

Lumbar Spinal Stenosis

Interventional & Surgical Management

An Evidence-Based Update for Primary Care Physicians

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Agenda

1. Definition & Pathophysiology
2. Etiology & Epidemiology
3. Clinical Presentation
4. Diagnostic Criteria and Workup
5. Treatment Options
6. Practical Algorithm

Definition & Pathophysiology

- Adult LSS is a pathological narrowing of the central canal, lateral recesses or foramina leading to neural compression.

Classification:

Central canal stenosis – narrowing of the main spinal canal.

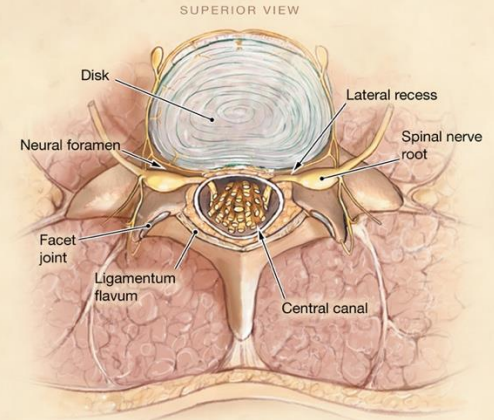
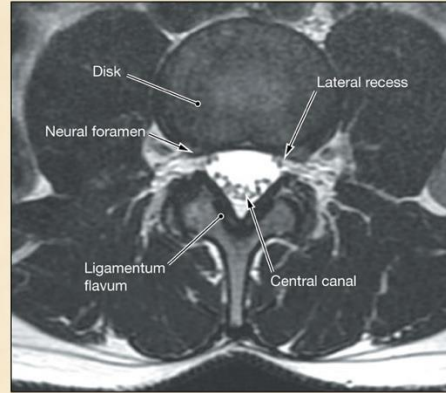
Lateral recess stenosis – narrowing medial to the pedicle affecting the traversing root.

Foraminal stenosis – narrowing of the intervertebral foramen involving the exiting root.

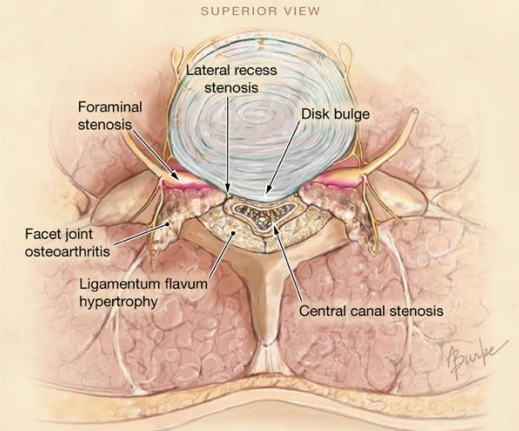
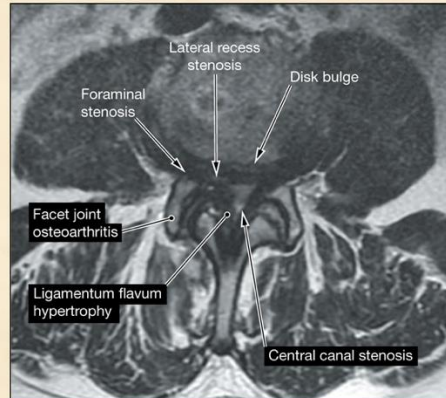
- Degenerative cascade: disc dehydration → facet osteoarthritis/osteophytes → ligamentum flavum hypertrophy & buckling.
- Spondylolisthesis or instability further reduce space.

Does This Older Adult With Lower Extremity Pain Have the Clinical Syndrome of Lumbar Spinal Stenosis?. *Jama*. 2010;304(23):2628-36.
doi:10.1001/jama.2010.1833.

A Normal lumbar spine



B Degenerative changes in lumbar spinal stenosis (LSS)



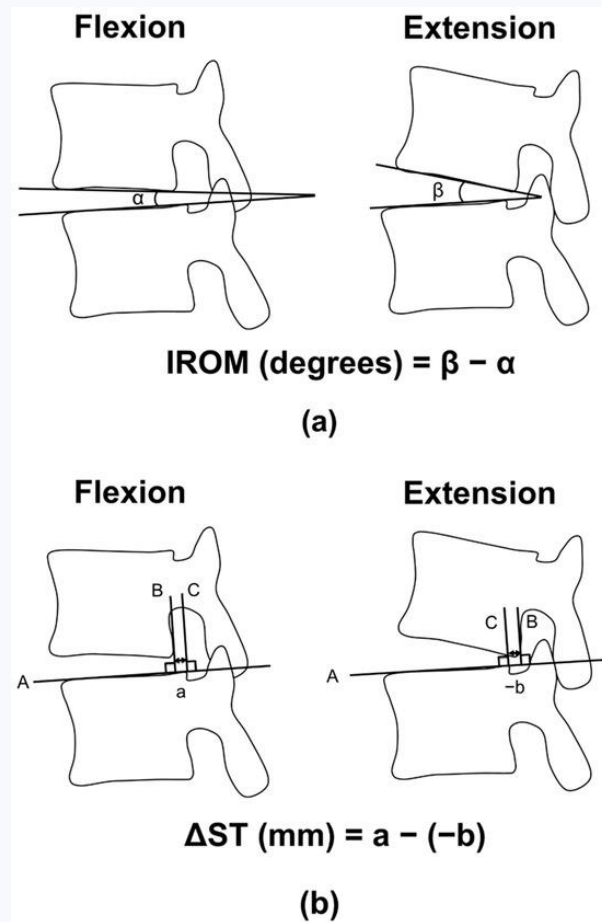
Dynamic vs Fixed Stenosis

Fixed stenosis is due to congenital or degenerative narrowing that remains constant regardless of posture.

