Subclinical Neurodynamic Restrictions as a Contributor to Chronic Low Back Pain: A Theoretical and Clinical Perspective

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Abstract

Background: Chronic low back pain (CLBP) is a multifactorial condition and a leading cause of disability worldwide [4,5,55–57,60–63,77,78]. Despite guideline-based care, a substantial subgroup remains symptomatic, suggesting overlooked peripheral contributors [4–6,55–57,60–63,64–66,69–71,77]. Neurodynamics research indicates that peripheral nerves are mechanical structures that must slide, adapt, and tolerate limited strain during movement [1–3,7–9,11–13,15–19,27–29,33,36,39,41,42,45,51–53,72–76]. Subtle impairments in this behavior—here termed subclinical neurodynamic restrictions (SNR)—may provoke mechanosensitivity, protective muscle tone, and altered motor control without producing frank neuropathic signs [1–3,9–13,15–19,22–24,27–29,33,36,38–41,44,45,47–49,50–53,58–63,69–71,72–76,78–80].

Objective: To articulate a coherent theoretical and clinical framework for SNR as a contributor to CLBP; to operationalize assessment criteria using established neurodynamic tests; and to outline a pragmatic, progression-based management pathway for manual and rehabilitation clinicians [1–3,7–9,11–13,19,22,24,29,33,36,39–43,44,45,50–53,55–57,60–63,69–71,72–76,77,80–87,93–100].

Conceptual Approach: Drawing on foundational neurodynamic literature and clinical observation, this paper synthesizes evidence on neural excursion, perineural interface mechanics, and mechanosensitivity [1–3,7–9,11–13,15–19,27–29,33,36,38,39,41,42,45,46,51–53,72–76]. It proposes explicit SNR case criteria centered on symptom modification with neurodynamic sensitizers during the straight leg raise (SLR), Slump, and prone knee bend (PKB) tests [1–3,7,11–13,19,29,33,36,39,41–43,45,51–53,73–76]. The framework links restricted excursion/stiffer interfaces to heightened afferent input, protective hypertonicity (e.g., hamstring or gluteal guarding), and maladaptive loading patterns in the lumbopelvic region [15–18,20–24,27,28,30,31,39–41,44,45,50,58–63,67–71,80–87,88–92]. Two brief case vignettes (sciatic and cluneal presentations) illustrate application and early response to targeted care [20,21,31,39–41,44,45,88–92].

Clinical Pathway: Assessment emphasizes bilateral comparison and symptom behavior rather than range alone [1–3,7,11–13,19,29,33,36,39,41–43,45,51–53,69–71,72–76,93–99]. Decision-making is guided by irritability grading, with attention to mechanosensitivity, after-effects, and psychosocial load [22–26,33,36,39–43,47–

49,51–53,55–57,60–63,69–71,72–76,77,78,93–100]. Initial management prioritizes nerve sliders (low tensile load, high excursion) alongside gentle perineural soft-tissue/interface techniques [2,3,7–9,11–13,19,27–29,33,36,38,39,41–43,45,51–53,72–76]. As irritability decreases, programs progress to motor control and proprioceptive training, with tensioners introduced cautiously in low-irritability contexts [22,24,30,37,39–43,50,58–63,67–71,80–87]. Outcome tracking uses NPRS, ODI, PSFS, and within-test change (e.g., SLR angle, Slump/PKB symptom modulation), interpreted using established core outcome and minimal important change guidance [93–100].

Implications: The SNR framework offers clinicians a practical way to identify a plausible, under-recognized driver of persistent CLBP and to individualize care beyond generic strengthening or mobility prescriptions [1–3,4–6,22,24,29,33,36,39–43,44,45,50–53,55–57,58–63,64–71,72–76,77,80–87,93–100]. By specifying operational criteria and a progression algorithm, it supports more consistent clinical reasoning and creates testable hypotheses for future research [3,4–6,33,36,39–43,44–46,51–53,55–57,60–63,69–71,72–76,77,80–87,93–100].

Future Directions: Priorities include (1) establishing inter-rater/test–retest reliability of SNR classification; (2) quantifying peripheral nerve excursion changes (e.g., ultrasound) alongside symptom/functional outcomes; and (3) randomized trials assessing the added value of sliders and interface-focused care versus usual management [1–3,7,11–13,19,29,33,36,37,39–41,44–46,51–53,55–57,60–63,69–71,72–76,77,80–87,93–100]. If validated, SNR-targeted strategies could refine patient stratification and improve outcomes in a meaningful subset of individuals with CLBP [3–6,22–26,33,36,39–43,44–46,50–53,55–57,58–63,64–71,72–76,77,78,80–87,93–100].

1. Introduction

Chronic low back pain (CLBP) is a leading global health challenge, affecting an estimated 7.5% of the world's population (over 570 million individuals) and contributing to significant disability, healthcare costs, and reduced quality of life [4,5,55–57,60–63,77,78]. The 2018 Lancet series on low back pain highlights its multifactorial etiology, including biomechanical dysfunction (e.g., disc pathology, facet joint arthritis), psychosocial factors (e.g., fear-avoidance, catastrophizing), and central sensitization [4–6,25,26,55–57,60–63,77,78,93–100]. Despite extensive research and therapeutic advances, many patients experience persistent symptoms that resist conventional interventions, such as physical therapy, pharmacological management, or surgical procedures [4–6,55–57,60–63,69–71,72–76,77]. This therapeutic gap underscores the need for novel perspectives that address overlooked mechanisms, particularly at the interface between peripheral neural tissues and the lumbopelvic musculoskeletal system [1–3,15–19,22,24,27–29,31,39–43,44–46,50–53,58–63,69–71,72–76,80–87,88–92].

This paper introduces the hypothesis that subclinical neurodynamic restrictions (SNR)—mild limitations in peripheral nerve mobility and/or heightened mechanosensitivity without classical neuropathic symptoms—play a significant role in a subset of CLBP presentations, building on Shacklock's foundational concepts in clinical neurodynamics [1,2,7,8,29]. Unlike overt neuropathies, which present with clear neurological deficits, subclinical restrictions are subtle impairments that alter neural mechanics, influencing pain and motor behaviour without producing frank neurological signs [1–3,7–9,11–13,15–19,22–24,27–29,33,36,38–41,44,45,47–49,50–53,58–63,69–71,72–76,78–80]. The nervous system is not only a conduit for electrical signalling but also a dynamic mechanical structure that must glide, stretch, and adapt to movement [1–3,7,9,11–13,15–19,27–29,33,36,39,41,42,45,46,51–53,72–76]. Restrictions in neural mobility, even if minor, may provoke protective muscle tone, alter movement patterns, and sustain nociceptive input, creating a feedback loop that perpetuates pain chronicity [11,12,15–18,22–24,27,28,39–41,44,45,50–53,58–63,69–71,72–76,80–87]. Pain is understood here in line with the current IASP definition, as an unpleasant sensory and emotional experience associated with actual or potential tissue damage [47,48,78].

The lumbopelvic region, with its intricate network of nerves (e.g., sciatic, femoral, superior and inferior cluneal nerves), is particularly susceptible to such restrictions due to its anatomical and biomechanical complexity [20,21,31,39–41,44,45,80–82,88–92]. For example, the sciatic nerve's long course through the pelvis and posterior thigh makes it vulnerable to mechanical constraints from hypertonic muscles (e.g., piriformis, hamstrings) or fascial adhesions [20,21,31,39–41,44,45]. Similarly, the cluneal nerves, which innervate the posterior pelvis, may be compressed by gluteal hypertonicity or fascial restrictions, contributing to localized pain [20,21,88–92]. This paper aims to elucidate the theoretical basis for SNR, their role in CLBP, and their clinical implications, drawing on foundational work in neurodynamics and manual therapy [1–3,7–9,11–13,19,22,24,29,33,36,39–43,51–53,72–76]. By integrating these insights, we seek to provide a framework for clinicians to identify and address these restrictions, potentially improving outcomes for patients with recalcitrant CLBP [3–6,22–24,33,36,39–43,44–46,50–53,55–57,58–63,69–71,72–76,77,80–87,93–100].

1.1 **Global Burden, Natural History, and Clinical Heterogeneity**

CLBP remains a top cause of years lived with disability and a major driver of healthcare utilisation and indirect economic loss, including absenteeism and reduced productivity [4,5,55–57,60–63,77,78]. The condition is heterogeneous in onset (insidious vs. post-episode), clinical pattern (mechanical, movement-evoked, or mixed), and trajectory (episodic vs. persistent) [4–6,22,24,55–57,58–63,69–71,77,78,93–100]. Many patients cycle between transient relief and relapse, while a clinically meaningful subgroup reports persistent limitations despite exposure to guideline-concordant care [4–6,55–57,60–63,64–71,72–76,77]. The Lancet series underscored that no single modality dominates across all phenotypes, advocating person-centred, multimodal care and careful de-medicalisation where appropriate [4–6,55–57,60–63,64–66,69–71,77,78]. Yet, even with contemporary care models, residual pain and disability often persist—suggesting unaddressed contributors in peripheral, interface-level physiology that interact with central processes [1–3,15–

19,22–24,27–29,39–43,44–46,50–53,55–57,58–63,69–71,72–76,77,80–87,88–92,93–100].

1.2 **Why Look Beyond the Usual Suspects?**

Traditional explanations for persistent CLBP privilege spinal structures (discs, facets, ligaments) and psychosocial factors (beliefs, fear-avoidance, catastrophizing) [4–6,24–26,55–57,60–63,77,78,93–100]. While these are critical, they may not fully explain movement-specific symptom behaviour that is modulated by neural sensitizers—for example, pain that is provoked by end-range SLR but eases with ankle plantarflexion or cervical extension, or discomfort during Slump that attenuates when the neck is extended [1–3,11–13,19,29,33–37]. Such patterned changes are not well captured by joint-centric or purely psychosocial models, but they are coherent within a neurodynamic frame where nerve excursion and interface compliance matter [1–3,12,19,27–29,33,36,39–43,45,51–53,72–76]. When peripheral neural tissues glide sub-optimally, mechanosensitive afferents can respond excessively to normal loads, fostering protective hypertonicity (e.g., hamstring or gluteal guarding), altered recruitment patterns, and maladaptive movement strategies—features commonly observed in persistent CLBP [11,12,22–25,30,31,39–41,44,50,58–63,67–71,80–87].

1.3 **The Nervous System as a Mechanical Tissue**

Clinical neurodynamics emphasises that peripheral nerves must slide, elongate modestly, and undergo transverse excursion relative to surrounding tissues during everyday movements [1,13–16,27–29,33,36,39,41,42,45,51–53,72–76]. Perineural connective tissues (epineurium, perineurium) and interfascial planes enable low-friction movement and distribute stress [15,16,27,28,36,45,46]. If these interfaces become stiffer or adherent, or if regional muscle tone impairs the available “slack,” then a given limb/trunk motion may impose higher local strain at specific neural segments, sensitising afferents and altering reflex behaviour [17,18,19,22–24,27,28,39–41,44,50,58–63]. Importantly, these mechanical changes can occur without producing objective neurological deficits—hence the designation subclinical [1–3,7–9,11–13,15–19,27–29,33,36,38–41,44,45,47–49,50–53,58–63,69–71,72–76,78–80]. Clinically, this may manifest as movement-provoked, familiar pain that changes predictably with neurodynamic sensitizers (e.g., ankle dorsiflexion/plantarflexion; cervical flexion/extension) during SLR and Slump, or with hip extension/knee flexion during PKB [1–3,7,11–13,19,29,33–37,39,41–43,51–53,73–76].

1.4 **Lumbopelvic Neuroanatomy and Vulnerable Interfaces**

Anatomical and biomechanical features of the lumbopelvic region create “pinch points” where modest alterations in tissue compliance can have outsized clinical effects [20,21,31,39–41,44,45,80–82,88–92]. The sciatic nerve navigates the deep gluteal region and posterior thigh where it lies in close relation to the piriformis, short

external rotators, and hamstrings; regional hypertonicity or fascial stiffness can reduce excursion and increase local stress during hip flexion and knee extension [20,21,31,39–41,44,45]. The superior and inferior cluneal nerves traverse the posterior iliac crest and gluteal fascia, a zone where mechanical load and soft-tissue tension frequently concentrate; tenderness and symptom reproduction here can mimic facet or SIJ-related pain [21,39–41,44,45,88–92]. The femoral nerve and anterior thigh interfaces may be sensitive during prone knee bend manoeuvres and hip extension, particularly in patients with anterior pelvic tilt or iliopsoas hypertonicity [32,33,36,37,39–41]. These regional facts provide plausible mechanical substrates for subclinical restrictions to interact with motor control and symptom behaviour [20,21,30–32,39–41,44,45,50,58–63,67–71,80–87,88–92].

1.5 **From Mechanobiology to Clinical Behaviour**

Mechanosensitive afferents (C-fibres, A-delta fibres) within peripheral nerves respond to stretch and compression [17–19,27,28,36,38,39,41,45,51–53,72–76,78]. Low-grade inflammation and micro-adhesion formation within perineural tissues increase stiffness and reduce glide, lowering the threshold for nociceptive signalling during ordinary movement [17–19,27,28,36,38,39,41,44,45,50–53,58–63,69–71,72–76]. Over time, this promotes protective strategies: increased baseline tone in agonist/antagonist pairs, stiffness-dominant motor solutions (e.g., excessive erector spinae co-contraction), and reduced segmental mobility [22–25,30,39–41,50,58–63,67–71,80–87]. Patients then move “around” the problem—using trunk flexion instead of hip flexion, or avoiding terminal ranges—amplifying regional load on lumbopelvic tissues [22–25,30,39–41,50,58–63,67–71,80–87]. These patterns often persist despite generic strengthening or mobility work unless neural interfaces are addressed, at least for the subset where neurodynamic signs are present [1–3,11,12,29,33–36,39–43,44,45,50–53,58–63,69–71,72–76].

1.6 Defining Subclinical Neurodynamic Restriction (Working Criteria)

For the purposes of this paper, SNR is defined as:

1.Symptoms: CLBP ≥3 months without objective neurological deficit, dermatomal pain, progressive weakness, or reflex change; [4–6,47–49,55–57,60–63,77,78]

2.Neurodynamic signs: Reproduction of familiar lumbar/pelvic symptoms during SLR/Slump/PKB that are modified by neurodynamic sensitizers (e.g., ankle dorsiflexion/plantarflexion, cervical flexion/extension, hip position) [33–37];

3.Clinical pattern: Features consistent with protective tone, altered movement strategies, and localized mechanosensitivity along neural pathways, in the absence of frank neuropathy [11,12,22,24,31,34,39–41,44,50,58–63,80–87].

These criteria emphasise symptom modulation over range endpoints alone and align with pragmatic clinical decision-making in manual therapy and rehabilitation [1–3,7–9,11–13,15–19,22,24,27–29,31,33–37,39–42,44,45,47–49,51–53,58–63,69–71,72–76].

1.7 Distinguishing SNR from Other Phenotypes
SNR should be differentiated from:

-Mechanical LBP without neurodynamic features: pain behaviours not modifiable by neural sensitizers;

-Radiculopathy/neuropathy: objective neurological deficit and dermatomal distribution;

-Nociplastic/central mechanisms: disproportionate pain, diffuse hyperalgesia, limited relation to mechanical loading (though central processes can co-exist and be influenced by peripheral inputs) [5,6,24–26,47–49,60–63,77,78,93–100].

This differentiation matters because treatment emphasis changes: SNR suggests early prioritisation of sliders and interface techniques, with careful dosing to minimise flare, then progressive motor control and proprioception as irritability decreases [1–3,11,12,22–25,30,31,33–42,44,45,50,58–63,67–71,72–76,80–87].

1.8 Why SNR May Be Missed in Routine Care

Several practical factors obscure SNR in day-to-day clinics:

-Over-reliance on imaging: Many patients with persistent CLBP have normal or age-typical imaging; absence of a structural lesion can prematurely shift focus away from mechanical neural interfaces that are not visible on standard scans.

-Range-centric testing: Clinicians may record SLR “degrees” without probing symptom change under sensitizers, losing the key neurodynamic signal [33–37].

-Generic programming: Strengthening and flexibility programs that do not explicitly restore excursion or modulate mechanosensitivity may underperform in SNR-positive patients [1–3,11,12,39–42].

-Irritability mis-management: Over-loading early (e.g., tensioners too soon) can provoke flares, reinforcing avoidance and undermining adherence [33,39–41].

Together, these factors help explain why a subset of CLBP patients remain symptomatic despite guideline-concordant, but non-phenotype-specific, care [3–6,11,12,29,33–42,44,45,55–57,60–63,67–71,72–76,77].

1.9 Clinical Signal: What SNR Looks Like in the Room

Typical SNR-consistent patterns include:

-Movement-provoked pain at or near terminal ranges in SLR/Slump, attenuated by releasing a sensitizer (e.g., plantarflexion, cervical extension) [33–36];

-Hamstring/gluteal guarding that improves within-session when sliders/interface work is applied, with small-but-meaningful increases in comfortable range [31,39,40];

-Task-level adaptations: e.g., substituting lumbar flexion for hip flexion in bending, shortened stride due to posterior chain tension, or avoidance of prolonged sitting/standing.

These are not pathognomonic, but their consistency and modifiability under neurodynamic principles support SNR classification and guide treatment emphasis [1–3,11,12,20–22,24,30,31,33–42,44,45,50,58–63,67–71,72–76,80–87,88–92].

1.10 Integrating SNR with the Biopsychosocial Model

SNR is not a rival to the biopsychosocial paradigm—it slots inside it. Peripheral interface compromise can provide a bottom-up nociceptive driver that interacts with beliefs, expectations, and central processing [5,6,24–26,47–49,60–63,77,78,93–100]. Education should normalise symptoms (“nerves are living tissues that need to slide”), reduce threat, and explain why short, gentle movements (sliders) are more helpful initially than aggressive stretching. In practice, blending low-irritability neurodynamic work with graded activity, motor control, and simple self-calibration rules enhances adherence and reduces flare-related fear [1–3,11,12,33–42,44,45,50,58–63,67–71,72–76,80–87].

1.11 Contribution of This Paper

This work adds three pragmatic elements:

1.Operational Criteria for SNR tailored to CLBP—grounded in symptom modulation, not degrees alone;

2.A Decision Algorithm tied to irritability (high → micro-sliders/interface; moderate → standard sliders + gentle control; low → consider tensioners + task loading);

3.Dosage/Progression Rules that are simple to deliver, easy to document (NPRS, ODI, PSFS, within-test change), and compatible with routine care [33–41,93–100].

Together, these components support consistent clinical reasoning and provide testable propositions for research [1–3,4–6,11,12,33–41,44,45,51–53,55–57,60–63,67–71,72–76,77].

1.12 Scope, Assumptions, and Delimitations

Scope: Adults with CLBP ≥3 months; non-radicular presentations likely to exhibit neurodynamic modulation. Primary nerves considered: sciatic, femoral, cluneal [4–6,20–22,31,39–41,44,45,58–63,88–92]. Assumptions: Subtle interface changes (e.g., low-grade inflammation, micro-adhesions, increased tone) can meaningfully alter excursion and mechanosensitivity [17–19,22,24,27,28,31,39–41,44,45,50,58–63,69–71,72–76]. Delimitations: This paper does not cover surgical care, acute radiculopathy, or serious spinal pathology; psychosocial interventions are included only as adjuncts, not as a primary focus [5,6,24–26,47–49,55–57,60–63,77–79].

1.13 Aims and Structure of the Manuscript

Aim: To articulate a coherent theoretical and clinical framework for SNR in CLBP, operationalise assessment, and outline a progression-based management pathway [1–3,5,6,33–41,51–53,55–57,60–63,67–71,72–76,77].
Structure: Section 2 reviews nerve mechanics, mechanosensitivity, and clinical testing; Section 3 develops the SNR model and testable propositions; Section 4 provides operational definitions and documentation; Section 5 details the intervention strategy; Section 6 proposes research priorities; Section 7 summarises clinical implications [1–3,5,6,33–41,55–57,60–63,67–71,72–76,77].

1.14 Anticipated Clinical and Research Impact

Clinically, identifying SNR refines patient stratification, focusing effort on restoring glide and down-regulating mechanosensitivity before chasing range or strength. This

alignment can yield faster within-session wins (e.g., symptom-modified SLR/Slump), which in turn improve engagement and adherence [1–3,11,12,33–41,44,45,50,58–63,67–71,72–76,80–87]. For researchers, explicit criteria and a simple algorithm enable reproducible classification and intervention delivery, facilitating reliability studies, mechanism-focused cohorts, and feasibility RCTs that compare sliders + usual care versus usual care [3,12,33–41,51–53,72–76,93–100].

1.15 Summary

CLBP is common, costly, and complex. A proportion of persistent cases likely reflect subclinical neurodynamic restrictions that conventional joint-centric or generic programs do not fully address. By framing nerves as mechanical tissues that must slide and adapt—and by prioritising symptom modulation under neurodynamic testing—clinicians can identify a coherent treatment target. The remainder of this manuscript specifies the rationale, definitions, assessment steps, and management pathway to make that target actionable in everyday practice [1–3,4–6,11,12,17–19,20–22,24,25,31,33–41,44,45,50,51–53,55–57,58–63,67–71,72–76,77–79,93–100].

1.16 Epidemiology, Natural History, and Economic Burden

Low back pain ranks among the top contributors to years lived with disability across all age groups and regions, with CLBP (≥3 months) responsible for the bulk of persistent disability [4–6,55–57]. Point prevalence estimates near 7–8% translate into hundreds of millions of affected adults globally, and lifetime prevalence is substantially higher [4–6,55–57]. Natural history is heterogeneous: many individuals experience recurrent, fluctuating symptoms with inter-episode recovery, while a sizable subgroup develops a persistent pattern characterized by pain-related interference with work, caregiving, and leisure [4–6,55–57,60–63]. The economic burden includes direct medical costs (primary care, imaging, pharmaceuticals, injections, procedures) and indirect costs (absenteeism, presenteeism, early retirement), with downstream societal effects on productivity and caregiver time [4–6,55–57]. Critically, the mismatch between spending and outcomes persists: high utilization of imaging and procedure-heavy pathways often fails to improve functional endpoints in non-specific presentations [5,6,55–57,77]. This gap invites more precise phenotyping—including identification of subgroups like SNR-positive CLBP—so that management pathways can be better aligned to underlying mechanisms [3–6,11,12,33–41,44,45,51–53,60–63,67–71,72–76,77–79].

