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Lumbar Spinal Stenosis: Multidisciplinary Care Pathway for Radiology, Nursing & Physical Therapy

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Abstract

Background: Cracked tooth syndrome (CTS) is a prevalent, diagnostically challenging condition with heterogeneous **Background:** Lumbar spinal stenosis (LSS) is a leading cause of pain, gait limitation, and surgery in older adults. Because symptoms reflect both fixed anatomic narrowing and posture-dependent dynamics, single-modality decisions underperform.

Aim: To outline a multidisciplinary care pathway integrating radiology, nursing, and physical therapy (PT) to improve diagnostic accuracy, functional outcomes, and value.

Methods: Narrative synthesis of core domains—anatomy, etiology, epidemiology, pathophysiology, history/physical, evaluation, treatment, prognosis, complications, patient education, and team operations—emphasizing actionable links between imaging phenotypes (central, lateral recess, foraminal, extraforaminal) and bedside/rehabilitation decisions.

Results: Flexion widens canal/foramina; extension worsens crowding—an insight that aligns patient education and PT toward flexion-biased programs. MRI remains first-line; axial-loading MRI and CT/myelography resolve discrepancies or contraindications. Standardized MRI grading (e.g., Schizas/Chen Jia/Lee) plus functional metrics (SPWT, ODI) enable decision-ready reports. Epidural injections provide short-term relief with modest effects on walking; medial branch radiofrequency helps facet-predominant pain. When needed, decompression—open, minimally invasive, or endoscopic—improves pain and function; fusion is reserved for instability/deformity. Interspinous spacers benefit selected extension-sensitive, one-to-two-level disease without osteoporosis/instability.

Conclusion: A pathway that matches mechanism to modality—precise radiology, nurse-led safety/education, and PT-led graded conditioning—yields faster, safer diagnosis and individualized care, limiting unnecessary procedures while preserving surgical benefit for appropriate candidates.\

Keywords: lumbar spinal stenosis; neurogenic claudication; MRI; axial-loading imaging; physical therapy; epidural injection; decompression; multidisciplinary pathway.

1. Introduction

Within the framework of a multidisciplinary care pathway for radiology, nursing, and physical therapy, a precise grasp of vertebral anatomy is foundational for diagnosis, bedside assessment, and rehabilitation planning. Macroscopically, the vertebral column is organized into anterior and posterior structural regions (see Image. Lumbar Vertebral Anatomy). The anterior spine is formed by a column of cylindrical vertebral bodies separated by intervertebral (IV) disks and stabilized longitudinally by the anterior and posterior longitudinal ligaments. Each IV disk is a composite structure with a gelatinous nucleus pulposus centrally, encased by the fibrocartilaginous annulus fibrosus (see Image. Intervertebral Disk). The cervical and lumbar segments possess the greatest disk height and surface area, reflecting their enhanced ranges of motion and load-bearing demands. Functionally, this anterior complex behaves as the primary shock-absorbing apparatus for axial loading, attenuating forces transmitted during posture changes, gait, and lifting—features directly relevant to physical therapy programs that target segmental stabilization and controlled loading. The posterior spine is constituted by the vertebral arch and its processes. Each arch comprises

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paired anterior pedicles and posterior laminae (see Image. Lumbar Vertebra, Superoposterior View). Projecting from the arch are two lateral transverse processes, a single midline spinous process, and paired superior and inferior articular processes whose apposition forms the zygapophyseal (facet) joints. Together, the vertebral bodies and IV disks anteriorly, with the vertebral arches posteriorly, delineate the spinal canal, which encloses the thecal sac and neural elements. Segmental nerve roots emerge through the intervertebral canals (neural foramina), typically traveling superior to their eponymous vertebral body, an anatomical relationship of direct consequence for radiologic localization of foraminal and extraforaminal pathology and for nursing neurologic screening at the bedside. Bridging adjacent laminae is the ligamentum flavum, a dense, elastic structure whose hypertrophy in degenerative states contributes to dorsal encroachment on neural space.

Of particular clinical importance for lumbar spinal stenosis is the lateral recess, an anatomically constrained corridor bounded anteriorly by the vertebral body and disk, posteriorly by the ligamentum flavum and arch, laterally by the pedicle, and medially by the thecal sac. This recess, by virtue of its narrow baseline dimensions, is a common locus for nerve root compression when degenerative or space-occupying changes arise. In aggregate, the posterior elements safeguard the neural axis and serve as anchor points for musculoligamentous attachments—considerations that guide both physical therapy strategies to optimize paraspinal endurance and nursing education on posture, body mechanics, and activity pacing. For radiologists, nuanced knowledge of these compartments—central canal, lateral recess, foraminal, and extraforaminal—underpins precise reporting that aligns with surgical and rehabilitative decision-making across the pathway.

Lumbar Spinal Stenosis

Lumbar spinal stenosis (LSS) denotes pathologic narrowing within the lumbar spine that may involve the central canal, lateral recess, or neural foramina [1]. Central canal compromise can compress the thecal sac and multiple bilateral nerve rootlets; in advanced cases this pattern produces bilateral neurogenic manifestations consistent with diffuse central encroachment. By contrast, lateral recess or foraminal restriction typically impinges a single exiting or traversing root and therefore presents unilateral lumbar radicular symptoms and signs [2]. In the context of a multidisciplinary pathway, these anatomic–clinical correlations inform radiologic mapping (central vs lateral/foraminal), nursing triage of laterality and red flags, and physical therapy dosing of flexion-bias or decompressive positions tailored to the symptomatic root distribution. Central canal stenosis commonly evolves from dorsal ligamentum flavum hypertrophy in concert with ventral disk bulging, a degenerative coupling that narrows the anteroposterior canal diameter. The L4–L5 level is most frequently affected, reflecting its high mobility and cumulative mechanical load. Lateral recess stenosis is typically driven by facet arthropathy and osteophyte formation, which constrict the zone traversed by the nerve root before it reaches the foramen. Foraminal stenosis arises with disk height loss, foraminal disk protrusion, or marginal osteophytes, collectively reducing the cross-sectional area available to the exiting root within the intervertebral foramen. Beyond the foramen, extraforaminal stenosis most often reflects far-lateral disk herniation, compressing the nerve root after it has exited laterally [3]. These mechanistic distinctions translate directly to imaging protocols (supine MRI with attention to sagittal and axial foraminal sequences; consideration of weight-bearing or extension CT/MRI where available), nursing counseling (activity modification to avoid sustained extension that accentuates facet loading and recess narrowing), and physical therapy selection of flexion-biased exercises and positional decompression to maximize the remaining neural reserve.

From a population-health perspective, LSS constitutes a major source of disability among older adults and is one of the leading indications for spinal surgery in individuals over 65 years of age [4][5][6]. This epidemiologic burden mandates coordinated screening for functional decline, gait instability, and falls risk within nursing assessments; imaging stratification and standardized reporting lexicons within radiology services; and conservative care pathways within physical therapy emphasizing graded activity, trunk endurance, and gait training. Historically, Henk Verbiest delineated relative and absolute canal stenosis using midsagittal diameter thresholds of <12 mm and <10 mm, respectively—metrics that, while heuristically valuable, do not capture the full clinical heterogeneity. Indeed, despite the global prevalence of LSS, there remains no universally accepted clinical or radiologic case definition, and formal diagnostic criteria are not standardized [7][8]. Consequently, multidisciplinary teams must integrate symptom provocation patterns (e.g., neurogenic claudication improved by flexion), objective functional impairment, and imaging correlates rather than relying on single-dimension measurements alone.

Radiology plays a central role in phenotyping the stenotic compartment(s), quantifying canal and foraminal compromise, and identifying contributory features—disk contour, facet joint orientation and hypertrophy, capsular cysts, ligamentum flavum thickness, and dynamic factors suggested by posture-dependent changes. Structured reports that explicitly label central, lateral recess, foraminal, and extraforaminal involvement map directly onto surgical planning and physical therapy emphasis (e.g., flexion relief patterns), while also guiding nursing education about symptom trajectories and red flags (progressive weakness, saddle anesthesia, bladder changes).

Nursing integrates anatomical knowledge with patient-facing surveillance. Focused neurologic examinations that document dermatomal pain distribution, motor strength, and reflexes provide a baseline to monitor progression. Education emphasizes postural hygiene—limiting prolonged lumbar extension and heavy axial loading—safe ambulation strategies and pacing to reduce claudication. Nurses also coordinate conservative measures (heat/ice, analgesic timing, sleep positioning in slight flexion) and ensure adherence to follow-up when new deficits or red-flag symptoms emerge, facilitating timely escalation.

Physical therapy operationalizes anatomy into movement: programs prioritize flexion-biased mobilizations, trunk and hip strengthening, neuromuscular control, and endurance training to redistribute loads away from compromised posterior elements. Manual therapy and neural mobilization may complement strengthening where appropriate, while graded walking in slightly flexed postures (e.g., using a walker or treadmill incline) leverages the known canal-widening effect of lumbar flexion. Therapists translate radiologic compartmental findings into exercise selection, e.g., cautious facet loading in lateral recess disease—and partner with nursing on patient-specific activity plans. Finally, across the pathway, teams should recognize that symptom severity and disability do not correlate perfectly with static diameter thresholds. A comprehensive approach—clinical pattern recognition, functional assessment, and high-quality imaging interpretation—is therefore paramount. When conservative care does not achieve goals, radiology's targeted characterization supports procedural planning (e.g., interlaminar vs transforaminal injections, decompression levels), nursing prepares patients for peri-procedural care and expectation management, and physical therapy transitions toward postoperative rehabilitation or persistent conservative optimization. In this way, the anatomical and pathophysiological insights outlined above directly animate the multidisciplinary care pathway for radiology, nursing, and physical therapy, aligning daily actions with patient-centered outcomes in lumbar spinal stenosis [1][2][3][4][5][6][7][8].

Etiology

Lumbar spinal stenosis (LSS) arises from a spectrum of congenital and acquired processes, with degenerative spondylosis representing the predominant pathway in routine practice. Within the multidisciplinary care pathway that unites radiology, nursing, and physical therapy, understanding *why* the canal narrows—i.e., the mechanical and biologic drivers of constriction in the central canal, lateral recess, and neural foramina—directly informs imaging protocols, bedside surveillance, and movement-based rehabilitation strategies. Aging and cumulative microtrauma accelerate intervertebral disk dehydration and height loss, shifting axial load posteriorly toward the facet joints and posterior elements. This load redistribution fosters posterior disk bulging and stimulates reactive bone formation along the vertebral ring apophyses, generating posterior vertebral osteophytes. Simultaneously, the facet capsules undergo laxity and hypertrophy; synovial facet cysts may emerge from degenerated joints; and the ligamentum flavum thickens and buckles into the canal as elastin fragments and collagen proliferates. The aggregate effect is progressive encroachment upon central and lateral neural compartments, a pathoanatomic cascade that explains the clinical shift from positional discomfort to neurogenic claudication as reserve space is consumed. From an anatomic–biomechanical standpoint, intervertebral disk degeneration is the inciting event that increases posterior element loading. Posterior annular weakening allows mild posterior protrusion, which narrows the ventral canal and reduces foraminal height. As the disk collapses, the superior articular process migrates cephalad into the foramen, while the ligamentum flavum infolds dorsally; these coupled changes decrease the cross-sectional area available to the traversing and exiting roots. For the radiologist, this cascade maps to reproducible findings on MRI—disk desiccation and bulge, facet arthropathy with capsular hypertrophy, synovial cysts of variable T2 signal, and ligamentum flavum thickening—often most conspicuous at L4–L5, the segment with the greatest mobility and shear exposure. For nurses and physical therapists, the same cascade justifies a flexion-bias in activity planning, because lumbar flexion transiently enlarges canal and foraminal dimensions by unloading the facets and tensioning the posterior longitudinal structures.