Dynamic stenosis occurs when spinal canal dimensions change with posture. Flexion opens the canal and alleviates symptoms, while extension narrows the canal and exacerbates discomfort.

White & Panjabi Criteria: IROM > 10° - 20° or Δ ST > 3 – 4 mm

Lumbar instability is defined by abnormal segmental motion beyond normal physiological limits on flexion–extension radiographs (Suzuki et al., 2024).



Etiology & Causes

Degenerative (most common):

- Disc dehydration/bulging
- Facet arthropathy & osteophytes
- Ligamentum flavum hypertrophy
- Degenerative spondylolisthesis

Congenital/Developmental:

- Short pedicles (idiopathic)
- Achondroplasia or skeletal dysplasias

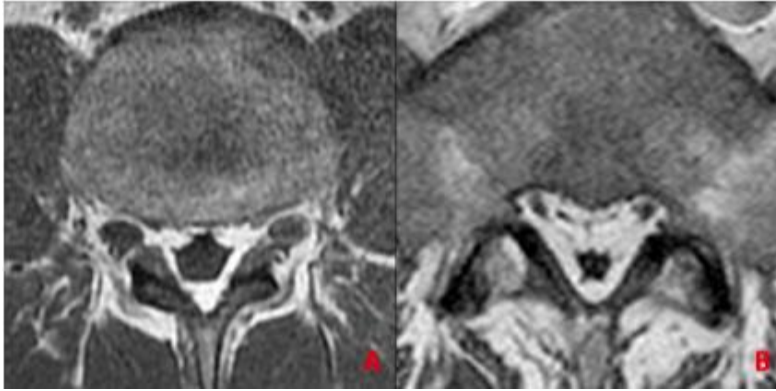
Other causes:

- Epidural lipomatosis (steroid therapy, hypothyroid, obesity)
- Synovial (facet) cyst
- Neoplasms (meningioma, schwannoma, metastases)
- Infections (epidural abscess, TB)
- Traumatic fractures & dislocations
- Iatrogenic: post-laminectomy fibrosis / arachnoiditis, adjacent segment degeneration

Neurovascular Compromise: Mechanical compression, impaired arterial inflow/venous drainage, and inflammatory mediators contribute to pain and neurogenic claudication.

Images

Epidural Lipomatosis



Arachnoiditis



Lumbar Facet Cyst



Retropulsed Vertebral Burst Fracture



Epidemiology & Risk Factors

Age-Dependent Prevalence

In the general population, the prevalence of severe stenosis is 8% rising to 20% in patients over age 60.

Prevalence: ~11% of US adults with symptomatic LSS; increases to 19% in adults >60.

Age >60 years is the strongest risk factor; degenerative changes accelerate after midlife.

Other risks:

- Female gender
- Obesity
- Smoking
- Congenital narrow canal
- Trauma or surgery
- Endocrine disorders (e.g. steroid use)
- Occupations with heavy axial load

Age Group (years)	Relative LSS Prevalence (%)	Absolute LSS Prevalence (%)
<40	20	4
60-69	47	19
≥55 (moderate)	21-30 (moderate)	6-7 (severe)

Relative LSS = moderate LSS [< 12 mm]

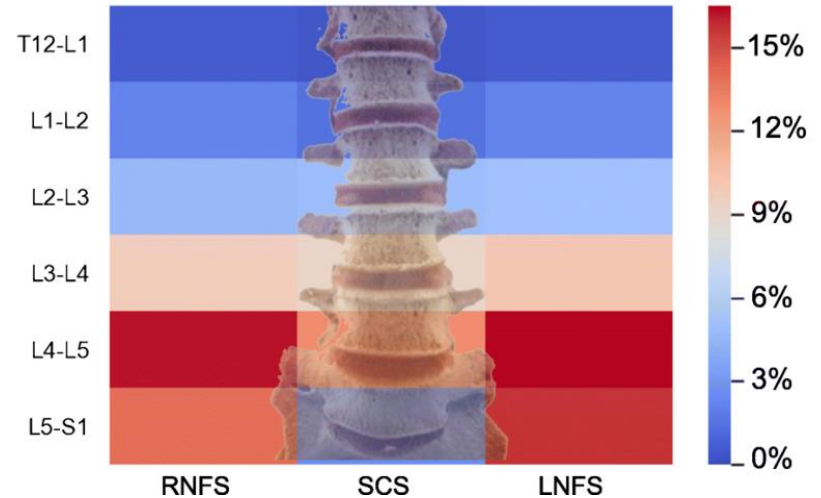
Absolute LLS = severe stenosis [<10 mm]

Kalichman L, Cole R, Kim DH, et al. Spinal stenosis prevalence and association with symptoms: The Framingham Study. *Spine J.* 2009;9(7):545-550.
doi:10.1016/j.spinee.2009.03.005

Epidemiology

Lumbar stenosis above L1-2 is fairly uncommon, and rare above T12

Spinal Canal and Neuroforaminal Stenosis Prevalence (n = 43255)



Kaiser R, Weber M, Götschi T, et al. Effects of age and sex on the distribution and symmetry of lumbar spinal and neural foraminal stenosis: a natural language processing analysis of 43,255 lumbar MRI reports. *Eur Radiol.* 2023;33(3):1733-1742. doi:10.1007/s00330-022-09044-6