1.17 Neuroanatomy of the Lumbopelvic Region: Sites of Mechanical Vulnerability

1.17.1 Sciatic Nerve and Deep Gluteal Interfaces

The sciatic nerve traverses the greater sciatic notch beneath (most commonly) the piriformis and between short external rotators before descending beneath the gluteus maximus into the posterior thigh [20,21,31]. Here it lies within dense fascial envelopes and interfaces intimately with the hamstring origin. Regional hypertonicity (piriformis, deep rotators, proximal hamstrings) or increased fascial stiffness can reduce local excursion during hip flexion/knee extension combinations, the very motions taxed by SLR and Slump sequences [20,21,31,33–36]. Even in the absence of entrapment neuropathy, micro-adhesions and postural/motor biases may raise local strain at a given range, lowering the threshold for symptom provocation [13–19,22,24,27,28,31,39–41,44,45,50].

1.17.2 Superior/Inferior Cluneal Nerves and Posterior Iliac Crest

The superior cluneal nerves cross the posterior iliac crest through osteofibrous tunnels and penetrate the thoracolumbar fascia to innervate skin of the posterior iliac crest and upper buttock [21]. Mechanical pinch points over the crest, coupled with gluteal fascial tension or thickening, can sensitize these cutaneous branches; palpation and functional loading may reproduce familiar, localized pain that can mimic facet or SIJ pain patterns [21,39,40,88–92]. Gentle transverse gliding and targeted interface work often modulate symptoms in this territory, reinforcing the interface hypothesis [21,39–41,44,45,88–92].

1.17.3 Femoral Nerve and Anterior Thigh Interfaces

The femoral nerve passes beneath the inguinal ligament into the femoral triangle; proximal neural interfaces interact with iliopsoas and anterior hip capsule. In prone knee bend (PKB), combined knee flexion and hip extension bias the femoral pathway [32,37]. Patients with anterior pelvic tilt, iliopsoas hypertonicity, or reduced anterior hip capsular compliance may show anterior thigh symptoms that modify with cervical position or hip adjustments—again consistent with neurodynamic contribution rather than pure muscle stretch [32,33,37,39–41,44,45,80–82]. These patterns help define a femoral-biased SNR phenotype when considered alongside global clinical features [1–3,11,12,32,33,37,39–41,44,45].

1.18 Mechanobiology of Peripheral Nerves: Glide, Strain, and Interface Stiffness

Peripheral nerves tolerate modest strain through hierarchical architecture: endoneurial microenvironment, perineurial lamellae conferring stiffness and barrier function, and epineurial sheaths permitting longitudinal/transverse movement [15,16,27,28,36,45,46]. In healthy conditions, movement is accommodated primarily by glide (sliding) rather than large elongation; when glide is reduced, a greater portion of motion is absorbed as strain within the nerve and its vasculature [13–

16,27,28,36,38,39,41,45,46,51–53]. Basic science and in vivo work indicate that low-grade inflammation and fibrosis increase perineural stiffness and reduce excursion, sensitizing mechanosensitive afferents (C-fibre/A-delta) and lowering thresholds for nociceptive signalling [17,18,19,27,28,36,38,39,41,44,45,50–53,69–71,72–76]. Repetitive micro-loading—prolonged sitting, repetitive lumbar flexion/extension, constrained hip strategies—can accumulate interface changes (micro-adhesions, viscosity increases), making previously innocuous ranges symptomatic [11,12,17–19,22,24,27,28,30,31,39–41,44,45,50,58–63]. Within this context, sliders aim to restore relative movement across interfaces at low tensile loads; tensioners (used later) dose controlled strain when irritability is low, with the goal of normalizing viscoelastic behaviour [1–3,7–9,29,33,39–42,44,45,51–53,72–76].

1.19 Central–Peripheral Interplay: Why Peripheral Interfaces Still Matter

While central sensitization frameworks explain persistent pain without clear lesion, peripheral inputs remain capable of driving and maintaining central changes [5,6,25,26,47–49,60–63,77,78]. Protective motor adaptations—heightened baseline tone, synergistic co-contraction—alter proprioceptive feedback and sensorimotor representations, potentially reinforcing pain and stiffness perceptions [22–25,30,39–41,50,58–63,67–71,80–87]. Addressing peripheral sources of mechanosensitivity can therefore provide bottom-up relief, enabling motor recalibration and more effective integration of education and graded exposure [1–3,11,12,33–42,44,45,50,58–63,67–71,72–76,80–87]. In clinical terms, achieving within-session symptom modulation with sliders or interface work can reduce threat value, enhance self-efficacy, and make higher-level rehabilitation more tolerable—allowing biopsychosocial care to work synergistically rather than competitively [5,6,25,26,39–43,55–57,77–79,93–100].

1.20 Differential Diagnosis and Phenotype Delineation: Practical Decision Points

Distinguishing SNR from competing explanations improves targeting:

-Facet/SIJ-dominant pain: Often provoked by extension/rotation (facet) or specific load transfer tests (SIJ); lacks consistent sensitizer-driven symptom change in neurodynamic tests.

-Myofascial pain without neural contribution: Local taut bands and trigger points respond primarily to myofascial techniques/stretching without predictable modulation by ankle/cervical changes in SLR/Slump.

-Radiculopathy/neuropathy: Dermatomal distribution, reflex/sensory/motor deficits; neurodynamic tests may reproduce distal symptoms but objective neurological signs are present.

-Predominantly nociplastic/central: Diffuse hyperalgesia, sleep disturbance, inconsistent relation to load/movement; neurodynamic tests may be uncomfortable but lack consistent sensitizer responses [5,6,24–26,47–49,60–63,77,78,93–100].

Rule-in SNR when: (i) familiar pain is elicited near end-range SLR/Slump/PKB and changes predictably with neural sensitizers; (ii) no objective neurological deficits; (iii) clinical picture includes protective tone and movement substitutions that ease as mechanosensitivity is down-regulated [1–3,11,12,21,31,33–37,39–42,44,45,50,58–63,67–71,72–76,80–87,88–92].

1.21 Measurement, Documentation, and Clinically Meaningful Change

What to record:

-NPRS, ODI, PSFS at baseline and planned re-assessments (e.g., weeks 2–3 and 6) [93–100].

-SLR angle at symptom onset; change with ankle DF; change with cervical flexion/extension [33,36].

-Slump sequence: exact order, symptom behaviour, modification with reversing sensitizers [33–36].

-PKB: knee flexion angle at onset; change with hip extension or cervical position [32,37].

-Irritability grade (high/moderate/low) and flare rules [33,39–41].

Meaningful change: Small but consistent improvements in end-range tolerance (e.g., +10–15° SLR over several sessions) and predictable symptom easing with sensitizer release signal progress. Clinicians should pair these with functional gains on PSFS to capture real-world improvement [33,36,39–41,44,45,50,58–63,67–71,93–100].

1.22 Why Generic Programs Underperform in SNR-Positive Patients

General strengthening/flexibility programs may miss the mark when they do not restore excursion or inadvertently increase tensile stress early (e.g., aggressive hamstring stretching in a high-irritability sciatic SNR). Without first down-regulating mechanosensitivity via sliders and addressing interfaces (gluteal/hamstring fascial stiffness, iliopsoas tone), patients can flare, disengage, or adopt more guarded

movement strategies [11,12,22–25,30,31,39–41,44,45,50,58–63,67–71,80–87]. Incorporating low-load, high-repetition sliders, gentle interface work, and graded motor control early aligns the stimulus with the biology of the problem and eases entry into progressive loading [1–3,7–9,29,33–42,44,45,51–53,72–76].

1.23 Terminology and Conceptual Clarity

"Subclinical" denotes absence of frank neuropathic signs (dermatomal pain, objective sensory/motor loss, reflex changes), not that the problem is trivial [4–6,9,10,47–49,60–63,78,79]. "Restriction" refers to reduced relative movement (excursion) or increased interface stiffness that functionally elevates mechanosensitivity during everyday loading [13–19,22,24,27,28,31,36,38,39,41,44,45,50–53,58–63,69–71,72–76]. "Mechanosensitivity" captures lowered thresholds and increased response to mechanical stimuli (stretch/compression) at peripheral receptors—distinct from, yet interactive with, central sensitization [17,18,25,27,28,36,38,39,41,44,45,47–49,60–63,72–76,78]. These distinctions matter for communication, patient education, and research reproducibility [1–3,5,6,24–26,33–41,47–49,55–57,77–79,93–100].

1.24 Clinical Vignettes (Introductory Signals)

-Posterior chain dominant CLBP: A 50-year-old teacher with non-dermatomal posterior discomfort shows SLR onset at 60°; ankle plantarflexion reduces familiar pain, cervical flexion increases it. After 2–3 weeks of sliders + gluteal/hamstring interface work, SLR tolerates 75° with less guarding and improved PSFS for sitting tolerance—pattern consistent with SNR [33,36,39,40,44,45,50,58–63,67–71].

-Posterior iliac crest pain: A 42-year-old nurse reports focal tenderness over the posterior iliac crest. Slump is uncomfortable but modifies with neck extension; transverse gliding over the crest and graded sliders reduce local symptoms and improve standing tolerance—cluneal interface signal [21,39,40,44,45,88–92].

These vignettes illustrate modifiability—a cornerstone for identifying SNR and guiding care [1–3,11,12,20–22,24,30,31,33–42,44,45,50,58–63,67–71,72–76,80–87,88–92].

1.25 Limitations of Current Evidence and Rationale for a Structured Framework

Evidence linking peripheral neural mechanics to CLBP spans mechanistic, observational, and interventional domains but remains fragmented: methods for

SLR/Slump/PKB vary; some studies emphasize range rather than symptom modification; and few trials stratify patients by neurodynamic phenotype [1–3,11,12,19,29,33–41,44,45,51–53,72–76]. Without operational criteria and a simple decision algorithm, clinicians struggle to apply neurodynamics consistently, and researchers cannot easily compare studies or build cumulative evidence [3–6,12,33–41,44,45,51–53,55–57,60–63,67–71,72–76,77–79]. This paper addresses these deficits by specifying workable criteria, scripts, and dosage rules, thereby creating a platform for reliability studies and feasibility trials.

1.26 Aims (Restated) and Integration with the Remainder of the Manuscript

-Aim: Provide a practical, defensible framework for identifying and managing SNR-positive CLBP, grounded in neurodynamics and compatible with biopsychosocial care [1–3,5,6,24–26,33–41,51–53,55–57,60–63,67–71,72–76,77–79].

-Integration: Section 2 supplies the literature foundation; Section 3 formalizes the SNR logic model and hypotheses; Section 4 operationalizes clinical tests and decision-making; Section 5 presents the graded management pathway (sliders → interface → motor control/proprioception → task loading); Section 6 outlines concrete research next steps; Section 7 concludes with implications for practice [1–3,5,6,33–41,44,45,51–53,55–57,60–63,67–71,72–76,77].

1.27 Working Summary

A non-trivial subset of persistent, non-radicular CLBP likely reflects subclinical neurodynamic restrictions—reduced excursion and/or increased interface stiffness that heighten mechanosensitivity. These patients often display predictable symptom modulation with neurodynamic sensitizers, protective tone, and movement substitutions. By identifying SNR through symptom-modified SLR/Slump/PKB and by dosing interventions according to irritability, clinicians can pursue a biologically plausible, low-risk pathway that integrates smoothly with education and graded activity [1–3,5,6,11,12,17–19,20–22,24,25,31,33–41,44,45,50,51–53,55–57,58–63,67–71,72–76,77–79,93–100]. Establishing clear criteria, scripts, and progression rules is a necessary precursor to robust measurement work and trials capable of testing the added value of SNR-targeted care [3,12,33–41,51–53,72–76,93–100].

1.28 Detailed Biomechanics of the Sciatic Pathway in Functional Tasks

During forward bending, sit-to-stand, and gait, the sciatic pathway must accommodate a complex choreography of glide and modest strain. In hip flexion with knee

extension—mirrored clinically by SLR and components of the Slump—the sciatic nerve normally displaces distally in the thigh while perineural tissues buffer load [13–16,27,28,36,39,41,45,46]. When posterior chain stiffness (proximal hamstrings, deep gluteal muscles) and gluteal fascial tension reduce interface compliance, relative movement is reallocated: less excursion occurs in the interface and more intraneural strain accumulates at a given joint angle. Mechanosensitive afferents (C and A-delta) are then recruited earlier, and the perceived end-range arrives sooner with familiar pain [17,18,19,27,28,36,38,39,41,44,45,50–53,58–63,69–71,72–76]. Clinically, symptom behaviour that improves instantly when a sensitizer is released (plantarflexion, cervical extension) argues for a neural mechanical constraint rather than pure muscle shortness—because changing a spinal/ankle component alters symptoms without lengthening the hamstrings themselves [33–36]. These dynamics explain why sliders—which trade tension at one end for slack at the other in rhythm—often yield within-session relief in SNR-positive patients [1–3,7–9,29,33–36,39–42,44,45,51–53,72–76].

1.29 Femoral Pathway Mechanics and the Role of the Iliopsoas–Capsular Interface

The femoral nerve must traverse under the inguinal ligament into the femoral triangle, gliding relative to the iliopsoas tendon and anterior hip capsule. Prone knee bend (PKB) increases anterior thigh neural load via knee flexion and, when combined with gentle hip extension, further biases the pathway [32,37]. In patients with anterior pelvic tilt, iliopsoas hypertonicity, or capsular stiffness, perineural sliding may be impeded, producing early anterior thigh discomfort that is modulated by cervical or hip adjustments—again pointing toward a neurodynamic component [32,33,37,39–41,44,45,80–82]. Progression rules therefore favour early low-amplitude femoral sliders and interface soft tissue work, adding hip extension loading later, once irritability is low [1–3,7–9,29,32,33,37,39–42,44,45,51–53,72–76].

1.30 Cluneal Nerves and Posterior Iliac Crest: Small Branches, Big Signals

The superior cluneal nerves cross osteofibrous tunnels at the posterior iliac crest and can be sensitive to shear or focal compression from thickened fascia and local tissue tension [21,88–92]. Although cutaneous, cluneal irritation can mimic deep lumbopelvic pain due to overlap and convergence in dorsal horn processing. Reproduction of familiar symptoms with palpation/transverse gliding over the crest and modification during Slump with cervical adjustments is consistent with an interface-dominant contribution rather than facet/SIJ alone [21,39,40,44,45,88–92]. Gentle transverse interface mobilization, desensitization, and graded loading of gluteal fascia are therefore plausible first steps, integrated with global sliders when SNR signs are present [1–3,11,12,20–22,24,30,31,33–42,44,45,50,58–63,67–71,72–76,80–87,88–92].

1.31 Central–Peripheral Interdependence: A Two-Way Street

Peripheral input from sensitized neural interfaces can maintain or amplify central sensitization via sustained nociceptive drive and altered proprioceptive signalling [22–25,48,49,60–63,78,79]. Conversely, central mechanisms can lower peripheral thresholds, making ordinary interface loads symptomatic [25,26,48,49,60–63,77–79]. Practically, this mutual influence argues for dual targeting: (i) bottom-up reduction of mechanosensitivity through sliders/interface work and (ii) top-down strategies (education, graded exposure, expectation management) [1–3,5,6,24–26,33–42,44,45,51–53,55–57,60–63,67–71,72–76,77–79]. Within-session predictable modulation (e.g., Slump easing with neck extension) provides a concrete, embodied learning experience that reduces perceived threat and facilitates motor recalibration—often a prerequisite for patients who otherwise brace or avoid motion [1–3,11,12,25,26,33–37,39–42,44,45,50,58–63,67–71,72–76,80–87].

1.32 A Flow-Based Differential and Decision Logic (Textual Algorithm)

Step 1: Screen red flags and frank neuropathy (dermatomal pattern, objective deficit) [4–6,47–49,55–57,60–63,77–79].
Step 2: Baseline NPRS, ODI, PSFS; irritability grading (high/moderate/low) [93–100].
Step 3: Provocation with Sensitizers

-SLR to first familiar symptom → add ankle DF → add cervical flexion.
If symptoms increase with sensitizers and ease when reversed → neurodynamic contribution likely [1–3,11,12,33–37].

-Slump with staged spine, neck, knee, and ankle positions; reverse sensitizers and observe symptom modification [33–36].

-PKB for anterior pathway; modify with hip or neck position [32,37].

Step 4: Classify

-SNR-positive: consistent symptom modulation; no objective neuro deficit.

-SNR-uncertain: inconsistent modulation; consider re-test after interface warm-up.

-SNR-negative: no consistent modulation; pursue other phenotypes (facet/SIJ/myofascial/nociplastic) [5,6,21,24–26,33–37,48,49,60–63,77,78,88–92].

Step 5: Match Intervention to Irritability

-High: micro-sliders (pain ≤3/10), gentle interface de-loading, brief motion snacks, education; avoid tensioners [1–3,11,12,33–37,39–42,44,45,50,58–63,67–71,72–76].

-Moderate: standard sliders, light motor control; test small tension doses only if next-day response is clean [1–3,11,12,33–37,39–42,44,45,50,58–63,67–71,72–76].

-Low: consider tensioners, progress amplitude/frequency, add task-specific loading and proprioception [1–3,32,33,37,39–42,44,45,50,58–63,67–71,72–76,80–87].

Step 6: Reassess at 2–3 and 6 weeks: NPRS/ODI/PSFS + within-test changes (SLR angle, Slump/PKB modulation). Adjust dosage with the 24-hour rule (no >24 h flare) [33–37,39–41,44,45,50,58–63,67–71,72–76,93–100].

1.33 Why "Symptom Modulation" Beats "Degrees Alone"

Degrees of motion (e.g., SLR angle) are informative but non-specific—they shift with hamstring extensibility, pelvic control, fear, and testing style [22–25,30,31,33–37,58–63]. In contrast, predictable change under sensitizers (cervical/ankle/hip) is a patterned, mechanistic clue that a neural pathway is bearing load it cannot comfortably accommodate. This is why the working definition of SNR centres on modulation: it reflects the property of the system (excursion allocation across the chain) rather than just an endpoint number [1–3,13–19,22–25,27,28,31,33–37,39–42,44,45,51–53,69–76].

1.34 Patient Education Language (Clinic-Ready)

-"Nerves are living cables." They don't like being yanked; they prefer sliding through their sleeves [1–3,13–19,27,28,36,39–42,44,45,51–53,69–76].

-"We'll move the stress around." Sliders share the load between joints so the nerve glides without a big stretch [1–3,7–9,29,33–37,39–42,44,45,51–53,69–76].

-"Green, yellow, red." Green = mild, easing quickly; Yellow = tolerable, fades within hours; Red = lingers >24 h—we dial back [33,39–41,44,45,50,58–63].

-"Little and often." Short bouts (30–60 seconds) beat long stretches early on [1–3,33–37,39–42,44,45,51–53,72–76].

-"Wins you can feel." If your pain eases when we release the ankle or neck, that tells us we're on the right track [33–37,39–42,44,45,50,58–63,67–71,72–76,80–87].

Provide a one-page handout summarizing these ideas and the self-calibration rules (reduce amplitude/frequency if next-day soreness >3/10 or lingers >24 h; use heat/breathing; resume at the last comfortable setting) [93–100].

1.35 Common Pitfalls and How to Avoid Them

-Too much tensile load too soon: Aggressive hamstring stretching in a high-irritability sciatic SNR provokes flares. Start with sliders [1–3,11,12,22–25,30,31,33–37,39–42,44,45,50,58–63,67–71,72–76].

-Ignoring interfaces: Skipping gluteal/hamstring or iliopsoas interface work leaves excursion bottlenecks unaddressed [15–19,20–22,31,39–42,44,45,50,58–63,80–82,88–92].

-Range-only thinking: Recording SLR degrees without sensitizers misses the neurodynamic signal [33–37].

-No 24-hour check: Lack of next-day monitoring blunts dosage learning; use symptom diaries or a simple traffic-light log [33,39–41,44,45,50,58–63,93–100].

-One-size-fits-all dosing: Progress amplitude/frequency only when irritability permits; regress quickly after a flare [1–3,11,12,33–37,39–42,44,45,50,58–63,67–71,72–76].

1.36 Documentation Cheatsheet (What to Chart in <90 Seconds)

-NPRS / ODI / PSFS (with one specific functional goal) [93–100].

-SLR L/R: angle at first familiar symptom; change with DF (+/0/−); change with cervical flexion (+/0/−) [33,36].

-Slump: provocation (+/0/−) with sequence; symptom change when reversing sensitizers [33–36].

-PKB: angle and symptom change with hip/cervical adjustments [32,37].

-Irritability: High / Moderate / Low; 24-h rule outcome from last session [33,39–41].

-Today’s dose: sliders reps × sets; interface region; motor-control drill; home program tweaks [1–3,11,12,33–42,44,45,50,58–63,67–71,72–76].

1.37 Glossary of Key Terms (Working)

-Subclinical Neurodynamic Restriction (SNR): Reduced neural excursion and/or increased interface stiffness causing mechanosensitivity without objective neurological deficit; identified by symptom modulation with neurodynamic sensitizers [1–3,4–6,9–12,13–19,22–25,27,28,31,33–37,39–42,44,45,47–49,51–53,58–63,69–76,78,79].

-Neurodynamic Sensitizers: Positional changes that shift neural load (e.g., ankle dorsiflexion/plantarflexion, cervical flexion/extension, hip position) during SLR/Slump/PKB [1–3,11,12,33–37].

-Sliders: Alternating movements at adjacent joints to promote longitudinal nerve glide with low tensile load [1–3,7–9,29,33–37,39–42,44,45,51–53,69–76].

-Tensioners: Movements that increase tensile load across the neural pathway; used later at low irritability [1–3,7–9,29,33,39–42,44,45,51–53,69–76].

-Interface Techniques: Manual/movement strategies targeting tissues that surround nerves (gluteals, hamstrings, fascia, iliopsoas) to restore relative movement and reduce local compression/shear [15–19,20–22,31,39–42,44,45,50,58–63,80–82,88–92].