A second, clinically salient etiologic pathway is degenerative spondylolisthesis, in which accumulated degeneration can be accompanied by, or progress to, defects of the pars interarticularis with ensuing segmental instability. This instability permits anterior translation (anterolisthesis) of one vertebral body on another—most commonly at L4–L5—further narrowing the canal and foramina by telescoping posterior elements and aggravating ligamentum flavum infolding. The dynamic component of this process explains the frequent extension-provoked exacerbation of symptoms and the partial relief with flexion or sitting. Radiology contributes by quantifying slip percentage, facet orientation, and dynamic motion on flexion–extension radiographs when indicated; nursing integrates fall-risk screening and red-flag monitoring (new weakness, bladder changes); and physical therapy prioritizes trunk and hip stabilization to diminish shear across the involved level while coaching patients to avoid sustained extension postures that accentuate stenosis. Although degeneration and spondylolisthesis account for the majority of LSS seen in older adults, a range of less common acquired causes can also constrict the canal. Space-occupying lesions (e.g., synovial cysts, epidural masses), post-surgical epidural fibrosis, rheumatologic disorders, and skeletal diatheses such as ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis (DISH) may all produce fixed or dynamic narrowing and symptomatology [9]. In postsurgical patients, scar-mediated tethering can reduce dorsal epidural compliance, producing recurrent claudication despite adequate bony decompression. In inflammatory spondyloarthropathies, enthesopathic new bone and capsular thickening stiffen the posterior elements, functionally shrinking recess and foraminal corridors. These etiologies require radiologic discrimination—distinguishing fibrosis from recurrent disk on contrast-enhanced MRI, identifying inflammatory versus degenerative patterns—and careful nursing history to surface prior procedures or systemic inflammatory disease, while physical therapy adapts load-management and mobility work to the underlying pathobiology (e.g., gentle mobility for inflammatory stiffness versus graded stabilization for instability-driven disease).

Congenital contributors to LSS are rarer but clinically important. Conditions such as achondroplasia produce inherently short pedicles and medially positioned facets, yielding a congenitally narrowed canal that may decompensate with superimposed degenerative changes [9]. In these patients, modest posterior hypertrophy or disk bulge can have outsized symptomatic consequences because baseline reserve space is minimal. Radiologic recognition of the congenital architecture prevents over-attribution to minor degenerative findings; nursing teams anticipate earlier or more severe symptom onset; and physical therapists dose exercise intensity conservatively, avoiding abrupt extension loads that further diminish canal diameter. Across this etiologic landscape, three themes align with the multidisciplinary care pathway. First, phenotype the stenosis: central, lateral recess, foraminal, and extraforaminal patterns emerge from distinct combinations of disk, facet, ligament, and alignment changes, and each pattern carries implications for imaging sequences, clinical monitoring, and exercise prescription. Second, recognize dynamics: extension often worsens canal compromise by facet impaction and ligamentum flavum buckling, whereas flexion can transiently improve caliber—guiding both patient education (nursing) and graded, flexion-biased programming (physical therapy). Third, contextualize comorbidity and history: prior surgery, systemic inflammatory disease, or skeletal dysplasias alter both the mechanism and the response to conservative care, and radiology's precise characterization enables tailored plans rather than one-size-fits-all algorithms.

In sum, LSS can be congenital or acquired, with degenerative spondylosis and degenerative spondylolisthesis as principal acquired mechanisms that interact with age, cumulative loading, and, at times, pars defects to narrow neural pathways. Additional contributors—space-occupying lesions, post-surgical fibrosis, rheumatologic disorders, and skeletal diseases—as well as congenital dysplasias like achondroplasia—round out the etiologic differential [9]. Translating these causes into practice, the radiologist delineates compartments and dynamics; the nurse tracks risk factors, educates on posture and red flags, and coordinates care; and the physical therapist leverages biomechanics to reduce symptomatic load while building capacity.

Epidemiology:

Epidemiologic characterization of lumbar spinal stenosis (LSS) remains challenging because no single, universally accepted clinical–radiologic definition exists. This definitional gap yields heterogeneity across studies in case ascertainment, imaging thresholds, and symptom criteria, thereby complicating prevalence and incidence estimates and, in turn, the planning of services across radiology, nursing, and physical therapy. Morphometric descriptors (e.g., canal midsagittal diameter or cross-sectional area), qualitative grading on MRI or CT, and symptom-driven classifications (neurogenic claudication, radicular pain) are variably employed, and each captures a different slice of the LSS construct. Against this backdrop, several population and clinic-based investigations still provide important anchors for the multidisciplinary care pathway.

In structural terms, an ancillary analysis from the Framingham cohort reported that 19.4% of participants aged 60–69 years exhibited an internal canal diameter <10 mm, a threshold historically associated with "absolute" stenosis [10]. While this measure reflects an anatomic substrate rather than clinical impairment, it highlights the substantial reservoir of individuals with potentially limited neural reserve—information that is directly actionable for radiologists when standardizing reports and for nurses and physical therapists when counseling older adults about posture, pace, and load-management strategies. Complementing these morphometric data, a Japanese population-based survey documented age-related increases in symptomatic LSS: 1.9% (40–49 years), 4.8% (50–59), 5.5% (60–69), and 10.8% (70–79) [10]. These gradients underscore how degenerative remodeling accumulates over decades, progressively narrowing the canal and foramina and raising the likelihood of neurogenic claudication that becomes clinically relevant to triage and conservative rehabilitation.

From a health-services perspective, LSS affects more than 200,000 individuals in the United States and is the most common indication for spinal surgery in patients >65 years, emphasizing its public-health footprint and the need for disciplined, stepwise care to avoid premature procedural escalation [11]. For the care pathway, these figures translate into pragmatic imperatives: radiology must provide precise compartmental phenotyping that can guide nonoperative planning; nursing must implement systematic screening for functional decline, gait limitation, and falls risk; and physical therapy should offer accessible, flexion-biased programs that mitigate symptoms and delay or obviate the need for surgery in appropriate candidates. Etiologic heterogeneity also extends to host biology. A genetic component has been identified, with aberrant gene expression implicated in pathways that drive osteophyte proliferation of vertebral bodies and facets, ligamentum flavum hypertrophy, and intervertebral disk degeneration [12]. For clinicians, this reinforces that LSS is not purely a mechanical consequence of aging and load; inherent tissue remodeling propensities may predispose some individuals to earlier or more severe stenosis. Radiologists may encounter disproportionate hypertrophy or osteophytosis relative to chronological age, nurses can integrate family history and systemic features into risk screening, and physical therapists can tailor expectations when progression reflects biology as much as biomechanics.

A striking feature of LSS epidemiology is the discordance between radiologic stenosis and symptoms. Among adults older than 40, the radiologic prevalence of moderate and severe stenosis can reach ~80% and ~40%, respectively; in the U.S., ~11% of older adults are affected clinically, 20% of those >60 have imaging evidence of LSS, and paradoxically ~80% of such individuals remain asymptomatic [13]. This gap has profound consequences for the multidisciplinary pathway. For radiology, it cautions against equating imaging severity with symptomatic disease; structured reports should contextualize findings with potential clinical relevance. For nursing, it supports careful correlation of imaging with patient-reported function, not just pain intensity. For physical therapy, it validates trialing conservative management—even in the presence of “severe” imaging—when red flags are absent, because symptoms often respond to flexion-biased conditioning and gait modification irrespective of static canal dimensions. Synthesis efforts mirror this complexity. A systematic review estimated the pooled prevalence of LSS at ~11% in the general population and 25–39% in clinical settings, reflecting selection enrichment as symptomatic individuals present for care [14]. Downstream utilization patterns further illustrate burden: among patients with lumbar degeneration, 5.9 per 100 undergo lumbar fusion within one year of diagnosis—a metric that underscores both disease severity in a subset and the importance of optimizing nonoperative care pathways prior to surgery [14]. For our pathway, these data argue for robust front-end triage, standardized conservative protocols, and clear criteria for escalation, thereby aligning imaging interpretation, nursing surveillance, and therapy dosing with value-based care.

Several additional points sharpen epidemiologic interpretation for practice. First, modality and posture matter: most prevalence estimates derive from supine MRI/CT, which may underestimate dynamic stenosis that manifests during upright extension—an insight guiding radiologists to describe features suggestive of posture sensitivity (facet hypertrophy, ligamentum flavum infolding) and guiding physical therapists to leverage flexion postures that increase canal and foraminal caliber. Second, comorbidity clusters—hip/knee osteoarthritis, obesity, deconditioning—likely influence symptom expression and functional limitation despite similar imaging, reinforcing nursing’s role in holistic assessment and fall-prevention planning. Third, geographic and cultural differences in activity patterns and healthcare access may partly explain variation across studies, as suggested by the contrast between the Japanese symptomatic data and U.S. service utilization [10][11][14]. In sum, the epidemiology of LSS is best conceived as a continuum that spans anatomic narrowing common in aging populations, variable symptom penetration, and heterogeneous care trajectories. Anchoring management in a *Multidisciplinary Care Pathway for Radiology, Nursing & Physical Therapy* reconciles these layers: radiology delineates where and how the space is narrowed; nursing adjudicates clinical significance through function and safety screens; and physical therapy deploys graded, flexion-biased interventions that address impairment while monitoring for progression. By integrating population signals—prevalence gradients with age [10], national burden and surgical indications [11], genetic predisposition [12], high radiologic prevalence with frequent asymptomatic status [13], and pooled estimates with notable procedural rates [14]—teams can calibrate resource allocation, patient education, and escalation thresholds, ultimately delivering care that is both evidence-aligned and person-centered.

