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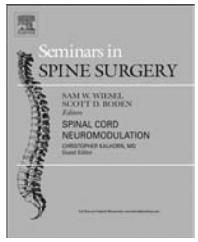


# Diagnostic modalities and nonoperative treatment of lumbar spinal stenosis

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A B S T R A C T

Lumbar spinal stenosis is a common and disabling condition with increasing prevalence in the United States. It is seen in the setting of degenerative lumbar pathology such as degenerative disc disease, spondylolisthesis or degenerative scoliosis and typically results in symptoms of neurogenic claudication – pain in the buttocks or legs that occurs with walking or standing and resolves with sitting or lumbar flexion. While diagnosis can typically be made with a thorough history and physical exam, functional tests such as walking exercise treadmill protocols can be used to increase specificity. Plain radiography with flexion-extension views are the initial imaging modality and demonstrate alignment, instability, bone density, and overall degenerative findings. Magnetic resonance imaging (MRI) is the imaging test of choice since it provides excellent detail of the bony anatomy as well as neural elements without the use of ionizing radiation. Canal cross-sectional area less than 100 to 130 $mm^2$ is generally the threshold for central stenosis, whereas a lateral recess height $\leq$ 2 mm, lateral recess depth $\leq$ 3 mm, or a lateral recess angle $< 30^{\circ}$ are indicative of lateral recess stenosis. Computed tomography (CT) is obtained when MRI is contraindicated in patients and provides superior resolution of bony anatomy, but poorer resolution of the neural elements. Nonoperative treatment is the first line strategy, with the use of anti-inflammatories, analgesics, muscle relaxants, neuromodulators, opioids, as well as physical therapy, steroid injections, multidisciplinary rehabilitation and lifestyle modification. Other treatments that have been explored include calcitonin, methylcobalamin, and alternative medicine strategies. Given that rapid deterioration is rare and symptoms often wax and wane, nonoperative treatment can provide an effective initial management strategy.



## 1. Introduction

Lumbar spinal stenosis (LSS) is defined as narrowing of the lumbar spinal canal with subsequent compression of the thecal sac resulting in radicular leg pain or neurogenic claudication - pain in the buttocks or legs that occurs with walking or standing and resolves with sitting or lumbar flexion.[1] Patients may also experience numbness, tingling, or weakness in the legs and buttocks. While the exact mechanism remains unclear, symptoms are generally thought to arise from mechanical compression or ischemia of nerve roots in the lumbosacral spine.[2] Degenerative lumbar pathology resulting in osteoarthritic facet joints, bulging discs, loss of disc height, and infolding of the ligamentum flavum contributes to a decreased cross-sectional area within the spinal canal for the lumbar nerve roots.

LSS is a prevalent and disabling condition, with approximately 400,000 individuals affected in the United States.[3] Typically, it affects patients over the age of 60 and is being increasingly diagnosed in the aging population with the advent of better diagnostic modalities.[4] While the diagnosis of LSS may seem straightforward, in clinical practice it requires complex judgment. The integration of symptoms,

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physical exam findings, and imaging along with consideration of relevant comorbidities is required to make an accurate diagnosis.[2] In general, patients often present with neurogenic claudication, lumbar radiculopathy or both. Due to the lack of an objective diagnostic standard, patients with these overlapping symptoms may be grouped under the same category of LSS.[2] In addition, LSS is the final manifestation of different types of degenerative lumbar pathology including degenerative changes with normal alignment, isolated degenerative spondylolisthesis, or degenerative scoliosis. As LSS can be a dynamic pathology, there also may not be clear findings on imaging studies. Therefore, judicious use of diagnostic modalities such as physical examination and imaging using plain radiographs, computed tomography (CT), CT myelogram, or magnetic resonance imaging (MRI) is needed.