-Irritability: Symptom severity and persistence after loading; used to dose interventions safely [33,39–41,44,45,50,58–63].

-Symptom Modulation: Predictable change in familiar symptoms with sensitizer application/release—primary clinical signal of SNR [1–3,11,12,33–37,39–42,44,45,50,58–63,67–71,72–76].

1.38 Conceptual Figure (Caption Text You Can Paste Under a Diagram)

Figure 1. Conceptual model of subclinical neurodynamic restriction (SNR) in CLBP.
Panel A: In healthy conditions, movement is accommodated primarily by neural glide within compliant interfaces (epineurium/perineurium, fascia), keeping intraneural strain low [13–19,27,28,36,38,39,41,45,46].
Panel B: With interface stiffness (micro-adhesions, low-grade inflammation, muscle hypertonicity), the same movement produces earlier mechanosensitive afferent recruitment, protective muscle tone, and altered motor strategies [17–19,22–25,27,28,30,31,36,38,39,41,44,45,50–53,58–63,69–71,72–76,80–87].
Panel C: Clinical testing (SLR, Slump, PKB) reveals predictable symptom modulation with neurodynamic sensitizers; early management prioritizes sliders and interface techniques, followed by motor control/proprioception and cautious tensioning as

irritability declines [1–3,11,12,20–22,32,33–37,39–42,44,45,50–53,58–63,67–71,72–76,80–87,88–92].
Panel D: Outcome tracking (NPRS, ODI, PSFS, within-test changes) guides dosage. If validated, SNR-targeted care may improve stratified outcomes in a subset of CLBP [3–6,11,12,33–41,44,45,51–53,55–57,58–63,67–71,72–76,77–79,93–100].

1.39 Structured Differential: Feature-by-Feature Comparison

Phenotype	Typical Provocation	Neurodynamic Sensitizer Effect (SLR/Slump/PKB)	Neurological Signs	Palpation/Interface Findings	First-Line Emphasis
SNR-positive CLBP	End-range combined movements (e.g., hip flexion + knee extension; seated slump)	**Predictable modulation**: ↑ with DF/cervical flexion, ↓ with PF/cervical extension	**Absent** (no objective deficit)	Focal tenderness along neural course; myofascial/interface stiffness (gluteals/hamstrings/iliopsoas; posterior iliac crest)	**Sliders**, interface techniques; graded motor control; cautious tensioners later
Facet-dominant LBP	Extension/rotation/loaded compression	Minimal/variable; not systematically modulated by sensitizers	Absent	Paraspinal tenderness; articular signs	Segmental techniques, movement control, graded exposure
SIJ-related pain	Load transfer tasks (e.g., single-leg stance, hip shear)	Minimal; not systematically modulated	Absent	SIJ provocation pattern; pelvic ring dysfunction	Ring stability, hip strategy training
Myofascial pain (no neural component)	Stretch, compression of specific muscles	Discomfort may occur but **not** systematically modified by sensitizers	Absent	Taut bands/trigger points; local referral	Myofascial release, graded flexibility, strength
Radiculopathy/neuropathy	Cough/sneeze/valsalva; SLR/Slump	May provoke distal symptoms; **plus objective deficit**	**Present** (sensory/motor/reflex)	Nerve root signs	Pathway per guidelines; consider medical referral
Predominantly nociplastic	Diffuse, inconsistent with load;	Variable; often non-	Absent	Widespread tenderness	Education, graded

Phenotype	Typical Provocation	Neurodynamic Sensitizer Effect (SLR/Slump/PKB)	Neurological Signs	Palpation/Interface Findings	First-Line Emphasis
	sleep/fatigue issues	patterned			exposure, sleep/stress, activity pacing

Representative supporting references for the phenotypes and first-line emphases in this table: neurodynamics and SNR [1–3,11–13,29,31,33–37,42,51–54,72–76]; CLBP epidemiology/guidelines [4–6,55–57,77]; motor control and proprioception [22,24,30,50,58–59,80–87]; nociplastic/central sensitisation and psychosocial factors [25–26,47–49,60–63,78–79,100]; cluneal/interface-related pain [20–21,88–92]; outcome measures [93–99].

Clinical rule-in for SNR: familiar pain reliably changes with neural sensitizers in the absence of deficit, plus interface findings compatible with neural course involvement [33–37].

1.40 Epidemiology and Cost—Expanded Context

Global point prevalence near 7–8% translates to an enormous absolute burden; recurrence after an acute episode is common, and transition to chronicity occurs in a meaningful subset [4–6]. Indirect costs (absenteeism/presenteeism, reduced participation, caregiver time) typically exceed direct medical costs. Importantly, high spend on imaging/procedures in non-specific LBP does not track with better outcomes [5,6,55–57]. More precise subgrouping—e.g., identifying SNR-positive patients—aims to redirect care toward lower-cost, mechanism-aligned strategies (education + sliders + interface work + graded control), with escalation only as needed [1–3,33–41,55–57].

1.41 Where SNR Sits Relative to Nociplastic Pain

SNR and nociplastic frameworks are complementary. Nociplastic pain emphasizes central amplification and altered pain modulation; SNR spotlights peripheral interface mechanics that can feed central gain [5,6,25,26,48,49,60–63,78,79]. In practice:

-If neurodynamic modulation is strong and reproducible, prioritize peripheral down-regulation (sliders/interface), while embedding brief top-down strategies (expectation

setting, graded exposure).

-If nociplastic signs dominate (diffuse tenderness, sleep disturbance, poor load-symptom coupling), expand psychosocial emphasis and use neurodynamic work as tolerability builders rather than primary drivers [25,26,48,49,60–63,78–80].

1.42 Limitations of the SNR Model (and How This Paper Mitigates Them)

1.Measurement heterogeneity: Neurodynamic tests vary by sequence and end-point.
Mitigation: Provide scripts, emphasize symptom modulation over degrees, and propose reliability checks [33–37].

2.Attribution risk: Symptom change with sensitizers could reflect non-neural factors (e.g., muscle tone shifts).
Mitigation: Use converging signs: modulation + interface findings + reproducibility + functional carryover [22,24,31,33–37,39–41].

3.Irritability sensitivity: Early overloading (tensioners too soon) can flare symptoms.
Mitigation: Employ 24-hour rule, start with sliders, and titrate [1–3,33,39–41].

4.Comorbidity/overlap: SNR can co-exist with facet/SIJ/myofascial or nociplastic features.
Mitigation: Use the flow-based differential (Section 1.32); treat the dominant driver while respecting co-drivers [4–6,21,22,24,31,48,49,55–57].

1.43 Ethical, Feasibility, and Safety Considerations

The SNR pathway relies on low-risk first-line strategies: gentle sliders, interface soft-tissue, and graded control [1–3,39–42]. Safety is anchored to:
-Irritability grading and the 24-hour rule (no persistent >24 h exacerbation).
-Early de-escalation if flares occur; re-establish tolerable amplitude/frequency.
-Screening for red flags and progressive deficits before neurodynamic work.
These principles are compatible with routine outpatient practice and with research protocols (pilot reliability/feasibility studies) [4–6,33,39–41,55–57,77].

1.44 From Theory to Testable Propositions (Operationalized)

-H1 (Classification reliability): SNR classification via scripted SLR/Slump/PKB shows ICC ≥0.75 across raters; SEM and MDC reported [33–37].

-H2 (Mechanistic sensitivity): SNR-positive patients demonstrate larger within-session symptom modulation (effect size) than SNR-negative CLBP on standardized

neurodynamic sequences [1–3,11–12,33–37].

-H3 (Treatment moderation): Sliders + interface-focused care yield greater improvements in NPRS/ODI/PSFS at 6 weeks for SNR-positive vs. SNR-negative CLBP [3,11–12,33–41,51–53,72–76].

-H4 (Biomechanical change): Ultrasound measures of sciatic excursion increase after a 4-week slider program and correlate with symptom/function gains [13–19,39–41,46].

1.45 Integration with Multimodal Rehabilitation Pathways

SNR-directed care layers cleanly into evidence-based CLBP management:

1.Education (threat reduction; “nerves need to slide”).

2.Sliders + interface (dose to irritability; quick “wins”).

3.Motor control (neutral spine, hip hinge, gait mechanics).

4.Proprioception & graded tasks (return-to-function).

5.Psychosocial adjuncts (graded exposure, expectancy alignment).
This staged plan respects clinical capacity and can be delivered in brief sessions with simple home programming [4–6,22–26,39–43,55–57,67–71,77–80].

1.46 Anticipated Objections and Brief Rejoinders

-“This is just hamstring tightness.”
Rejoinder: Tight muscle does not typically show predictable symptom change with ankle/cervical sensitizers; SNR does [33–36].

-“Neurodynamic tests lack specificity.”
Rejoinder: True for range-only endpoints. This framework elevates symptom modulation plus interface findings and reproducibility, improving clinical signal [1–3,33–37].

-“Patients flare with nerve work.”
Rejoinder: Over-tensioning early is the issue. Start with sliders and calibrate by 24-hour rule; progression is conditional on clean next-day response [1–3,33,39–41,51–

53,72–76].

-“Central factors trump everything.”
Rejoinder: Central and peripheral mechanisms are bidirectional; reducing peripheral drive can enable top-down gains [5,6,22–26,48,49,60–63,78–80].

1.47 Reporting Checklist for Clinical Use (Quick Reference)

When documenting an SNR-positive presentation, include:

-Symptom modulation details in SLR/Slump/PKB (which sensitizer changed what).

-Interface regions treated and rationale.

-Irritability grade; dose (reps × sets; amplitude); 24-hour response.

-Outcome trio: NPRS, ODI, PSFS; within-test change (SLR angle, symptom behaviour).

-Education points delivered and patient’s self-calibration plan.
This checklist improves reproducibility across clinicians and aligns with research reporting [33–37,39–41,93–99].

1.48 Practical Takeaways (for the Busy Clinician)

-Think glide first: early sliders over stretching/tensioners.

-Chase modulation, not degrees: document how symptoms change with sensitizers.

-Treat the interfaces (gluteals/hamstrings/iliopsoas; posterior iliac crest) that bottleneck excursion.

-Dose by irritability; obey the 24-hour rule.

-Integrate motor control and graded function as mechanosensitivity quiets.
-Keep NPRS/ODI/PSFS plus within-test changes to demonstrate progress [1–3,21–25,31,33–41,51–53,67–71,93–99].

1.49 How This Introduction Supports the Remainder of the Work

Sections 1.16–1.48 provided the epidemiologic rationale, anatomical/mechanobiological plausibility, central–peripheral interplay, a structured differential, and operational tools (scripts, decision logic, documentation). This scaffolding feeds directly into:

Chapter 2: detailed literature synthesis on neural mechanics and mechanosensitivity.

Chapter 3: formal SNR logic model and hypotheses.

Chapter 4: standardized assessment and decision algorithm.

Chapter 5: graded treatment pathway (with dosage and progression rules).

Chapter 6: research agenda (reliability, mechanisms, feasibility RCTs).
This structure is designed to be both clinically pragmatic and research-ready in the context of contemporary CLBP science [1–6,11–19,22–26,31–41,44,45,51–57,60–63,67–71,77–80,93–99].

1.50 Closing Orientation

CLBP's complexity demands mechanism-aware care. Subclinical neurodynamic restrictions offer a tractable clinical target that is detectable at the bedside through symptom modulation and addressable with low-risk interventions. By codifying definitions, scripts, and progression rules, this Introduction sets the stage for systematic application and evaluation of SNR-informed practice. The next chapter elaborates the empirical and theoretical foundations that justify this approach and identify the most promising levers for clinical change [1–6,11–19,22–26,31–41,44,45,51–57,60–63,67–71,77–80,93–100].

Introduction Review: Reliability, Validity, and Clinical Utility of Neurodynamic Testing in CLBP

Scope and purpose

Neurodynamic tests—including the straight leg raise (SLR), Slump, and prone knee bend (PKB)—are core to identifying movement-provoked symptom behaviour that may implicate neural tissues in chronic low back pain (CLBP). In this mini-review we summarize (i) reliability of common test procedures, (ii) aspects of validity (construct, discriminative, and responsiveness), and (iii) practical considerations for clinical utility and documentation. The emphasis is on symptom modulation with sensitizers

(e.g., ankle dorsiflexion/plantarflexion, cervical flexion/extension) rather than range endpoints alone, consistent with the subclinical neurodynamic restriction (SNR) construct developed in this manuscript [1–3,11–12,33–37].

Why reliability matters for an SNR phenotype

For SNR to be clinically meaningful and researchable, clinicians must reproduce findings within and between sessions and across examiners. Traditional critiques of neurodynamic testing point to variability in end-point selection (first onset vs. maximum tolerable), sequencing of sensitizers, and inconsistent recording of test angles. These concerns are legitimate when tests are used range-only. However, when procedures are scripted, end-points are standardized, and clinicians document symptom modulation (change with application and release of sensitizers), reliability improves and the tests say more about the property of the neural system rather than joint flexibility per se [33–37].

Straight Leg Raise (SLR): reliability and interpretation

The SLR remains the most widely used neurodynamic test for the posterior chain. Studies in musculoskeletal populations show moderate-to-good intra-rater reliability for angle measurement when using clear end-points and inclinometers or goniometers; inter-rater reliability is typically moderate but improves with standardized instruction and the use of symptom-first end-points rather than "stretch" [33,36]. Adding ankle dorsiflexion or cervical flexion as sensitizers changes neural load distribution; a predictable increase in familiar symptoms with these sensitizers—and easing with plantarflexion or cervical extension—supports a neurodynamic contribution [33–36]. Importantly, this behaviour is more informative for SNR than absolute angle because it reflects load sensitivity of the neural pathway and its interfaces [1–3,11–13,36]. From a mechanistic perspective, in vivo and experimental studies confirm that nerves undergo longitudinal sliding and modest strain during SLR; perineural stiffness and interface adhesions reduce glide and raise mechanosensitive afferent recruitment at earlier ranges [13–19,27,28,45,46]. This aligns with clinical observations that patients with SNR-consistent signs reach "symptom onset" at lower angles yet display immediate improvement when a sensitizer is released—an effect not well explained by hamstring length alone [31,33–37,39–41].

Slump test: reliability, staging, and value of sensitizers

The seated Slump incorporates spinal and dural loading via staged flexion, knee extension, and ankle dorsiflexion, typically finalized by cervical flexion. Reliability work suggests that staging (spine → neck → knee → ankle) and consistent reversal of sensitizers markedly improve agreement on both symptom provocation and symptom change [33–35]. Inter-rater reliability for presence/absence of familiar symptoms and their modulation tends to be moderate-to-good when a scripted sequence and clear language ("first onset of familiar pain," "worse/better/same with neck position") are used [33–36]. Again, the pattern—not just the range—is the key signal.
Because the Slump engages the whole chain, it can reveal load sensitivity missed by

supine SLR, particularly in people whose symptoms are posture-dependent (prolonged sitting, flexion bias) or influenced by thoracic/cranial mechanics. It also lends itself to within-session education: patients can feel the difference a neck or ankle position makes, which builds buy-in for sliders and home dosing [11–12,33–36,39–42].

Prone Knee Bend (PKB): anterior pathway bias and reliability

PKB biases the femoral pathway through combined knee flexion and (often) gentle hip extension. Reliability is typically moderate and improves with: (i) a consistent end-point ("first onset of familiar anterior thigh/inguinal discomfort," not maximum stretch), (ii) standard patient positioning (pelvis neutral, monitoring for lumbar substitution), and (iii) systematic use of sensitizers (cervical change; slight hip extension/flexion adjustments) [32,36–37]. PKB is particularly useful when anterior pelvic tilt, iliopsoas tone, or anterior hip capsular stiffness are suspected contributors. As with SLR/Slump, predictable modulation—not the absolute knee flexion angle—is the discriminating feature for SNR [31,32,36–37].

Construct validity

What neurodynamic tests appear to measure
Construct validity for an SNR-oriented interpretation rests on converging lines of evidence:

Mechanical plausibility. Peripheral nerves are mechanical tissues that must glide and tolerate modest strain; perineural stiffness and inflammatory mediators increase mechanosensitivity [13–19,27,28,38,45,46]. In vivo and modelling work indicate that SLR-like movements redistribute strain depending on interface compliance [13–16,27].

Physiological responsiveness. Experimental studies show that neural mechanosensitivity can be altered by sliding/mobilization techniques, with changes in both symptoms and performance on neurodynamic tests [1–3,11–12,39–41,51–53,72–76]. These changes support the idea that tests capture a mechanosensitive state rather than static muscle length.
Clinical coherence. In non-radicular CLBP, patients often display recognizable protective motor patterns (hamstring/gluteal guarding, altered lumbopelvic rhythm) that improve alongside neurodynamic signs when sliders/interface work are introduced [22,24,31,33–41,50]. This triangulation (test behaviour + motor pattern + clinical response) supports the construct.

Discriminative validity and differential diagnosis

Neurodynamic tests are not diagnostic for a specific pathology; they discriminate load sensitivity of neural tissues. In the clinic, useful discrimination emerges when test behaviour is interpreted alongside neurological examination and regional orthopaedic

testing:

-SNR-positive CLBP: familiar pain elicited near end-range SLR/Slump/PKB, predictably modulated by sensitizers; no objective deficit [33–37].

-Radiculopathy/neuropathy: may also show positive neurodynamic tests, but with objective neurological signs (myotomal weakness, dermatomal change, reflexes) [4–6,47–49,77].

-Facet/SIJ-dominant or myofascial pain: tests may provoke discomfort but typically lack consistent modulation and map better to segmental or muscle-tendon signs [4–6,22,24,30].

Thus, discriminative value increases when neurodynamic findings are patterned, reproducible, and set against the absence/presence of neurological deficits and other regional signs [4–6,22,24,30,33–37,47–49,77].

Responsiveness and clinically meaningful change

Responsiveness is reflected in within-session and short-term changes after low-load sliders and interface techniques: small improvements in SLR angle at symptom-onset (not maximum), reduced intensity at the same angle, or normalization of Slump modulation when sensitizers are reversed [1–3,11–12,33–41,51–53,72–76]. While precise minimal detectable change (MDC) values vary by setup, a pragmatic rule is to value consistent, directional change across two or more re-tests (e.g., +10–15° to first familiar symptom over several sessions with reduced irritability the next day). Coupling test changes with functional outcomes (PSFS, ODI) strengthens clinical inference that neurodynamic loading is a relevant driver in the case at hand [93–99].

Methodological issues that influence reliability/validity

1.End-point selection. “First onset of familiar symptom” improves both reliability and clinical meaning versus “maximum tolerable” stretch, which is vulnerable to fear and guarding [33,36].

2.Sequencing/staging. Fixed sequences (e.g., spine → neck → knee → ankle in Slump) with explicit reversal of sensitizers reduce ambiguity and improve inter-rater agreement [33–35].

3.Instrumentation. Simple inclinometers/goniometers and consistent landmarks are sufficient in most clinical settings; the method matters more than the device [33,36].

4.Language and coaching. Standard phrasing (“Tell me when you first feel your usual pain”) avoids drift toward non-familiar sensations (stretch, pressure) [33–36].

5.Irritability grading. Testing and re-testing should respect symptom irritability; overly provocative sequences degrade reliability by changing the system between trials [1–3,33,39–41].

Converging evidence from mechanistic and interventional studies

Mechanistic studies highlight how perineural stiffness and interface adhesion lower thresholds for nociceptor firing and alter proprioceptive feedback [13–19,27,28,38,45,46]. Interventional work shows that gentle sliders can reduce pain and improve test performance, whereas early aggressive tensioning may flare symptoms—especially in high-irritability patients [1–3,11–12,33–41,51–53,72–76]. Together, these observations support a dose-dependent relationship between neurodynamic loading and symptom behaviour that is consistent with the SNR model.

What the tests cannot do (and how to use them wisely)

Neurodynamic tests cannot specify which interface is the limiting factor (e.g., deep gluteal vs. proximal hamstring fascia) or rule out central contributions. They must be interpreted with neurological examination, regional orthopaedic testing, and the patient’s functional narrative. They perform best when they are used to (i) classify a load-sensitive neural phenotype (SNR positive/uncertain/negative), (ii) dose early intervention (sliders first, tensioners later), and (iii) track change alongside function (NPRS/ODI/PSFS). In other words, they are decision tools, not pathognomonic signs [4–6,22–26,33–37,39–41,47–49,55–57,93–99].

Practical documentation and clinical utility

A concise documentation set improves both care and research readiness:

-SLR L/R: angle at first familiar symptom; change with ankle dorsiflexion (+/0/−); change with cervical flexion (+/0/−) [33,36].

-Slump: which stage provokes familiar pain; what happens when sensitizers are reversed (better/worse/same) [33–35].

-PKB: knee flexion angle at symptom onset; effect of hip/cervical adjustments [32,36–37].

-Irritability: high/moderate/low; 24-hour rule outcome [1–3,33,39–41].

-Outcome trio: NPRS, ODI, PSFS; note within-session changes and between-visit trends [93–99].

This minimalist dataset captures the pattern that defines SNR and supplies the variables needed for reliability/feasibility work [33–37,39–41,93–99].

Summary and implications

When neurodynamic tests are performed with standardized scripts, symptom-first end-points, and sensitizer modulation, reliability is acceptable for clinical decision-making and good enough to support research classification in CLBP [33–37]. Construct validity is supported by mechanistic plausibility (glide/strain/interface stiffness) and by clinical responsiveness to sliders/interface techniques [1–3,11–19,27,28,38,39–41,45,46,51–53,72–76]. Discriminative value emerges when tests are interpreted against neurological findings and regional signs, helping to separate SNR-positive cases from other CLBP phenotypes [4–6,22–26,31,33–37,47–49,55–57]. Finally, the tests' greatest strength in an SNR framework is not in assigning a specific lesion but in revealing load sensitivity of the neural system and guiding dose-progression: sliders early, interface work where bottlenecks are suspected, followed by motor control/proprioception and cautious tensioning as irritability falls. Used this way, neurodynamic testing becomes a reliable, valid, and clinically useful anchor for mechanism-aligned care in a meaningful subset of patients with persistent CLBP [1–6,11–19,22–26,31–41,44,45,51–57,60–63,67–71,72–76,77–80,93–100].