Pathophysiology:

Within the multidisciplinary care pathway for radiology, nursing, and physical therapy, the pathophysiology of lumbar spinal stenosis (LSS) is best understood as a progressive, load-redistribution phenomenon that remodels the motion segment and constricts neural passageways. Repeated microtrauma from everyday posture, gait, and occupational or recreational loading—especially in the presence of deconditioning and weakening of the axial musculature—accelerates intervertebral (IV) disk desiccation, a hallmark of degenerative disc disease. As disk height diminishes and hydrostatic properties decline, axial forces are shifted posteriorly onto facet joints and posterior ligamentous structures. This maladaptive load transfer precipitates facet arthropathy, marginal osteophyte formation, and synovial facet cyst development, while simultaneously promoting ligamentous thickening and buckling, collectively narrowing the central canal, lateral recesses, and neural foramina. The resulting anatomic encroachment explains the characteristic evolution from positional low back pain to activity-limited neurogenic claudication as canal "reserve" is consumed.

Intrinsic osseous dimensions modulate susceptibility to this degenerative cascade. Notably, the anteroposterior (AP) diameter of the bony canal—L5 in males and L4 in females—emerges as a dominant structural risk factor for degenerative LSS; individuals with smaller baseline AP diameters reach symptomatic thresholds earlier as superimposed degenerative changes accrue [15]. Moreover, combined morphometric features, including the interplay between canal caliber and vertebral body dimensions, further influence the likelihood and tempo of stenosis, reminding clinicians that absolute millimeter cutoffs incompletely capture risk. For radiologists, these relationships argue for standardized reporting that integrates both canal and foraminal metrics; for nurses and physical therapists, they validate education on posture modification and conditioning strategies that minimize extension loading in anatomically constrained spines. Among the soft-tissue contributors, ligamentum flavum hypertrophy represents a pivotal pathological variable driven by mechanical stress and repetitive shear across the posterior elements [16]. Histopathologic analyses demonstrate that thickening of this elastic ligament reflects multispectral remodeling—a convergence of fibrosis, chondroid metaplasia, and even amyloid deposition within the ligamentous matrix [17]. Microstructurally, investigators have documented elastic fiber degeneration, relative collagen overabundance, fibrotic scarring (cicatrization), and calcific change, all of which stiffen the ligament and promote dorsal infolding into the canal under extension loads [18]. These alterations reduce the dynamic compliance of the dorsal epidural space, potentiating symptom provocation during standing or walking and symptom relief with lumbar flexion. For the multidisciplinary team, this biology translates into concrete actions: radiologists should comment on ligamentum flavum thickness and crowding in neutral and extension-prone postures inferred from facet orientation; nurses can coach flexion-favoring activities of daily living; and physical therapists can prioritize flexion-biased exercise progressions and endurance training that unload the posterior compartment.

The neurophysiological substrate of symptoms in LSS integrates mechanical compression with microvascular compromise. Neural elements at stenotic levels are vulnerable to root compression within narrowed lateral recesses and foramina and to thecal sac crowding within the central canal. Symptom onset and fluctuation are therefore not purely mechanical; ischemic mechanisms and venous congestion likely contribute to axial back pain and neurogenic claudication, particularly during upright activity when canal caliber decreases and metabolic demand rises [19]. In lateral recess and foraminal disease, disk prolapse, facet hypertrophy, ligamentum infolding, and synovial cysts constrict the path of the traversing or exiting nerve, generating radicular pain with dermatomal distribution. Neural compromise can result from direct extrinsic pressure or from increased intrathecal pressure as global canal narrowing restricts CSF pulsatility, impeding perfusion to radicular vessels. Although a component of inflammatory neuritis has been proposed, prevailing evidence places inflammation secondary to the dominant compression–ischemia paradigm in most patients [20]. These mechanistic distinctions are clinically germane: nursing surveillance should track position-dependent leg symptoms, walking tolerance, and red flags (progressive weakness, bladder changes); physical therapy can harness flexion postures, gait aids, and graded walking to improve perfusion and reduce mechanical strain; radiology can delineate the compartment(s) of maximal compromise to align nonoperative plans and, when necessary, procedural targeting.

Host predisposition further shapes disease expression. Individuals with congenitally narrower canals or smaller AP diameters, as well as those with sagittal or coronal malalignment, may decompensate earlier under otherwise typical degenerative loads [15]. Superimposed spondylolisthesis adds an instability component, permitting anterior translation that telescopes posterior elements and accentuates ligamentous infolding; degenerative scoliosis introduces asymmetric facet loading and unilateral foraminal collapse. These alignment-driven phenomena propagate the same degenerative toolkit—osteophytes, disk protrusions, ligamentous hypertrophy, and facet overgrowth—but often in multilevel or asymmetric patterns that complicate symptom mapping and conservative dosing. Within the multidisciplinary pathway, radiology's role is to quantify slip, rotation, and coronal imbalance; nursing integrates fall-risk assessment and education about avoiding prolonged extension or uneven loading; and physical therapy addresses core stabilization, hip–pelvic mechanics, and postural re-education tailored to the deformity pattern.

In summary, the pathophysiology of LSS—central to a *Multidisciplinary Care Pathway for Radiology, Nursing & Physical Therapy*—arises from the interaction of degenerative disk failure, posterior element remodeling, and soft-tissue hypertrophy, all modulated by baseline osseous dimensions and segmental alignment. The cascade begins with disk desiccation and posterior load shift, advances through facet arthropathy, osteophytes, synovial cysts, and ligamentum flavum thickening [16][17][18] and culminates in compartment-specific narrowing that provokes symptoms via compression and ischemia

[19][20]. Recognizing these mechanisms allows radiologists to produce targeted, decision-ready reports; enables nurses to deliver precise, posture- and symptom-based counseling; and equips physical therapists to deploy flexion-biased, endurance-oriented programs that respect the underlying biomechanics. Ultimately, connecting microstructural change to macro-level function transforms pathophysiologic insight into practical, patient-centered care.

History and Physical:

A rigorous history and physical examination are the cornerstone of care pathways for lumbar spinal stenosis (LSS), enabling radiology, nursing, and physical therapy to align on diagnosis, triage, and individualized management. Classically, LSS manifests as combinations of axial low back pain, lower-extremity radicular symptoms, and—most characteristically—neurogenic claudication precipitated by ambulation and lumbar extension. Symptoms are often bilateral yet asymmetric; paresthesias (numbness and tingling) commonly involve much of the leg rather than a single dermatomal territory, and objective weakness occurs in a substantial subset of patients (≈43%) [21]. Patients frequently volunteer that functional activities in a forward-flexed posture—climbing stairs, leaning on a counter, or pushing a shopping cart—ameliorate discomfort; this “shopping cart sign” reflects the canal-widening effect of lumbar flexion. Relatedly, some adopt a compensatory “simian stance”—slight flexion at the hips and knees—to maintain symptom relief during gait. Nociceptive axial pain typically arises from facet arthropathy, whereas distal dysesthesias and fatigue are driven by root crowding and microvascular compromise within narrowed recesses and foramina.

Symptom patterns map to anatomic compartments and should be elicited with precision to guide imaging protocols and rehabilitation emphasis. Central canal stenosis most often produces neurogenic claudication—activity-dependent, often bilateral leg discomfort and weakness that improve with sitting or flexion. In contrast, lateral recess and foraminal narrowing favor radiculopathy, with unilateral leg pain, sensory change, and possibly myotomal weakness. Among historical features, radiating leg pain worsened by walking is one of the most sensitive clinical markers of LSS, whereas fixed, dermatomally restricted pain suggests superimposed disc herniation. A useful functional cue is directional difficulty on stairs: patients commonly report that going upstairs is easier than downstairs, because ascent encourages lumbar flexion while descent promotes relative extension. Symptom burden spans a continuum. Mild LSS may be asymptomatic, discovered incidentally on imaging. Moderate LSS has been pragmatically defined as up to 50% reduction in central canal or nerve-root canal dimensions, with preserved capacity to sit ≥50 minutes without pain and to ambulate ≥50 feet [22]. Severe LSS is associated with motor weakness, frank gait impairment, and abnormal postural sway that compromises balance and increases fall risk [22]. These gradations are operationally important for the interprofessional team: nursing integrates fall-risk mitigation and home safety counseling; physical therapy calibrates flexion-biased conditioning, gait training, and endurance work; radiology tailors protocol selections (e.g., high-resolution axial sequences for foraminal assessment) and communicates compartmental involvement for procedure planning.

Red-flag recognition is essential. LSS can, though uncommonly, progress to cauda equina or conus medullaris syndrome—new bowel or bladder dysfunction, saddle anesthesia, and acute or rapidly progressive bilateral lower-extremity weakness—which constitutes a medical emergency requiring immediate escalation. Clinicians should heighten vigilance in patients with pronounced stooping and restricted extension who develop sudden neurologic change. The physical examination should be systematic and reproducible, linking bedside findings to likely compartments of stenosis. Begin with posture and gait observation: forward-flexed antalgic postures, shortened stride, wide-based or cautious gait, and difficulty with heel- or toe-walking suggest functional compromise. Lumbar range-of-motion testing typically reproduces symptoms with extension and alleviates them with flexion. Neurologic screening documents dermatomal sensation, myotomal strength, and reflexes (patellar, Achilles), establishing a baseline for monitoring. In suspected foraminal stenosis, reproduction of unilateral radicular pain with passive or active lumbar extension—the Kemp sign—supports foraminal involvement [23]. Additional bedside markers include bilateral wasting of the extensor digitorum brevis, reflecting chronic L5/S1 compromise in some patients [23].

Importantly, the exam may be normal in asymptomatic LSS, and even in symptomatic patients classic maneuver positivity can be limited. The Valsalva maneuver—often provocative in disc herniation—commonly fails to exacerbate LSS-related radicular pain, helping differentiate the two entities. Likewise, the straight-leg raise test is positive in only ~10% of LSS cases, further underscoring the mechanistic difference from acute disc prolapse [24]. Because vascular claudication can mimic neurogenic claudication, clinicians should palpate pedal pulses and consider ankle–brachial indices or vascular referral when pulses are diminished or when history suggests exertional calf ischemia [24]. A quick functional benchmark, the five-repetition sit-to-stand (5R-STS), provides a pragmatic screen: completion in 10.4 seconds has been proposed as a threshold indicating no functional impairment [25]. The structured use of patient-reported outcome measures further anchors shared decision-making. Instruments such as the Oswestry Disability Index (ODI), Swiss Spinal Stenosis questionnaire, visual analog scale (VAS) for pain, Pain Disability Index, SF-36, and the Self-Paced Walking Test (SPWT) quantify baseline status and treatment response, inform therapy goals, and support payor and surgical candidacy determinations [26]. Within a multidisciplinary pathway, nursing can administer and track these tools at intake and follow-up, physical therapy can integrate scores into goal setting and progression criteria, and radiology reports can reference functional limitations to contextualize imaging findings.

