms of advanced LSS. Therefore, difficulties in walking and deformity of the lower extremities can be results of the neurological abnormality effects of LSS [47].

Van Akkerveeken recommended primary and secondary classifications of LSS on the basis of etiology [48]. Primary stenosis is caused by congenital malformations or defects in postnatal development, with an incidence of only 9%. Secondary LSS is acquired and is a result of degenerative changes, spondylolisthesis, lumbar intervertebral disc herniation, or a combination of these factors. LSS is a result of a dynamic and structural component. The extension of the spine may cause posterior protrusion of the intervertebral discs and bulging of the ligamentum flavum. This results in a narrowing of the central and lateral canals. In a normal spine, this reduction is 9%, whereas in LSS this can increase to 67%. When there is a greater structural narrowing, the relative narrowing during extension will also be greater. Axial loading may also cause narrowing of the spinal canal. Schonstrom et al. found a slightly more pronounced effect on the cross-sectional area of the spinal canal with loading than with spinal extension [49].

Treatment Options

Lifestyle Modification

Improvements and alterations in lifestyle such as regular exercise, core strengthening, balanced diet, and maintaining ideal body weight have been often recommended for overall spinal health. There have not yet been any clinical trials that support this, however. The natural progression of LSS begins slowly with minimal symptoms, followed by a steep increase as critical stenosis is reached. Once LSS symptoms are noticed, it is often too late for lifestyle modifications to be significantly effective.

Epidural Injections (with/Without Steroids)

Targeted injection of local anesthetic with or without corticosteroids has been widely utilized in the treatment of neurogenic claudication due to LSS. When conservative treatments have been exhausted, patients are given the option of interventional pain management, often in the form of spinal epidural injections before surgery. Injected local anesthetics are often combined with corticosteroids to provide pain relief by reduction of local inflammation and ischemia, which are caused by the stenosis. This practice has been accepted as an alternative to more invasive surgical decompression or to help delay surgery by providing short- to medium-term pain relief. The North American Spine Society (NASS) has developed a set of evidence-based guidelines for the diagnosis and treatment of lumbar spinal stenosis from their Degenerative Lumbar Spinal Stenosis Work Group. There is a Grade B recommendation (with suggestion of a Level I study and additional Level II or III studies) for the application of interlaminar epidural steroid injections to provide short-term (two weeks to six months) relief of neurogenic claudication. The long-term efficacy of interlaminar epidural steroid injections was considered controversial. Transforaminal and caudal approaches obtained a Grade C recommendation in providing medium-term (three to 36 months) pain relief. Additional evidence for spinal epidural injections to treat LSS came from two randomized controlled trials by Manchikanti et al. showing statistically significant pain relief and improvement of disability in patients with LSS undergoing both interlaminar and caudal epidural injections [50–52]. Conflicting data were later published in the *New England Journal of Medicine* suggesting minimal benefit of epidural steroids compared with local anesthetics alone [53]. This article was highlighted for its controversial results and was criticized for study design, inclusion criteria, data analysis, and conclusions, which led to confusion among patients and providers regarding epidural injections for spinal stenosis. Additional systematic reviews of the literature have shown modest benefits in both short- and long-term pain relief for epidural injections in treating LSS. A comprehensive review of 10 studies that included 1,010 subjects indicated a minimal difference between the use of injected epidural steroids vs local anesthetic only.

Physiotherapy and Rehabilitation

Multidisciplinary rehabilitation can be effective for mild to moderate LSS. The efficacy of physical therapy and manipulation may vary widely due to inconsistent patient participation [9]. Often this treatment modality is

overlooked entirely as LSS patients seek more interventional treatment options that offer quicker results or less labor-intensive options such as electric scooters. The therapeutic role for rehabilitation in treating chronic back pain and LSS has been validated. In the older LSS patient population, multidisciplinary treatments such as strength and endurance training, flexibility exercises, lifestyle modification, and environment modifications have shown positive results. The NASS has developed a set of evidence-based guidelines for the diagnosis and treatment of lumbar spinal stenosis from their Degenerative Lumbar Spinal Stenosis Work Group [31]. Currently there is insufficient evidence to support the use of physical therapy/exercise or manipulation treatment for spinal stenosis. Interestingly, from a secondary analysis of the cohort in the Spine Patient Outcomes Research Trial (SPORT), there was a positive association between physical therapy and long-term outcomes of LSS patients [54]. Specifically, subjects in the SPORT trial receiving physical therapy within the first six weeks after enrollment noted better self-rating of improvement, improved physical functioning, and reduced likelihood of surgery at one-year follow-up.