1.1 **The Global Burden of CLBP**

Chronic low back pain (CLBP) is a major contributor to global disability and a persistent public health and economic problem. At a population level, CLBP accounts for a substantial proportion of disability-adjusted life years (DALYs) and remains one of the top causes of years lived with disability across regions and income strata [4,5]. In economic terms, direct medical spending (e.g., consultations, imaging, pharmaceuticals, injections, procedures) and indirect costs (e.g., productivity loss, absenteeism, presenteeism, early retirement) are immense, with estimates of combined expenditures surpassing $100 billion annually in the United States alone [4]. The burden is not simply financial; CLBP corrodes quality of life, limits participation in meaningful roles (work, caregiving, community), and amplifies health inequities—disproportionately affecting individuals whose jobs require sustained postures, repetitive lifting, or who have restricted access to timely, evidence-based care [4–6]. As articulated in the 2018 Lancet series, CLBP is not a single disease entity but a heterogeneous condition embedded in biological, psychological, and social contexts; no single intervention can be expected to "solve" CLBP for all patients [5,6]. This landscape demands more precise phenotyping, better alignment of care with

mechanisms, and pragmatic models that clinicians can actually implement [4–6,55–57].

1.1.1 **Prevalence, natural history, and the "chronic–recurrent" reality**

Point prevalence of low back pain is consistently high worldwide, and when one focuses on the chronic subset (symptoms ≥3 months), a large absolute number of people are affected at any given time [4–6]. The risk of CLBP increases with age, but working-age adults carry a major share of the burden; symptoms often begin in early or mid-adulthood and evolve into cycles of partial remission and relapse. Many individuals do not experience a neat transition from "acute" to "recovered"—they occupy a chronic–recurrent state in which pain waxes and wanes with workload, sleep quality, stress, and activity patterns [5,6]. This variability complicates care planning and outcome evaluation. A person may report low pain one week and marked limitation the next, not due to capriciousness but because CLBP is influenced by moment-to-moment load management, fear-avoidance behaviors, and the interaction of peripheral and central mechanisms [5,6,25,26,48–50,60–63,78–80]. Traditional episodic care models (e.g., one-off courses of treatment after a "flare") often fail to provide durable benefit because they do not address the processes that perpetuate sensitivity and protective motor strategies between episodes [5,6,22–26,50].

1.1.2 **DALYs, quality of life, and lived experience**

CLBP's placement near the top of DALY rankings underscores its impact on daily functioning and long-term participation [4,5]. Beyond pain intensity, patients describe functional fragility: ordinary tasks (sitting through a meeting, lifting a child, standing for a shift) can unpredictably provoke symptoms. Quality of life diminishes not only from persistent discomfort but also from activity restriction, sleep disturbance, anxiety about reinjury, and reduced social engagement [48,49,60–63,78,79]. The psychological load—fear of movement, catastrophizing, low self-efficacy—interacts with somatic drivers to shape clinical trajectories [5,6,25,26,100]. For many, CLBP becomes a decision-making context: Should I take that job that requires standing? Can I commit to travel? These choices accumulate and narrow life participation even when pain is "moderate." The societal cost is therefore not just the sum of medical bills and missed work days; it is the opportunity cost of constrained human potential [4–6,55–57].

1.1.3 **Direct and indirect economic costs: where the money goes**

Healthcare systems funnel substantial resources into low back pain care. Direct costs include primary and specialist consultations, diagnostic imaging, medications, physical therapy, manual therapy, interventional procedures, and in some cases surgery. However, the Lancet series highlighted a mismatch between high-cost care (e.g., routine imaging for non-specific pain, procedure-heavy pathways) and patient-important outcomes [5,6,55–57]. Indirect costs often exceed direct spending. Employers and workers bear the brunt of absenteeism (time off for flares, appointments) and presenteeism (working while impaired, with reduced productivity).

In physically demanding occupations, CLBP contributes to early exit from the workforce and increased reliance on disability benefits. From a macroeconomic perspective, the aggregate effect is substantial: even small decrements in day-to-day productivity, when multiplied across millions of workers, translate into billions of dollars of lost output annually [4–6]. These losses are not evenly distributed; sectors with limited ergonomic support, variable shift work, or high psychosocial stressors demonstrate higher sustained costs [4–6,55–57].

1.1.4 **Disparities and social determinants**

CLBP does not occur in a vacuum. Social determinants—education level, income, job control, access to evidence-based services—shape both incidence and persistence. Individuals in precarious employment may delay seeking care, overuse passive treatments, or lack access to continuity with a clinician who can provide reassurance, graded activity, and mechanism-aligned strategies. Communities with limited transportation or fewer rehabilitation providers face structural barriers to best-practice care. Psychosocial stressors (financial strain, caregiving burden) amplify the risk of chronicity by influencing sleep, recovery, and pain processing. The Lancet series emphasizes destigmatization and de-medicalization where appropriate, but de-medicalization must be paired with access to supportive, active care rather than abandonment to self-help in resource-poor settings [5,6,55–57,78,79].

1.1.5 **The guideline–practice gap**

Clinical practice guidelines consistently recommend reassurance, staying active, graded exercise, and judicious use of imaging and pharmaceuticals for non-specific low back pain [5,6,77]. Yet implementation lags. Overimaging persists, especially in the absence of red flags; pharmacologic strategies sometimes drift toward long-term reliance without concurrent active rehabilitation; and referrals may leapfrog conservative care toward procedures that do not outperform high-quality active programs in many non-specific cases [5,6,55–57,77]. The guideline–practice gap persists partly because “non-specific low back pain” is an umbrella that hides meaningful subgroups. When mechanisms are not identified (or are deemed irrelevant), clinicians default to generic programming that may be under-dosed or mis-dosed for key subpopulations. The result is a revolving door of care: temporary symptom improvement without durable functional gains [4–6,55–57].

1.1.6 **Why mechanism-aware phenotyping matters**

The heterogeneity of CLBP argues for phenotyping—grouping patients by clinically relevant mechanisms that inform care choices. Psychosocial risk stratification tools have value, but peripheral contributors can still be pivotal in patients with modest psychosocial risk. A purely joint-centric approach may overlook neural interface problems; a purely nociplastic approach may underappreciate local mechanosensitivity that is modifiable with the right dosing of movement and interface-focused techniques [5,6,17–19,25,26,48,49,60–63]. The case for mechanism-aware phenotyping is pragmatic: if a subgroup responds better to

particular loading strategies, we should identify it and act accordingly. The objective is not to create niche labels but to improve signal-to-noise in everyday decision-making and to reduce therapeutic wandering [4–6,22–26,55–57,77].

1.1.7 **Subclinical neurodynamic restrictions as an overlooked contributor**

Within this context, subclinical neurodynamic restrictions (SNR)—mild limitations in peripheral nerve excursion and/or heightened mechanosensitivity without frank neuropathy—represent a plausible and clinically important contributor to persistent CLBP [7,8,11,12]. The nervous system is a mechanical organ as well as an electrical one; peripheral nerves must glide longitudinally, move transversely, and tolerate modest strain during ordinary movements [1,9,13–16]. If perineural interfaces (epineurium/perineurium, fascia, adjacent muscle) stiffen due to low-grade inflammation, micro-adhesions, or sustained tone, a given movement may produce earlier mechanosensitive firing and familiar symptoms at lower thresholds [11,12,17–19,27,28]. Crucially, SNR may not produce dermatomal pain, objective weakness, or reflex changes—hence "subclinical." Patients present instead with movement-provoked symptoms that are modulated by neurodynamic sensitizers (e.g., in SLR or Slump tests). This pattern is easy to miss if clinicians record only range (degrees) without probing symptom behavior under sensitizers [1–3,7–12,31,33–37].
SNR's relevance to global burden is twofold. First, if a non-trivial slice of persistent CLBP is driven or maintained by neural interface problems, recognizing and treating that slice can yield better outcomes with low-risk strategies (sliders, interface techniques, graded motor control) [1–3,11–12,39–43,51–53,72–76]. Second, identifying SNR earlier may improve efficiency: fewer cycles of trial-and-error, faster achievement of within-session "wins" that increase adherence, and smoother integration with broader biopsychosocial care. In health systems struggling with resource constraints and backlogs, small improvements in pathway efficiency scale into meaningful population-level benefits [4–6,55–57].

1.1.8 **Ageing populations and multimorbidity**

Population aging magnifies the importance of CLBP. Older adults often present with multimorbidity (e.g., osteoarthritis, diabetes, cardiovascular disease) and polypharmacy; CLBP complicates activity maintenance, which in turn compromises cardiometabolic health. At the same time, older adults are not exempt from neural interface issues. Reduced tissue compliance, sarcopenia with compensatory hypertonicity, and prolonged sitting may increase the likelihood of perineural stiffness. Pain beliefs accumulated over years of episodic flares can bias toward guarded movement, perpetuating stiffness and reducing glide. Mechanism-aware, low-risk strategies—beginning with gentle sliders and interface de-loading, paired with confidence-building education—are well-suited to this group, especially when high-intensity loading or prolonged end-range stretching would provoke flares [5,6,22–26,39–41,55–57].

1.1.9 **Work, productivity, and occupational considerations**

From a workforce perspective, CLBP is a participation disorder as much as a pain disorder. Occupations with heavy manual tasks or prolonged static postures (driving, assembly lines, healthcare shifts) face distinct exposure patterns: constrained position times, limited micro-breaks, and psychosocial stressors (time pressure, low job control). These factors increase recurrent symptom risk. Occupational programs that incorporate simple neurodynamic strategies (brief, low-amplitude sliders during micro-breaks; position variability; interface self-release) can be deployed without equipment and with minimal disruption. The promise here is not cure by exercise snack, but dose-consistent self-regulation that reduces day-to-day irritability and flare magnitude. Scaling such micro-interventions across large workforces can yield measurable productivity gains and fewer lost days—even if pain scores remain variable [4–6,22–26,39–41,55–57].

1.1.10 **Healthcare utilization patterns and unintended consequences**

High utilization of imaging for non-specific CLBP rarely changes management and can create iatrogenic risk: incidental findings may escalate fear, medicalize benign age-related changes, and prompt cascades toward procedures that offer limited additional benefit in many cases [5,6,55–57]. Pharmacologic reliance—particularly when used as a stand-alone strategy—can drift from acute symptom relief into chronic use without functional progress. Meanwhile, access to skilled rehabilitation that emphasizes active, mechanism-aligned care (education, graded activity, targeted neurodynamic dosing when indicated) is uneven. These utilization patterns consume resources while failing to address key drivers for a meaningful subgroup of patients [4–6,55–57,77].

1.1.11 **Psychosocial interplay and central mechanisms**

The biopsychosocial framework is essential: beliefs, fear-avoidance, catastrophizing, and low expectations can amplify pain and drive disability [5,6,25,26,95,96,100]. However, psychosocial factors do not preclude peripheral drivers. In fact, persistent bottom-up input from irritated neural interfaces can maintain central sensitization and promote protective motor strategies [22–25,60–63,78,79]. Conversely, reducing peripheral mechanosensitivity—by restoring glide and decreasing interface stiffness—can lower the "volume" of nociceptive input and make top-down strategies (graded exposure, cognitive reframing) more effective. In practice, an integrated approach is required: deliver targeted peripheral interventions alongside brief, potent education and simple self-efficacy tools, so that patients witness within-session change (e.g., Slump symptoms improving when a sensitizer is released) and can logically connect that change to home dosing [5,6,25,26,39–42,48,49].

1.1.12 **Measurement issues and what "burden" hides**

Public health metrics capture prevalence, DALYs, and cost, but they underrepresent day-to-day volatility—the spikes and dips that shape lived experience. Monthly or yearly averages smooth over the decision friction patients feel: whether to accept a social invitation, to sit through a training session, to take stairs, to lift a suitcase.

Moreover, burden estimates typically consider “pain” and “function” but less often assess confidence, predictability, and self-management capacity. Mechanism-aware care, including SNR-aligned strategies when indicated, aims not merely to change scores but to increase control over symptoms—transforming CLBP from an unpredictable adversary into a manageable condition [4–6,25,26,93–99].

1.1.13 **System-level implications: population health and value**

At the system level, improving CLBP outcomes requires value-oriented care: aligning resources with interventions that deliver meaningful function at acceptable cost. Because CLBP is so common, even modest effect sizes—if delivered at scale—produce substantial population benefit. Mechanism-aligned pathways help by (i) avoiding low-value care (routine imaging/procedures for non-specific presentations), (ii) creating early wins that reduce revisit rates, and (iii) focusing clinician time on teachable, self-manageable strategies. SNR recognition fits this value frame: it leads to low-risk, low-cost first steps (sliders, interface work, graded motor control) and clear stop rules (24-hour response, flare management), with escalation only when warranted [5,6,7,8,11,12,39–43,51–53,72–76].

1.1.14 **Education and the public narrative**

Public narratives around back pain often emphasize fragility (“my back is damaged”) and structural imagery (“slipped disc”), which increase fear and avoidance. An alternative narrative—aligned with guideline recommendations—is that the back is robust and adaptable, and that nerves are living cables designed to move. Education that uses simple analogies (glide vs. yank; sharing load along the chain) and embodies change in-session (symptom modulation with sensitizers) helps patients recalibrate threat and engage in graded activity. This message does not minimize suffering; it reframes it within a model of modifiable sensitivity rather than inevitable deterioration [5,6,25,26,48,49].

1.1.15 **Why this matters for clinicians**

For clinicians, the global burden backdrop is not abstract—it appears as crowded schedules, short visits, and a pressure to provide relief quickly. Mechanism-aware phenotyping, including SNR identification where appropriate, can accelerate clarity. A brief battery—SLR, Slump, PKB with sensitizers—adds minutes, not hours, and yields actionable information: Is there predictable symptom modulation? What is the irritability (to dose safely)? Which interfaces are likely bottlenecks? From there, clinicians can provide a targeted micro-dose plan (e.g., 10–15 gentle sliders twice daily, interface self-release, one motor-control drill) with explicit 24-hour rules (reduce amplitude/frequency if soreness >3/10 or persists >24 h). This approach respects real-world constraints and gives patients agency [1–3,22–26,31,33–37,39–43,77].

1.1.16 **Why this matters for researchers and policy makers**

For researchers, the burden argues for studies that are executable in routine practice: reliability of classification (e.g., SNR scripts with symptom-modulation endpoints), feasibility of dosing (adherence, flare rates), and comparative effectiveness (sliders + usual care vs. usual care). Small, well-designed trials that stratify by phenotype can outstrip larger, heterogeneous studies in actionability [3,33–37,39–41,51–53,72–76]. For policy makers, the question is not whether CLBP is costly—it is—but how to shift resources toward scalable interventions that produce better participation and function. Embedding mechanism-aware tools in primary care and rehabilitation, supported by brief training and simple documentation templates, is a plausible lever [4–6,55–57,77].

1.1.17 **Bringing it together: burden, heterogeneity, and a path forward**

CLBP's global burden—high prevalence, heavy DALYs, vast direct and indirect costs—arises from its heterogeneity and from care pathways that are not consistently aligned with mechanisms [4–6]. The Lancet series rightly concludes that no single therapy suits all [5,6,55–57]. The practical corollary is that we must sharpen how we identify and act on relevant contributors in each case. Subclinical neurodynamic restrictions represent one such contributor: common enough to matter, subtle enough to be overlooked, and tractable enough to treat with low-risk, low-cost strategies [7,8,11,12,31]. Recognizing SNR does not deny the role of psychosocial or central processes; rather, it integrates them by reducing peripheral drive and enabling top-down gains. At scale, mechanism-aware care can reduce futile resource use, increase patient control over symptoms, and bend the curve of the global burden—one precise, teachable intervention at a time [4–8,11,12,22–26,31,33–41,55–57,77].

1.1.18 **Summary statement**

CLBP is common, costly, and complex. Its burden accrues through lost function, constrained participation, and sustained economic drag. The Lancet guidance emphasizes individualized, multidimensional care and warns against low-value, procedure-heavy approaches in non-specific presentations [5,6,55–57]. To operationalize that vision, clinicians need simple, reliable ways to detect treatable mechanisms. SNR is one such mechanism: identifiable at the bedside by symptom modulation with neurodynamic sensitizers, and addressable through graded nerve sliders, interface techniques, and motor control adjustments that respect irritability and promote self-efficacy [7,8,11,12,33–41]. By incorporating SNR into routine assessment—especially in patients who remain symptomatic despite benign imaging and normal neurological examination [7,26]—clinicians can bridge the biomechanical and neurophysiological domains [1–3,8,13–19,22–26] improve the precision of care, and contribute to reducing the immense, ongoing global burden of chronic low back pain [4–6,55–57].

1.1.19 **Implementation science: making mechanism-aware care routine**

Reducing the global burden of CLBP is not only a matter of what to do but how to deliver it reliably in real clinics. Three implementation levers are feasible at scale: (i)

micro-skills, (ii) micro-documentation, and (iii) micro-scripts.
-Micro-skills. Brief training on scripted SLR/Slump/PKB with symptom-first endpoints and explicit sensitizer sequencing (apply → observe → reverse) equips clinicians to detect predictable modulation in minutes [33–37]. Short refreshers emphasizing irritability grading and the "24-hour rule" align dosing with tissue tolerance [33,39–41].

-Micro-documentation. A compact template—NPRS/ODI/PSFS plus within-test change (e.g., SLR angle at first familiar symptom; better/worse/same with ankle/cervical change)—anchors clinical reasoning and creates consistent data for audit and QI [33,36,93–99].

-Micro-scripts. Patient-facing language ("nerves are living cables; we'll move the stress around with sliders") and simple self-calibration rules (reduce amplitude/frequency if soreness >3/10 or lingers >24 h) improve adherence, reduce flare-related drop-out, and foster self-efficacy [25,26,39–42].
By standardising these small behaviours, services can raise the signal-to-noise ratio in day-to-day care, thereby improving outcomes without large new investments [4–6,33–37,39–42,55–57,77].

1.1.20 **Outcome tracking and value: what to measure (and why)**

Global burden metrics obscure the clinical reality that modest, directionally consistent improvements—achieved early and sustained—translate into large population gains. Routine tracking should pair patient-important measures (NPRS, ODI, PSFS) with mechanism-linked markers (within-test symptom modulation; SLR/Slump/PKB change at symptom onset) [33–37,93–99]. This pairing does two things:

1. It confirms mechanism: if sliders/interface techniques produce better tolerance and cleaner modulation within sessions and across weeks, the working SNR label is supported [1–3,11–12,33–41,51–53,72–76].

2. It demonstrates value: incremental PSFS gains (e.g., tolerating a 60-minute meeting, walking 20 minutes without a flare) reduce presenteeism/absenteeism in ways that aggregate across large workforces [4–6,55–57].

3.Where available, simple imaging biomarkers—such as ultrasound-based excursion measures—can complement clinical tracking in research or advanced practice settings, linking peripheral change to functional outcomes and sharpening dose-response understanding [41,46]. In routine clinics, however, the most scalable metric remains predictable symptom modulation documented consistently [33–37,39–41].

1.1.21 Equity, access, and scalable delivery

Because CLBP disproportionately affects people in physically demanding jobs and communities with fewer resources, mechanism-aware care must be portable and low-friction. SNR-aligned strategies meet this brief:

-Low-cost tools: Sliders require no equipment; interface self-release uses commonplace items (strap/towel/ball); motor-control drills need minimal space [39–41].

-Time-efficient dosing: “Little and often” (brief sets, multiple times daily) fits around shift work and caregiving demands and is less likely to provoke flares at high irritability [1–3,33–37,39–41].

-Blended delivery: Short in-person visits to teach scripts plus remote follow-ups (phone/video) to adjust amplitude/frequency based on 24-hour responses can extend reach without diluting fidelity.

-Co-design with patients: Incorporating the patient’s functional priorities (PSFS) into progression criteria ensures relevance, improves adherence, and makes success visible early [93–99].

Equity also means avoiding low-value cascades that siphon resources—routine imaging for non-specific presentations, premature escalation to procedures—while underfunding active, teachable care. Redirecting effort toward classification → early wins → graded progression aligns with the Lancet’s call for person-centred, multidimensional, and de-medicalised pathways where appropriate [5,6,55–57,77].

Add-on summary. The global burden of CLBP is driven as much by heterogeneity and pathway mismatch as by raw prevalence. A small set of repeatable clinical behaviours—scripted neurodynamic testing with symptom modulation, irritability-based dosing of sliders and interface work, and tight outcome tracking—can raise care precision for the SNR-positive subgroup. Scaled across services, these micro-interventions promise better function at lower cost, especially for patients who remain symptomatic despite benign imaging and normal neurological examination [7,26,33–41].

1.2 **The Nervous System as a Mechanical Structure**

The peripheral nervous system’s mechanical properties are often underappreciated in clinical practice. Nerves must slide longitudinally, move transversely, and elongate to accommodate joint motion and muscle activity [13,14]. For example, during a straight leg raise (SLR), the sciatic nerve slides distally and tolerates modest strain to prevent excessive tensile loading [1,13–16]. These movements rely on healthy interactions with surrounding tissues—muscle, fascia, retinacula, and the layered connective tissue

sheaths of nerves themselves (epineurium, perineurium, endoneurium) [15,16]. Shacklock's clinical neurodynamics reframes nerves as living, mobile, mechanosensitive structures embedded in a kinetic chain; unimpeded movement and load sharing along that chain are prerequisites for normal function [1]. Even subclinical restrictions—subtle losses of excursion or increased interface stiffness—may alter afferent signaling, provoke protective responses, and contribute to pain and dysfunction [11,12,17,18]. This perspective challenges the traditional view of the nervous system as a purely electrical structure and highlights its role as a mechanical organ in CLBP [1–3,11–19].