Clinical Presentation: Neurogenic vs Vasogenic Claudication

Characteristic	Neurogenic	Vascular
Pain distribution	Proximal→distal, may include buttocks and thighs	Distal→proximal (calves)
Quality	Neuropathic, burning, tingling	Cramping, tightness
Triggers	Walking downhill/extension	Exertion regardless of posture
Relief	Sitting, walking uphill or flexion (shopping-cart sign)	Standing still or rest
Pulses	Normal	Reduced/absent
Cycling ability	Unimpeded (flexion)	Provokes pain

Clinical Presentation: Neurogenic Claudication vs Facet Pain

Feature	Neurogenic Claudication	Facet Joint Pain
Location	Buttocks, posterior thighs, calves; bilateral	Localized low back or paraspinal region
Radiation	Often leg radiation with walking	Rarely radiates below the knee
Positional effects	Worse with extension/walking; relief with flexion	Worse with extension and rotation; relief lying down
Neurologic deficits	Possible numbness/weakness	Typically absent
Facet loading test \ 30 second extension	May reproduce leg symptoms	Reproduces back pain on extension

Diagnosis: Clinical Criteria

N-CLASS (≥ 10 suggest LSS):

Item	Score
Age > 60	4
+ve 30 seconds extension test	4
Patient reports pain in both legs	3
Patient reports leg pain relieved by sitting	3
Patient reports leg pain decreased by forward leaning or flexing the spine	3
Negative straight leg raise (SLR) test <60° of passive hip flexion	2

Other Screening Tools:

- LSS-DST: 8-item decision support tool (Sensitivity 91%, Specificity 76%)
- LSS-SSHQ: Self-administered self-report questionnaire (Sensitivity 84%, Specificity 58%)
- NASS Criteria: Sensitivity 64%, Specificity 90%

Test	Sensitivity (95% CI)	Specificity (95% CI)	Positive LR (95% CI)	Negative LR (95% CI)
Historical features				
Age, y				
Age >65 (vs ≤65) ³⁸	0.77 (0.64-0.89)	0.69 (0.53-0.85)	2.5 (1.4-4.2)	0.34 (0.19-0.61)
>70 ^{58a}	NA	NA	2.0 (1.6-2.5)	
<60 ^{58a}	NA	NA	0.40 (0.29-0.57)	
Comorbidities				
Orthopedic disease ³⁰	0.18 (0.13-0.24)	0.91 (0.87-0.95)	2.0 (1.2-3.5)	0.90 (0.83-0.98)
Pain locations				
Bilateral buttock or leg ³⁷	0.51 (0.40-0.62)	0.92 (0.87-0.97)	6.3 (3.1-13)	0.54 (0.43-0.68)
Pain below buttocks ³⁸	0.88 (0.79-0.98)	0.34 (0.18-0.51)	1.4 (1.0-1.8)	0.34 (0.13-0.88)
Thigh ³⁷	0.95 (0.90-1.0)	0.14 (0.07-0.21)	1.1 (1.0-1.2)	0.36 (0.12-1.1)
Gluteal ³⁷	0.84 (0.75-0.92)	0.05 (0.01-0.09)	0.88 (0.79-0.98)	3.3 (1.2-8.8)
Symptoms reproduced by specific actions				
No pain when seated ³⁸	0.47 (0.32-0.61)	0.94 (0.85-1.0)	7.4 (1.9-30)	0.57 (0.43-0.76)
Burning sensation around the buttocks, intermittent prapism associated with walking, or both ³⁵	0.06 (0.03-0.09)	0.99 (0.98-1.0)	7.2 (1.6-32)	0.95 (0.92-0.98)
Improvement when bending forward ³⁵	0.62 (0.45-0.78)	0.92 (0.88-0.95)	6.4 (4.1-9.9)	0.52 (0.46-0.60)
Neurogenic claudication ³⁵	0.82 (0.77-0.87)	0.78 (0.73-0.83)	3.7 (2.9-4.8)	0.23 (0.17-0.31)
Improve when seated ³⁵	0.51 (0.36-0.66)	0.84 (0.72-0.97)	3.3 (1.4-7.7)	0.58 (0.41-0.81)
Exacerbation when standing up ³⁵	0.68 (0.62-0.74)	0.70 (0.65-0.76)	2.3 (1.8-2.8)	0.46 (0.37-0.56)
Exacerbated while standing up ³⁵	0.92 (0.88-0.96)	0.21 (0.15-0.27)	1.2 (1.1-1.3)	0.38 (0.21-0.69)
Other symptoms				
Urinary disturbance ³⁵	0.14 (0.09-0.19)	0.98 (0.96-1.0)	6.9 (2.7-17)	0.88 (0.83-0.93)
Numbness of perineal region ³⁵	0.05 (0.02-0.07)	0.99 (0.97-1.0)	3.7 (1.0-13)	0.97 (0.94-1.0)
Bilateral plantar numbness ³⁵	0.27 (0.21-0.33)	0.87 (0.83-0.92)	2.2 (1.4-3.2)	0.84 (0.76-0.92)
Treatment for symptoms needs to be repeated every year ³⁵	0.40 (0.33-0.47)	0.80 (0.75-0.86)	2.0 (1.5-2.8)	0.75 (0.65-0.86)
Wake up to urinate at night ³⁵	0.86 (0.81-0.91)	0.27 (0.21-0.33)	1.2 (1.1-1.3)	0.50 (0.33-0.78)
Physical examination				
Provocative tests				
No pain with flexion ³⁸	0.79 (0.67-0.91)	0.44 (0.27-0.61)	1.4 (1.0-2.0)	0.48 (0.24-0.96)
Symptoms induced by having patients bend forward ³⁵	0.18 (0.13-0.23)	0.63 (0.57-0.69)	0.48 (0.34-0.66)	1.3 (1.2-1.5)
Neuromuscular tests				
Wide-based gait ³⁸	0.42 (0.27-0.57)	0.97 (0.91-1.0)	13 (1.9-95)	0.60 (0.46-0.78)
Abnormal Romberg test result ^{38b}	0.40 (0.25-0.54)	0.91 (0.81-1.0)	4.2 (1.4-13)	0.67 (0.51-0.87)
Vibration deficit ³⁸	0.53 (0.39-0.68)	0.81 (0.68-0.95)	2.8 (1.3-6.2)	0.57 (0.40-0.82)
Pinprick deficit ³⁸	0.47 (0.32-0.61)	0.81 (0.68-0.95)	2.5 (1.1-5.5)	0.66 (0.48-0.91)
Weakness ³⁸	0.47 (0.32-0.61)	0.78 (0.64-0.92)	2.1 (1.0-4.4)	0.69 (0.49-0.96)
Absent Achilles reflex ³⁸	0.47 (0.32-0.61)	0.78 (0.64-0.92)	2.1 (1.0-4.4)	0.69 (0.49-0.96)