Medication Therapy

Medication therapy for spinal stenosis symptoms has primarily fallen under the same guidelines as those for treating chronic low back pain. Although the pathophysiology of neurogenic claudication is distinctly different from spondylosis or lumbar radiculopathy, the medications used to treat include systemic corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), anticonvulsants, antidepressants, muscle relaxers, and opioids. Short-term use of systemic corticosteroids for acute radiculopathy has been studied, and their role in chronic neurogenic claudication has not been established. Long-term use of NSAIDs has been noted to cause gastrointestinal and cardiovascular events. Furthermore, for chronic back pain with radicular symptoms, there was no significant difference between NSAIDs and placebo [12, 55]. In the same systematic review by Chou, only a few trials showed small improvements in pain scores with the use of gabapentin and topiramate. The off-label use of gabapentinoids for chronic neuropathic pain has steadily increased since the introduction of gabapentin in 1993, followed by pregabalin in 2004. Both drugs are analogs of the gamma-aminobutyric acid (GABA) molecule and share pharmacokinetic/pharmacodynamic properties. By targeting the alpha-2-delta subunit of the voltage-gated calcium channel, they can alter the downstream neurotransmitter release, which gives them the proposed antinociceptive properties. Pregabalin is currently listed as a Schedule V drug by the Drug Enforcement Agency; there is an ominous global trend of misuse and abuse of gabapentinoids [56]. In the midst of the prescription opioid epidemic, misuse and abuse of noncontrolled substances such as gabapentin and less controlled substances such as pregabalin have surged, leading to significant risk to patients. Gabapentinoids can be used to potentiate the effects of opioids, and the supratherapeutic use of gabapentin among patients with substance use disorder (SUD) can lead to a 60% increase in the odds of opioid-related death compared with opioids alone [57]. Currently there is insufficient evidence to support the use of pharmacological treatment for spinal stenosis based on the NASS guidelines [31].

Surgical Treatment

Surgery for lumbar spinal stenosis is typically performed when nonsurgical treatment, such as those listed previously, does not sufficiently relieve a patient's symptoms. It is the most common reason for spinal surgery among patients aged 65 years or older [58]. The objective of the surgery is to increase the cross-sectional area of the spinal canal at the level of the stenosis, in an effort to decrease the pressure on the affected nerves. In most cases, the surgery is considered elective, as its purpose is to alleviate symptoms and improve function, rather than prevent neurologic impairment. Exceptions include patients who are myelopathic or have symptoms of cauda equina and therefore require surgery to prevent neurologic decline.

In a study of patients with lumber spinal stenosis who originally opted for nonsurgical treatment, approximately 30% subsequently requested surgery [59, 60]. However, a recent systematic review of 26 published studies, including five randomized controlled trials, was unable to conclude whether lumbar spinal stenosis is best treated using a surgical or a more conservative approach, primarily due to a lack of well-designed research [61].

Patient characteristics predictive of a good clinical outcome following surgery for spinal stenosis vary significantly between studies [62–64]. However, a systematic review of 21 studies reported that negative predictors included depression, a concomitant disorder influencing walking capacity, cardiovascular comorbidity, and scoliosis [65]. Smoking also seems to be associated with negative surgical outcomes [66]. In contrast, positive predictors were male gender, younger age, better walking ability, better self-rated health, less comorbidity, and more pronounced canal stenosis [65].

Decompressive Laminectomy Without Fusion

Decompressive laminectomy without fusion is considered the gold standard surgical procedure for patients with lumbar spinal stenosis in the absence of other complicating spine pathology that is refractive to conservative treatment [67]. With the patient in a prone position, imaging is used to guide the exposure and partial removal of the lamina, spinous process, facet joints, and soft tissue. Preservation of at least 50% of each facet joint and sufficient pars is necessary to avoid iatrogenic instability [68].

An updated, evidence-based clinical guideline for the diagnosis and treatment of lumbar spinal stenosis found sufficient evidence to recommend surgical intervention for moderate to severe lumbar stenosis following failure of nonsurgical treatment [31]. The SPORT, a multicenter randomized controlled trial (N = 289) with an observational cohort (N = 365) that evaluated surgical vs nonoperative treatment of stenosis, reported similar results at up to four years of follow-up. Patients who were treated surgically had significantly greater improvement in pain and function than did patients who were treated more conservatively [69, 70]. However, long-term analysis of patients in the randomized trial suggested diminishing benefits of surgery between four and eight years of follow-up, whereas outcomes for the observational cohort remained stable [71].