Nonoperative care is usually the initial treatment strategy for patients with LSS. If patients continue to have persistent symptoms despite nonoperative treatment for several months, interventional treatment may be considered. The natural course of LSS remains largely unknown given that patients with severe symptoms seek treatment, however the current North American Spine Society (NASS) guidelines suggest that one-third to one-half of patients do well with nonoperative treatment in the long-term.[5] Given that rapid deterioration is rare and symptoms often wax and wane or even gradually improve, surgery is almost always elective.[2,6] Treatment modalities for nonoperative treatment range from the use of orthoses, analgesics, anti-inflammatories, neuromodulators, lifestyle modifications, physical therapy, and epidural injections, and other pain relieving treatments (heat, ice, electrical stimulation, massage, ultrasound) with varying success reported in literature.[7] The aim of this review is to describe available diagnostic modalities and nonoperative treatment strategies for LSS.

## 2. Diagnostic modalities

### 2.1. History and physical exam

The chief complaint in patients presenting with lumbar spinal stenosis is buttock and leg pain with prolonged standing or walking.[8–10] Typically, pain is not present while seating and increases with activity. Patients frequently describe relief of pain when bending forward while standing, also known as the stoop test.[10] They may find walking downhill or active lumbar extension uncomfortable, however riding a bicycle usually does not cause pain due to the flexed position.[10] Patients may also ambulate with a wide-based gait, however this is nonspecific and can be found in other spinal pathologies.

Pain due to neurogenic claudication should be differentiated from other types of pain that may also affect the buttocks and legs with activity. For example, intermittent claudication that occurs in the presence of peripheral vascular disease, also causes increased pain in the buttocks and thighs with activity. However, with intermittent claudication pain is relieved with rest while standing, as compared to bending forward or sitting for LSS. Additionally, referred pain from the low back may produce pain in the buttocks or thighs and can also be aggravated with walking.

Presently, dynamic functional tests have been developed to diagnose and quantify the severity of existing spinal stenosis by calculating the walking capacity. These include various walking exercise treadmill protocols, such as the gait loading test and the self-paced walking test.[11–14] In one study assessing the gait loading test, the mean time to symptoms while walking on a treadmill at a predetermined speed was noted to be 1.8 minutes, with mean total ambulation time to be 6.9 minutes.[12] The authors note that this is a safe, easily administered, and quantifiable method for assessing functional status. Similarly, a self-paced protocol that allows patients to walk for a predetermined distance at their own speed found that it is an easy and reproducible means for quantifying the severity of spinal stenosis.[13] The lumbar extension-loading test is another proposed provocative maneuver where the patient is asked to hold a standing position with moderate lumbar extension for as long as possible.[15] Finally, the use of accelerometers to identify changes in posture, gait, and overall performance is a new and emerging technology that may prove useful in the future.[16]

### 2.2. Electromyography

The use of electromyography (EMG) for the diagnosis of LSS has been investigated in a few studies.[17,18] EMG may detect physiological alterations in the muscle as a result of affected lumbar nerve roots in LSS, but is also helpful in detecting change due to many other disorders such as polyneuropathy or distal nerve compression. Its principal use is in the differentiation of from other causes of back, buttock and thigh pain including spinal cord tumors, vascular disease, or hip osteoarthritis when the physical exam and imaging may not be consistent with LSS. Haig et al. showed that with the use of paraspinal mapping, EMG was able to differentiate patients with clinical stenosis from asymptomatic patients and those with mechanical back pain.[18] The authors concluded that consultation with an EMG may be a helpful adjunct, especially when MRI findings are equivocal.

### 2.3. Plain radiography

Plain radiographs are usually the initial imaging modality in patients presenting for lumbar spine issues (Table 1). They are cheap, widely available, and can provide significant information about the lumbar spine including the presence of disc degeneration, vacuum phenomenon, osteoporosis, or other bone-forming diseases such as ankylosing spondylitis (AS) and diffuse idiopathic skeletal hyperostosis (DISH). Dynamic radiographs with load-bearing flexion and extension views are a helpful adjunct as they may highlight the presence or severity of spondylolisthesis and degenerative scoliosis. Unrelated causes of back pain may also be identified, including vascular disease (calcification) or sacroiliac joint pathology. However, plain radiography does not provide information regarding the soft tissue, including the neural elements.