Practical pearls knit the history and physical together for interprofessional execution:

- Provocative–relief patterning: Extension (standing, downhill walking) typically worsens, and flexion (sitting, leaning forward, uphill walking) improves symptoms—core education points for nurses and therapists to translate into activity modification and home programs.
- Laterality and distribution: Bilateral, asymmetric leg symptoms with diffuse sensory change favor central compromise; strictly unilateral dermatomal pain suggests focal recess/foraminal disease, guiding both targeted rehabilitation and imaging emphasis.
- Fatigability and endurance: Time-limited walking with recovery in flexion typifies neurogenic claudication; documenting walking tolerance (e.g., SPWT distance) provides an objective anchor for progression [26].
- Comorbidity adjudication: Peripheral arterial disease, hip/knee osteoarthritis, and neuropathies can confound presentation; pulse examination, joint screening, and sensory mapping help disentangle contributors (nursing), while therapy targets compensatory mechanics that aggravate symptoms.

Finally, the synthesis of clinical data should explicitly inform imaging and therapy. A history consistent with central stenosis and neurogenic claudication prioritizes high-quality sagittal and axial sequences at likely culprit levels, whereas suspected foraminal disease merits meticulous foraminal cuts and correlation with Kemp sign reproduction. Physical therapy leverages this mapping to prioritize flexion-biased exercise, trunk endurance, hip hinge retraining, and graded walking (e.g., slight forward-lean on a treadmill or with a rollator). Nursing reinforces pacing, fall prevention, and red-flag education, ensuring seamless escalation if progressive weakness, sphincter disturbance, or saddle anesthesia emerges. In sum, the history and physical examination in LSS—centered on flexion-relieved, extension-provoked leg symptoms; careful delineation of central versus foraminal patterns; judicious application of bedside maneuvers such as Kemp sign; and standardized functional metrics—provide a robust clinical scaffold. When this scaffold is shared across radiology, nursing, and physical therapy, it streamlines imaging selection, sharpens nonoperative plans, and accelerates safe escalation for the rare but critical emergencies of cauda equina or conus medullaris syndromes [21][22][23][24][25][26].

Evaluation:

Because there is not universally accepted clinical–radiologic definition of lumbar spinal stenosis (LSS), the evaluation phase in a multidisciplinary care pathway must integrate symptoms, functional status, and multimodality imaging rather than rely on any single metric. In practice, neuroimaging is indicated for low-back pain with red-flag features (eg, new neurologic deficit, bowel/bladder change, trauma, infection, malignancy risk) and whenever lumbosacral radiculopathy or clinical spinal stenosis is suspected on history and examination. Radiology establishes the anatomic substrate; nursing adjudicates urgency, safety, and patient education; and physical therapy quantifies functional limitation and informs conservative plans that respect the anatomic pattern of narrowing.

Plain radiography and dynamic assessment

Lumbar plain x-rays remain a low-cost, accessible, first look at axial loading effects on spinal biomechanics. Typical degenerative findings include osteophyte formation and reduced intervertebral disk height. As a coarse canal screen, the lower limit of normal anteroposterior (AP) diameter of the lumbar spinal canal on x-ray is ~15 mm. Moreover, interlaminar space measurements on routine films can help predict LSS and prompt cross-sectional imaging when borderline or asymmetric [27]. Equally important, dynamic (flexion–extension) radiographs interrogate instability—a determinant of both symptoms and surgical planning—by revealing translation or angular motion that may necessitate fixation with decompression rather than decompression alone.

Computed tomography (CT)

CT refines osseous anatomy and quantifies canal and foraminal geometry. Commonly used thresholds include spinal sac cross-sectional areas <75 mm² (absolute LSS) and <100 mm² (relative LSS). Lateral recess stenosis is likely when the AP recess measures <4 mm, while foraminal height <15 mm typically correlates with foraminal stenosis and may be present clinically with gluteal pain. On axial CT, advanced central narrowing often produces the characteristic "trefoil" or cloverleaf canal configuration. A crucial pitfall is relying on sagittal measurements alone, which underestimate lateral stenosis and have been associated with poorer operative outcomes; therefore, meticulous axial assessment of the recesses and foramina is essential. Beyond human interpretation, emerging tools such as a CT-aided LSS-VGG16 deep-learning classifier have reported ~90% diagnostic efficacy, foreshadowing decision-support aids that could standardize screening and triage across radiology workflows.

MRI and CT myelography

Non-contrast MRI of the lumbosacral spine is the imaging modality of choice for suspected LSS because of its superior soft-tissue resolution (disks, ligamentum flavum, neural elements) and sensitivity to spinal nerve lesions. When MRI is contraindicated or non-diagnostic, CT myelography offers a valuable alternative by outlining the thecal sac and nerve root sleeves under contrast to reveal focal constrictions [28]. In MRI-based grading, many authors use intraspinal canal area <76 mm² to denote severe stenosis and <100 mm² for moderate stenosis; AP canal diameters <10 mm also frequently support the diagnosis. The "nerve root sedimentation" sign improves reader confidence: in supine patients without LSS, dependent dorsal sedimentation of cauda equina roots is typically seen; absence of sedimentation favors stenosis [29]. Multiple MRI-based classification systems achieve high diagnostic accuracy and facilitate cross-disciplinary communication. The Schizas, Chen Jia, and Braz systems grade central canal compromise on axial T2 imaging, while the Lee grading system, which emphasizes cauda equina morphology, has been linked to surgical decision-making [30]. For a pathway that must translate images into actions, structured radiology reports should (1) identify compartmental involvement (central, lateral recess, foraminal, extraforaminal), (2) provide quantitative or graded severity using one of these schemas, and (3) correlate the anatomic pattern with the likely symptomatic root(s) to guide targeted therapy and, when needed, procedural planning.

Axial-loading MRI and measurement nuances

Standard, supine MRI images the spine under minimal load and can underestimate dynamic stenosis that emerges with upright posture or extension. Axial-loading MRI more closely simulates physiologic conditions and is more effective for evaluating clinically significant narrowing in some patients [34]. In fact, conventional supine MRI may overestimate lateral recess dimensions by ≈13%, potentially explaining discordance between images and exertional symptoms. When available, axial-loading studies—combined with clinical markers such as neurogenic claudication, dural cross-sectional area, and Chen Jia grade—can sharpen surgical triage and personalize nonoperative plans. Parallel advances include machine-learning algorithms for canal segmentation and deep-learning systems trained to evaluate canal stenosis and facet arthropathy on MRI, which have shown promising performance for reproducible quantification and could reduce inter-reader variability in busy clinical settings [35].

Electrodiagnostics and functional assessment

Imaging does not stand alone. Electromyography (EMG) and nerve conduction studies (NCS) can differentiate LSS from clinical mimics (eg, peripheral neuropathy, plexopathy) and document chronic denervation in a myotomal pattern when root compromise is longstanding [36]. In addition, gait analysis combined with EMG provides a functional lens on severity, correlating exertional fatigability and muscle activation patterns with patient-reported claudication and walking limits [37]. For nursing and physical therapy teams, these tools validate objective deficits, inform assistive-device prescriptions, and anchor goal-oriented rehabilitation (eg, improving self-paced walking test distance or sit-to-stand performance).

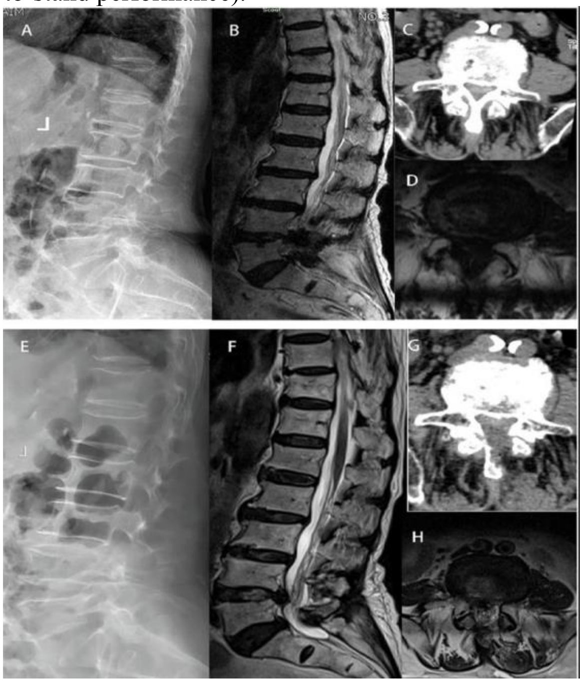

Figure-1: Imaging of patient suffering from spinal lumbar stenosis.

Interprofessional synthesis for decision-ready evaluation

Within the *Multidisciplinary Care Pathway for Radiology, Nursing & Physical Therapy*, evaluation is not a sequence of disconnected tests but a **closed loop** that starts with history and physical findings and ends with a decision-ready synthesis:

- Radiology: Select modality based on clinical question and constraints (plain films and dynamic views for alignment/instability; CT for osseous detail and recess/foraminal metrics; MRI for soft tissues and nerve visualization; CT myelography when MRI is contraindicated) [27][28]. Report on where the narrowing is, how severe it is (quantitative area/diameter and/or grading system), and which roots are plausibly affected. When symptoms outstrip supine MRI findings, consider axial-loading MRI to reconcile the discrepancy [34].

- Nursing: Triage red flags, ensure safety (falls-risk, bladder/bowel changes), and educate patients on flexion-relief strategies pending definitive imaging. Capture functional baselines (eg, self-paced walking time, 5-repetition sit-to-stand) and coordinate logistics for imaging (screening for MRI contraindications), electrodiagnostics, and follow-up.
- Physical Therapy: Translate anatomic findings into movement diagnostics—identifying extension-provoked patterns, laterality, and endurance limits—and initiate flexion-biased conditioning, trunk/hip stabilization, and graded gait with forward-lean or assistive support as appropriate, while monitoring for deterioration that would prompt re-imaging or escalation.

Practical pearls and pitfalls

1. Do not over-rely on a single dimension. AP diameter cut-offs and canal areas offer helpful anchors but must be paired with axial evaluation of lateral recess and foramina to avoid missing clinically dominant narrowing.
2. Correlate with the clinic. Radiating leg pain worsened by walking and relieved by flexion strongly supports LSS; when imaging and symptoms diverge, consider dynamic factors or comorbid conditions (vascular claudication, hip/knee OA, neuropathy).
3. Choose the right alternative. In patients with pacemakers or severe claustrophobia, CT myelography is a practical substitute that still delineates constrictions and root sleeve compromise [28].
4. Lean on standardized grading. Using Schizas, Chen Jia, Braz, or Lee systems improves communication across disciplines and supports prognostication and surgical planning [30].
5. Embrace objective function. Augment imaging with EMG/NCS for diagnostic clarity and gait/EMG assessment to quantify impairment and track response to therapy [36][37].

In sum, evaluating LSS within a multidisciplinary framework requires accurate anatomic mapping, recognition of dynamic stenosis, and objective functional corroboration. Plain films and dynamic views screen alignment and instability; CT delivers precise osseous and recess/foraminal metrics; MRI (with axial-loading when indicated) visualizes the soft-tissue drivers of crowding and enables standardized grading; CT myelography fills gaps when MRI is not feasible; and electrodiagnostics plus gait analysis clarify neural compromise and real-world limitation. When radiology, nursing, and physical therapy close this loop together—anchoring each step in evidence and patient goals—the result is faster, safer diagnosis and a clearer path to effective, personalized care [27][28][29][30][34][35][36][37].