Although a more recent systematic review and meta-analysis of nine randomized clinical trials that studied the effectiveness of conservative vs surgical treatment for lumbar spinal stenosis found that the latter group had better clinical outcomes at one year, they also had a higher complication rate throughout the follow-up period. Further, there was a wide range of surgical and conservative methods used among the studies, and the authors were unable to conclusively recommend one approach over another [72].

Another challenge regarding the surgical treatment of spinal stenosis relates to whether single-level or multilevel laminectomy is more appropriate for multilevel disease. Two recent studies have suggested that single-level surgery is the preferred procedure for such patients. More specifically, in a prospective, multicenter cohort study of 141 patients, 23% of whom underwent single-level laminotomy and 77% of whom underwent multilevel decompression, the latter group was associated with significantly less favorable stenosis symptoms and function score, with no significant difference with respect to all other outcomes of interest between the two groups [73]. Similarly, a retrospective study of 114 patients by Adilay and Guclu found that those undergoing single-level laminectomy (N = 48) experienced better recovery, in terms of disability score, pain score, and walking duration, than did those undergoing multilevel laminectomy (N = 64) [74]. The authors also reported that complications and postoperative spondylolisthesis were higher in the latter group.

Other Decompression Techniques

Because of the challenges associated with decompressive laminectomy, there is interest in developing minimally invasive lumbar decompression techniques, including uni- or bilateral laminotomy and spinous process–splitting laminectomy. Such procedures are typically performed through a small incision using an endoscope or microscope, permitting preservation of soft tissue and bony anatomy. They have been the focus of small, uncontrolled, and generally single-center studies. For example, two different randomized trials comparing micro-endoscopic decompressive laminectomy with conventional open laminectomy found that the former was associated with less operative blood loss, shorter hospital stay and time to mobilization, lower likelihood of needing opioids for postoperative pain, less muscle destruction, and less low back and leg pain at final follow-up [75, 76]. However, data regarding long-term efficacy are limited.

Even in patients with lumbar spinal stenosis (LSS) associated with stable low-grade degenerative spondylolisthesis, lower reoperation and fusion rates, less progression of listhetic slip, and greater patient satisfaction were seen in patients undergoing minimally invasive decompression compared with those undergoing open surgery [77].

Decompressive Laminectomy with Fusion

Single- or multilevel decompressive laminectomy with fusion is typically reserved for patients whose stenosis is exacerbated by spondylolisthesis, a condition where a vertebral body slips anteriorly or posteriorly relative to the adjacent vertebral body. However, a recent prospective cohort study (N = 83) found that patients with lumbar stenosis and degenerative spondylolisthesis who were treated using spinal process osteotomy without fusion experienced significant postoperative improvement in pain and disability, suggesting that fusion is not always necessary. Only 10% of the cohort required subsequent fusion at a mean follow-up of three months [78].

The Spinal Laminectomy vs Instrumented Pedicle screw (SLIP) study was a randomized prospective trial at five centers comparing laminectomy vs laminectomy with fusion for stenosis with Grade 1 spondylolisthesis. Overall, patients treated with laminectomy plus fusion had superior health-related quality of life outcomes at two, three, and four years after surgery. Those patients treated with simple lumbar spine decompression had a one-in-three risk of a second operation to fuse the spine at the level of the prior decompressive laminectomy. The patients treated with fusion had a 14% rate of reoperation associated with the development of adjacent-level disease. Overall, this study demonstrates that patients are better served with the addition of fusion, but that 70% of patients treated with decompression alone also did well [25].

According to an analysis of a Medicare claims database, the use of decompression plus fusion to treat spinal stenosis procedures increased 15-fold between 2000 and 2007, as did postsurgical complications, reoperation rates, and cost [58]. A recent systematic review of 24 randomized controlled trials focusing on patients with lumbar stenosis in the absence or presence of mild spondylolisthesis concluded that decompression with fusion does not appear to be superior to decompression without fusion. Patients in the latter group experienced

significantly less perioperative blood loss and required shorter surgical procedures, although there was no difference in the number of reoperations between the two groups [79]. When fusion was augmented with instrumentation, the success rate improved, but there was no clear impact on clinical outcomes, and the complication rate increased.