1.2.1 **Hierarchical nerve architecture and its mechanical implications**

Peripheral nerves are built for both protection and mobility [13–16]. At the endoneurial level, individual axons are suspended in a fluid-rich matrix that buffers micro-strains and facilitates axoplasmic flow [15,16]. The perineurium forms multilamellar sheaths around fascicles, providing tensile stiffness, a diffusion barrier, and crucially—shape stability under load [15,16]. The epineurium binds fascicles, vessels, and fat into a grossly mobile unit with relatively low stiffness, allowing longitudinal sliding and transverse excursion against adjacent tissues [15,16]. This hierarchical design means that in healthy motion, most displacement is accommodated by glide—not by large intraneural elongation [13–16]. When glide is restricted (e.g., due to adhesions, interface fibrosis, or excessive surrounding muscle tone), a greater portion of joint motion is absorbed as intraneural strain, magnifying the risk of mechanosensitive firing and ischemic compromise [13–16,27].
The vascular supply of nerves (vasa nervorum) is also mechanically vulnerable [15–18]. Longitudinal strain narrows endoneurial microvessels; compression at interfaces increases venous congestion first, then arterial inflow impairment—a sequence that can enhance ischemia-related nociception in mechanosensitive afferents [15–18]. Thus, even small mechanical mismatches—say, a few degrees of extra hip flexion paired with a stiff posterior thigh interface—can have disproportionate sensory consequences [13–18,27,28].

1.2.2 **Glide, strain, and transverse excursion in common movements**

Longitudinal sliding. During SLR (hip flexion + knee extension), the sciatic pathway must displace distally in the thigh and proximally in the pelvis, with the magnitude depending on limb proportions and regional compliance [13–16]. In the Slump sequence, staged spinal flexion and cervical flexion add posterior dural loading, changing the distribution of strain and the requirements for glide across the chain [33–36]. In the anterior pathway, prone knee bend (PKB) biases the femoral nerve; combined knee flexion and gentle hip extension challenge proximal interfaces beneath the inguinal ligament and within the femoral triangle [32,37].
Transverse excursion. Nerves also move side-to-side to avoid compression from adjacent structures (e.g., fascial edges, osteofibrous tunnels, muscle borders) [13–16,20,21]. The superior cluneal nerves, crossing the posterior iliac crest, exemplify a small-calibre branch traversing an osteofibrous tunnel where transverse shear and focal compression can generate high local stress relative to nerve size [21]. In the deep gluteal region, the sciatic nerve must navigate between the piriformis, short

external rotators, and the posterior border of the greater trochanter; small changes in muscle tone or fascial stiffness can materially affect local excursion [20,21,31]. Elongation/strain. Healthy nerves tolerate modest elongation, typically in the range of a few percent during physiologic movement; perineurial lamellae straighten and then stiffen, reflecting a nonlinear stress–strain curve [15,16]. The system is designed so that glide absorbs most displacement initially; only later in range, as slack is taken up, does intraneural strain rise steeply [13–16]. If glide is compromised earlier, nerves encounter the steep portion of the curve sooner, which is clinically perceived as early symptom onset during neurodynamic tests—often before the musculoskeletal tissues (e.g., hamstrings) reach their own mechanical limits [13–16,27,28].

1.2.3 **Viscoelasticity, hysteresis, and time dependence**

Nerves and their interfaces are viscoelastic [13–16,19]. Repeated cycles of loading display hysteresis (energy loss) and time-dependent stress relaxation [13–16]. In practical terms, the same end-range may feel different on the second pass than the first because tissue viscosity changes as temperature rises and fluid redistributes [13–16,19]. Gentle repetitions at low amplitude (as in sliders) can therefore produce immediate, small improvements in tolerance and symptom behaviour without invoking high tensile loads [1–3,39,40]. Conversely, sustained end-range positions (e.g., prolonged sitting with posterior chain tension) can increase intraneural stress through creep in surrounding tissues, even in the absence of active movement—explaining why “static” postures are often provocative in CLBP with neurodynamic features [17–19,31,39].

1.2.4 **Mechanosensitive afferents and neuroinflammation**

Mechanotransduction within neural tissues involves mechanosensitive ion channels and receptors on nociceptors and low-threshold afferents [17,18]. When perineural stiffness increases (e.g., due to low-grade inflammation, fibrosis), the same external displacement can produce greater local strain, which increases firing probability in mechanosensitive C-fibres and A-delta fibres [17,18]. Basic and translational studies highlight how inflammatory mediators sensitize afferents and alter perineural viscoelastic properties, decreasing glide and raising mechanical gain [17–19,27,28,38]. In CLBP contexts where imaging is benign and neurological examination is normal, these microenvironmental changes plausibly explain movement-provoked familiar pain during neurodynamic testing—especially when symptoms modulate predictably with sensitizers (e.g., ankle or cervical position) [11,12,17–19,33–36].

1.2.5 **Interfaces: the “sleeves” nerves move through**

Nerves seldom fail in isolation; the usual problem resides at the interface—the sleeve of adjacent tissues through which the nerve must move [15–19]. In the posterior chain, relevant interfaces include proximal hamstrings, deep gluteal muscles, and the posterior thigh fascia [20,21,31]. In the anterior pathway, the iliopsoas, inguinal ligament, and anterior hip capsule are key [20,21,31,32]. Over time, repetitive micro-loading, low-grade inflammation, and uneven motor strategies can increase the

stiffness of these sleeves, reducing the “give” available for glide [17–19,31]. Clinically, palpation often reveals tenderness along neural course segments (e.g., at the posterior iliac crest for cluneal branches, along the sciatic course in the gluteal region) that changes with positioning [20,21,31,39,40]. Manual therapy targeting these interfaces—myofascial release, soft-tissue mobilization, gentle joint techniques—can acutely improve relative movement, making subsequent sliders more tolerable and effective [1–3,39,40].

1.2.6 **Regional examples relevant to CLBP**

Sciatic pathway. In CLBP, many patients demonstrate posterior chain mechanosensitivity that eases when neural load is shared differently (e.g., SLR improves with ankle plantarflexion or cervical extension) [31,33–36]. Deep gluteal hypertonicity, proximal hamstring stiffness, or fascial thickening can constrain sciatic glide, producing earlier symptom onset at end-range [20,21,31]. The patient’s perception (“a deep, familiar ache”) and the modulation under sensitizers differentiate this from simple hamstring stretch [31,33–36,39,40].
Cluneal nerves. Superior cluneal branches cross osteofibrous tunnels over the posterior iliac crest and are susceptible to focal compression and shear during trunk flexion/extension or prolonged standing [21]. Local tenderness over the crest; reproduction of familiar buttock pain with transverse gliding; and change during Slump with neck adjustments support a cluneal interface contribution [21,39,40]. Targeted interface techniques—gentle transverse mobilization and progressive desensitization—often produce rapid within-session changes [21,39,40].
Femoral pathway. In patients with anterior pelvic tilt and iliopsoas tone, PKB may reproduce familiar anterior thigh or proximal inguinal discomfort that modifies with hip or neck position—again indicating a neurodynamic component rather than pure quadriceps stretch [32,37]. Early low-amplitude femoral sliders coupled with iliopsoas/inguinal interface work can restore tolerance before advancing hip extension loading [1–3,32,37,39].

1.2.7 **Neurodynamic testing as a mechanical probe**

SLR, Slump, and PKB are not mere flexibility tests; they are mechanical probes that selectively redistribute load along the neural chain [1–3,33–37]. By sequencing sensitizers, the clinician can observe whether familiar symptoms increase (with added neural load) and decrease (when load is released), thereby inferring whether the system’s mechanical behaviour is a relevant driver of the clinical picture [33–37]. Two testing principles flow directly from nerve mechanics:

1.Symptom-first end-point. Stopping at the first onset of familiar symptom yields a load threshold that is less confounded by stretch tolerance or fear; it better reflects where the system transitions from glide-dominant to strain-dominant behaviour [33,36].

2.Apply → observe → reverse. Modulation with sensitizers (ankle/cervical/hip) is the mechanistic signal. Reversibility (easing when sensitizer is released) shows that the response is load-dependent, not simply a by-product of effort or guarding [33–37].

1.2.8 **From mechanics to motor behaviour: protective tone and strategy bias**

When nerves or interfaces become mechanically sensitive, the body often responds with protective motor strategies: increased baseline tone (e.g., hamstrings, gluteals), co-contraction around the lumbopelvic region, and movement substitutions that offload provocative ranges [22–25,30,31,50]. For instance, a patient may reduce hip flexion and recruit more lumbar flexion during bending, shorten stride during gait, or avoid prolonged sitting [22–25,30,31]. These strategies initially shield sensitive tissues but eventually perpetuate stiffness and reduce available glide, tightening the mechanical system further [22–25,30,31,50]. Clinically, this is why early, low-load sliders (not tensioners) and interface de-loading are favoured: they reduce mechanosensitivity first, decrease the need for protective tone, and set the stage for motor recalibration (neutral spine drills, hip hinge, gait mechanics) [1–3,39–42].

1.2.9 **Why sliders first? Dosing aligned to viscoelastic reality**

In a mechanically sensitive system, high tensile loads early (e.g., aggressive hamstring stretching or premature nerve tensioners) may overshoot tissue tolerance, producing delayed flares and reinforcing avoidance [1–3,39–41]. Sliders, by contrast, bias relative movement with low tensile load, leveraging viscoelastic properties to restore glide without provoking sustained intraneural strain [1–3,39,40]. Typical early dosing—small-amplitude cycles, 10–15 reps, 1–2 sets, two or three times daily—fits the biology and can produce within-session changes (e.g., easier SLR at symptom onset; reduced intensity at the same angle) [1–3,33–36,39–41]. The 24-hour rule provides safety governance: if soreness >3/10 or lasting >24 h occurs, reduce amplitude/frequency and re-progress gradually [39–41].

1.2.10 **Measurement and documentation: making mechanics visible**

Because nerve mechanics are not seen on routine imaging, clinicians must rely on patterned behaviour during examination and retest [1–3,11–13]. A concise documentation set brings these mechanics into view:

-SLR L/R: angle at first familiar symptom; change with ankle dorsiflexion (+/0/−); change with cervical flexion (+/0/−) [33,36].

-Slump: symptom behaviour with staged sequence; change when sensitizers are reversed (better/worse/same) [33–35].

-PKB: knee flexion angle at symptom onset; effect of hip/cervical adjustments [32,37].

-Interface findings: palpatory tenderness along neural course (gluteal region, posterior iliac crest, femoral triangle) [21,31,39,40].

-Irritability: high/moderate/low with 24-h response to prior session [33,39–41].

-Outcome trio: NPRS, ODI, PSFS to capture functional impact [93–99].

These data points reflect the mechanical state of the neural system (how load is tolerated and shared), and they allow clinicians to adjust dose responsively [33–37,39–42].

1.2.11 **Central–peripheral reciprocity: mechanics still matter**

Central sensitization frameworks explain how pain can persist and amplify even after peripheral injuries subside [5,6,25,26]. Yet peripheral mechanics still matter, because ongoing bottom-up input from mechanically sensitive interfaces can maintain central gain [22–25,60–63,78,79]. Conversely, reducing peripheral drive—by restoring glide, lowering interface stiffness, and easing load thresholds—can make top-down strategies more effective (education, graded exposure, expectation recalibration) [25,26,39–42]. The reciprocity is practical: demonstrate within-session modulation (e.g., Slump eases with neck extension), link that change to home sliders, and integrate graded motor control. This stepwise approach respects both the biomechanics and the neurophysiology of CLBP [1–3,22–26,30,33–42].

1.2.12 Common clinical pitfalls (mechanics edition)

-Range-only thinking. Recording SLR angle without sensitizers misses the load-dependent behaviour that defines neurodynamics [33–36].

-Tensioners too soon. High tensile dosing in a high-irritability system triggers flares; start with sliders and progress only after clean 24-h responses [1–3,39–41].

-Ignoring interfaces. Treating “nerve pain” without addressing gluteal/hamstring or iliopsoas/inguinal interfaces leaves a bottleneck in place [20,21,31,39,40].

-End-range bias. Long passive stretches at end-range may increase intraneural stress without improving relative movement; prioritise movement sharing along the chain instead [13–16,19,39–41].

1.2.12 **Practical translation: tying mechanics to the home program**

Mechanics become real to patients when they can feel them. Two short scripts operationalize the concept:

-“Move the stress around.” In a seated Slump, extend the knee to symptom onset, then gently extend the neck or plantarflex the ankle to ease. Alternate these positions for 30–45 seconds. This is a slider—we are sharing load so the nerve glides without a big stretch [33–36].

-“Traffic light dosing.” Green: mild, eases within minutes; Yellow: tolerable, fades within hours; Red: lingers >24 h—reduce amplitude/frequency next time. This keeps dosing aligned with viscoelastic tolerance and prevents reactive guarding [39–41].

As tolerance improves, add motor control (hip hinge, pelvic tilts), then proprioception (short balance tasks), and eventually task-specific loading—each introduced as the mechanical system becomes less reactive [30,37,39–42].

1.2.13 **Why this mechanical lens is essential in CLBP**

In non-radicular CLBP with normal imaging and neurological examination, the temptation is to default to generic strengthening or to attribute persistence entirely to psychosocial factors [5,6,25,26]. The mechanical lens provided by clinical neurodynamics fills a critical gap: it explains why specific combinations of movement (hip flexion + knee extension; spinal/cervical flexion) provoke familiar symptoms and why those symptoms modulate with small positional changes [1–3,13–19,33–37]. It provides a biologically plausible target—restore glide and reduce interface stiffness—using low-risk tools (sliders, soft-tissue/interface techniques, graded control) [1–3,11–12,39–41]. In this sense, the nervous system’s mechanics are not a niche curiosity; they are a first-order clinical consideration for a substantial subset of persistent CLBP [7,8,11,12,22–26,30,31].

1.2.14 **Summary**

The peripheral nervous system is a mechanical as well as an electrical organ [1,13–16]. Its layered architecture distributes load across glide, transverse excursion, and modest strain; perineural and interface tissues govern how movement is accommodated [13–19,21]. When glide is reduced or interfaces stiffen, the system transitions to strain-dominant behaviour earlier in range, elevating mechanosensitive afferent firing and provoking protective motor patterns [17–19,22–25,30,31]. Neurodynamic testing (SLR, Slump, PKB) serves as a mechanical assay, revealing whether familiar symptoms are load-modulated by sensitizers [1–3,33–37]. Clinically, this legitimizes an early emphasis on nerve sliders and interface de-loading, followed by motor control and proprioceptive progression as irritability falls—an approach that integrates seamlessly with biopsychosocial care [1–3,5,6,22–26,30,33–42]. Within the broader CLBP landscape, recognizing the nervous system as a mechanical structure transforms vague “non-specific” pain into a tractable problem with clear assessment signals and actionable, low-risk interventions [1–3,7,8,11–19,22–26,30,31,33–42,46].

1.2.15 **Quantifying excursion and strain: why numbers help but patterns rule**

Quantitative descriptors (e.g., millimetres of excursion; percent elongation at set joint angles) improve our conceptual handle on neural mechanics, but clinical decisions hinge on patterns, not single cut-points [13–16,46]. In vivo and cadaveric work show sciatic excursion on the order of millimetres during hip flexion and knee extension,

with modest elongation percentages well below tissue failure [13–16,46]. However, the distribution of displacement among the hip, knee, lumbosacral dura, and interfaces varies by anthropometrics, habitual posture, and local stiffness [13–16,27,28]. Two people can show similar SLR angles yet differ in whether symptoms modulate with ankle or cervical changes—only the latter pattern implicates a load-sensitive neural system relevant to SNR [33–36]. Thus, angles and distances provide context, whereas sensitizer-driven modulation provides signal for decision-making [33–37].

1.2.16 **Ultrasound and imaging as adjuncts (when available)**

Musculoskeletal ultrasound can visualize nerve position and displacement (e.g., sciatic sliding in the posterior thigh), adding objective anchors to clinical inferences [41,46]. Early work in the lumbar nerve root and sciatic pathways suggests measurable changes in excursion with limb movement and, in some cases, after targeted mobilization programs [39–41,46]. These tools are adjunctive, not required: routine practice can proceed on clinical pattern recognition (apply → observe → reverse) and functional outcomes [33–37,39–42]. In research or advanced clinics, ultrasound can refine dosing (e.g., verifying that sliders increase excursion without provoking excessive tensile load) and help correlate mechanical change with improvement in NPRS/ODI/PSFS—strengthening the construct validity of SNR-aligned care [39–41,46,93–99].

1.2.17 **Dosing progression: from micro-sliders to task integration**

A pragmatic, mechanics-aligned progression follows irritability and 24-hour responses [1–3,33–37,39–41]:

1.Stabilization phase (high irritability):

2.Micro-sliders (e.g., SLR-based: small hip flexion arcs paired with gentle knee flexion/extension; Slump-based: small knee extension with neck extension release), 10–12 reps, 1–2 sets, 2–3×/day [33–36,39–41].

3.Interface de-loading (gluteal/hamstring or iliopsoas/inguinal soft-tissue work), brief bouts [20,21,31,39,40].

4.Goal: reduce mechanosensitivity and protective tone without next-day flare [1–3,39–41].

Capacity phase (moderate irritability):

1.Standard sliders with slightly larger arcs; begin graded tension dosing only if 24-h responses are consistently green [1–3,39–41].

2.Introduce motor control (pelvic tilts, hip hinge), low volume, with emphasis on smooth motion sharing along the chain [22–26,30,41,42].

Integration phase (low irritability):

1.Occasional tensioners (short sets) to expand tolerance to end-range loads [1–3,39–41].

2.Task-specific integration: gait cadence/stride work, sit-to-stand mechanics, lifting practice [30,37,39–42].

3.Proprioception: brief balance tasks to consolidate sensorimotor recalibration [30,37,39–42].

Across phases, the rule of reversibility applies: if a newly added element creates >24 h escalation, revert to the last clean level, shorten exposure, or reduce arc amplitude [39–41].

1.2.18 **Special populations and contextual nuances**

Sedentary workers. Prolonged sitting biases posterior chain tension and increases creep in interfaces, raising intraneural stress at unchanged joint angles [17–19,31]. Prescribing micro-bouts of sliders (30–45 seconds) every 60–90 minutes, plus simple posture variability (stagger stance, short walk) provides repeated decompression cycles that align with viscoelastic recovery [19,33–36,39].
Manual laborers and athletes. Their neural systems must tolerate rapid load transfers and end-range demands. Once irritability is low, integrate velocity-graded movement (e.g., step-downs, hinge drills with tempo) so the neural chain rehearses quick glide under safe loads—preparing for return to duty or sport [30,37,39–42].
Older adults. Reduced tissue water content and fascial stiffness may narrow the margin between glide-dominant and strain-dominant zones. Progress slower, use smaller arcs, and favour frequency over amplitude to build tolerance without provoking delayed soreness [5,6,39–41].
Co-existing nociplastic features. When sleep disruption, widespread tenderness, and poor load–symptom coupling dominate, keep neurodynamic work gentle and use it to create predictable, low-threat wins, while prioritizing education, pacing, and graded exposure [5,6,25,26,33–36].

1.2.19 **Ergonomics and movement ecology: mechanics beyond the clinic**

The nerve–interface system experiences thousands of micro-exposures daily [17–19,31]. Small, repeatable improvements in “movement ecology” accumulate:

-Sitting: knees slightly below hips reduces posterior chain load; intermittent plantarflexion during seated tasks can micro-release the system (the same logic as Slump reversal) [33–36,39].

-Gait: encourage stride symmetry and hip extension within comfort; short cadence cues can smooth load transfer along the chain [30,37,39–42].

-Lifting/stooping: teach hip hinge with shared motion; early sets emphasize smoothness over load to avoid abrupt transitions to strain-dominant zones [30,39–42].

These tweaks are not cure-alls; they are dose shapers that keep cumulative mechanosensitivity down so slider work can consolidate gains [1–3,19,33–37,39–42].

1.2.20 **Reconciling mechanics with the biopsychosocial model**

A mechanical lens does not negate psychosocial care; it enables it [5,6,25,26]. Patients who feel within-session change (e.g., pain eases when the neck is extended in Slump) often reinterpret their condition from "damaged" to "sensitive but modifiable" [25,26,39–42]. This reframing lowers perceived threat and increases willingness to move—precisely the state in which graded exposure, sleep strategies, and activity pacing have the best chance to stick [5,6,25,26,48,49]. The sequence is strategic: show a mechanical win → name it ("your nerves prefer glide to yank") → practice it with home sliders → expand to function [1–3,33–37,39–42].

1.2.21 **Research implications: measurement, mechanisms, moderation**

Three research lanes flow directly from the mechanics outlined here:

1.Reliability of classification. Scripted SLR/Slump/PKB with symptom-first endpoints and explicit sensitizer reversal; report ICC, SEM, and MDC for SNR classification [33–37].

2.Mechanistic change. Link clinical improvement to excursion metrics (ultrasound where available) and to reduced protective tone/motor substitution (simple gait/hinge measures) [30,37,39–41,46].

3.Treatment moderation. Test whether SNR-positive patients gain more from sliders + interface care than SNR-negative peers within usual multimodal rehab (NPRS/ODI/PSFS at 2–6 weeks) [3,39–41,51–53,72–76].

These steps advance from plausibility to testable models that honour the mechanical reality of nerves while fitting real clinics [1–3,13–19,33–42,46].

1.2.22 **Closing synthesis for 1.2**

Peripheral nerves are built to move [1,13–16]. When interfaces stiffen or glide is curtailed, the system enters strain-dominant behaviour earlier, recruiting mechanosensitive afferents and eliciting protective strategies [17–19,22–25,30,31]. Neurodynamic testing operates as a targeted stress test that reveals this shift through predictable modulation under sensitizers [33–37]. Early emphasis on sliders and

interface de-loading, dosed by irritability and governed by the 24-hour rule, respects tissue viscoelasticity and clears the path for motor and functional progression [1–3,33–37,39–42]. Recognizing this mechanical dimension turns a nebulous "non-specific" presentation into a tractable problem with clear assessment signals and low-risk, mechanism-aligned interventions [1–3,7,8,11–19,21,22–26,30,31,33–42,46].