Diagnostic Workup

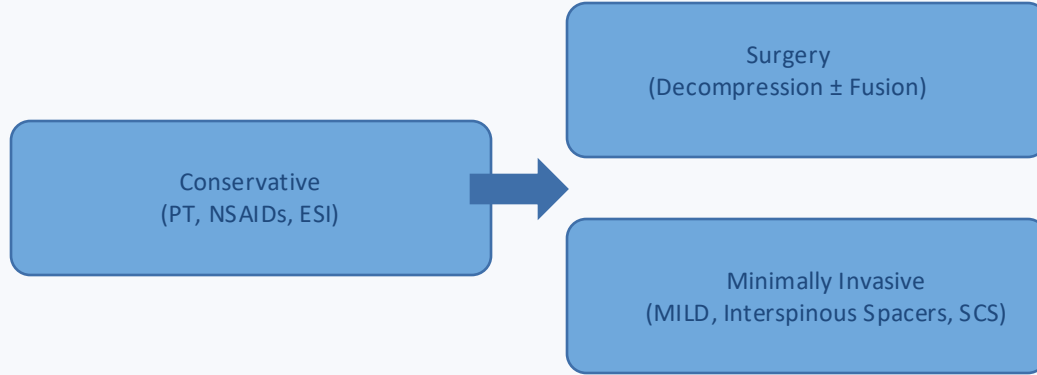
History & Physical:

- Symptom onset, posture dependence, walking tolerance
- Neurologic exam: 30 second extension test, sensory deficits, motor weakness, reflex change, wide-based gait [$>4''$ to prevent falls], Romberg's
- Vascular exam: pulses or ABI to rule out PA

Investigations:

- MRI lumbar spine: gold standard to assess canal and nerve root compression
- CT myelography: useful if MRI contraindicated
- Electromyography: evaluate radiculopathy or peripheral neuropathy
- Consider vascular studies if vasogenic claudication suspected
 - ABI < 0.9 is indicative of PAD
 - Duplex ultrasound

Treatment Overview



Start with non-operative care

Escalate to minimally invasive interventions when symptoms persist despite conservative measures

Consider open decompression if significant neurologic deficit, severe stenosis or failure of other therapies

Conservative Management

- Education & lifestyle: weight loss, avoid extension activities
- Physical therapy: core strengthening, flexion-based exercise, and **cardiovascular fitness [recumbent bike]**.
- Pharmacologic: NSAIDs, neuropathic agents, short-course opioids
- **Epidural steroid injections:**
Long-term relief with acute radicular symptoms
Short-term [~3-6 months] relief with chronic symptoms;

* Treating inflammatory vs mechanical radicular pain.
- Over 3 to 10 years, most patients with moderate symptoms experience either stable or improved symptoms. Only 10–20% experienced worsening.
- Trial of conservative care for ≥ 3 to 6 months before escalating

Evidence Summary

Conservative therapy may provide modest symptomatic improvement but rarely reverses stenosis. Many patients ultimately require interventional or surgical treatments.

MILD Procedure – Minimally Invasive Lumbar Decompression

- Minimally invasive lumbar decompression via a percutaneous portal – Incision the size of an **ASPIRN**
- Target: debulk hypertrophic ligamentum flavum at stenotic level
- Performed under fluoroscopic guidance using tissue sculptor and portal system
- Outpatient procedure; preserves bony architecture and stability
- Indications: **Moderate LSS** with relief in flexion after ≥ 6 months of conservative care and imaging confirmation
- Appropriate for non-surgical candidates or as a bridge to surgery; long-term outcomes and cost-effectiveness under investigation.



Figure 8a: Lateral view MRI reveals a thickened ligamentum flavum relative to the spinal canal



Figure 8b: Axial view MRI also demonstrates a thickened ligamentum flavum relative to the spinal canal

Figure 15: Positioning and Use of the mild Bone Rongeur



Figure 16a



Figure 16b



Figure 16c

MILD Outcomes (MOTION Study)

- 3 Year follow up data: 150 patients.
- Leg/back pain reduction maintained through 3 years
- Reoperation rate ~5.6%
- Minimal Clinically Important Difference [MCID] is an ODI of 10 for spine studies.