Another analysis of patients from a Medicare claims database (N = 1,672) included a focus on perioperative complications and reported that 36% of subjects treated with posterolateral spinal fusion for spinal stenosis and spondylolisthesis required reoperation and/or epidural injection within two years of surgery, and almost 25% required readmission for a procedure-related complication [80]. Some of the most common complications at two-year follow-up included urinary tract infection (40%), postlaminectomy syndrome (25%), and pneumonia (13%).

Adjacent segment disease is another risk associated with spinal fusion. The 10-year prevalence of adjacent segment disease requiring surgical treatment following lumbar spinal fusion ranges from 22% to 27%, with a 2.5% incidence per year [81, 82]. There is some thought that issues related to spinopelvic alignment (e.g., significant pelvic incidence lumbar lordosis mismatch) or other intrinsic risk factors contribute to the likelihood of adjacent segment disease after fusion [83].

Staartjes and Schoder maintain that, although laminectomy with fusion may not be an appropriate treatment for the majority of patients with spinal stenosis accompanied by Grade 1 spondylolisthesis, there remains a subset of patients who would benefit from a combination of the two procedures. They assigned patients to treatment with laminectomy (N = 51) or laminectomy plus fusion (N = 51) using a decision-making protocol based on clinical history, location of nerve root compression, and facet angles/facet effusion to assign patients to receive one or both procedures [84]. Results indicated a low rate of revision surgery for iatrogenic spondylolisthesis after laminectomy alone and positive patient-reported outcomes in both groups. Similar findings by Austevoll et al. suggested that a considerable number of patients with lumbar spinal stenosis and degenerative spondylolisthesis can be treated with decompression alone [85].

Decompression laminectomy with fusion is significantly more expensive than laminectomy alone. For example, a cost-benefit analysis compared 320 patients with stenosis who underwent laminectomy with 344 patients with stenosis and spondylolisthesis who underwent laminectomy plus fusion and found that, over a two-year postsurgery period, the cost of the former procedure was $77,000 per quality-adjusted life-year (QALY) gained, whereas the cost of the latter procedure was $115,000 per QALY gained. The authors note that $100,000 per QALY gained is often considered to be the maximum at which a procedure is considered to be cost-effective [86].

Decompression with Interspinous/Interlaminar Spacer

Interspinous process "spacer" devices provide an additional treatment option within the continuum of care for LSS when conservative therapy and epidural steroid injections have failed to provide sustained pain relief before more invasive open decompression surgery. Several implantable devices have been developed. Some are rigid expanders such as the X-stop (Medtronic Spine, Minneapolis, MN, USA), which is no longer commercially available in the US market. Others are flexible load-sharing devices such as the Wallis implant (Zimmer Spine, Warsaw, IN, USA). The Dynesy implant (Zimmer Spine, Warsaw, IN, USA) requires the placement of pedicle screws. The Diam implant (Medtronic Spine, Minneapolis, MN, USA) is a viscoelastic device intended to load-share and prevent compression. The Coflex device (Paradigm Spine, New York, NY, USA) is a spring-loaded spacer that is designed to decompress without fusion. More recently, the Superion device (Vertiflex, Carlsbad, CA, USA) was introduced as a minimally invasive standalone spacer to serve as a back stop preventing compression of the spinal canal and lateral recess during extension of the spine. The Superion device is the only minimally invasive application that does not require additional surgical resection, fixation, or decompression during implantation. Superion interspinous process decompression (IPD) was shown to have five-year durable clinical improvement of neurogenic claudication in 84% of study patients in at least two of three applications of the Zurch Claudication Questionnaire. This application of IPD has also been shown to improve quality of life in patients with LSS. In this study, 189 patients treated with the Superion IPD device were evaluated with SF-12. At two years, 81% of subjects showed maintenance or improvement in Physical Component Summary scores, and Mental Component Summary scores improved [87].

Minimally invasive options compared with open surgical decompression allow for shorter procedure time with reduced anesthetic risk. The Superion IPD procedure can be performed the same day as surgery with minimal recovery time. Indirect decompression in the posterior elements requires adequate bone strength. Patients with advanced osteoporosis are contraindicated. Spinous process fractures and device migrations were mostly reported in the first two years of implantation [87]. IPD is not indicated for treatment of the L5/S1 segment due to anatomical limitations.