Myelography is the administration of intrathecal contrast for evaluation of the spinal canal. In 1954, Verbiest provided the first description of LSS in a group of male patients with normal radiographs but with signs of extradural compression on myelography.[19] He noted that the form of the lower part of the

**Table 1 – Imaging modalities and diagnostic criteria for lumbar spinal stenosis.**

| Imaging modality | Findings |
|---|---|
| Plain radiographs | Preferred initial imaging modality – cheap, widely available |
| | Degenerative pathology: |
| | - Decreased disc height |
| | - Osteoarthritic facet joints |
| | - Spondylolisthesis |
| | - Degenerative scoliosis |
| Myelography | Not currently used for spinal imaging. Initially used to describe extradural compression by Verbiest et al. |
| MRI | High sensitivity for identifying anatomic narrowing of central canal and lateral canal. |
| | Only moderate specificity for diagnosis of lumbar spinal stenosis. |
| | Diagnostic measurements for central stenosis: |
| | $< 100\ mm^2$ cross-sectional area on axial imaging |
| | $< 10$ mm AP diameter on sagittal cut indicative of absolute central stenosis |
| | $< 12$ mm AP diameter on sagittal cut indicative of relative central stenosis |
| | Diagnostic measurements for lateral canal stenosis: |
| | $\leq 2$ mm for lateral recess height |
| | $\leq 3$ mm for lateral recess depth |
| | $\leq 30^\circ$ for lateral recess angle |
| | Diagnostic measurements for foraminal stenosis: $\leq 3$ mm |
| CT Myelography | Preferred if MRI contraindicated in patient. |
| | Intrathecal contrast agent administered via lumbar puncture can highlight points of extradural compression |
| CT | Performed if CT myelography contraindicated |
| | Superior bony resolution compared to MRI |
| | Inferior soft tissue contrast compared to MRI |
| | Similar diagnostic measurements as MRI |

*MRI* = Magnetic Resonance Imaging, *CT* = Computed Tomography, *AP* = antero-posterior

column of radio-opaque fluid seemed to suggest a gradual narrowing of the vertebral canal, which was confirmed during later surgery.[19] It was also the first imaging modality to quantify stenosis by measuring the antero-posterior (AP) diameter of the contrast column at the level of stenosis. Verbiest suggested that 10–12 mm constituted relative stenosis, while < 10 mm constituted absolute stenosis.[19] While myelography has been replaced by CT myelography for imaging spine pathology, the principles of administration of a radiopaque contrast and outlining extradural compression remain the same.

### 2.4. *Magnetic resonance imaging*

MRI is a noninvasive, multi-planar imaging modality that provides excellent detail of the bony lumbar anatomy as well as the neural elements (Table 1). It has the highest sensitivity in the diagnosis of LSS, does not use ionizing radiation, and is thus considered the imaging modality of choice.[20] Several common causes of anatomic narrowing are easily assessed with MRI, including disc herniations, facet hypertrophy, and ligamentum flavum hypertrophy. Less common causes may include epidural lipomatosis, or synovial cysts. Many studies have attempted to quantify the amount of central and lateral stenosis on MRI.[21] The AP diameter and the cross-sectional area of the spinal canal are the most often applied criteria for assessing central canal stenosis. Definitions of central stenosis vary between 10, 12 or 15 mm for the AP diameter and 100 $mm^2$ or 130 $mm^2$ depending on the study and the method used for measurement.[22–25] Lateral stenosis (lateral recess, foramen) is assessed using the height and depth of the lateral recess and the foraminal diameter. The depth of the lateral recess is measured as the distance on an axial cut between the superior articular facet and the top of the pedicle, whereas the lateral recess height is defined as the distance between the anterior most point of the superior articular facet and the posterior aspect of the vertebral body.[21] The lateral recess angle is defined as the angle between the horizontal and the roof of the lateral recess. Typically, a lateral recess height ≤ 2 mm, lateral recess depth ≤ 3 mm, or a lateral recess angle < 30° has been considered diagnostic for lateral recess stenosis.[21] Foraminal stenosis is quantified using the diameter of the foramen, with a diameter less than 2–3 mm considered indicative of stenosis.[26]