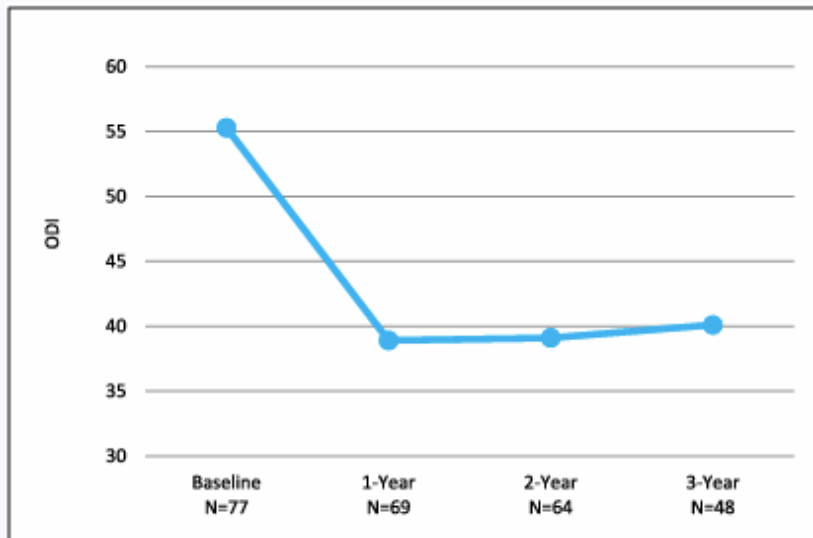


Fig. 2. Oswestry Disability Index (ODI) mean outcomes for the mild + CMM group.

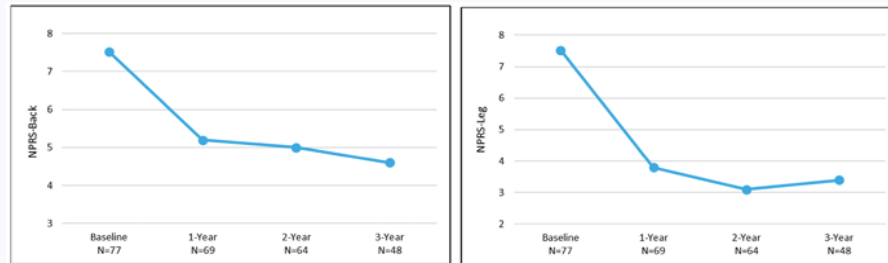
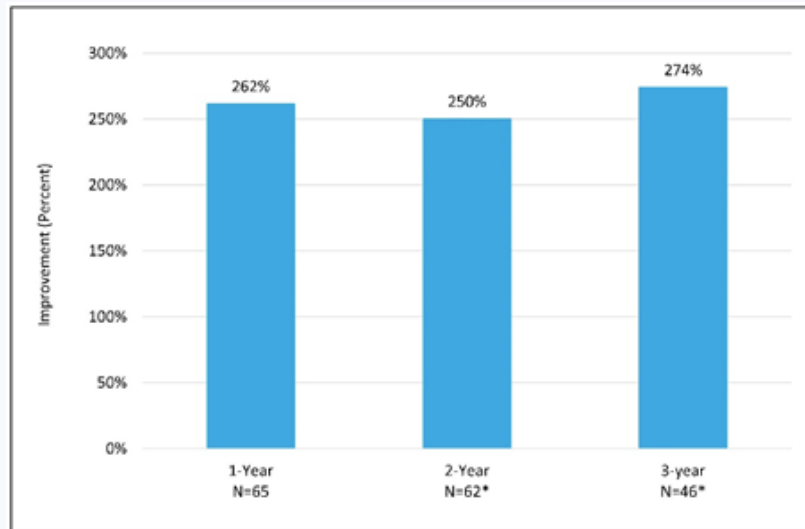


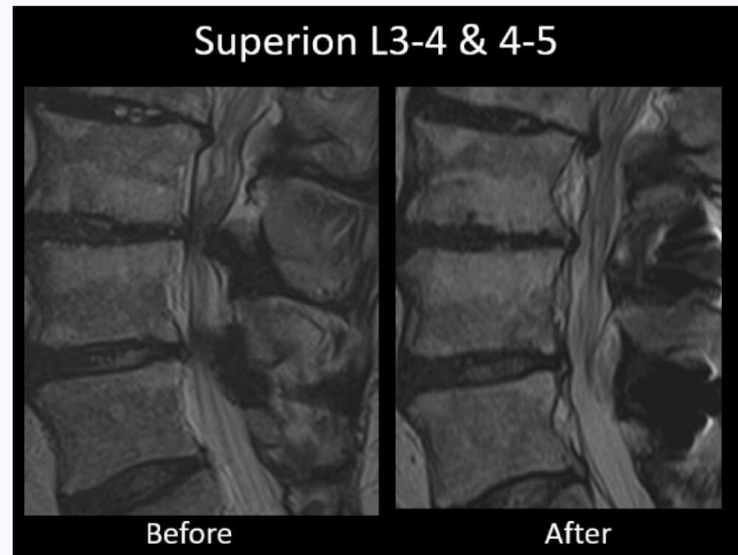
Fig. 3. Numeric Pain Rating Scale (NPRS) mean outcomes for back and leg pain for the mild + CMM group.



* Walking Tolerance Test (WTT) was not recorded during the follow-up visit for two patients

Interspinous Spacers

- Indications: 25% - 50% reduction in canal or foraminal width who receive relief with flexion and have moderate physical function impairment on some disability scale.
- Mechanism: distracts spinous processes, flexes the segment, unloads facets and increases canal area.
- Minimally invasive; can be performed under local or general anesthesia.
- May serve as a bridge for those unfit for or unwilling to undergo open surgery
- 3.5% revision rate within 1 year.
- 5%-15% reoperation at 2 – 5 years. 25% reoperation rate after 5 years. This is higher than surgical decompression.