Minimally Invasive Lumbar Decompression

Ligamentum flavum hypertrophy causing compression and stenosis and resulting intermittent neurogenic claudication can be treated by a minimally invasive method of removal of a small amount of laminae and thinning of the ligament. To be a candidate for this procedure, the ligament should be >2.5 mm and should be a major contributing factor to the stenosis. It should be noted that

comorbidities can be present, and the use of this technique is not limited to solitary disease states of the ligament. Guidance for using this technique including patient selection and outcomes, is outlined in the Minimally Invasive Spine Treatment protocol guidelines paper [88].

Conclusions

It is clear that LSS presents an economic and medical challenge to both patients and physicians. The disorder can be asymptomatic for an extended period, and once the patient is exhibiting signs of pain, it may be too late for conservative treatment. Effective treatment for LSS is particularly challenging due to the requirement for accurate diagnosis. However, when medical interventions are indicated, the present work demonstrates that there are many techniques at the physician's disposal. As medical imaging improves, with new technological advancements and increasing resolution, it would be the hope that identifying LSS accurately will become easier and that surgical techniques and tools will improve to most effectively reduce the patient's pain and restore function.

References

1. Sachs B, Fraenkel J. Progressive ankylotic rigidity of the spine (spondylose rhizomelique). J Nerv Ment Dis 1900;27(1):1–15.
2. Verbiest H. Stenosis of the lumbar vertebral canal and sciatica. Neurosurg Rev 1980;3(1):75–89.
3. Social Security Administration. Disability evaluation under social security. Soc Secur Bull 2010;70(3):70.
4. Ke J, Rosen I, Uden A. The natural history of lumbar spinal stenosis. Clin Orthop 1992;279:82–6.
5. Djurasovic M, et al. Contemporary management of symptomatic lumbar spinal stenosis. Orthopedic Clinics 2010;41(2):183–91.
6. Young PM, Berquist TH, Bancroft LW, et al. Complications of spinal instrumentation. Radiographics 2007;27(3):775–89.
7. Taher F, Essig D, Lebl DR, et al. Lumbar degenerative disc disease: Current and future concepts of diagnosis and management. Adv Orthop 2012;2012:14–7.
8. Parker SL, Chotai S, Devin CJ, et al. Bending the cost curve—establishing value in spine surgery. Neurosurgery 2017;80(3S):S61–9.
9. Fritz JM, Delitto A, Welch WC, et al. Lumbar spinal stenosis: A review of current concepts in evaluation, management, and outcome measurements. Arch Phys Med Rehabil 1998;79(6):700–8.
10. Goldman S, Funk J, Christensen V. Spinal stenosis. A common cause of podiatric symptoms. J Am Podiatr Med Assoc 1997;87(3):117–24.
11. Konno S, Hayashino Y, Fukuhara S, et al. Development of a clinical diagnosis support tool to identify patients with lumbar spinal stenosis. Eur Spine J 2007;16(11):1951–7.
12. Deyo RA, et al. Trends, major medical complications, and charges associated with surgery for lumbar spinal stenosis in older adults. JAMA 2010;303 (13):1259–65.
13. De Villiers PBE; Fibrous Spinal Stenosis. A report on 850 myelograms with a water-soluble contrast medium. Clin Orthop Relat Res 1976;115:140–4.
14. Fanuele JC, Birkmeyer NJ, Abdu WA, et al. The impact of spinal problems on the health status of patients: Have we underestimated the effect? Spine 2000;25(12):1509–14.
15. Kalichman L, Cole R, Kim DH, et al. Spinal stenosis prevalence and association with symptoms: The Framingham Study. Spine J 2009;9(7):545–50.
16. Yabuki S, Otani K, Sekiguchi M, et al. Prevalence of lumbar spinal stenosis, using the diagnostic support tool, and correlated factors in Japan: A population-based study. J Orthop Sci 2013;18(6):893–900.
17. Ishimoto Y, Yoshimura N, Muraki S, et al. Prevalence of symptomatic lumbar spinal stenosis and its association with physical performance in a population-based cohort in Japan: The Wakayama Spine Study. Osteoarthritis Cartilage 2012;20 (10):1103–8.
18. Jarvik JJ, Hollingworth W, Heagerty P, et al. The longitudinal assessment of imaging and disability of the back (LAIDBack) study: Baseline data. Spine 2001;26 (10):1158–66.
19. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994;331 (2):69–73.
20. Schonstrom N, Bolender N-f, Spengler DM. The pathomorphology of spinal stenosis as seen on CT scans of the lumbar spine. Spine 1985;10(9):806–11.
21. Arnoldi C, et al. Lumbar spinal stenosis and nerve root entrapment syndromes. Clin Orthop Relat Res 1976;115:4–5.
22. Sirvanci M, Bhatia M, Ganiyusufoglu KA, et al. Degenerative lumbar spinal stenosis: Correlation with Oswestry Disability Index and MR imaging. Eur Spine J 2008;17(5):679–85.
23. Spivak JM. Current concepts review-degenerative lumbar spinal stenosis. J Bone Joint Surg Am 1998;80 (7):1053–66.
24. Yukawa Y, Lenke LG, Tenhula J, et al. A comprehensive study of patients with surgically treated lumbar spinal stenosis with neurogenic claudication. J Bone Joint Surg Am 2002;84(11):1954–9.
25. Ghogawala Z, Dziura J, Butler WE, et al. Laminectomy plus fusion vs laminectomy alone for lumbar spondylolisthesis. N Engl J Med 2016;374 (15):1424–34.
26. Harrop JS, Youssef JA, Maltenfort M, et al. Lumbar adjacent segment degeneration and disease after arthrodesis and total disc arthroplasty. Spine 2008;33 (15):1701–7.