Treatment / Management:

The overarching purpose of treatment for lumbar spinal stenosis (LSS) within a multidisciplinary care pathway is to attenuate pain, expand walking tolerance, and restore meaningful function while minimizing risk. Because symptomatic narrowing arises from a spectrum of anatomic drivers and patient phenotypes, management is necessarily individualized and phased. Across phases, radiology supplies decision-ready anatomic clarification, nursing orchestrates safety, education, and monitoring, and physical therapy translates pathoanatomy into graded movement strategies that improve endurance and reduce extension-provoked symptoms. Although many modalities are available—analgesics, bracing, physical therapy, transcutaneous electrical nerve stimulation, neuromodulation, epidural steroid injection (ESI), interspinous spacers, and a range of decompressive operations—high-quality comparative evidence is uneven, and concrete, universally accepted guidelines remain limited; medical management therefore emphasizes short-term symptom relief while longer-range plans are titrated to response and risk.

Pharmacologic care in LSS is best conceived as supportive rather than curative. Nonsteroidal anti-inflammatory drugs (NSAIDs) are first-line agents for nociceptive axial pain and activity-provoked discomfort. In selected patients with refractory pain or prominent nocturnal symptoms, clinicians may use opioids, short courses of muscle relaxants, gabapentin, vitamin B12, or calcitonin as adjuncts; hemp-derived cannabidiol has also demonstrated improvement in pain scores in some cohorts [38]. Nonetheless, the evidence base for the long-term use of these medications is limited, and risk–benefit must be reassessed regularly, with nurses reinforcing safe dosing, adverse-effect surveillance, and fall-prevention counseling. As with all pharmacologic strategies in older adults, polypharmacy risks, renal function, and coagulopathy require attention, and radiology's reporting of the dominant pain generator (facet arthropathy, lateral recess crowding, foraminal collapse) can help the team align medication choice (e.g., anti-inflammatories for facet flares) with the most likely mechanistic source of symptoms.

Physical therapy is a cornerstone of conservative management, yet the literature shows low-certainty evidence for PT alone in improving pain and function; by contrast, there is moderate evidence that PT confers physiologic stability 3–6 months after surgery, supporting its routine use in postoperative pathways [39]. In practice, the therapeutic lens is biomechanical: flexion increases canal and foraminal caliber, whereas extension accentuates posterior element impaction and ligamentum flavum infolding. Accordingly, programs emphasizing core muscle stretching and strengthening, hip–pelvis coordination, and flexion-biased conditioning can correct posture and reduce extension-provoked symptoms [39]. Therapists often begin with short bouts of forward-lean walking (e.g., slight treadmill incline or a rollator), seated cycling, relative trunk flexion drills, and graded sit-to-stand practice, then progress endurance and load as tolerance improves. Nursing complements therapy by coaching home safety, pacing, and symptom diaries; radiology's precise compartmental mapping (central, lateral recess, foraminal, extraforaminal) informs exercise selection and patient education about provocations to avoid.

Several adjunctive conservative modalities are used variably. Flexion–distraction manipulation therapy can provide short-term symptom relief but has not shown durable benefits, and standardized protocols are lacking. Evidence supporting semirigid lumbosacral bracing, transcutaneous electrical nerve stimulation (TENS), acupuncture, or spinal manipulation remains limited for LSS specifically. Nonetheless, lumbosacral braces and corsets may temporarily increase walking distance by restricting extension and providing proprioceptive feedback, which can be valuable during acute flares or while conditioning takes hold. Neuromodulation has a more focused role: it is preferentially considered in failed back surgery syndrome, where recurrent or persistent neuropathic pain persists despite anatomically adequate decompression, and is usually contemplated after a structured review by an interprofessional panel to ensure mechanical causes of symptoms are not being overlooked. As lifestyle modification threads through all phases, counseling on weight control, glycemic and lipid management, smoking cessation, and graded daily activity improves general health and may reduce systemic contributors to deconditioning that magnify the disability of claudication.

Epidural steroid injection occupies an intermediate tier between purely conservative care and operative intervention. In the ideal course, an ESI provides at least three months of significant pain relief and functions as both diagnostic (does dampened inflammation improve radicular reproduction?) and therapeutic (does walking distance rise while the anti-inflammatory effect persists). Steroid may be delivered via interlaminar or transforaminal routes. Approximately 65% of patients undergo at least one epidural steroid injection, yet typical relief ranges from two weeks to six months, and a systematic review found minimal improvement in walking capacity in LSS patients treated with epidural injections. Furthermore, epidural anesthetic alone is not statistically different from anesthetic plus corticosteroid for short-term outcomes, questioning the additive value of steroid in some settings [40]. In carefully selected cases of severe LSS where foraminal access limits steroid spread into the canal, steroid plus botulinum toxin type A injected into bilateral facet joints has been reported as more effective for symptom control than transforaminal ESI, plausibly because facet-driven inflammation is addressed at its source. Caudal ESI combined with ozone has been associated with significant improvement in walking distance index, although wider adoption awaits larger, methodologically rigorous trials [41]. For patients with spondylosis and facet arthropathy, medial branch blocks and radiofrequency ablation can reduce posterior element nociception, with benefit reported in nearly 70% of individuals with mild-to-moderate stenosis. These interventions are best folded into a broader plan that also builds capacity through physical therapy; nurses can monitor analgesic use, educate about post-injection expectations, and flag red-flag symptoms; radiology assists by localizing culprit levels for targeted delivery.

When conservative measures fail or progressive neurologic deterioration supervenes, surgical decompression becomes appropriate. Outside of emergencies such as cauda equina syndrome, surgery is typically elective, with the unifying dictum to achieve adequate neural decompression while preserving or restoring spinal stability. The liberal laminectomy—a wide pedicle-to-pedicle decompression of the canal—is the most frequently performed operation and remains the age-old standard. Open lumbar decompressive laminectomy benefits approximately 80% of patients with severe LSS and is considered after 3–6 months of optimized nonoperative care fails to control persistent, refractory, or progressive pain, particularly when progressive neurologic decline is documented. The MIST (minimally invasive spinal treatment) guidelines endorse open decompression with or without fusion in cohorts with progressive deficits because the risk-to-benefit ratio is favorable. Absolute surgical contraindications include spinal instability and coagulopathy, whereas relative contraindications encompass concurrent scoliosis, kyphosis, or spondylolisthesis grade ≥2, in which case stabilization strategies may be required. In comparative effectiveness work, the SPORT trial demonstrated that, in patients with LSS without spondylolisthesis, surgery provided sustained improvements in function and pain relative to conservative strategies, although careful patient selection and expectation setting are essential.

The durability of benefit matters to patients and payors alike. A randomized trial has shown that individuals undergoing laminectomy experience greater symptomatic improvement than those treated nonsurgically, though the magnitude of benefit diminishes over time, likely as adjacent segments degenerate or scar tissue accrues [42]. Where instability is present or when iatrogenic destabilization is anticipated (e.g., wide facetectomy), laminectomy with fusion is indicated, particularly in degenerative or isthmic spondylolisthesis and degenerative scoliosis.

Here the literature is nuanced: the Swedish Spinal Stenosis Study and similar investigations found no clinical advantage to adding fusion over decompression alone in many patients, whereas the SLIP study reported better quality of life and lower reoperation rates when instrumented fusion accompanied decompression. In SLIP, decompression with fusion was 2.55 times more effective in improving Oswestry Disability Index (ODI) scores than decompression without fusion, but the combined operation incurred greater blood loss, longer hospital stay, and higher cost. Shared decision-making should thus weigh symptom drivers, alignment, bone quality, and comorbidity against the heightened physiologic demands of fusion; radiology's comment on facet orientation, pars integrity, and dynamic motion helps clarify the stability question, while nursing and therapy teams prepare patients for the different recovery arcs.

In the last decade, minimally invasive and endoscopic decompressions have matured and broadened options for patients and surgeons. Percutaneous lumbar decompression, particularly for patients with hypertrophied ligamentum flavum of ≥2.5 mm, can be performed outpatient, reduces soft-tissue trauma, and may accelerate convalescence; however, it is technically demanding and carries a notable learning curve [43]. Minimally invasive unilateral or bilateral decompression using microscopic or endoscopic tubular retractors strives to achieve equivalent canal enlargement through smaller corridors, often translating to less intraoperative blood loss and fewer complications, while relying on meticulous technique and advanced instrumentation [43]. Unilateral biportal endoscopy (UBE) laminectomy has emerged as a safe and effective option in multiple series [44][45]. Comparative work suggests that microscopic unilateral laminotomy with bilateral decompression (ULBD) and UBE-ULBD both relieve symptoms, yet UBE-ULBD may yield shorter hospital stays and greater pain improvement than microscopic ULBD [46][47]. A recent meta-analysis reported equivalent efficacy between UBE and ULBD, with UBE conferring less intraoperative bleeding and shorter stays [48]. These differential recovery profiles are material to perioperative planning: nurses tailor early mobilization and analgesia protocols to incision size and drain usage, therapists begin flexion-friendly gait training earlier when feasible, and radiology ensures postoperative imaging is only obtained when clinically indicated, avoiding unnecessary radiation or artifact-laden scans that do not alter care.

Another minimally invasive tool, minimally invasive lumbar decompression (MILD), targets posterior element hypertrophy through limited access with the promise of minimal blood loss and paraspinal muscle preservation, making it attractive in outpatient settings. Yet, long-term benefits have not been definitively established in high-quality trials, and the technique entails a steep learning curve and radiation exposure that must be conscientiously managed. For patients whose symptoms are chiefly extension-provoked and multilevel degenerative change is modest, the interspinous spacer offers a motion-sparing alternative: by reducing lumbar extension at the implanted level(s), these devices can enlarge the canal during standing and walking. Interspinous spacers are safe, cost-effective, and approved for one or two lumbar levels, with outcomes comparable to decompression for intermittent claudication in selected patients [49]. Approximately 50% of recipients achieve clinically meaningful benefit. Device-specific data suggest Supirion implants may have fewer complications than X-STOP, and, in contrast to laminectomy, some series report lower reoperation rates with spacers. Contraindications are crucial: osteoporosis and dynamic instability argue against spacer use due to risk of spinous process fracture or progressive listhesis. Here again, radiology's measurement of bone density proxies, facet alignment, and dynamic motion, paired with nursing's falls-risk appraisal and physical therapy's assessment of flexion-relief patterns, guides prudent selection.