While MRI carries a high sensitivity for identifying radiographic findings of LSS, it only has a moderate specificity. Studies of asymptomatic volunteers have shown that significant central canal stenosis can be present on MRI and that these findings increase with age.[27,28] In addition, a multitude of studies have not found a correlation between the presence of central or lateral stenosis on imaging with claudication pain, disability, or functional capacity.[18,29,30] NASS guidelines state that there is insufficient evidence to recommend for or against a correlation between evidence of stenosis on imaging and clinical symptoms. One unique finding on MRI that may serve as a marker of symptomatic LSS is the nerve root sedimentation sign, as described by Barz and colleagues.[31] On a supine MRI, the lumbar nerve roots normally migrate dorsally. However, in patients with LSS, these nerve roots may demonstrate an absence of sedimentation to the dorsal thecal sac on axial MRI cuts at a level above or below the stenosis. These findings were present even when no correlation was identified between cross-sectional area and disability in this cohort of patients.[31] While it is clear that MRI provides a superior imaging modality, this radiographic information alone is insufficient to make a diagnosis of LSS and must be taken into consideration with the entire clinical picture.

### 2.5. *Computed tomography*

CT is recommended when MRI is unobtainable or contraindicated in patients (Table 1). CT provides superior resolution of lumbar bony anatomy compared to MRI but has poorer resolution of the neural elements and less soft tissue contrast. In addition, it is less preferable due to patient exposure to ionizing radiation. Despite this, previous studies have shown good agreement between CT and MRI in identification of pathology.[32] Some conditions where CT may provide greater detail than MRI include bone-forming diseases that can encroach on the epidural space such as ossification of the posterior longitudinal ligament (OPLL), DISH, or AS. One study showed a more narrowed canal cross-sectional area on axial CT cuts compared to MRI, indicating that a multidetector CT may be able to discriminate cortical bone from soft tissue such as the ligamentum flavum with increased detail.[33] Another scenario in which a CT scan may provide valuable information is with pre-surgical planning.

CT myelography was first used in 1976, only a few years after the advent of the first CT scanner. It combined the principles of conventional myelography with CT to outline points of epidural compression and other spinal pathology.[34] It is preferable when the patient has a contraindication to an MRI scan and is able to tolerate a lumbar puncture. CT myelography provides superior resolution of the central and lateral canal and is thus preferable to the use of CT alone. Intravenous contrast does little to improve diagnostic capability. The use of myelography is not without adverse effects however, as intrathecal puncture and introduction of a foreign agent pose the risk of pain, muscle spasms, paresthesias, arachnoiditis, pseudotumor formation, and seizures.

### 2.6. *Nonoperative treatment strategies*

Nonoperative treatment is the first line strategy for patients with LSS (Table 2). There are many different modalities that are available to reduce pain and increase functional capacity ranging from medications such as nonsteroidal anti-inflammatory drugs (NSAIDs), analgesics, opioids, muscle relaxants, and neuromodulators to epidural steroid injections, physical therapy, multidisciplinary rehabilitation, and lifestyle modification. Other treatments that have been explored include calcitonin and methylcobalamin. Some patients also may opt to use non-traditional medicine such as chiropractic treatment, acupuncture, homeopathic medicine, or herbal medicine, however this is beyond the scope of this review.

Oral medications are easy to administer and may be effective as an initial treatment for patients with mild symptoms. Oral medications may include NSAIDs, prostaglandins, analgesic medications, or neuromodulatory agents. Examples of frequently sued NSAIDs include ibuprofen, naproxen, celecoxib, etodolac, and diclofenac. These medications work by blocking the Cox-1 and Cox-2 enzymes in the inflammatory pathway and theoretically reduce inflammation of lumbar nerve roots. Prostaglandin analogues such as Limaprost (prostaglandin E1) have been shown to improve LSS symptoms and are potentiated with the concurrent use of NSAIDs.[35] It is thought that they exhibit an effect via their vasodilatory properties, increasing blood flow in the case of neurovascular ischemia in LSS. Commonly prescribed analgesic medications include acetaminophen and tramadol. Acetaminophen works via a yet to be fully elucidated pathway but is a weak inhibitor of prostaglandin production. Tramadol functions via a combined opioidergic and noradrenergic mechanism. While it is intuitive that anti-inflammatories would be more effective than these analgesic medications in LSS, comparative studies have found no difference.[2] Opioids exert powerful analgesic properties by acting on centrally located opioid receptors, and while they may be effective in chronic pain, they should rarely be used for the treatment of LSS given the habit-forming potential and other side effects.[36] Muscle relaxants may also be prescribed for pain, however they work similarly to analgesics to reduce symptoms and