27. Foris LA, Dulebohn SC. Spinal Stenosis and Neurogenic Claudication. Florida: StatPearls Publishing; 2018.
28. Floman Y. Isthmic spondylolisthesis and degenerative spondylolisthesis. 2019. Available at: https://www.spineuniverse.com/conditions/spondylolisthesis/isthmic-spondylolisthesis-degenerative-spondylolisthesis (accessed September 1, 2019).
29. Burton MR, Mesfin FB. Isthmic Spondylolisthesis. Florida: StatPearls Publishing; 2019.
30. Talekar KS, et al. Imaging Spinal Stenosis. Scotch Plains, NJ: Anderson Publishing, Inc; 2017.
31. Kreiner DS, Shaffer WO, Baisden JL, et al. An evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis (update). Spine J 2013;13(7):734–43.
32. Steurer J, et al. Quantitative radiologic criteria for the diagnosis of lumbar spinal stenosis: A systematic literature review. BMC Musculoskelet Disord 2011;12(1):175.
33. Katz JN, Harris MB. Lumbar spinal stenosis. N Engl J Med 2008;358(8):818–25.
34. 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):1919–24.
35. Thomas SA. Spinal stenosis: History and physical examination. Phys Med Rehabil Clin 2003;14(1):29–39.
36. Hall S, et al. Lumbar spinal stenosis: Clinical features, diagnostic procedures, and results of surgical treatment in 68 patients. Ann Intern Med 1985;103(2):271–5.
37. Agrawal Y, et al. The modified Romberg balance test: Normative data in US adults. Otol Neurotol 2011;32(8):1309–11.
38. Lentell GL, Katzman LL, Walters MR. The relationship between muscle function and ankle stability. J Orthop Sports Phys Ther 1990;11(12):605–11.
39. Stucki G, Daltroy L, Liang MH, et al. Measurement properties of a self-administered outcome measure in lumbar spinal stenosis. Spine 1996;21(7):796–803.
40. Fukui M, Chiba K, Kawakami M, et al. JOA back pain evaluation questionnaire: Initial report. J Orthop Sci 2007;12(5):443–50.
41. Fukui M, Chiba K, Kawakami M, et al. Japanese Orthopaedic Association Back Pain Evaluation Questionnaire. Part 2. Verification of its reliability: The Subcommittee on Low Back Pain and Cervical Myelopathy Evaluation of the Clinical Outcome Committee of the Japanese Orthopaedic Association. J Orthop Sci 2007;12(6):526–32.
42. Fukui M, Chiba K, Kawakami M, et al. Japanese Orthopaedic Association Back Pain Evaluation Questionnaire. Part 3. Validity study and establishment of the measurement scale. J Orthop Sci 2008;13(3):173–79.
43. Hashizume H, Konno S-i, Takeshita K, et al. Japanese Orthopaedic Association Back Pain Evaluation Questionnaire (JOABPEQ) as an outcome measure for patients with low back pain: Reference values in healthy volunteers. J Orthop Sci 2015;20(2):264–80.
44. Choi Y-S. Pathophysiology of degenerative disc disease. Asian Spine J 2009;3(1):39.
45. Amundsen T, Weber H, Lilleås F, et al. Lumbar spinal stenosis. Clinical and radiologic features. Spine 1995;20(10):1178–86.
46. Wiesel SW, Tsourmas N, Feffer HL, et al. A study of computer-assisted tomography. I. The incidence of positive CAT scans in an asymptomatic group of patients. Spine 1984;9(6):549–51.
47. Lurie JD, Tosteson AN, Tosteson TD, et al. Reliability of readings of magnetic resonance imaging features of lumbar spinal stenosis. Spine 2008;33(14):1605–10.
48. Van Akkerveeken P. Lumbar spinal stenosis. Classification and clinical presentation. Orthopade 1993;22(4):253–65.
49. Schönström N, Lindahl S, Willén J, et al. Dynamic changes in the dimensions of the lumbar spinal canal: An experimental study in vitro. J Orthop Res 1989;7(1):115–21.
50. Manchikanti L, Cash KA, McManus CD, Pampati V, Fellows B. Results of 2-year follow-up of a randomized, double-blind, controlled trial of fluoroscopic caudal epidural injections in central spinal stenosis. Pain Physician 2012;15:371–84.
51. Laxmaiah Manchikanti M, Candido KD. Randomized trial of epidural injections for spinal stenosis published in the New England Journal of Medicine: Further confusion without clarification. Pain Physician 2014;17:E475–87.
52. Newark ND, et al. Lumbar interlaminar epidural injections in central spinal stenosis: Preliminary results of a randomized, double-blind, active control trial. Pain Physician 2012;15:51–63.
53. Friedly JL, Comstock BA, Turner JA, et al. A randomized trial of epidural glucocorticoid injections for spinal stenosis. N Engl J Med 2014;371(1):11–21.
54. Fritz JM, Lurie JD, Zhao W, et al. Associations between physical therapy and long-term outcomes for individuals with lumbar spinal stenosis in the SPORT study. Spine J 2014;14(8):1611–21.
55. Chou R, Huffman LH. Medications for acute and chronic low back pain: A review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. Ann Intern Med 2007;147(7):505–14.
56. Evoy KE, Morrison MD, Saklad SR. Abuse and misuse of pregabalin and gabapentin. Drugs 2017;77(4):403–26.
57. Peckham AM, Ananickal MJ, Sclar DA. Gabapentin use, abuse, and the US opioid epidemic: The case for reclassification as a controlled substance and the need