Because patients and conditions vary, algorithmic care is best presented as a narrative progression rather than a rigid checklist. Most individuals start with lifestyle modification, physical therapy, and pain medication, a phase that establishes foundational conditioning and identifies those in whom symptoms respond to posture and endurance training. When pain precludes progress or radicular features dominate, the plan may incorporate ESI, facet injections, or medial branch radiofrequency ablation, targeted by imaging and clinical correlation. If relief is partial or transient—particularly in complex anatomy or after prior surgery—an interdisciplinary review by radiologists, interventional pain physicians, and spine surgeons can adjudicate the potential value of neuromodulation, repeat ESI, or a tissue-sparing decompressive option. For patients whose symptoms are clearly extension-sensitive and whose imaging supports level-limited disease without instability, interspinous spacers may be considered. When functional goals remain unmet, or progressive deficits occur, the pathway advances to stepwise decompression, from MILD or percutaneous approaches to open laminectomy with or without fusion, as dictated by stability, alignment, and patient preferences. Throughout, nursing anchors safety and education, physical therapy calibrates graded exposure and measures progress objectively, and radiology ensures that imaging—pre-intervention and, when needed, post-intervention—answers focused clinical questions rather than being obtained reflexively.

Even as multiple options exist, several cross-cutting principles enhance outcomes. First, match treatment to mechanism: patients whose pain is dominated by facet-mediated extension flares may benefit from posterior-element interventions and flexion-biased conditioning, while those with crisp, level-specific radiculopathy from foraminal collapse may respond best to targeted decompression. Second, track function, not pain alone: walking distance, the self-paced walking test, stair negotiation, and sit-to-stand times capture meaningful change better than numeric ratings alone and help the team judge whether an intervention is restoring participation. Third, sequence rather than stack: because the long-term benefit of many injections and adjuncts is limited, each step should be given a clear time-boxed trial with predefined success criteria; if those are not met, escalation or a pivot should follow rather than repeating the same modality indefinitely. Fourth, plan perioperative rehabilitation proactively: the moderate evidence for PT-mediated physiologic stability at 3–6 months post-surgery justifies early referral and coordinated discharge planning [39]. Finally, communicate clearly across disciplines: radiology should specify compartments and likely affected roots; nursing should document red flags, teach flexion-relief strategies, and reconcile medications; physical therapy should share objective milestones and barriers so that interventionalists and surgeons can time procedures when patients are most likely to benefit.

In conclusion, LSS management is a phased, individualized endeavor that aims to reduce pain and expand function while safeguarding patients from unnecessary risk. Short-term medical management—centered on NSAIDs and carefully selected adjuncts—provides symptomatic relief but should be coupled with flexion-biased physical therapy to address biomechanics [38][39]. ESI and related injections can modulate inflammation or nociception for weeks to months, but walking gains are modest, and anesthetic alone may match steroid in impact [40][41]. When anatomy and symptoms warrant, decompression—via open, minimally invasive, or endoscopic routes—remains the definitive means of enlarging neural passageways, with fusion reserved for instability or deformity; trial data show superiority of surgery over nonoperative care in appropriate candidates, albeit with benefits that may attenuate over time [42]. Percutaneous decompressions, UBE, ULBD, and MILD broaden options, trading smaller incisions and quicker discharge for technical demands and, in some instances, less mature long-term evidence [43][44][45][46][47][48]. Interspinous spacers serve carefully selected, extension-sensitive cases with one to two diseased levels but require intact bone and stability [49]. The most reliable predictor of success is not a single image or procedure; it is a coordinated, interprofessional process that matches mechanism to modality, measures function, and revisits the plan based on transparent goals.

Table 1. Stepwise Management Ladder and Expected Effects.

Step	Modality & examples	Expected benefit window	Primary goal(s)	Notes for escalation
1	Education, NSAIDs/adjuncts, lifestyle, **flexion-biased PT**	Weeks to months	Pain control, endurance, gait tolerance	Advance if red flags or failure of functional gains
2	**ESI** (interlaminar/transforaminal), facet injections; medial branch block/RFA	2 weeks–6 months (variable)	Short-term relief to enable rehab	Limited walking gains overall; consider if facet-predominant pain
3	Interdisciplinary review; neuromodulation in FBSS	Patient-selected	Refractory neuropathic pain	Ensure no remediable mechanical compression
4	**Interspinous spacer** (1–2 levels, no instability/osteoporosis)	Months–years in responders	Extension control, claudication relief	For extension-sensitive disease; monitor for spinous fracture
5	**Decompression** (open/MIS/endoscopic ± fusion for instability)	Years; may attenuate with time	Definitive space enlargement	Fusion when instability/deformity; plan peri-op rehab early

Differential Diagnosis

Distinguishing lumbar spinal stenosis (LSS) from common mimics relies on careful synthesis of history, examination, and targeted testing. Vascular claudication can present with bilateral calf or thigh symptoms that worsen on standing and ambulation and may appear to improve with a flexed "shopping cart" posture; however, confirmation depends on vascular imaging and an ankle–brachial index to document flow-limiting disease. Peripheral neuropathy typically produces a stocking-and-glove sensory disturbance that is present at rest and often disrupts sleep; the non-dermatomal, length-dependent pattern and reduced distal reflexes help differentiate it from root compression. Lumbar spondylosis with acute or subacute disc involvement is suggested by a positive straight-leg raise or Lasègue test (L4–S1) and, for upper lumbar involvement, a positive reverse straight-leg (Ely) test (L2–L4). Lumbar plexopathies may manifest as sensorimotor deficits with minimal pain depending on etiology, emphasizing the value of electrodiagnostics when localization is uncertain. Hip and knee osteoarthritis commonly cause activity-related joint line pain and tenderness without neurologic signs; restricted range of motion, crepitus, and provocative joint maneuvers support the diagnosis. Metabolic neuropathies from alcohol misuse or vitamin deficiencies produce symmetric sensorimotor changes with systemic clues. A thorough evaluation—integrating vascular assessment, focused neurologic testing, joint examination, and, when indicated, electrodiagnostics—usually separates these entities from LSS.

Table 2. Compartment–Symptom–Imaging–Care Mapping

Stenosis compartment	Typical clinical pattern	Imaging priorities	Nursing focus	PT strategy
Central canal	Bilateral, asymmetric neurogenic claudication relieved by sitting/flexion	MRI with axial/sagittal grading (Schizas/Chen Jia/Lee); consider axial-loading MRI if discordant	Red-flag surveillance; pacing, flexion-relief education	Flexion-biased gait (incline/rollator), trunk endurance, graded walking
Lateral recess	Dermatomal leg pain ± weakness with extension provocation	High-resolution axial MRI; CT for osseous detail	Fall-risk mitigation; flare management	Neural mobilization as tolerated; anti-extension drills; hip hinge retraining
Foraminal	Unilateral radicular pain, Kemp sign positive	Foraminal cuts on MRI; CT for osteophytes/height	Sleep/position coaching; analgesic timing	Posture correction, pelvic control, flexion-friendly mobility
Extraforaminal	Far-lateral radiculopathy	MRI/CT with far-lateral coverage	Targeted education for activity triggers	Local mobility, graded loading avoiding end-range extension

Prognosis

LSS is a major source of pain and disability, and additional spinal segments become involved over time in roughly half of patients. Despite this, the natural history is favorable for many: approximately 33% to 50% of individuals with mild-to-moderate disease improve or remain stable, and the North American Spine Society similarly reports a favorable course in about half of symptomatic, mild-to-moderate cohorts. With conservative management, symptom progression has been reported in about 15% at five years and nearly 30% at ten years, while improvement occurs in about 70% and 30% of these cohorts over the same intervals. Overall, 20% to 40% of patients with mild-to-moderate stenosis ultimately require surgery within ten years. Conditions most strongly associated with eventual operation include cauda equina syndrome, degenerative scoliosis, spondylolisthesis, and persistent refractory symptomatology; importantly, severe canal narrowing on imaging does not mandate surgery, as many patients remain asymptomatic, cautioning against overreliance on MRI quantitative parameters alone when making clinical decisions [50].

Preoperative symptom intensity is prognostic: a higher baseline visual analog scale (VAS) score independently predicts recovery trajectory [51]. In moderate-to-severe central canal stenosis, lumbar decompression improves VAS scores and functional outcomes [52]. In medically managed cohorts, polypharmacy rates may reach 70%, whereas decompression surgery can substantially reduce medication burden [53][54]. After microsurgical decompression, improvements in lumbar kyphosis and sagittal balance are often sustained for up to five years, although about one-third experience subsequent deterioration in global alignment thereafter [55]. Psychosocial and demographic factors also influence outcomes and costs; advanced age, preoperative depression, and discharge to rehabilitation facilities are associated with higher resource use and less favorable results, whereas preoperative smoking cessation and weight reduction are beneficial [56]. Instability is common when spondylolisthesis or facet cysts coexist. In multilevel disease, selective microendoscopic laminotomy confined to symptomatic levels may lower reoperation risk [57]. Semirigid polyetheretherketone (PEEK) constructs have shown advantages in physiologic motion, fusion rates, complication reduction, and adjacent-segment protection [58], whereas conventional instrumentation continues to yield low hardware failure, substantial pain relief, and high fusion rates even in osteoporosis [59]. When cervical and lumbar stenosis coexist, a staged two-step decompression is commonly recommended; combined cervical–thoracic disease may be addressed in one stage [60]. Across techniques, leg pain generally improves more than axial back pain, a counseling point that aligns expectations with typical postoperative trajectories [61][62][63][64][65].

Complications

Left untreated, LSS can lead to chronic back and lower-limb pain, diminished exercise tolerance, reduced mobility and function, disuse muscle atrophy, mood disorders such as depression and anxiety, and overall decline in quality of life; in rare but critical cases, progressive narrowing precipitates cauda equina or conus medullaris syndrome requiring emergent care. Interventions themselves carry risk. Open and minimally invasive treatments share potential complications that include epidural hematoma, dural tear, surgical site infection, iatrogenic neurovascular injury, retained hemostatic materials, postoperative instability, bony regrowth with recurrent stenosis, failed back surgery syndrome, and, in open laminectomy series, a reported mortality ranging from 0.5% to 2.3% [61]. Patients should be instructed to seek immediate evaluation for new bowel or bladder dysfunction, saddle anesthesia, rapidly progressive weakness, fever with severe back pain, or intractable escalation of symptoms to facilitate timely intervention and prevent sequelae [63][64][65].

Patient Education

Although LSS reflects age-related degeneration for many, modifiable behaviors can lower risk and blunt exacerbations. Patients should be encouraged to maintain regular aerobic and strengthening exercise for overall fitness; practice body-mechanics strategies that avoid sustained lumbar extension underload; interrupt prolonged sitting with movement breaks; wear supportive, comfortable footwear; and cultivate posture awareness throughout daily activities. Ergonomic seating and workstations reduce repetitive strain, while smoking cessation, adequate hydration, and routine stretching support spinal health. Annual health evaluations facilitate early detection of cardiometabolic comorbidities that aggravate deconditioning, and prompt clinical assessment is advised for low-back pain accompanied by sensorimotor changes or limb pain. For diagnosed LSS, interventions should proceed stepwise: conservative modalities—education, analgesics, and flexion-biased physical therapy—precede operative consideration unless red flags compel faster escalation. When surgery is contemplated, minimally invasive strategies are preferentially considered before open procedures when anatomically and clinically suitable. Education must span both physical and psychological pain management to optimize adherence and outcomes [62] [64].