Interspinous Spacers – Outcomes

4-year Superion study: 40% improvement in ZCQ scores; 73% leg pain relief; 69% back pain relief; 61% ODI improvement

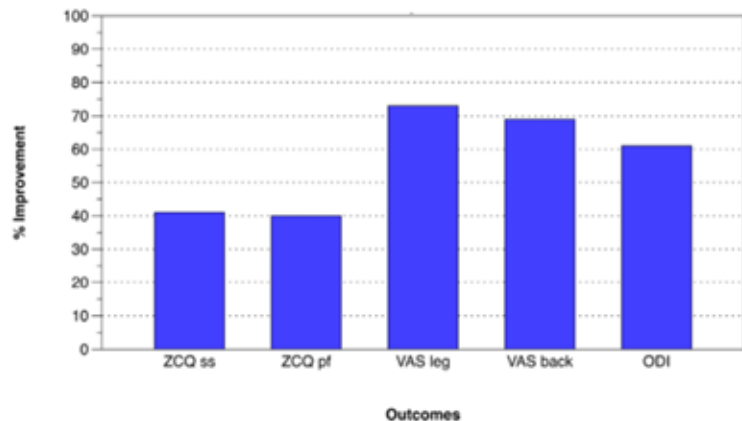


Figure 4. Percentage improvement for each outcome at 4 years compared with preoperative levels. All changes were statistically significant ($P < 0.001$). ZCQ, Zurich Claudication Questionnaire; ss, symptom severity; pf, physical function; VAS, visual analog scale; ODI, Oswestry Disability Index.

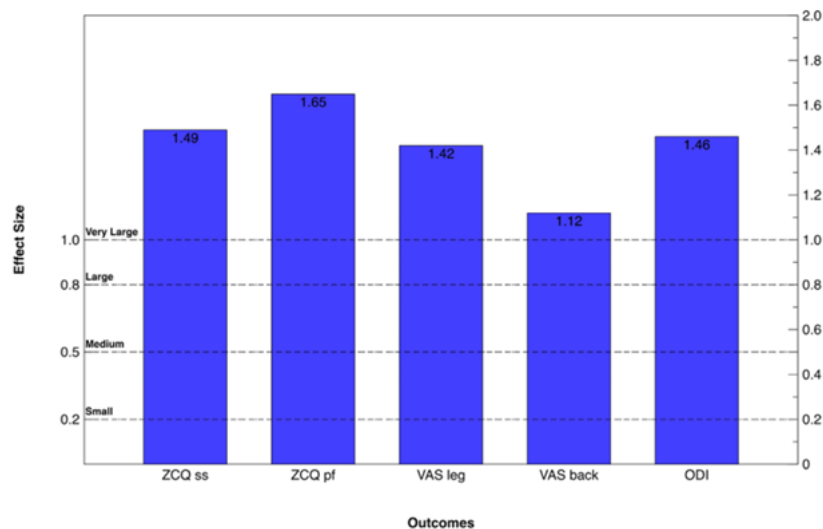


Figure 5. Within-group effect sizes for each outcome at 4 years. All effect sizes exceeded the very large threshold and were highly statistically significant

($P < 0.0001$). ZCQ, Zurich Claudication Questionnaire; ss, symptom severity; pf, physical function; VAS, visual analog scale; ODI, Oswestry Disability Index.

Spinal Cord Stimulation

- Limited studies (3) that investigate SCS to treat lumbar stenosis.
- Established as beneficial when treating CRPS, DPN, Persistent Spinal Pain Syndrome Type I [No Prior Surgery] and Type II [Failed Back Surgery Syndrome]
- Epidural leads deliver electrical pulses to dorsal columns to modulate pain pathways
- IPG implanted in gluteal or flank region; various waveforms (sub-perception and paresthesia-based)
- Trial phase precedes permanent implantation



SCS: Key Studies & Waveforms

Study / Design	Population / Indication	Waveform	Key Findings
Van Buyten 2013 (Prospective)	82 chronic back pain patients	HF 10 kHz	88% responders; VAS 8.4→2.7
Kapural 2016 (RCT)	198 chronic back/leg pain patients	HF 10 kHz vs tonic [40-60 Hz]	76% vs 49% responders at 24 months. 67% vs 41% pain relief
Pain Physician 2021 (Prospective)	118 LSS patients with neurogenic claudication	Mixed (paresthesia + HF)	86% trial success; 80% sustained relief at 27 months
Hara 2022 (RCT)	50 post-surgery radiculopathy patients	Burst (sub-perception)	No significant benefit over sham
SENZA-PDN 2023 (RCT)	216 painful diabetic neuropathy	HF 10	80% mean pain reduction; 90% ≥50% relief. Mean decrease of over 65% in the pain and sleep questionnaire

DISTINCT Trial: BurstDR SCS vs CMM

- Multicentre randomized controlled trial comparing passive recharge BurstDR SCS with conventional medical management in chronic low back pain patients without surgical options
- 115 enrolled; 50 patients received permanent paddle leads after successful trial
- Pain reduction: mean NRS decreased from 7.8 at baseline to ~2.0 at 6–12 months
- Disability improvement: ODI dropped from 54.4 to ~20; 85.7% achieved ≥ 13 -point improvement and 76.2% achieved ≥ 20 -point improvement
- High satisfaction and safety: 93% of patients reported moderate to much improvement; adverse event rate was low (~4% explant, 2% revision)