for pharmacovigilance. Risk Manag Healthc Policy 2018;11:109–16.
58. Deyo RA. Treatment of lumbar spinal stenosis: A balancing act. Spine J 2010;10(7):625–7.
59. Amundsen T, Weber H, Nordal HJ, et al. Lumbar spinal stenosis: Conservative or surgical management?: A prospective 10-year study. Spine 2000;25 (11):1424–36.
60. Chang Y, Singer DE, Wu YA, et al. The effect of surgical and nonsurgical treatment on longitudinal outcomes of lumbar spinal stenosis over 10 years. J Am Geriatr Soc 2005;53(5):785–92.
61. Zaina F, et al. Surgical versus non-surgical treatment for lumbar spinal stenosis. Cochrane Database Syst Rev 2016;1.
62. Freedman MK, Hilibrand AS, Blood EA, et al. The impact of diabetes on the outcomes of surgical and nonsurgical treatment of patients in the spine patient outcomes research trial. Spine 2011;36(4):290.
63. Memtsoudis SG, Vougioukas VI, Ma Y, et al. Perioperative morbidity and mortality after anterior, posterior and anterior/posterior spine fusion surgery. Spine 2011;36(22):1867.
64. Radcliff K, Curry P, Hilibrand A, et al. Risk for adjacent segment and same segment reoperation after surgery for lumbar stenosis: A subgroup analysis of the Spine Patient Outcomes Research Trial (SPORT). Spine 2013;38(7):531–39.
65. Aalto TJ, Malmivaara A, Kovacs F, et al. Preoperative predictors for postoperative clinical outcome in lumbar spinal stenosis: Systematic review. Spine 2006;31(18):E648–63.
66. Pearson A, Lurie J, Tosteson T, et al. Who should have surgery for spinal stenosis?: Treatment effect predictors in SPORT. Spine 2012;37(21):1791–1802.
67. Overdevest GM, et al. Effectiveness of posterior decompression techniques compared with conventional laminectomy for lumbar stenosis. Cochrane Database Syst Rev 2015;3.
68. Weng Dennis Hey H, et al. Post-laminectomy spondylolisthesis—a review of the posterior elements and their contribution to the stability of the lumbar spine. Open Spine J 2012;4(1).
69. Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical versus non-operative treatment for lumbar spinal stenosis four-year results of the Spine Patient Outcomes Research Trial (SPORT). Spine 2010;35(14):1329.
70. Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical versus nonsurgical therapy for lumbar spinal stenosis. N Engl J Med 2008;358(8):794–810.
71. Lurie JD, Tosteson TD, Tosteson A, et al. Long-term outcomes of lumbar spinal stenosis: Eight-year results of the Spine Patient Outcomes Research Trial (SPORT). Spine 2015;40(2):63–71.
72. Ma X-l, Zhao X-w, Ma J-x, et al. Effectiveness of surgery versus conservative treatment for lumbar spinal stenosis: A system review and meta-analysis of randomized controlled trials. Int J Surg 2017;44:329–38.
73. Ulrich NH, Burgstaller JM, Held U, et al. The influence of single-level versus multilevel decompression on the outcome in multisegmental lumbar spinal stenosis. Clin Spine Surg 2017;30(10):E1367–75.