**Table 2 – Nonoperative treatment modalities for lumbar spinal stenosis.**

| Nonoperative treatment | Findings |
|---|---|
| Oral medications | Preferred:<br>- NSAIDs (ibuprofen, naproxen, etodolac, diclofenac, etc.)<br>- Prostaglandin analogues<br>- Analgesic medications (tylenol, tramadol)<br>- anti-convulsants (gabapentin, pregabalin)<br>Further evidence needed:<br>- tricyclic antidepressants<br>- oral corticosteroids<br>- calcitonin<br>- methylcobalamin<br>Not preferred:<br>- opioids |
| Physical therapy | Postural correction, core strengthening, flexibility exercises<br>Cycling<br>Body-weight supported treadmill walking<br>Aquatic therapy<br>Other modalities: Heating, icing, TENS, ultrasound treatment |
| Epidural injections | Maybe administered via transforaminal, interlaminar, or caudal approach<br>Can be administered with local anesthetic only vs. local anesthetic plus corticosteroid |
| Alternative medicine | Limited evidence to support chiropractic treatment with spinal manipulation, acupuncture, homeopathic and naturopathic medicine |

promote muscle relaxation at a downstream level. Similar to opioids, they have not been shown to be more effective than analgesics.[2] Neuromodulatory drugs such as membrane-stabilizing anti-convulsants (gabapentin, pregabalin) may reduce neuropathic pain by stabilizing excitatory nerves in patients with LSS.[36] One randomized trial comparing patients with gabapentin versus those without observed a greater walking distance, reduced pain and reduction in sensory deficits in the gabapentin group.[37] Finally, both oral corticosteroids and tricyclic anti-depressants are also prescribed for patients with this condition, however evidence regarding their efficacy with well-controlled studies is lacking.[2]

Other medications that have been trialed in the treatment of LSS include calcitonin and methylcobalamin. Calcitonin is a peptide hormone produced by the parafollicular cells of the thyroid gland is involved in calcium homeostasis. It is typically administered to patients with a history of Paget's disease with bone-related pain, osteoporosis, or cancer related hypercalcemia. Aside from this metabolic function, calcitonin is thought to have a direct analgesic effect via the release of $\beta$-endorphin or a direct central nervous system effect.[38] An indirect effect that has been postulated is that it can direct vascular supply from the bone to the compromised neural tissues.[39] Previously, researchers have attempted to use of calcitonin to treat pain secondary to LSS and have found mixed results. Two recent meta-analyses have found little evidence to suggest that calcitonin is more effective than placebo in the reduction of pain due to LSS.[7,40] Since high doses of B-vitamins have been used previously in nerve entrapment syndromes, methylcobalamin (methyl-vitamin B12) has been investigated in the treatment of LSS.[39,41] However, to date only one randomized trial has been conducted, which found no significant difference between groups in terms of pain, motion, or neurological findings but improved ambulation in patients receiving methylcobalamin.[41]