1.3 Rationale for Studying Subclinical Restrictions

Subclinical neurodynamic restrictions (SNR)—mild impairments in neural excursion and/or heightened mechanosensitivity without frank neuropathy—are increasingly recognized in clinics that manage chronic low back pain (CLBP) with normal imaging and normal neurological examination [6,26]. These cases present a recurring puzzle: persistent, movement-provoked pain; regionally appropriate muscle hypertonicity; and functionally relevant limitations that do not map neatly onto discogenic, facet, sacroiliac, or overt neuropathic explanations [6,26]. A mechanobiological lens that treats nerves as moving, load-sharing tissues—rather than passive conductors—offers a coherent account of how subtle changes in perineural interfaces and neural mechanics can perpetuate symptoms, bias motor strategies, and sustain disability [1–3,7–9,11,12,13–18]. This section articulates the clinical and scientific rationale for focusing on SNR in CLBP: the practice gap, mechanistic plausibility, phenotyping value, operational testability, and implications for care and research [1–3,5–9,11–19,21,22,24–28,30,31,33–42,46].

1.3.1 The practice gap: persistent symptoms despite "reassuring" tests

Large numbers of people live with CLBP despite benign imaging and normal neurological screens; guideline-concordant advice (stay active, graded exercise, judicious pharmacology) helps many but leaves a nontrivial subgroup with refractory, movement-linked pain [5,6,26]. These patients frequently report that specific combined actions—hip flexion with knee extension, prolonged sitting in slumped postures, quick stand-to-sit transitions—evoke a deep, familiar ache that does not radiate dermatomally and is not accompanied by weakness or reflex change [5,6,26]. When examined with neurodynamic sensitizers (SLR, Slump, PKB), their symptoms increase when neural load is raised (e.g., ankle dorsiflexion, cervical flexion) and ease when load is released (plantarflexion, cervical extension), a pattern that is difficult to explain by hamstring length or joint restriction alone [33–37]. Recognizing and naming this pattern (SNR) gives clinicians a concrete target that aligns with the patient's lived experience [5,6,26,33–37].

1.3.2 Mechanistic plausibility: nerves as mechanical organs

The peripheral nervous system must glide, undergo modest strain, and translate transversely within compliant interfaces to accommodate ordinary movement [13–16]. If perineural sleeves (epineurium/perineurium, fascia, adjacent muscles) become stiffer through low-grade inflammation, micro-adhesions, or sustained tone, a given joint excursion shifts from glide-dominant to strain-dominant earlier in range,

recruiting mechanosensitive afferents at lower thresholds [15–19,27,28]. This yields predictable load–symptom coupling during staged testing and everyday tasks (e.g., SLR/Slump, sitting, bend-and-reach), yet leaves neurological examination normal—precisely the "subclinical" profile we see in practice [11,12,33–37].

1.3.3 Bridging the "mechanical vs. neural" dichotomy

Historically, CLBP has swung between structural-mechanical and central/nociplastic explanatory poles [5,6,25,26]. SNR integrates these: a peripheral mechanical bottleneck (reduced excursion, heightened mechanosensitivity) can feed central gain via sustained nociceptive input and degraded proprioception; conversely, central amplification can lower peripheral thresholds so that ordinary loads become symptomatic [22–26,30]. Addressing SNR therefore acts as a bottom-up lever that complements top-down strategies (education, graded exposure) [22–26,30].

1.3.4 Phenotyping value: from "non-specific" to actionable subgroup

The Lancet series emphasizes that no single treatment suits all and calls for mechanism-aligned care [5,6]. SNR offers a rule-in clinical phenotype: (i) familiar pain provoked near end-range by SLR/Slump/PKB; (ii) predictable modulation with sensitizers (worse with ankle DF/cervical flexion, better with PF/cervical extension); (iii) no objective neurological deficit; (iv) compatible interface findings (tenderness along neural course; stiff gluteal/hamstring or iliopsoas sleeves) [21,31,33–37,39,40]. Classifying this subgroup guides early choices toward sliders and interface strategies rather than generic stretching or premature tensioning [1–3,33–37,39–42].

1.3.5 Operational testability in routine clinics

A key rationale for SNR is that it is testable at the bedside with minimal equipment [33–37]. Using scripted sequences and symptom-first endpoints, clinicians can elicit load-dependent patterns within minutes [33–37]. Re-testing after a brief dose of sliders or interface de-loading frequently demonstrates within-session change (reduced intensity at the same angle; later symptom onset), which both (a) supports the working diagnosis and (b) provides an embodied rationale for home dosing [1–3,39–41].

1.3.6 Explaining common clinical puzzles

SNR helps resolve otherwise vexing findings:

-"Tight hamstrings" that don't lengthen with stretching. If early symptoms in SLR disappear when ankle plantarflexion or cervical extension is added, the problem is neural load sharing, not sarcomere length. Stretching harder often flares; sliders restore glide without high tensile load [33–36,39,40].

-Posture-provoked pain in “strong” patients. Prolonged sitting increases posterior chain tension and interface creep, shifting the system toward strain-dominant behaviour; micro-bouts of sliders act as decompression cycles [19,33–36].

-Guarded movement patterns. Protective tone (hamstrings, gluteals, iliopsoas) emerges to protect sensitive neural tissues; reducing mechanosensitivity with sliders/interface work often unlocks motor control training [22–24,31,39–42].

1.3.7 Risk–benefit calculus: low risk, high plausibility

Early SNR-aligned care (gentle sliders, interface soft-tissue work, graded motor control) is low risk and low cost, particularly compared with imaging cascades and procedure-heavy pathways that rarely improve long-term outcomes in non-specific CLBP [5,6,39–41]. The safety anchor is irritability-based dosing and the 24-hour rule (reduce amplitude/frequency if soreness >3/10 or lasts >24 h) [1–3,39–41].

1.3.8 Expected moderators of response

Not every CLBP case is SNR-driven [5,6,25,26]. The rationale includes recognizing moderators:

-Higher likelihood: posture-provoked familiar pain; end-range combined movements; clear sensitizer modulation; focal interface tenderness (gluteal region, posterior iliac crest, femoral triangle) [21,31,33–37,39,40].

-Lower likelihood or co-drivers dominate: diffuse, non-patterned pain with sleep disruption and widespread tenderness (nociplastic tilt) [25,26,48,49]; robust facet/SIJ signs without neurodynamic modulation; objective neuro deficits (radiculopathy) needing different pathways [5,6,25,26]. Identifying moderators ensures SNR care is targeted, not universal [5,6,25,26,33–37].

1.3.9 Mechanistic chain: from restriction to disability

A plausible logic model:

1.Interface stiffness (micro-adhesions, low-grade inflammation, hypertonicity) reduces capacity for glide [15–19,27,28].

2.Movement reaches the steep portion of the nerve’s stress–strain curve earlier; mechanosensitive afferents fire at lower thresholds [13–18].

3.The CNS responds with protective motor strategies (co-contraction, substitutions) that initially reduce load but perpetuate stiffness and degrade proprioception [22–24,30,50].

4.Day-to-day function becomes fragile: ordinary tasks provoke symptoms; activity is avoided; fitness drops; sleep worsens; perceived threat increases—locking in chronicity [5,6,25].

5.SNR-aligned care (sliders/interface → motor control → graded function) reverses steps 1–3, reducing peripheral drive and enabling top-down gains [1–3,39–42].

1.3.10 Why symptom modulation outranks raw range

Degrees alone are noisy—affected by limb length, fear, and inconsistent end-points [33–37]. Modulation with applied and reversed sensitizers is the mechanistic signature of load-sensitive neural tissues [33–37]. This repeatable pattern is central to SNR classification and is more reliable when scripts are standardized [33–37].

1.3.11 Alignment with existing evidence streams

Converging strands support the SNR rationale [1–3,13–19,22–24,30,31,39–41,46]:

-Mechanical/physiology: hierarchical nerve architecture, viscoelastic behaviour, vascular sensitivity to strain/compression [13–18].

-Experimental/clinical neurodynamics: sliders can change symptoms and test performance in mechanosensitive states; early aggressive tensioning is provocative—underscoring the need for dose intelligence [1–3,11,12,39–41].

-Sensorimotor literature: CLBP is associated with impaired proprioception and altered motor control; reducing mechanosensitivity can normalize movement sharing and lower protective tone [22–24,30,31].

1.3.12 Health-system rationale: value and scalability

Because CLBP is common, even modest per-patient gains scale [4–6]. SNR care requires no expensive equipment, can be delivered briefly, and is amenable to micro-dosing between visits (short slider sets, interface self-care), improving equity and access [39–41]. Avoiding low-value cascades (routine imaging/procedures for non-specific cases) while providing mechanism-aligned self-management represents the value shift urged by contemporary guidelines [5,6,39–41].

1.3.13 Ethical rationale: making invisible mechanisms visible

Patients deserve explanations that fit their symptoms and do not pathologize normal scans [5,6,25]. Demonstrating real-time change—for example, Slump pain easing with neck extension—turns an abstract theory into a felt experience and validates the person's report [25,33–37,39–41]. This fosters self-efficacy and reduces stigma ("it's all in your head"), an ethical gain in itself [25].

1.3.14 Research-ready propositions

-H1 (Classification reliability): SNR scripts using symptom-first end-points and sensitizer reversal achieve ICC ≥0.75 with acceptable SEM/MDC [33–37].

-H2 (Mechanistic change): A 4–6 week slider + interface program increases tolerance (later symptom onset; reduced intensity) and improves functional outcomes (NPRS/ODI/PSFS) in SNR-positive CLBP [3,39–41].

-H3 (Moderation): SNR-positive patients benefit more from slider-first care than SNR-negative peers under the same multimodal plan [3,39–41].

-H4 (Biomarker linkage, exploratory): Where available, ultrasound shows increased excursion after treatment, correlating with clinical improvement [41,46].

1.3.15 Practical corollaries for the clinic

-Start with sliders, not stretches. Bias glide over tensile load early, especially at high irritability [1–3,39–41].

-Treat interfaces. Address gluteal/hamstring or iliopsoas/inguinal sleeves that bottleneck excursion [21,31,39,40].

-Dose by irritability and obey the 24-hour rule. Avoid flares that reinforce avoidance [1–3,39–41].

-Document modulation. Make mechanics visible (apply → observe → reverse) and track the trio (NPRS/ODI/PSFS) with within-test notes [33–37,39–41].

-Integrate motor control and proprioception as mechanosensitivity falls, then load functionally [30,41,42].

1.3.16 Anticipated critiques and responses

-"Neurodynamic tests lack specificity." Range-only endpoints do, but modulation with sensitizers plus interface findings and reproducibility yields a more specific signal for load-sensitive neural tissues [33–37].

-“This is just hamstring tightness.” Hamstring length does not systematically change with cervical/ankle sensitizers; SNR does [33–37].

-“Central factors dominate CLBP.” Often true; SNR care reduces peripheral drive and facilitates top-down strategies—complementary, not competing [25,26].

-“Evidence is heterogeneous.” Hence the call for standardized scripts, reliability studies, and phenotype-stratified trials—feasible, low-risk, and practice-relevant [3,39–41].

1.3.18 Measurement error, irritability, and the case for symptom-first endpoints

Angle-only endpoints are vulnerable to expectation, guarding, and tester cueing [33–37]. Symptom-first endpoints (“tell me when you feel your usual pain begin”) plus sensitizer modulation convert a fuzzy biomechanical readout into a binary/ordinal pattern that is easier to agree upon across raters, even when irritability fluctuates [33–37]. The 24-hour rule (shrink amplitude/frequency if soreness >3/10 or persists >24 h) stabilizes between-session biology for reliable re-tests [1–3,39–41].

1.3.19 Operational taxonomy of irritability

-High: early symptoms, long after-sensations; strong modulation; obvious protective tone. → Micro-sliders, interface de-loading, no tensioners [1–3,39–41].

-Moderate: mid-range symptoms; short after-sensations. → Progress sliders; cautious tensioning only after clean 24-h responses; begin motor control [41,42].

-Low: end-range or rapid-task symptoms; minimal modulation. → Functional loading, occasional tensioners, proprioception and task integration [30,41,42].

This taxonomy translates biology into dosing rules that can be applied consistently in busy clinics [1–3,33–37,39–42].

1.3.20 Comparator frameworks: where SNR adds unique value

Facet/SIJ and myofascial models do not explain predictable sensitizer effects; nociplastic models do not account for tight load–symptom coupling and rapid within-session change with sliders [25,26,33–37]. SNR fills this explanatory gap as a distinct rule-in phenotype [25,26,33–37].

1.3.21 Micro-vignettes that clarify rationale

Posterior chain desk worker: SLR 52° reproduces familiar ache; ankle DF/cervical flexion worse, PF/extension better; gluteal sciatic tenderness. After 60 s sliders, onset ~60° with lower intensity; clean 24-h response → sliders + interface, not stretching [31,33–36,39,40].
Anterior pathway standing intolerance: Early PKB discomfort worsens with hip extension, eases with flexion; modest worsening with cervical flexion; iliopsoas tone. → Femoral sliders + iliopsoas interface, then hip-extension control [32,37,39–41].

1.3.22 Health economics: expected value of early SNR recognition

Early SNR recognition prevents low-value cascades (routine imaging, premature procedures) and substitutes low-cost sliders/interface strategies [4–6,39–41]. Small per-patient gains scale via fewer missed days and reduced presenteeism; at population level, this is a meaningful economic lever [4–6,39–41].

1.3.23 Education scripts that embody the rationale

-“Glide, not yank.” We redistribute load along the chain; change with ankle/neck proves load sensitivity [33–37,39–41].

-“Traffic-light dosing.” Green/Yellow/Red governs amplitude and frequency to avoid flares [1–3,39–41].

-“Wins you can feel.” Re-test SLR/Slump after sliders to make change visible and reinforce self-efficacy [33–36,39–41].

These scripts translate complex mechanics into actionable, patient-friendly rules [25,33–37,39–41].

1.3.24 Common pitfalls (and how SNR prevents them)

Over-stretching “tight” hamstrings; escalating to procedures after generic programs; ignoring day-to-day biology. SNR fixes: prove neural modulation; dose by irritability; 24-hour rule; interface focus; objective re-tests [33–41].

1.3.25 Training and fidelity: how to scale SNR in services

Deliver micro-workshops on scripted SLR/Slump/PKB (apply → observe → reverse), provide pocket templates (angle at symptom onset; sensitizer +/-; interface notes; irritability; 24-h response; NPRS/ODI/PSFS), and run brief audit cycles for documentation fidelity [33–37].

1.3.26 Research agenda sharpened by SNR

-Reliability: multi-site ICC/SEM/MDC for SNR classification with and without modulation fields [33–37].

-Feasibility: 4–6-week slider-first program; adherence, flare rates, PSFS/NPRS/ODI change; qualitative acceptability [3,39–41].

-Mechanistic: ultrasound excursion changes in a subset and correlation with clinical improvement [41,46].

-Moderation: SNR-positive vs. SNR-negative differential response within multimodal rehab [3,39–41].

Together, these lines of work move SNR from plausible construct to rigorously tested clinical phenotype [3,33–37,39–41,46].

1.3.27 Boundary conditions: when SNR is not the main lever

Objective neuro deficit → radicular pathway; clear facet/SIJ dominance without modulation → segmental/ring strategies; strong nociplastic tilt → heavier emphasis on education, sleep, pacing, graded exposure, with gentle sliders for predictability and confidence [25,26]. Clarifying these boundaries reduces over-application and keeps care mechanism-aligned [5,6,25,26,33–37].

1.3.28 Consolidated case for SNR

SNR targets a common but overlooked contributor to CLBP—peripheral neural systems that are mechanically load-sensitive yet neurologically "normal." It is plausible, observable, low-risk, scalable, and research-ready. It converts a slice of "non-specific" pain into a tractable problem with measurable within-session change and sustainable self-management [1–3,6–9,11–12,13–19,21,22,24–28,30,31,33–42,46].

1.3.29 Red flags, differential diagnosis, and safe selection for SNR-focused care

A strong rationale includes knowing when not to pursue an SNR-first pathway [5,6,25,26]. Classic red flags—progressive neurological deficit, saddle anesthesia, fever/unexplained weight loss, history of cancer, significant trauma—mandate medical work-up before any neurodynamic loading [5,6,25,26]. Even in their absence, some differentials deserve attention:

-True radiculopathy. Dermatomal pain, myotomal weakness, reflex changes, or marked positive neural tension with distal paresthesia point to nerve-root pathology; SNR-style sliders may still be used, but medical evaluation and radicular algorithms take priority [5,6,25,26].

-Facet/SIJ-predominant pain. Localized paraspinal pain with extension/rotation bias, clear segmental provocation, and no sensitizer modulation on SLR/Slump/PKB suggests spinal segmental drivers [5,6,33–37].

-Hip pathology. If hip internal rotation/FABER/FADIR strongly reproduce symptoms independent of sensitizers, prioritize hip-first pathways [5,6].

-Nociplastic tilt. Widespread tenderness, sleep disturbance, and poor load–symptom coupling mean neurodynamic work should be gentle and brief, while education, graded exposure, and sleep strategies carry more weight [25,26,48,49].

This triage respects safety, improves face validity with patients, and prevents misapplication of SNR [5,6,25,26,33–37].

1.3.30 Interpreting imaging and electrodiagnostics through the SNR lens

Normal MRI and nerve-conduction studies often reassure yet do not falsify SNR because SNR lives below the detection threshold of macro-structural tests [7,8,11,12]. The relevant signals for SNR are functional patterns: predictable sensitizer modulation (apply → observe → reverse) and within-session change after sliders/interface work [33–37,39–41]. When imaging does show structural changes (disc bulge, facet arthropathy) but symptoms behave like SNR (clean modulation, no neuro deficit), it is reasonable to treat SNR mechanisms as co-drivers [5–8,11,12,33–37]. Electrodiagnostics are typically normal in SNR; this normality should be framed for patients as: “good news—no nerve damage; what we’re addressing is sensitivity and movement-sharing of living neural tissues” [7,8,11,12,25,39–41].

1.3.31 A clinician-facing algorithm (operational rationale)

The SNR rationale becomes practical when encoded in a short algorithm [33–37,39–41]:

1.Screen & triage. Red flags absent? If present, refer. If objective neuro deficit → radicular pathway [5,6,25,26].

2.Scripted tests. SLR/Slump/PKB with symptom-first endpoints and staged sensitizers (ankle/cervical/hip) [33–37].

Classify.

1.SNR-likely: familiar pain reproduced near end-range + predictable modulation (worse with DF/CF; better with PF/CE); compatible interface tenderness; neuro exam normal [33–37,39–41].

2.SNR-unlikely: no modulation; strong segmental/hip signs; diffuse non-patterned pain [25,26,33–37].

3.Dose by irritability (high → micro-sliders + interface; moderate → sliders → add small-dose tensioners after clean 24-h; low → integrate function, occasional tensioners) [1–3,39–42].

4.Re-test each visit. Look for later symptom onset or reduced intensity at the same angle; adjust amplitude/frequency accordingly [33–37,39–41].

5,Progression. As mechanosensitivity falls, layer motor control, proprioception, and task practice [30,41,42].

This small set of decisions captures the why and how of SNR without heavy cognitive load [33–37,39–42].

1.3.32 Interprofessional integration: making SNR play well with others

SNR is not a silo. It integrates with:

-Pain education & graded exposure: Within-session modulation provides a hands-on demonstration that supports reconceptualization ("sensitive, not damaged") and reduces threat, making exposure more acceptable [25,26,33–37,39–42].

-Strength & conditioning: Once sliders normalize tolerance, posterior-chain strength (hip hinge, split squats) and trunk endurance (anti-rotation, carries) consolidate gains; loads are introduced after irritability calms to avoid re-sensitization [30,39–42].

-Ergonomics & pacing: Micro-bouts of sliders every 60–90 minutes for desk workers reduce posterior-chain creep and stabilize day-to-day biology [19,33–36,39].

-Manual therapy: Brief interface techniques (gluteal/hamstring or iliopsoas/inguinal) improve relative movement before active sliders, especially early in care [39,40].

-Behavioral health & sleep: Where nociplastic features are present, referral for CBT-I or brief ACT can amplify gains; slider programs serve as experiential anchors for capability-building [25,26].

The rationale strengthens when patients experience coherent messages from the whole team [5,6,25,26,33–37,39–42].

1.3.33 Defining meaningful change and responder profiles

A good rationale specifies what success looks like [33–37,39–42]. Pair standard patient-important outcomes with mechanism-linked markers:

-Patient-important: NPRS (average & worst), ODI, PSFS (1–3 priority tasks) [5,6].

-Mechanism-linked: angle at first familiar symptom in SLR/PKB, Slump symptom intensity at matched knee angle, sensitizer modulation consistency (present/absent; direction), and palpatory interface tolerance [33–37,39–41].

Responder profile (typical): in 2–4 weeks, SLR symptom onset shifts 10–15° later and Slump intensity at a matched angle drops ≥2/10, with clean 24-h responses to home sliders; PSFS improves ≥2 points on at least one task; NPRS average drops 1–2 points. These are realistic and clinically meaningful thresholds in routine practice, acknowledging variability [3,33–37,39–42].

1.3.34 Safety governance, flare management, and patient self-calibration

SNR interventions are low risk when dosed intelligently [1–3,39–41]. The 24-hour rule (reduce amplitude/frequency if soreness >3/10 or lingers into next day) is the bedrock [1–3,39–41]. Additional safety practices:

-"Yellow flag buffer." In high irritability, keep repetition low (8–10) and arcs small; stop rounds early if form deteriorates or symptoms begin to spread [1–3,39–41].

-Flare rescue plan. If a flare occurs: 24–48 hours of range-only sliders (very small arcs), resume prior level only after symptoms return to baseline; maintain walking and gentle breathing to avoid global withdrawal [1–3,39–41].

-Communication cues. Patients rehearse two lines: "Glide, not yank" (stay smooth) and "Better now or within hours" (don't chase soreness). These cues encode the rationale into actionable behaviours [25,39–41].

1.3.35 Limitations of the current rationale (and how to address them)

Honest rationales acknowledge limitations [3,5,6,33–37,39–41,46]:

-Heterogeneous tests. Neurodynamic procedures vary by clinic; solution: standardize scripts (apply → observe → reverse; symptom-first endpoints) and document modulation rather than angles alone [33–37].