Enhancing Healthcare Team Outcomes

Given its rising prevalence with age and frequent coexistence of cardiopulmonary and metabolic disease, LSS benefits from a coordinated interprofessional model. Primary care physicians typically identify the syndrome, initiate early management, and coordinate referrals. Radiologists provide the anatomical roadmap—clarifying central, lateral recess, foraminal, and extraforaminal compromise—and thereby shape both conservative and operative plans. Spine surgeons determine candidacy for decompression—with or without fusion—and perform minimally invasive or open procedures when indicated; neurosurgeons offer expertise for emergencies such as cauda equina or conus medullaris syndromes. Neurologists assist with localization and with mimics such as plexopathy or neuropathy, while pain specialists deliver interventional options and perioperative anesthesia care. Pharmacists optimize pharmacotherapy and counsel patients on efficacy and toxicity avoidance. Physical therapists design individualized programs that strengthen the core, enhance flexibility, and restore stability and walking endurance, aligning exercises with flexion-relief biomechanics. Psychologists address the cognitive and emotional dimensions of chronic pain, fostering coping skills that improve participation. Nurses knit the pathway together by ensuring comfort, administering medications, coordinating diagnostics and follow-up, and reinforcing education on adherence, safety, and red-flag recognition. Effective, timely communication among these disciplines, anchored in shared goals and clear role delineation—underpins comprehensive, patient-centered care and measurably improves outcomes [63] [64].

Conclusion:

Lumbar spinal stenosis exemplifies a condition in which anatomy, biomechanics, and host factors intertwine to produce fluctuating impairment. Effective care therefore cannot hinge on a diameter threshold or a single intervention. The multidisciplinary pathway presented here operationalizes first principles: radiology must identify the compartment(s) of crowding, quantify severity with reproducible schemes, and acknowledge dynamics that explain clinic–image mismatch; nursing must triage red flags, reduce falls risk, teach posture and pacing strategies that leverage flexion relief, and coordinate conservative and interventional logistics; physical therapy must convert pathoanatomy into graded, flexion-biased conditioning that improves endurance and walking tolerance while monitoring for deterioration. Interventions should be sequenced, not stacked: medications and injections provide short-term relief but rarely change natural history; durable improvement follows from restoring capacity and, where necessary, mechanically enlarging neural passageways by decompression, reserving fusion for instability or deformity. Objective functional measures (e.g., self-paced walking test, sit-to-stand, ODI) should anchor decisions and expectations. By linking mechanism to modality and function to follow-up, the pathway improves timeliness, safety, and personalization of care, reduces unwarranted procedures, and preserves the clear benefits of surgery for the right patient at the right time.

References:

1. Bagley C, MacAllister M, Dosselman L, Moreno J, Aoun SG, El Ahmadieh TY. Current concepts and recent advances in understanding and managing lumbar spine stenosis. F1000Res. 2019;8
2. Ciol MA, Deyo RA, Howell E, Kreif S. An assessment of surgery for spinal stenosis: time trends, geographic variations, complications, and reoperations. J Am Geriatr Soc. 1996 Mar;44(3):285-90.
3. Jenis LG, An HS. Spine update. Lumbar foraminal stenosis. Spine (Phila Pa 1976). 2000 Feb 01;25(3):389-94.
4. Zaina F, Tomkins-Lane C, Carragee E, Negrini S. Surgical Versus Nonsurgical Treatment for Lumbar Spinal Stenosis. Spine (Phila Pa 1976). 2016 Jul 15;41(14):E857-E868.
5. Lee SH, Choi HH, Chang MC. The Effectiveness of Facet Joint Injection with Steroid and Botulinum Toxin in Severe Lumbar Central Spinal Stenosis: A Randomized Controlled Trial. Toxins (Basel). 2022 Dec 23;15(1)
6. Laiwalla AN, Ratnaparkhi A, Zarrin D, Cook K, Li I, Wilson B, Florence TJ, Yoo B, Salehi B, Gaonkar B, Beckett J, Macyszyn L. Lumbar Spinal Canal Segmentation in Cases with Lumbar Stenosis Using Deep-U-Net Ensembles. World Neurosurg. 2023 Oct;178:e135-e140.
7. Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, Hunter DJ. Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine J. 2009 Jul;9(7):545-50.
8. Deer TR, Grider JS, Pope JE, Lamer TJ, Wahezi SE, Hagedorn JM, Falowski S, Tolba R, Shah JM, Strand N, Escobar A, Malinowski M, Bux A, Jassal N, Hah J, Weisbein J, Tomycz ND, Jameson J, Petersen EA, Sayed D. Best Practices for Minimally Invasive Lumbar Spinal Stenosis Treatment 2.0 (MIST): Consensus Guidance from the American Society of Pain and Neuroscience (ASPN). J Pain Res. 2022;15:1325-1354.
9. Binder DK, Schmidt MH, Weinstein PR. Lumbar spinal stenosis. Semin Neurol. 2002 Jun;22(2):157-66.
10. Yabuki S, Fukumori N, Takegami M, Onishi Y, Otani K, Sekiguchi M, Wakita T, Kikuchi S, Fukuhara S, Konno S. Prevalence of lumbar spinal stenosis, using the diagnostic support tool, and correlated factors in Japan: a population-based study. J Orthop Sci. 2013 Nov;18(6):893-900
11. Diwan S, Sayed D, Deer TR, Salomons A, Liang K. An Algorithmic Approach to Treating Lumbar Spinal Stenosis: An Evidenced-Based Approach. Pain Med. 2019 Dec 01;20(Suppl 2):S23-S31.
12. Xu J, Si H, Zeng Y, Wu Y, Zhang S, Shen B. Transcriptome-wide association study reveals candidate causal genes for lumbar spinal stenosis. Bone Joint Res. 2023 Jun 26;12(6):387-396.
13. Walter KL, O'Toole JE. Lumbar Spinal Stenosis. JAMA. 2022 Jul 19;328(3):310.
14. Buser Z, Ortega B, D'Oro A, Pannell W, Cohen JR, Wang J, Golish R, Reed M, Wang JC. Spine Degenerative Conditions and Their Treatments: National Trends in the United States of America. Global Spine J. 2018 Feb;8(1):57-67.
15. Abbas J, Yousef M, Peled N, Hershkovitz I, Hamoud K. Predictive factors for degenerative lumbar spinal stenosis: a model obtained from a machine learning algorithm technique. BMC Musculoskelet Disord. 2023 Mar 23;24(1):218.
16. Wang S, Qu Y, Fang X, Ding Q, Zhao H, Yu X, Xu T, Lu R, Jing S, Liu C, Wu H, Liu Y. Decorin: a potential therapeutic candidate for ligamentum flavum hypertrophy by antagonizing TGF-β1. Exp Mol Med. 2023 Jul;55(7):1413-1423.
17. Yabe Y, Hagiwara Y, Tsuchiya M, Minowa T, Takemura T, Hattori S, Yoshida S, Onoki T, Ishikawa K. Comparative proteome analysis of the ligamentum flavum of patients with lumbar spinal canal stenosis. JOR Spine. 2022 Dec;5(4):e1210.
18. Jain M, Sable M, Tirpude AP, Sahu RN, Samanta SK, Das G. Histological difference in ligament flavum between degenerative lumbar canal stenosis and non-stenotic group: A prospective, comparative study. World J Orthop. 2022 Sep 18;13(9):791-801.
19. Altun S, Alkan A, Altun İ. LSS-VGG16: Diagnosis of Lumbar Spinal Stenosis With Deep Learning. Clin Spine Surg. 2023 Jun 01;36(5):E180-E190.
20. Jinkins JR. Gd-DTPA enhanced MR of the lumbar spinal canal in patients with claudication. J Comput Assist Tomogr. 1993 Jul-Aug;17(4):555-62.
21. Hall S, Bartleson JD, Onofrio BM, Baker HL, Okazaki H, O'Duffy JD. Lumbar spinal stenosis. Clinical features, diagnostic procedures, and results of surgical treatment in 68 patients. Ann Intern Med. 1985 Aug;103(2):271-5.
22. Ujigo S, Kamei N, Yamada K, Nakamae T, Imada H, Adachi N, Fujimoto Y. Balancing ability of patients with lumbar spinal canal stenosis. Eur Spine J. 2023 Dec;32(12):4174-4183.
23. Munakomi S, Kumar BM. Wasting of Extensor Digitorum Brevis as a Decisive Preoperative Clinical Indicator of Lumbar Canal Stenosis: A Single-center Prospective Cohort Study. Ann Med Health Sci Res. 2016 Sep-Oct;6(5):296-300.
24. Katz JN, Dalgas M, Stucki G, Katz NP, Bayley J, Fossel AH, Chang LC, Lipson SJ. Degenerative lumbar spinal stenosis. Diagnostic value of the