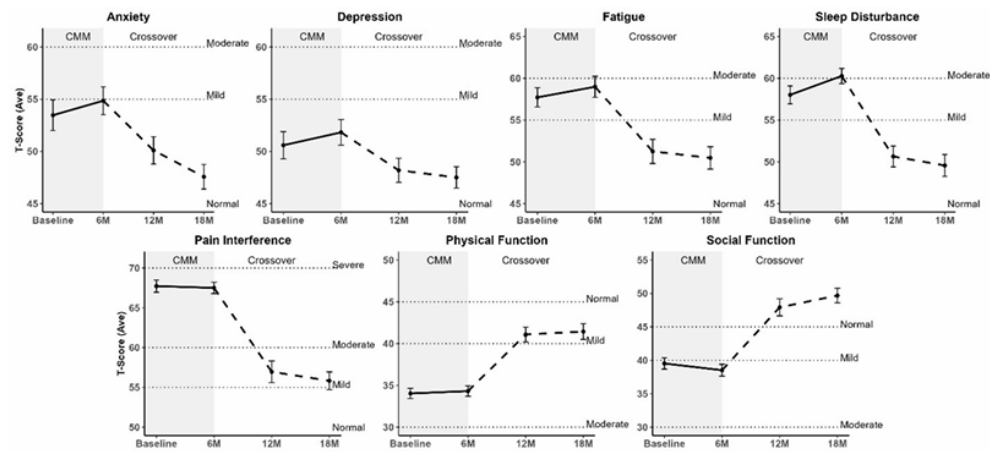


Figure 5 Patients in CMM group reported improvements in all PROMIS 29 domains when compared to their baseline and 6M scores. Patients reported normal scores for Anxiety, depression, Fatigue, Sleep disturbance and social function. Patients reported mild symptoms for pain interference and physical function.

SCS: Clinical Takeaways

- Candidate profile: chronic back/leg pain after failed conservative therapy or prior surgery; no significant mechanical instability or severe stenosis.
- High-frequency (10 kHz) therapy demonstrates superior responder rates and pain reduction over conventional tonic SCS
- Sub-perception burst SCS has mixed evidence in LSS; not superior to sham in one trial of post-operative patients.
- SCS serves as a bridge for non-surgical candidates or as salvage for persistent pain after decompression
- Discuss risks (lead migration, infection, reoperation) and long-term costs during shared decision-making

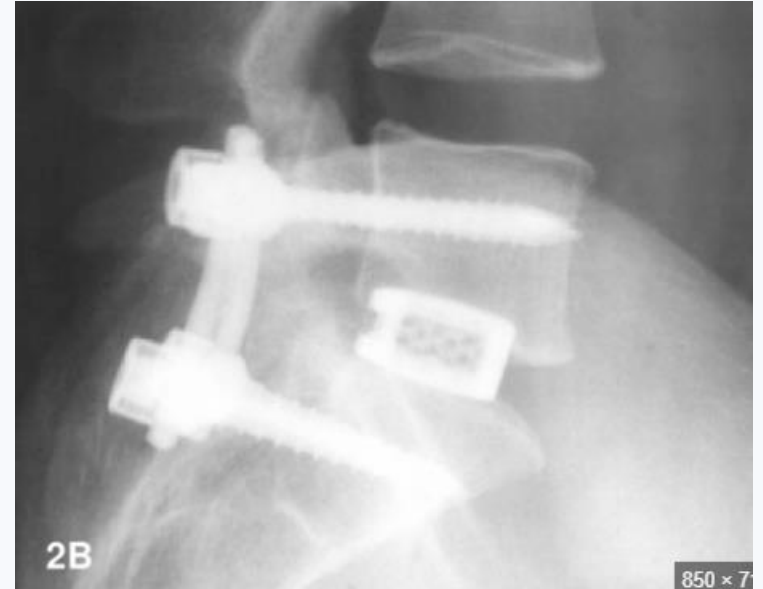
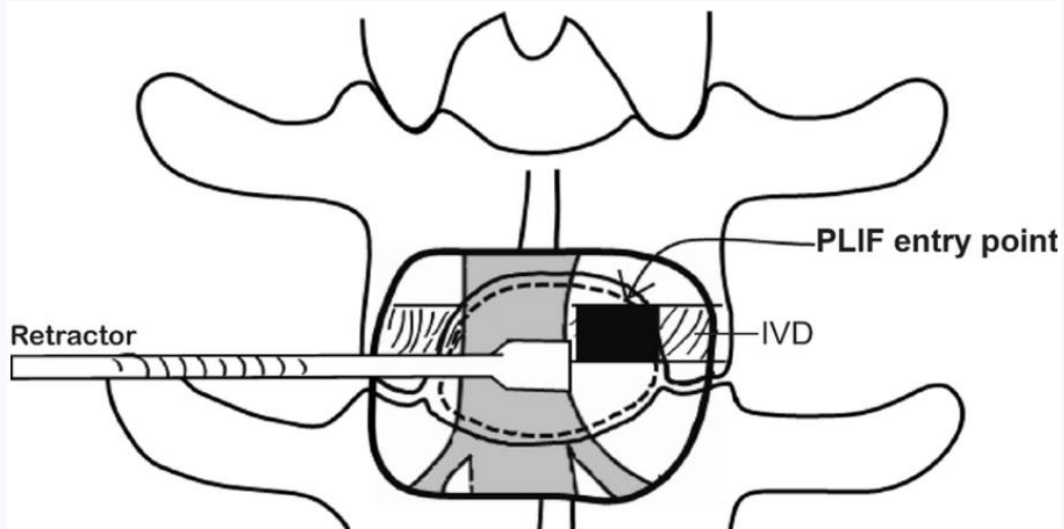
Surgical Management: Indications & Procedures