-Evidence granularity. Much support is mechanistic/observational; solution: conduct reliability studies, feasibility trials, and phenotype-stratified comparisons (SNR-positive vs. SNR-negative) with pragmatic outcomes [3,39–41].

-Attribution error. Improvement could stem from non-specific effects; solution: embed within-session re-tests tied to the mechanism and, where feasible, use simple objective adjuncts (e.g., ultrasound excursion) in sub-studies [41,46].

-Over-application risk. Not all CLBP is SNR; solution: adhere to moderators/boundaries outlined earlier; if no modulation or progress after 2–3 visits despite clean dosing, pivot the working diagnosis [3,33–37,39–41].

1.3.36 Summary rationale

SNR focuses attention on a treatable, testable contributor to CLBP that is easy to miss when clinicians rely solely on imaging, neurological screens, or range-only testing [5–8,11,12,33–37]. It provides a unifying explanation for posture-provoked pain, end-range symptom behaviour, and protective motor patterns in patients who lack overt neuropathy [22–26,30,31]. The approach is mechanistically plausible, clinically actionable, low-risk, and research-ready—and it integrates cleanly with biopsychosocial care [1–3,5,6,25,26,33–42]. In short, studying SNR is warranted because it promises to convert a subset of "non-specific" CLBP into a tractable problem with clear assessment signals and scalable, mechanism-aligned interventions [1–3,6–9,11–12,13–19,21,22,24–28,30,31,33–42,46].

1.4 Historical Context of Neurodynamics

The modern concept of neurodynamics—the study of how neural tissues move, deform, and respond to mechanical load within the musculoskeletal system—emerged from clinical observation, basic science, and a long lineage of manual and rehabilitation practice. Its development can be traced through four overlapping phases: (1) pre-formative insights about "nerve stretch" and adverse postures, (2) the formalization of neural mechanobiology in the late 1980s–1990s, (3) methodological maturation and early mechanistic/clinical studies in the 2000s, and (4) integration with contemporary pain science and phenotype-based rehabilitation in the 2010s–present. Foundational contributions by Butler and Shacklock codified a mechanical view of the nervous system and generated assessment/treatment frameworks that are still used today [1,7,9]. Subsequent experimental and clinical research—by Coppieters and colleagues, among others—linked small changes in neural mobility to altered mechanosensitivity and motor behavior, providing a physiologic substrate for clinical observations [2,11,12,27,28]. While early work centered on overt neuropathies (e.g., radiculopathy), later literature broadened the lens to include subclinical impairments that modify symptom behavior and function without frank neurological deficit [7,8,11,33,43]. This historical pathway underpins the present paper's focus on subclinical neurodynamic restrictions (SNR) as a contributor to chronic low back pain (CLBP) within the broader epidemiologic and guideline landscape highlighted by Balagué, Hartvigsen, Maher, and others [4–6,26,55–57,77].

1.4.1 Pre-formative threads: nerves as living tissues, not wires

Long before “neurodynamics” was named, clinicians noted that certain limb and spine positions provoked familiar symptoms, and that small adjustments could ease them. Early orthopaedic and neurological texts referenced “nerve stretching” tests and “tension signs,” particularly in the context of sciatica and radicular pain. As cadaveric and animal models accumulated, the peripheral nerve began to be described as a viscoelastic, vascularized tissue whose function depends on both electrophysiology and mechanics. Work on intraneural microcirculation and fascicular architecture (endoneurium, perineurium, epineurium) clarified that nerves are designed to glide and tolerate modest strain, with the perineurium providing shape stability and the epineurium allowing relative movement against surrounding tissues [13–16]. These insights foreshadowed the later clinical emphasis on sliding (excursion), transverse excursion, and strain partitioning during everyday movement [1–3,13,14,27,46].

1.4.2 The formal turn: Butler’s “adverse mechanical tension” model

A decisive moment came with Butler’s synthesis, which framed “adverse mechanical tension in the nervous system” as a clinical problem amenable to assessment and treatment [9]. Butler’s model proposed that symptoms can arise not only from chemical or compressive insults but also from mechanical dysfunction—situations in which normal movement places disproportionate stress on neural tissues because of reduced glide, altered interfaces, or sensitized afferents [9,17,18,28]. Crucially, Butler articulated neural mobilization strategies and linked position changes (so-called sensitizers) to predictable symptom modulation. This gave clinicians a language and a set of procedures to test whether a patient’s pain was load-dependent in neural tissues. Butler’s later educational work popularized the idea that the nervous system is a “continuum” whose mechanical load can be redistributed along the chain by adjusting joint positions at a distance (e.g., ankle or cervical spine during a Slump test), anticipating the structured test sequences used today [1–3,9,33–37].

1.4.3 Shacklock’s clinical neurodynamics: from concept to clinical method

Shacklock’s contributions in the mid-1990s further systematized the field. In Physiotherapy (1995), he argued that neurodynamics is inherently clinical: the nervous system must be tested and treated as a mechanically sensitive structure embedded in a musculoskeletal environment [1,7]. He described how specific sequences (e.g., straight leg raise [SLR] with addition or removal of ankle dorsiflexion and cervical flexion) can help distinguish muscular from neural contributions to symptoms by observing predictable increases or decreases in pain or stiffness at symptom onset—an “apply → observe → reverse” logic still central to practice [1,33–37]. In parallel, Shacklock emphasized that nerve tissues prefer glide to excessive tension, recommending early use of sliders (longitudinal movement with low tensile load) and cautious, later use of tensioners in irritable systems [1,3,7,73]. His 1995 paper in the Australian Journal of Physiotherapy elaborated clinical reasoning around sequencing, irritability grading, and the interaction between neural mechanics and adjacent interfaces (muscle, fascia, retinacula) [7,29]. Shacklock also urged researchers to refine terminology and protocols, calling for improved application of neurodynamic testing and standardized reporting to reduce noise and misinterpretation [29,33].

1.4.4 Early clinical signals: tests as mechanical probes, not flexibility screens

Clinical adoption of SLR, Slump, and prone knee bend (PKB) predates neurodynamics, but within this paradigm the tests were re-interpreted as mechanical probes of the neural system rather than generic flexibility screens. Studies explored their reliability and clinical utility when performed with standardized sequencing and clear endpoints [33–37]. Importantly, clinicians began to attend less to absolute angles and more to whether familiar symptoms were reproduced at symptom onset and modulated by remote joint positioning (e.g., ankle or cervical changes). This switch reflects the core historical insight: the "signature" of a neural contribution is not mere end-range discomfort but load-dependent modulation that is reversible within the test sequence [1–3,33–37].

1.4.5 Mechanistic consolidation in the 2000s: imaging, physiology, and mobility

The early 2000s saw a wave of mechanistic and clinical studies that validated and refined neurodynamic thinking:

– Imaging and excursion. Ultrasound and biomechanical studies documented nerve excursion during limb and spine movements and demonstrated that excursion varies with joint position and local interface conditions [27,41,46]. Dilley and colleagues quantified nerve sliding and examined how movement cycles influence mechanosensitive responses, giving object-level support to the clinical idea that glide matters [27,46].

– Physiology of mechanosensitivity. Basic neurophysiology work highlighted how afferents respond to mechanical deformation and how inflammation can sensitize these responses, altering perineural viscoelastic properties and lowering activation thresholds [17,18,28,38,45]. This reinforced a plausible biological pathway from reduced glide → increased strain at lower ranges → enhanced firing of mechanosensitive nociceptors.

– Experimental pain and neural loading. Coppieters and co-authors showed that seemingly minor restrictions or altered loading can change pain sensitivity and motor behavior, bridging the gap between cadaveric/mechanical measures and perception/movement in humans [2,11,12,39,44].

– Clinical procedures and reliability. Topp et al., Hall et al., Herrington, Nee and colleagues operationalized test procedures and examined measurement properties, strengthening the case for structured sequences and standardized endpoints [33–37]. Nee and Butler added applied clinical insights regarding dosing and irritability management [33,42].

Collectively, these studies advanced neurodynamics from concept to operational science: the neural system moves and is sensitive to mechanical load; we can measure its behavior; and we can influence it with graded movement strategies aligned to irritability [1–3,11,12,17–19,27,28,38–42,44–46,51–53,74–76].

1.4.6 Convergence with pain neuroscience and motor control

As pain science evolved, neurodynamics intersected with cognitive-affective and sensorimotor frameworks. Moseley's work emphasized that pain is an emergent experience influenced by meaning, attention, and prior learning, while still allowing that peripheral inputs matter—especially when they are predictable and controllable [25,47]. Vlaeyen and Linton articulated how fear-avoidance and catastrophizing can entrench disability, suggesting that early predictable wins (e.g., symptom eases when neural load is reversed) could reduce perceived threat and foster movement confidence [26,48,49,100]. Nociplastic constructs and central sensitization research further highlighted how altered central pain modulation and sensitivity can sustain symptoms in CLBP, while still acknowledging a role for peripheral drivers [47–49,60–63,78,79]. In CLBP specifically, studies documented altered proprioception, supraspinal motor control changes, and movement pattern adaptations—patterns the neurodynamic model interprets as protective motor strategies that initially offload sensitive neural tissues but, if persistent, contribute to stiffness and dysfunctional movement [22,24,30,31,50,58,59,80–86]. Neurodynamics thus sat naturally inside biopsychosocial care: peripheral mechanics are addressed with sliders and interface techniques, while education and graded exposure target learning and avoidance [5,6,22–26,30,39–42,60–63,77].

1.4.7 From overt neuropathy to subclinical restriction

Historically, neurodynamic thinking was most comfortable in overt neuropathic contexts—radiculopathy with clear neurological findings, or entrapments with focal deficits. Over time, clinical reports and mechanistic studies made room for subclinical states: presentations without dermatomal pain, weakness, or reflex loss, yet with reliable load–symptom coupling during neurodynamic testing and compatible interface findings [7,8,11,33,43]. Schmid and colleagues' work on neuroinflammation and mechanosensitivity suggested that low-grade inflammatory milieus can alter perineural viscoelastic properties and lower activation thresholds for mechanosensitive afferents [18,38,45]. In such states, the system may switch from glide-dominant to strain-dominant behavior earlier in range—provoking familiar pain with specific combinations (e.g., hip flexion + knee extension + spinal/cervical flexion) while leaving traditional neurological screens normal [11,12,17–19,27,28,33–37]. This mechanistic reframing helped clinicians account for CLBP cases where imaging is benign, neurological examination is normal, and yet symptom behavior is predictably influenced by remote joint positions [4–6,26,33–37,39–41,74–76].

1.4.8 Interfaces as the practical bottleneck

A consistent historical theme is that nerves rarely "fail" in isolation; problems arise at interfaces—the sleeves and tunnels they move through. In the lumbopelvic region, the sciatic nerve's relationships with the deep gluteal muscles and proximal hamstrings, and the superior cluneal nerves' passage over the posterior iliac crest, are frequent bottlenecks [20,21,31,88–90]. Clinical literature emphasized palpation and functional observation to identify tender or stiff interfaces and the use of manual therapy (soft-tissue mobilization, gentle joint work) as adjuncts to active neurodynamic dosing [31,39,40]. Cluneal neuralgia and related entrapment syndromes further highlighted how small-calibre sensory branches traversing osteofibrous tunnels can mimic deep lumbopelvic pain and respond to targeted interface interventions [21,88–92]. Historically, this interface-first orientation distinguished neurodynamics from traditional stretching: rather than pulling harder at end-range, clinicians sought to restore relative movement at the interfaces so that nerves could slide freely under lower tensile load—especially early in care [1–3,7,39,40,73].

1.4.9 Reliability, endpoints, and the shift from range to modulation

By the late 2000s and early 2010s, methodological papers argued that the endpoints of neurodynamic tests must be standardized if clinicians and researchers are to speak the same language. Rather than recording large end-range angles (which vary with limb length, fear, and compliance), investigators recommended symptom-first endpoints (the first familiar symptom, not maximal stretch) and systematic use of sensitizer reversibility to confirm a neural signal [33–37]. This historical shift—away from "how far" and toward "does it behave like a neural problem?"—improved reliability and clarified clinical decisions (e.g., slider dosing versus tensioning; interface emphasis; when to progress to functional tasks) [1–3,11,12,33–37,39–41]. These developments also set the stage for contemporary trials and meta-analyses that incorporate standardized neurodynamic procedures within broader CLBP programs [3,39–42,51–54,74–76].

1.4.10 Clinical outcome work and pragmatic dosing

Clinical outcome papers on neural mobilization are heterogeneous, reflecting diverse populations, dosing, and co-interventions. Yet a pragmatic consensus emerged: begin with low-load sliders in irritable systems; consider tensioners later if 24-hour responses are clean; combine with interface techniques and motor control as mechanosensitivity falls; and anchor progression to within-session change and patient-important outcomes (NPRS, ODI, PSFS) [1–3,22–24,30,39–42]. Systematic reviews and meta-analyses suggest that neural mobilization can provide clinically meaningful benefits in various musculoskeletal pain conditions, including low back pain, particularly when integrated into multimodal programs [3,39–41,51–54,71,72,74–76]. This dosing logic mirrors the biological properties of neural tissues (viscoelasticity, hysteresis) and respects sensorimotor and cognitive-affective contributors to persistence [22–26,30,48,49,60–63,80–83].

1.4.11 The wider CLBP landscape: why neurodynamics matters

The Lancet series on low back pain underscored the multifactorial nature of CLBP and the limited effectiveness of single-modality interventions [4–6,55–57]. Contemporary guidelines similarly emphasize active, person-centred care, judicious imaging, and avoidance of low-value, procedure-heavy pathways in non-specific presentations [55–57,77]. Against that backdrop, neurodynamics offered something the field needed: a mechanism-aware approach that is teachable, low-cost, and testable at the bedside. In a condition where a large subset of patients have normal imaging and neurological examinations but persistent movement-linked pain, neurodynamics provided a way to phenotype an overlooked subgroup—those whose symptoms show predictable modulation with neural loading/unloading [4–6,26,33–37,39–41,74–76]. Historically, this positioned neurodynamics not as a panacea but as a precision lever inside comprehensive care that also addresses nociplastic and psychosocial contributors [47–49,60–63,78,79].

1.4.12 Expanding the evidence base: targeted reliability and mechanistic links

Contemporary work has continued in three directions shaped by the field's history:

1.Reliability and standardization. Multi-site efforts to script test sequences and endpoints, report ICC/SEM/MDC, and document modulation (worse/better/same) rather than angles alone [33–37].

2.Mechanistic linkage. Studies exploring whether changes in excursion or interface tolerance (where measurable, e.g., ultrasound) correlate with clinical improvements, supporting construct validity [39–41,44,46,74–76].

3.Moderation and stratification. Comparative work examining whether patients who display SNR-consistent patterns derive greater benefit from slider-first programs within multimodal rehabilitation than those who do not [3,39–42,51–54,74–76].

These lines echo early calls (e.g., Shacklock's) to sharpen the science behind application and to link mechanics to meaningful outcomes [1,7,9,29,33–37,39–42,46,51–54].

1.4.13 Influence across disciplines: physiotherapy, manual therapy, osteopathy, sports.

Neurodynamics has diffused across professions. In physiotherapy and manual therapy, it provided a structured way to evaluate and treat nerve-related symptoms without waiting for frank neuropathy [1–3,9,22–24,30,39–42,51–53,72,74–76]. In osteopathic practice, with its emphasis on regional interdependence and soft-tissue interfaces, neurodynamics offered technique rationales congruent with existing osteopathic

principles (restore motion, reduce strain, normalize function), now anchored to mechanical behavior of neural tissues [40]. In sports and occupational settings, where position-dependent symptoms and repetitive loading are common, neurodynamics offered proactive strategies to manage sensitivity while restoring motor control and task exposure [22,24,30,31,37,39–42,71].

1.4.14 Controversies and clarifications that shaped the field

Like any growing domain, neurodynamics has faced critiques:

– Specificity and over-interpretation. Critics worried that neurodynamic tests could be "positive" for non-neural reasons (e.g., hamstring tightness). Historically, this has been addressed by emphasizing sensitizer modulation and reversibility rather than raw range; consistent, directionally appropriate changes (worse with ankle dorsiflexion/cervical flexion; better with plantarflexion/extension) strengthen the inference of neural load-sensitivity [1–3,33–37].

– Dosing and flare risk. Early enthusiasm sometimes led to over-tensioning irritable systems. The field responded by clarifying irritability grading and the 24-hour rule, advocating sliders and interface work first—especially when central sensitization features are present [1–3,25,26,39–42,47–49,60–63,78].

– Heterogeneity of trials. Mixed results in outcome studies reflect diverse populations and poor standardization. The historical response has been a push for scripted protocols, phenotype stratification (e.g., SNR-positive vs SNR-negative), and pragmatic outcomes (NPRS/ODI/PSFS with within-session tests and meaningful change thresholds) [3,33–37,39–42,51–54,74–76,93–99].

– Central vs peripheral debate. Some argued that focusing on peripheral mechanics ignores central contributors. The integrated view—now mainstream—is that reducing peripheral drive via improved glide/interface tolerance can facilitate top-down strategies; these are complementary, not competing lenses [22–26,30,47–49,60–63,78–83].

These clarifications have made the field more cautious, precise, and clinically useful.

1.4.15 The road to subclinical neurodynamic restrictions in CLBP

Within this history, the present paper's focus on subclinical restriction is a logical evolution. Early neurodynamics described neural behavior across the full spectrum—from normal glide through sensitized states to overt entrapment [1–3,7,9]. CLBP often sits in the middle: patients have mechanically predictable symptom behavior without overt deficits, in a context where epidemiologic and guideline data emphasize heterogeneity and the need for mechanism-aligned care [4–6,26,55–57,77]. Schafer

and colleagues reported on neural mobility limitations in clinical cohorts; although not framed explicitly as subclinical, their findings support the idea that relatively small mechanical changes can meaningfully affect symptoms and function [8,43]. Coppieters and Butler showed that even minor restrictions or experimental manipulations can alter mechanosensitivity and motor behavior, strengthening plausibility for a subclinical construct [2,11,12,39,44,73]. Schmid and co-workers connected inflammation to mechanosensitivity and tissue mechanical properties, providing a biologic bridge from clinical observation to mechanism [18,38,45]. As the Lancet series and related work called for mechanism-aligned care in CLBP and emphasized nociplastic/central contributions, SNR emerged as a clinically useful phenotype: normal imaging and neuro exam, but repeatable load–symptom coupling that responds to slider-first, interface-aware dosing [4–6,26,33–37,39–42,51–54,60–63,74–76,78].

1.4.16 Where we are now: a mature but still-evolving clinical science

Today, neurodynamics is a mature clinical method with clear principles: (1) nerves move and share load with adjacent tissues; (2) tests should use symptom-first endpoints and sensitizer reversibility; (3) early treatment favors glide (sliders) over tension in irritable systems; (4) interfaces often bottleneck glide and deserve attention; and (5) progress is anchored to within-session change and patient-important outcomes [1–3,7–9,22–24,30,33–42,51–54,71–76,93–99]. The field continues to refine measurement (e.g., ultrasound excursion, mechanosensitivity assays) and to pursue stratified clinical trials that test whether SNR-positive subgroups respond preferentially to slider-first programs when embedded in comprehensive care [3,39–42,44,46,51–54,71–76].

1.4.17 Implications of the historical arc for this paper

This history justifies three strategic moves in the current work:

1.Adopt the mechanobiological lens of Butler and Shacklock—treat nerves as living, mobile tissues whose load tolerance depends on glide, strain distribution, and interface compliance [1,7,9,13–19,27,28,38,45,46].

2.Use operational testing shaped by later methodological advances—symptom-first endpoints, standardized sequencing, and explicit sensitizer reversal—to classify SNR in CLBP [4–6,8,11,26,33–37,39–41,43].

3.Favor early dosing consistent with tissue behavior—sliders and interface de-loading before tensioners; integrate motor control and proprioception as mechanosensitivity falls; and measure progress with both patient-important outcomes and within-test markers, aligned with contemporary outcome and guideline literature [1–3,22–24,30,33–42,50,51–54,71–76,77,93–99].

These choices are the practical distillation of three decades of development and position SNR as a research-ready, clinically relevant phenotype within the broader CLBP landscape.

1.4.18 A concise historical synthesis

From its roots in “tension signs” and position-dependent symptoms, neurodynamics evolved through Butler’s conceptualization of adverse mechanical tension and Shacklock’s clinical systematization into a method that examines and treats the nervous system as a mechanical participant in movement and pain [1,7,9]. Imaging and physiology in the 2000s confirmed that nerves slide, translate, and sustain modest strain; that interfaces can bottleneck glide; and that low-grade inflammation sensitizes mechanoreceptive afferents [11,12,15–19,27,28,38,44–46]. Methodological work clarified that modulation under sensitizers—rather than raw angles—provides the key clinical signal [33–37]. Within modern pain science, neurodynamics became a bottom-up complement to top-down strategies, offering predictable, low-risk ways to reduce peripheral drive, restore confidence, and re-train motor control in CLBP and related conditions [4–6,22–26,30,39–42,47–49,50,55–57,60–63,71–76,78–83]. In CLBP—a heterogeneous, costly condition highlighted by population and guideline work—this history culminates in a practical, phenotype-based idea: subclinical neurodynamic restriction as a targetable contributor for a subset of patients with normal imaging/neurology but repeatable load–symptom coupling [4–6,7,8,11,26,33–43,39–42,51–54,60–63,71–76]. The present paper builds on this lineage to articulate SNR’s mechanisms, clinical features, and treatment implications in CLBP.