history and physical examination. Arthritis Rheum. 1995 Sep;38(9):1236-41.
25. Staartjes VE, Schröder ML. The five-repetition sit-to-stand test: evaluation of a simple and objective tool for the assessment of degenerative pathologies of the lumbar spine. J Neurosurg Spine. 2018 Oct;29(4):380-387.
26. Oskouie IM, Rostami M, Moosavi M, Zarei M, Jouibari MF, Ataie H, Jafarieh A, Moghadam N, Kordi R, Khadivi M, Mazloumi A. Translation, cross-cultural adaptation and validation of the Persian version of selected PROMIS measures for use in lumbar canal stenosis patients. J Educ Health Promot. 2023;12:99.
27. Wang Y, Zhang P, Yan X, Wang J, Zhu M, Teng H. The correlation between lumbar interlaminar space size on plain radiograph and spinal stenosis. Eur Spine J. 2023 May;32(5):1721-1728.
28. Saifuddin A. The imaging of lumbar spinal stenosis. Clin Radiol. 2000 Aug;55(8):581-94.
29. Pant YR, Paudel S, Lakhey RB, Pokharel RK. Nerve Root Sedimentation Sign among Lumbar Canal Stenosis Patients Visiting the Department of Orthopaedics in a Tertiary Care Centre: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2022 Dec 01;60(256):1030-1032.
30. Ahn DY, Park HJ, Yi JW, Kim JN. To Assess Whether Lee's Grading System for Central Lumbar Spinal Stenosis Can Be Used as a Decision-Making Tool for Surgical Treatment. Taehan Yongsang Uihakhoe Chi. 2022 Jan;83(1):102-111.
31. Ko YJ, Lee E, Lee JW, Park CY, Cho J, Kang Y, Ahn JM. Clinical validity of two different grading systems for lumbar central canal stenosis: Schizas and Lee classification systems. PLoS One. 2020;15(5):e0233633.
32. Qian G, Wang Y, Huang J, Wang D, Miao C. Value of nerve root sedimentation sign in diagnosis and surgical indication of lumbar spinal stenosis. BMC Musculoskelet Disord. 2023 Apr 28;24(1):336. [
33. Lee GY, Lee JW, Choi HS, Oh KJ, Kang HS. A new grading system of lumbar central canal stenosis on MRI: an easy and reliable method. Skeletal Radiol. 2011 Aug;40(8):1033-9.
34. Fang X, Li J, Wang L, Liu L, Lv W, Tang Z, Gao D. Diagnostic value of a new axial loading MRI device in patients with suspected lumbar spinal stenosis. Eur Radiol. 2023 May;33(5):3200-3210.
35. Bharadwaj UU, Christine M, Li S, Chou D, Pedoia V, Link TM, Chin CT, Majumdar S. Deep learning for automated, interpretable classification of lumbar spinal stenosis and facet arthropathy from axial MRI. Eur Radiol. 2023 May;33(5):3435-3443.
36. Haig AJ, Tong HC, Yamakawa KS, Quint DJ, Hoff JT, Chiodo A, Miner JA, Choksi VR, Geisser ME. The sensitivity and specificity of electrodiagnostic testing for the clinical syndrome of lumbar spinal stenosis. Spine (Phila Pa 1976). 2005 Dec 01;30(23):2667-76.
37. Nüesch C, Mandelli F, Przybilla P, Schären S, Mündermann A, Netzer C. Kinematics and paraspinal muscle activation patterns during walking differ between patients with lumbar spinal stenosis and controls. Gait Posture. 2023 Jan;99:44-50.
38. Bakewell BK, Sherman M, Binsfeld K, Ilyas AM, Stache SA, Sharma S, Stolzenberg D, Greis A. The Use of Cannabidiol in Patients With Low Back Pain Caused by Lumbar Spinal Stenosis: An Observational Study. Cureus. 2022 Sep;14(9):e29196
39. Mazanec DJ, Podichetty VK, Hsia A. Lumbar canal stenosis: start with nonsurgical therapy. Cleve Clin J Med. 2002 Nov;69(11):909-17.
40. Fukusaki M, Kobayashi I, Hara T, Sumikawa K. Symptoms of spinal stenosis do not improve after epidural steroid injection. Clin J Pain. 1998 Jun;14(2):148-51.
41. Rayegani SM, Soltani V, Cheraghi M, Omid Zohor MR, Babaei-Ghazani A, Raeissadat SA. Efficacy of ultrasound guided caudal epidural steroid injection with or without ozone in patients with lumbosacral canal stenosis; a randomized clinical controlled trial. BMC Musculoskelet Disord. 2023 Apr 29;24(1):339.
42. Malmivaara A, Slätis P, Heliövaara M, Sainio P, Kinnunen H, Kankare J, Dalin-Hirvonen N, Seitsalo S, Herno A, Kortekangas P, Niinimäki T, Rönty H, Tallroth K, Turunen V, Knekt P, Härkänen T, Hurri H., Finnish Lumbar Spinal Research Group. Surgical or nonoperative treatment for lumbar spinal stenosis? A randomized controlled trial. Spine (Phila Pa 1976). 2007 Jan 01;32(1):1-8.
43. Süner HI, Castaño JP, Vargas-Jimenez A, Wagner R, Mazzei AS, Velazquez W, Jorquera M, Sallabanda K, Barcia Albacar JA, Carrascosa-Granada A. Comparison of the Tubular Approach and Uniportal Interlaminar Full-Endoscopic Approach in the Treatment of Lumbar Spinal Stenosis: Our 3-Year Results. World Neurosurg. 2023 May;173:e148-e155.
44. Zhang Y, Feng B, Su W, Liu D, Hu P, Lu H, Geng X. [Early-effectiveness of unilateral biportal endoscopic laminectomy in treatment of two-level lumbar spinal stenosis]. Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi. 2023 Jun 15;37(6):706-712.
45. Hu YT, Fu H, Yang DF, Wang X, Xu WB. [Comparative study of decompression of unilateral biportal endoscopic compared to

laminectomy with fusion and internal fixation in the treatment of severe lumbar spinal stenosis]. Zhonghua Yi Xue Za Zhi. 2022 Nov 08;102(41):3281-3287.

46. Lin GX, Yao ZK, Xin C, Kim JS, Chen CM, Hu BS. A meta-analysis of clinical effects of microscopic unilateral laminectomy bilateral decompression (ULBD) versus biportal endoscopic ULBD for lumbar canal stenosis. Front Surg. 2022;9:1002100.
47. Patel K, Harikar MM, Venkataram T, Chavda V, Montemurro N, Assefi M, Hussain N, Yamamoto V, Kateb B, Lewandrowski KU, Umana GE. Is Minimally Invasive Spinal Surgery Superior to Endoscopic Spine Surgery in Postoperative Radiologic Outcomes of Lumbar Spine Degenerative Disease? A Systematic Review. J Neurol Surg A Cent Eur Neurosurg. 2024 Mar;85(2):182-191.
48. Junjie L, Jiheng Y, Jun L, Haixiong L, Haifeng Y. Comparison of Unilateral Biportal Endoscopy Decompression and Microscopic Decompression Effectiveness in Lumbar Spinal Stenosis Treatment: A Systematic Review and Meta-analysis. Asian Spine J. 2023 Apr;17(2):418-430
49. Machado GC, Ferreira PH, Yoo RI, Harris IA, Pinheiro MB, Koes BW, van Tulder MW, Rzewuska M, Maher CG, Ferreira ML. Surgical options for lumbar spinal stenosis. Cochrane Database Syst Rev. 2016 Nov 01;11(11):CD012421.
50. Gupta S, Bansal T, Kashyap A, Sural S. Correlation between clinical scoring systems and quantitative MRI parameters in degenerative lumbar spinal stenosis. J Clin Orthop Trauma. 2022 Dec;35:102050.
51. Inose H, Kato T, Matsukura Y, Hirai T, Yoshii T, Kawabata S, Takahashi K, Okawa A. Factors influencing the long-term outcomes of instrumentation surgery for degenerative lumbar spondylolisthesis: a post-hoc analysis of a prospective randomized study. Spine J. 2023 Jun;23(6):799-804.
52. Yeung CM, Heard JC, Lee Y, Lambrechts MJ, Somers S, Singh A, Bloom E, D'Antonio ND, Trenchfield D, Labarbiera A, Mangan JJ, Canseco JA, Woods BI, Kurd MF, Kaye ID, Lee JK, Hilibrand AS, Vaccaro AR, Kepler CK, Schroeder GD. The Implication of Preoperative Central Stenosis on Patient-Reported Outcomes After Lumbar Decompression Surgery. World Neurosurg. 2023 Jun 19;
53. Nagai S, Inagaki R, Michikawa T, Kawabata S, Ito K, Hachiya K, Takeda H, Ikeda D, Kaneko S, Yamada S, Fujita N. Efficacy of surgical treatment on polypharmacy of elderly patients with lumbar spinal canal stenosis: retrospective exploratory research. BMC Geriatr. 2023 Mar 24;23(1):169.
54. Imai T, Nagai S, Michikawa T, Inagaki R, Kawabata S, Ito K, Hachiya K, Takeda H, Ikeda D, Yamada S, Fujita N, Kaneko S. Impact of Lumbar Surgery on Pharmacological Treatment for Patients with Lumbar Spinal Canal Stenosis: A Single-Center Retrospective Study. J Clin Med. 2023 Mar 20;12(6)
55. Yokoyama K, Ikeda N, Tanaka H, Ito Y, Sugie A, Yamada M, Wanibuchi M, Kawanishi M. Long-Term Changes in Sagittal Balance After Microsurgical Decompression of Lumbar Spinal Canal Stenosis in Elderly Patients: A Follow-Up Study for 5-Years After Surgery. World Neurosurg. 2023 Aug;176:e384-e390.
56. D'Antonio ND, Lambrechts MJ, Trenchfield D, Sherman M, Karamian BA, Fredericks DJ, Boere P, Siegel N, Tran K, Canseco JA, Kaye ID, Rihn J, Woods BI, Hilibrand AS, Kepler CK, Vaccaro AR, Schroeder GD. Patient-specific Risk Factors Increase Episode of Care Costs After Lumbar Decompression. Clin Spine Surg. 2023 Oct 01;36(8):E339-E344
57. Murata S, Nagata K, Iwasaki H, Hashizume H, Yukawa Y, Minamide A, Nakagawa Y, Tsutsui S, Takami M, Taiji R, Kozaki T, J Schoenfeld A, K Simpson A, Yoshida M, Yamada H. Long-Term Outcomes after Selective Microendoscopic Laminotomy for Multilevel Lumbar Spinal Stenosis with and without Remaining Radiographic Stenosis: A 10-Year Follow-Up Study. Spine Surg Relat Res. 2022 Sep 27;6(5):488-496.
58. Varol E, Etli MU, Avci F, Yaltirik CK, Ramazanoglu AF, Onen MR, Naderi S. Comparison of clinical and radiological results of dynamic and rigid instrumentation in degenerative lumbar spinal stenosis. J Craniovertebr Junction Spine. 2022 Jul-Sep;13(3):350-356.
59. Aghajanloo M, Abdoli A, Poorolajal J, Abdolmaleki S. Comparison of clinical outcome of lumbar spinal stenosis surgery in patients with and without osteoporosis: a prospective cohort study. J Orthop Surg Res. 2023 Jun 21;18(1):443.
60. Lu C, Qiu H, Huang X, Yang X, Liu D, Zhang S, Zhang Y. Meta-Analysis of Simultaneous versus Staged Decompression of Stenotic Regions in Patients with Tandem Spinal Stenosis. World Neurosurg. 2023 Feb;170:e441-e454.
61. Estefan M, Munakomi S, Camino Willhuber GO. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Aug 13, 2023. Laminectomy.
62. Shaygan M, Zamani M, Jaberi A, Eghbal K, Dehghani A. The impact of physical and psychological pain management training on pain intensity, anxiety and disability in patients undergoing lumbar surgeries. Spine J. 2023 May;23(5):656-664.
63. Gatam AR, Gatam L, Phedy, Mahadhipta H, Ajiantoro, Aprilya D. Full Endoscopic Lumbar Stenosis Decompression: A Future Gold Standard

in Managing Degenerative Lumbar Canal Stenosis. Int J Spine Surg. 2022 Sep;16(5):821-830.
64. Wu L, Munakomi S, Cruz R. Lumbar spinal stenosis. InStatPearls [Internet] 2024 Jan 30. StatPearls Publishing.
65. Kwon JW, Suk KS, Moon SH, Park SY, Kim N, Park SR, Shin JW, Kim HS, Lee BH. Lumbar spinal stenosis: current concept of management. Asian Spine Journal. 2025 Jun 13.