- Consider surgery after ≥ 6 months of conservative therapy when disabling neurogenic claudication or radicular pain persists
- Emergent surgery for progressive motor weakness or cauda equina syndrome
- Decompressive laminectomy/laminotomy is the gold-standard operation: improves pain, walking tolerance and function in appropriately selected patients
- Decompression alone yields outcomes comparable to decompression + fusion in most degenerative stenosis and low-grade spondylolisthesis. Fusion adds operative time, blood loss, hospital stay and morbidity without superior outcomes in most cases
- Fusion pursued when clear instability or deformity exists (e.g., high-grade spondylolisthesis or significant motion on flexion–extension)

Surgical Management: Consideration of Fusion

- **Spinal Instability:** Dynamic instability on flexion-extension X-rays (typically >3–4 mm translational movement or >10–15° angulation at a stenotic level). A patient with a mobile spondylolisthesis or gross instability (e.g. due to facet erosion or pars defect) would benefit from fusion to prevent progression of slip after decompression.
- **High-Grade Spondylolisthesis:** Grade II or higher degenerative spondylolisthesis (> 25% slip) at the affected level is often fused, as there is greater risk of slip progression or postoperative segmental instability after wide decompression.
- **Coronal or Sagittal Imbalance:** Patients with concomitant degenerative scoliosis (coronal curve) or sagittal imbalance may require fusion with deformity correction to address the global alignment issues along with stenosis.
- **Extensive Facet Resection:** If an adequate decompression necessitates removal of both facet joints at a level (which would destabilize that segment), then an instrumented fusion is indicated to maintain stability after laminectomy. For example, multilevel laminectomies in the presence of a scoliosis often include fusion because of the amount of facet resection needed.
- **Revision Surgery:** In cases of recurrent stenosis after prior laminectomy, especially if there is post-laminectomy instability (“flat-back” or iatrogenic spondylolisthesis), adding fusion is frequently recommended.

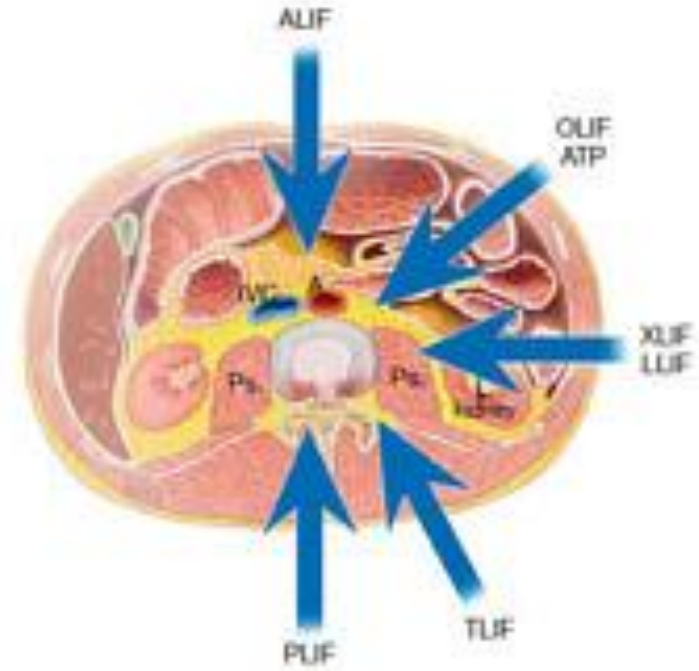
Decompression and Fusion



Cross-sectional view of lumbar fusion approaches

Interbody Fusion Approaches

- Technique choice depends on pathology, level, instability and deformity correction, as well as patient anatomy and comorbidity.
- ALIF: anterior abdominal approach; restores disc height and lordosis; best for L4–5 and L5–S1; risk of vascular injury; limited use at higher levels.
- LLIF/XLIF: lateral or extreme lateral approach through (or anterior to) the psoas; minimally invasive; **avoids posterior musculature**; not suitable for L5–S1; risk of lumbar plexus injury
- TLIF: transforaminal approach via **unilateral facetectomy**; reduces nerve retraction relative to PLIF; widely applicable across lumbar levels
- PLIF: posterior midline approach with laminectomy and bilateral nerve root retraction; allows bilateral decompression and robust fixation but incurs greater muscle disruption and blood loss



Cross-sectional view of lumbar fusion approaches

Surgical Management: Outcomes & Considerations

- Post-operative expectations: light activities within weeks; maximal improvement in 3–9 months
- Most patients maintain pain relief and functional gains; reoperation rates range from 10–22% at five+ years
- Recurrence or need for further intervention often due to progressive degeneration or inadequate decompression
- Long-term, fusion carries added risks of non-union, adjacent-segment disease, infection and hardware failure
- Interspinous spacers provide indirect decompression but have higher reoperation rates than open decompression
- Minimally invasive / endoscopic techniques reduce blood loss and hospital stay while achieving similar short-term results. There is a learning curve, added cost of equipment that remain as barriers.

Conclusions

- Conservative therapy first for ≥ 3 -6 months unless emergent deficit (cauda equina, progressive weakness). Typically, only successful when treating **moderate** stenosis.
- Decompressive laminectomy without fusion is sufficient for most degenerative LSS; add fusion only when instability or deformity present.
- Consider MILD or interspinous spacers for moderate stenosis with predominant ligamentum flavum hypertrophy when patients wish to avoid open surgery or are not candidate.
- SCS reserved for persistent pain in non-surgical candidates or after failed surgery; trial required before permanent implant.
- Shared decision-making is essential; discuss risks, benefits, and patient goals.

Thank you!

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