Physical therapy is the mainstay for treatment of LSS and is often prescribed in combination with oral medications to help patients participate effectively. Treatments related to physical therapy include general activity and conditioning exercises designed to increase strength, flexibility, and aerobic capacity. One specific exercise that is uniquely helpful to patients with lumbar stenosis is cycling, since it involves aerobic activity in the position of lumbar flexion. In this position, the spinal canal diameter increases and the patient is able to participate without being limited by claudication pain. Another technique useful for LSS is body weight supported treadmill walking, where a harness is used to reduce the amount of weight that is loaded onto the lumbar spine, thereby indirectly increasing the spinal canal diameter. One recent randomized trial found no difference between cycling and body weight treadmill walking in terms of pain reduction at the end of the treatment period.[42] Similar to supported treadmill walking is the use of aqua-therapy. In an aquatic environment, patients can relieve the amount of loading on the lumbar spine and can participate in strengthening and flexibility exercises. Other aspects of physical therapy may include muscle coordination training and specific core strengthening exercises. Some programs advocate for both flexion and extension exercises, while others include only flexion exercises to avoid exacerbating symptoms with extension.[43] Physical therapists may also recommend heating and icing modalities can help reduce pain and spasm, subsequently improving flexibility and participation in patients. Other modalities used by therapists may include transcutaneous electrical nerve stimulation (TENS) or ultrasound treatment, which have limited evidence. The use of orthoses, whether a soft or a hard lumbar corset, has been found to relieve symptoms by preventing excessive lumbar extension.[43] Due to the cumbersome nature of using an orthosis, this may play a limited role in and is probably best used as an adjunctive treatment.

Several randomized trials have attempted to address whether physical therapy is beneficial in the nonoperative management of LSS, however the quality of evidence is low and one systematic review found that no conclusions could be drawn regarding its effectiveness.[7] While physical therapy itself may not affect the underlying pathophysiology of LSS, it acts to increase patient functionality. A secondary analysis of the Spine Patient Outcomes Research Trial (SPORT) for LSS found no association with improvement in pain with physical therapy, but did find an improvement in self-reported functionality.[44] Since LSS is a dynamic condition, improving posture and coordination may confer benefits by improving the spinal canal diameter, thus lessening symptoms. To date, the optimal regimen for physical therapy has not been determined.[39]

Epidural injections are also commonly prescribed for LSS, and can be administered via a transforaminal, interlaminar, or caudal approach. Epidural injections with local anesthetic agents provide sympathetic blockade and vasodilatation, which may reduce transient ischemia related to LSS.[39] In addition, these local anesthetic agents may reduce neuronal sensitization by stabilizing pain pathways. Corticosteroid (methylprednisolone or triamcinolone) may be added to the injection to confer anti-inflammatory properties and potentially reduce edema at the injured nerve root, decrease sensitization in the dorsal horn neurons and suppress transmission of nociceptive C-fibers.[39] Several randomized trials have compared the use of epidural injections (local anesthetic only, local anesthetic + corticosteroid) vs. placebo and have found varying results, with only one of these trials showing superior effectiveness for corticosteroids and the others showing equal effectiveness.[39] There is limited evidence to show that an interlaminar approach may be more effective than a caudal approach.[2] Overall, the benefits of epidural injections are short-lived, usually lasting less than one month and are more effective for patients with radiculopathy than spinal stenosis. The addition of corticosteroid, while theoretically beneficial, has not been proven with well-designed studies. Therefore, despite early promising results, further studies are needed to clarify the role of epidural injections with or without corticosteroids.

Patients may also seek to pursue other forms of alternative medicine such as chiropractic treatment including spinal manipulation, acupuncture, homeopathic remedies, and herbal treatments. In the past, spinal manipulation was generally considered to be contraindicated in patients with degenerative changes and LSS.[43] Similarly, there is limited evidence to support acupuncture as an effective treatment for LSS. Overall, there is little rigorous data for alternative treatments and future well-designed studies are needed to draw conclusions regarding their effectiveness.

## 3. Conclusion

Lumbar spinal stenosis is becoming an increasingly common diagnosis with the aging population and better diagnostic modalities. While the pathophysiology of this condition is incompletely understood, patients present with a typical presentation of neurogenic claudication with standing, walking or lumbar extension that is relieved with lumbar flexion. Key diagnostic modalities include plain radiographs and MRI in combination with a thorough history and physical exam. Nonoperative management is the initial treatment strategy with NSAIDs, analgesics, and physical therapy. Other interventions include calcitonin, methylcobalamin and the use of a multi-modal treatment approach, however further research is needed.

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