74. Adilay U, Guclu B. Comparison of single-level and multilevel decompressive laminectomy for multilevel lumbar spinal stenosis. World Neurosurg 2018;111 :e235–40.
75. Mobbs RJ, et al. Outcomes after decompressive laminectomy for lumbar spinal stenosis: Comparison between minimally invasive unilateral laminectomy for bilateral decompression and open laminectomy. J Neurosurg Spine 2014;21(2):179–86.
76. Yagi M, et al. Postoperative outcome after modified unilateral-approach microendoscopic midline decompression for degenerative spinal stenosis. J Neurosurg Spine 2009;10(4):293–9.
77. Schöller K, Alimi M, Cong G-T, et al. Lumbar spinal stenosis associated with degenerative lumbar spondylolisthesis: A systematic review and meta-analysis of secondary fusion rates following open vs minimally invasive decompression. Neurosurgery 2017;80(3):355–67.
78. Ahmad S, Hamad A, Bhalla A, et al. The outcome of decompression alone for lumbar spinal stenosis with degenerative spondylolisthesis. Eur Spine J 2017;26 (2):414–9.
79. Machado GC, et al. Surgical options for lumbar spinal stenosis. Cochrane Database Syst Rev 2016;11.
80. Ong KL, Auerbach JD, Lau E, et al. Perioperative outcomes, complications, and costs associated with lumbar spinal fusion in older patients with spinal stenosis and spondylolisthesis. Neurosurgical Focus 2014;36 (6):E5.
81. Ghiselli G, Wang JC, Bhatia NN, et al. Adjacent segment degeneration in the lumbar spine. JBJS 2004;86 (7):1497–503.
82. Sears WR, Sergides IG, Kazemi N, et al. Incidence and prevalence of surgery at segments adjacent to a previous posterior lumbar arthrodesis. Spine J 2011; 11(1):11–20.
83. Tobert DG, Antoci V, Patel SP, et al. Adjacent segment disease in the cervical and lumbar spine. Clin Spine Surg 2017;30(3):94–101.
84. Staartjes VE, Schröder ML. Effectiveness of a decision-making protocol for the surgical treatment of lumbar stenosis with Grade 1 degenerative spondylolisthesis. World Neurosurg 2018;110:e355–61.
85. Austevoll IM, Gjestad R, Brox JI, et al. The effectiveness of decompression alone compared with additional fusion for lumbar spinal stenosis with degenerative spondylolisthesis: A pragmatic comparative non-inferiority observational study from the Norwegian Registry for Spine Surgery. Eur Spine J 2017;26(2):404–13.

86. Tosteson AN, et al. Surgical treatment of spinal stenosis with and without degenerative spondylolisthesis: Cost-effectiveness after 2 years. Ann Intern Med 2008;149(12):845.
87. Nunley PD, Patel VV, Orndorff DG, et al. Superion interspinous spacer treatment of moderate spinal stenosis: 4-year results. World Neurosurg 2017;104 :279–83.
88. Deer TR, Grider JS, Pope JE, et al. The MIST guidelines: The lumbar spinal stenosis consensus group guidelines for minimally invasive spine treatment. Pain Pract 2019;19(3):250–74.