2. Neural Mobility and Subclinical Restrictions

2.1 The Concept of Neural Mobility

Neural mobility, or neurodynamics, refers to the ability of peripheral nerves to glide, translate, and accommodate physiologic strain as joints move and tissues load during everyday function [13]. In simple terms, nerves must move with us—not only conduct impulses. This dynamic behavior encompasses three cooperative mechanical actions: longitudinal sliding (excursion along the nerve’s course), transverse excursion (side-to-side translation to avoid focal compression), and elongation/strain absorption (limited stretch buffered by viscoelastic sheaths) [14–16,27,46]. During a straight-leg raise (SLR), for example, the sciatic nerve slides distally—sometimes on the order of millimeters—to prevent a steep rise in tensile load; when the knee is flexed, a portion of that excursion is “paid back,” reducing tension [14,27,46]. Transverse translation helps a nerve skirt bony contours or fascial edges, a property that is especially salient in the lumbopelvic region with its complex curves and crossing interfaces [15,20,21,27]. Limited elongation (typically tolerable in the order of ~10–15% before injury thresholds are approached) is the final safeguard once slack has been consumed by glide [15,16,27,46]. These mechanical provisions are essential for preserving intraneural perfusion, protecting axons and their myelin, and maintaining a comfortable sensorimotor experience. Mechanosensitive nerve endings—nociceptors

and proprioceptors—respond to mechanical deformation and to the chemical milieu; when mobility is restricted, the system becomes load-sensitive earlier, altering both sensation and motor behavior [17,18,28,38,45]. Butler's work popularized this view, emphasizing that small restrictions in neural movement can have clinical consequences out of proportion to the apparent mechanical change [1,7,9]. A classic clinical example is restricted sciatic nerve mobility provoking protective hamstring guarding—biasing gait and increasing lumbar loading during daily tasks [31].

2.1.1 **From "wire" to "living mechanical organ"**

Historically, clinicians and even some texts treated nerves like passive "wires," where only conduction mattered. Contemporary mechanobiology updates that picture. Peripheral nerves are living mechanical organs embedded in a dynamic musculoskeletal environment. They must share load with muscles, fasciae, retinacula, and osseous tunnels as posture and movement change minute by minute. In healthy systems, most of the early range in a posture is handled by glide—longitudinal and transverse—which minimizes the amount of true tensile strain needed to achieve a task. When glide is constrained—by tight interfaces, fascial adhesions, repetitive postures, or low-grade inflammation—the same posture pushes the nerve earlier into a strain-dominant regime, recruiting mechanosensitive afferents at lower thresholds. Patients experience this as "pull," "sting," "deep ache," or "tightness" that modulates predictably when distal or proximal joints are adjusted (e.g., ankle dorsiflexion vs plantarflexion; cervical flexion vs extension), the hallmark of a neurodynamic signature [33–37].

2.1.2 **Microanatomy and viscoelastic behavior**

The microanatomy explains why nerves can move—and when they cannot. Axons and Schwann cells reside within the endoneurium; fascicles are wrapped by the multilaminar perineurium, a relatively stiff sheath that maintains shape and resists excessive deformation; multiple fascicles and blood vessels are embedded within the more compliant epineurium [15,16]. The vasa nervorum course through these layers, making perfusion sensitive to pressure and stretch. This composite architecture yields viscoelastic properties—time-dependent deformation (creep) and history dependence (hysteresis)—which means both the rate and repetition of loading matter [15,16,27]. Shear planes between fascicles and between nerve and interface enable sliding in multiple directions. When those planes lose compliance—because of perineural fibrosis, dehydration of connective tissue, or sustained protective muscle tone—sliding costs more stress, local endoneurial pressure rises faster, and mechanosensitive firing is facilitated at lower ranges [17,18,27,28]. This is why rapid end-range "yanks" can flare symptoms, whereas slow, smooth arcs paired with breath control often do not.

2.1.3 **A stress–strain landscape for nerves**

Clinically, it helps to picture a stress–strain curve. In the toe region (low strain), slack is taken up primarily by glide; symptoms are unlikely. In the linear region, glide

continues but some tensile contribution emerges; sensitive systems may experience symptom onset here, which modulates with remote joint changes (sensitizers). Beyond that lies a steep region, where small increases in load produce large stress jumps, and symptoms escalate rapidly. Subclinical neurodynamic restriction (SNR) is essentially a left-shift of this landscape: the steep region is encountered earlier in ordinary ranges (e.g., tying a shoe, prolonged sitting), even though neurological examination may remain normal [11,33–37]. Restoring glide (and thus moving the system back to the right) is the rationale for sliders and interface work early in care [1–3,39–41,51–53,71,72,74–76].

2.1.4 **Longitudinal sliding: the first line of defense**

Longitudinal sliding—excursion along the nerve's course—is the primary means by which neural tissues avoid abrupt tensile spikes. In SLR, the sciatic nerve can slide distally several millimeters; knee flexion partly reverses that excursion [14,27,46]. In the upper limb, median and ulnar nerves show similar behavior with wrist and elbow motion. Importantly, excursion is not uniform along the pathway; curves, branch points, and tunnels change where and how sliding occurs. This is why starting angles, limb positioning, and sequencing in testing matter. If the tissues that share motion with the nerve (gluteals, hamstrings, iliopsoas) are stiff or tonically active, longitudinal sliding is reduced, and symptom onset occurs at smaller hip flexion or knee extension angles. Patients describe this as "I feel it too soon," a pattern that often changes immediately when you modify ankle or cervical positions (sensitizers), confirming that you are dealing with load in the neural system rather than purely myofascial limitation [33–36].

2.1.5 **Transverse excursion: avoiding pinch points**

Nerves must also move side-to-side to avoid focal compression from bone, ligamentous bands, or fascial edges. In the lumbopelvic region, where the sciatic passes beneath/between deep gluteal structures and where cluneal branches cross the posterior iliac crest, transverse freedom prevents "pinch points" that can otherwise over-concentrate stress [20,21,31,88–92]. Subtle loss of transverse glide—through localized interface thickening or sustained postures—can make everyday positions irritating. Palpation may reveal tender tracks along the nerve's corridor; a short block of interface-focused manual therapy followed by re-testing often shifts the symptom onset angle or intensity, signaling restored transverse motion and validating the mechanism [39,40].

2.1.6 **Elongation and strain absorption**

Despite the priority of glide, some elongation is unavoidable. Healthy nerves tolerate limited strain—often described in the 10–15% range prior to tissue compromise—because fascicular architecture distributes load and because sliding reduces how much elongation is demanded locally [15,16,27,46]. Problems arise when glide is constrained early: the system enters a strain-dominant regime sooner, perfusion is challenged, and mechanosensitive endings fire at lower loads. Clinically, that

translates into familiar end-range symptoms during SLR, Slump, or PKB that resolve predictably when sensitizers are reversed. Education built around this physiology ("glide first, tension later, and only in small amounts") helps patients understand why sliders feel better than aggressive end-range stretching early on [1–3,33–37,51–53,71,72,74–76].

2.1.7 **Perfusion–mechanics coupling**

Intraneural blood flow is sensitive to tension (which narrows microvascular lumens) and compression (which raises endoneurial pressure). Smooth excursion and transverse translation prevent prolonged focal stress, preserving perfusion [15–18,27,28]. Rate matters: fast, jerky movements spike intraneural pressures and afferent firing; slow, even movements with controlled breathing produce lower mechanical peaks. Clinically, cueing "glide, not yank" and pairing motion with exhalation are not aesthetic choices—they are perfusion-friendly dosing strategies that reduce flares while still exposing the system to the movement it needs [1–3,39–41,51–53,71,72].

2.1.8 **Region-specific mechanics in the lumbopelvic chain**

The sciatic nerve depends on freedom within the deep gluteal corridor and proximal hamstrings; hip flexion and knee extension create a long posterior lever that demands excursion and transverse glide [20,21,31]. The femoral nerve traverses iliopsoas and the inguinal region; hip extension and prone knee flexion load this anterior pathway, which underpins the prone knee bend (PKB) test [32,37]. The superior cluneal nerves cross dense fascia over the posterior iliac crest and can be irritated by local stiffness or postural compression; targeted mobilization and positional off-loading are often helpful [21,88–92]. Appreciating these regional facts improves test selection (SLR/Slump for posterior pathways, PKB for anterior) and helps clinicians pick starting angles that bias the intended corridor [33–37].

2.1.9 **Evidence for mobility: what we can see and measure**

Ultrasound studies show measurable nerve excursion during limb motion and, in some cohorts, post-intervention increases consistent with improved glide [41,46]. Biomechanical experiments (e.g., Dilley and colleagues) illustrate how repeated cycles change mechanosensitive responses, supporting graded exposure principles fundamental to slider dosing [27,46]. Neurophysiology work confirms that afferents respond to combined mechanical and chemical inputs and that low-grade inflammation can lower thresholds and alter tissue mechanics [17,18,28,38,45]. Clinical and experimental neurodynamics research (e.g., Coppieters and co-authors) demonstrates that seemingly minor restrictions can shift pain sensitivity and motor behavior in humans, bridging cadaveric mechanics with lived experience [2,11,12,39,44,51–53,71,72,74–76]. Together, these strands validate the construct: nerves move; their movement matters; and small changes can be felt and measured.

2.1.10 **Neurodynamic tests as mechanical probes**

SLR, Slump, and PKB are targeted stress tests of the neural continuum rather than generic flexibility screens. Their interpretive power lies not in a single angle but in predictable modulation under sensitizers:

– SLR (sciatic-biased): familiar posterior chain symptoms that worsen with ankle dorsiflexion or cervical flexion and ease with plantarflexion or cervical extension suggest a neural load-sensitive contribution [33–36].

– Slump (dural bias): adding spinal and cervical flexion increases neural/dural load; reversing either element should reduce symptoms if neural load is the driver [33–36].

– PKB (femoral-biased): knee flexion in prone challenges the anterior pathway; symptom change with hip or cervical position helps confirm neural sensitivity [37].

Standardizing symptom-first endpoints (the first familiar symptom, not maximal stretch) improves reliability and ties the test to the patient's complaint [33–37]. Recording direction and magnitude of modulation (worse/better/none) provides decision-grade information.

2.1.11 **Interpreting "tightness": behavior beats end-feel**

Patients often report "tight hamstrings." The question is why they feel tight. If sensitizers (ankle or cervical) change the symptom angle or intensity, the limitation is more likely neural load sharing than purely myofascial. Static end-range stretching in such cases frequently flares irritability; sliders and interface de-loading improve tolerance and, secondarily, increase available range [33–36,39,40,51–53,71,72,74–76]. If sensitizers do not modulate symptoms and muscle length tests are isolated, targeted flexibility work may take precedence. Behavior under sensitizers—not end-range feel—should drive interpretation.

2.1.12 **Mobility and motor control: a reciprocal relationship**

When neural tissues are load-sensitive, the motor system deploys protective strategies: earlier hamstring activation, gluteal co-contraction, or trunk "holding" patterns that reduce motion sharing across the chain [22–24,30,31,50,58,59,80–83]. These strategies reduce exposure but at the cost of stiffness, reduced movement variability, and higher energy expenditure. Over time, they degrade proprioception and reinforce pain persistence, particularly when daily tasks repeatedly push the system into strain-dominant zones [22–24,30,31,50,58,59,80–87]. Restoring glide reduces peripheral mechanosensitive drive and unlocks motor re-education; conversely, improving motor control normalizes load distribution and reduces recurrent strain spikes on neural

tissues. The relationship is bidirectional and clinically exploitable: combine sliders with motor control as soon as irritability allows [30,50,67–70,80–83].

2.1.13 **Subclinical neurodynamic restriction (SNR): the gray zone**

SNR captures the common scenario where neural mobility is reduced enough to bias the stress–strain curve leftward but not enough to produce dermatomal loss, myotomal weakness, or reflex changes [11,33–37]. Clinically, SNR is suggested by: (i) reproduction of the patient's familiar symptoms with biasing postures; (ii) predictable modulation with sensitizers; (iii) compatible interface tenderness or stiffness; and (iv) a normal neurological screen. This phenotype is prevalent in CLBP with "benign" imaging and explains position-linked pain that would otherwise be deemed "non-specific." Its practical importance is that SNR is treatable with low-risk, mechanism-aligned strategies (sliders, interface work, graded motor exposure) [1–3,7,8,11,33–37,39–42,51–53,71,72,74–76].

2.1.14 **Dosing mobility: why sliders come first**

Because nerves prefer glide to tension—especially in irritable systems—early programs prioritize sliders:

– Posterior-chain slider (supine): small hip-flexion arc paired with knee flexion/extension; add/remove ankle dorsiflexion/plantarflexion and cervical flexion/extension to fine-tune load.

– Slump slider (seated): gentle knee extension performed with cervical extension (release); brief excursions into flexion as tolerated; small arcs, smooth rhythm.

– Femoral slider (prone/side-lying): small knee-flexion arcs with hip position controlled; bias early release with slight hip flexion.

Irritability-based dosing is crucial:

1.High irritability: micro-sliders (8–10 reps, tiny arcs) 1–2×/day; short interface work; strict 24-hour rule (if soreness >3/10 or persists to next day, reduce amplitude/frequency) [1–3,39–41,51–53,71,72,74–76].

2.Moderate: expand arc/reps; trial low-dose tensioners only after repeated "green" 24-hour responses; add simple motor control.

3.Low: integrate functional loading (hip hinge, squat patterning), proprioception, and occasional tensioners [30,39–42,50,67–70].

2.1.15 **Interfaces: where mobility is won (or lost)**

Neural mobility is often limited at interfaces—the tunnels and sleeves through which nerves pass. In the lumbopelvic region, gluteal/hamstring tone can tether the sciatic; iliopsoas/inguinal stiffness can bias femoral loading; posterior iliac crest fascial density can irritate cluneal branches [20,21,31,88–92]. Brief blocks of manual therapy and self-release at these bottlenecks can immediately shift neurodynamic test behavior (later symptom onset; lower intensity at matched angles), confirming that glide improved and guiding progression [39,40]. The practical sequence is: test → brief interface-focused intervention → re-test. If behavior changes in the expected direction, maintain the strategy and scale dose cautiously.

2.1.16 **Rate, rhythm, and respiration**

Because neural tissues are rate-dependent, how a patient moves matters. Slow, even arcs minimize stress peaks and reduce sympathetic arousal; pairing movement with exhalation can further lower background mechanosensitivity. Coaching phrases—"glide, not yank," "smooth in, smooth out"—translate complex tissue mechanics into memorable motor scripts patients can enact safely at home [1–3,39–41,51–53,71,72].

2.1.17 **Movement ecology: thousands of micro-loads per day**

Neural mobility is shaped by the ecology of daily movement: hours of sitting, repeated bending, prolonged standing. Small, repeatable adjustments accumulate benefit:

– Sitting: hips slightly open; knees not markedly above hips; every 60–90 minutes, perform micro-releases (20–30 seconds of ankle plantarflexion cycles) and a brief set of sliders to prevent posterior-chain creep [19,33–36].

– Gait: encourage relaxed stride with gentle hip extension within tolerance and symmetrical arm swing.

– Bending/lifting: teach hip hinge with shared motion across hips and lumbar spine; in early sets, prioritize smoothness over range to remain glide-dominant [30,41,42,67–70].

These low-friction changes reduce baseline mechanosensitive drive so formal slider practice can consolidate gains.

2.1.18 **Outcome anchors and meaningful change**

Tie mobility work to outcomes that matter. Pair patient-important measures—NPRS (average and worst pain), ODI, PSFS (patient-selected tasks)—with mechanism-linked markers: angle at first familiar symptom in SLR/PKB; Slump symptom intensity at a matched knee angle; presence/direction of sensitizer modulation; palpatory interface tolerance [33–37,93–99]. In typical responders over 2–6 weeks, expect later symptom onset (e.g., +10–15° SLR), lower Slump intensity at the same angle (≥2/10 reduction), stable 24-hour responses, and PSFS gains—patterns consistent with restored glide and delayed entry into the strain-dominant region [3,39–41,51–53,71,72,74–76].

2.1.19 **Clinical vignettes**

Posterior-chain SNR (sciatic-biased). A 50-year-old teacher with CLBP presents with SLR onset at ~60° and a deep, familiar ache. Ankle dorsiflexion worsens symptoms; plantarflexion eases; cervical flexion worsens; extension eases. After 60–90 seconds of micro-sliders and brief gluteal/hamstring interface work, onset shifts to ~70° with reduced intensity; the 24-hour response is clean. Interpretation: glide improved; early strain reduced. Plan: continue sliders, add hip-hinge drills, and progress amplitude/frequency as irritability allows [31,33–36,39–41,51–53,71,72,74–76].
Anterior pathway sensitivity (femoral-biased). A 38-year-old office worker reports CLBP worse on rising from sitting. PKB provokes anterior thigh discomfort early; slight hip extension worsens; cervical extension eases marginally. Femoral sliders and iliopsoas interface techniques produce within-session relief; progression to pelvic alignment and graded hip-extension control occurs after several clean 24-hour periods [32,37,39–41,51–53,71,72,74–76].

2.1.20 **Boundaries and differentials**

Apply the mobility lens with clinical judgment. Red flags (progressive neurological deficit, constitutional symptoms, trauma) demand medical pathways. Where objective myotomal weakness, dermatomal loss, or reflex change is present, treat as radiculopathy; neurodynamic care may assist comfort but is not primary. If neurodynamic tests show no sensitizer modulation and facet/SIJ signs dominate, prioritize segmental/ring strategies. If nociplastic features are prominent (widespread tenderness, sleep disturbance, poor load–symptom coupling), emphasize education, sleep, pacing, and graded exposure; keep neurodynamic work gentle, focusing on predictability and control [5,6,25,26,48,49,60–63,78,79].

2.1.21 **A clinic-ready protocol**

Screen & triage

– Red flags absent; neuro exam normal (if deficit → radicular pathway) [5,6,77].

– Pain is position-linked; imaging is non-explanatory (common in CLBP) [4–6,55–57,60–63,78,79].

Test (scripted, symptom-first)

1.SLR/Slump/PKB to first familiar symptom; note angle/intensity.

2.Add/remove sensitizers (ankle DF/PF, cervical flex/ext; hip ext/flex for PKB); log directional modulation (worse/better/none) [33–37].

3.Palpate likely interfaces (gluteal/hamstring, iliopsoas, posterior iliac crest) for tenderness/stiffness [20,21,31,88–92].

Classify

1.SNR-likely: familiar pain + predictable modulation + compatible interface + normal neuro exam [7,8,11,33–37,39–42].

2.SNR-unlikely: no modulation; strong facet/SIJ or hip-dominant signs.

Dose (irritability-based)

1.High: micro-sliders (8–10 reps, tiny arcs) 1–2×/day; short interface work; enforce 24-hour rule [1–3,39–41,51–53,71,72,74–76].

2.Moderate: expand arc/reps; trial low-dose tensioners only after repeated green 24-hour responses; begin motor control [30,39–42,50,67–70].

3.Low: integrate functional loading, proprioception, and occasional tensioners [30,39–42,50,67–70].

Progress & safety

– Within-session: later SLR onset or lower Slump intensity at a matched angle after a slider block.

– Between-sessions (2–6 weeks): +10–15° SLR onset, ≥2/10 Slump reduction at matched angle, stable 24-hour responses, and PSFS gains [3,39–41,51–53,71,72,74–76,93–99].

If no progress after 2–3 visits despite correct dosing, pivot the working diagnosis (segmental, hip, nociplastic emphasis) [25,26,30,55–57,60–63,78,79,100].

2.1.22 **Patient education that makes mechanics intuitive**

– "Living cables." "Your nerves are living cables that must slide. When sliding is limited, you feel load earlier. Changing ankle or neck position reroutes that load."

– "Glide, not yank." "We'll start with small, smooth movements that encourage glide. If soreness lingers into tomorrow, we made the arc too big—next time smaller."

– "Wins you can feel." "We'll test, glide for a minute, then re-test so you can feel the change."

These scripts reduce threat by demonstrating reversibility and control and increase adherence to home practice [25,26,33–36,39–41,51–53,71,72,74–76].

2.1.23 **Special populations**

Adolescents. Growth spurts temporarily change lever arms and tissue compliance; excursion slack can feel limited without pathology. Keep dosing conservative—brief micro-sliders, smooth cadence, and frequent movement snacks across the school day to offset sitting [33–36].
Pregnancy/postpartum. Hormonal changes alter connective-tissue compliance and fluid balance; posture shifts (e.g., increased lordosis) modify loading. Use symptom-first endpoints, positional supports (pillows), and emphasize sliders and off-loading; avoid high-tension work [1–3,39–41,51–53,71,72].
Older adults. Age-related changes in vascular elasticity and hydration reduce shear and increase pressure sensitivity. Tests remain informative when the clinician privileges modulation over angles, uses slow cadence, and observes longer 24-hour windows before progressing [15–18,27,28,39–41,51–53,71,72,74–76].

2.1.24 **Research agenda**

Three pragmatic fronts could sharpen the mobility–outcome link:

1.Protocol harmonization. Multi-site use of the same scripted sequences (apply → observe → reverse), symptom-first endpoints, and a shared modulation taxonomy (worse/better/none) to improve reliability and enable pooling [33–37].

2.Mechanism-anchored pragmatic trials. In CLBP cohorts, compare slider-first care vs usual care within a multimodal program, stratifying by SNR status. Co-primary outcomes should mix NPRS/ODI/PSFS with mechanism-linked markers (SLR onset angle, Slump intensity at matched angle, interface tolerance) to test moderation by phenotype [3,8,39–41,43,51–53,71,72,74–76,93–99].

3.Feasible objective adjuncts. Limited-region ultrasound excursion measures before/after a short slider block in sub-studies as a construct check; in routine practice, standardized within-session re-tests remain the most practical surrogate [41,46,74–76].

2.1.25 **Common pitfalls (and mobility-savvy solutions)**

– Pitfall: Treating “tight hamstrings” with aggressive static stretching.
Solution: Prove/disprove neural modulation first. If present, use sliders and interface work; postpone long holds until irritability settles [33–36,39,40,51–53,71,72,74–76].

– Pitfall: Chasing angles rather than behavior.
Solution: Document symptom-first endpoints and directional sensitizer effects; they are the clinical signature [33–37].

– Pitfall: Flaring irritability with early tensioners.
Solution: Obey the 24-hour rule; progress dose slowly and only after repeated green responses [1–3,39–41,51–53,71,72,74–76].

– Pitfall: Ignoring movement ecology.
Solution: Install micro-releases into the day and coach task form to keep the system glide-dominant [19,30,33–36,39–42,67–70].

Summary of 2.1:

Peripheral nerves are living mechanical organs that must slide, translate, and absorb modest strain within compliant interfaces to function comfortably. The clinical signature of a mobility problem is predictable symptom modulation under sensitizers during SLR/Slump/PKB, not a particular angle. Because nerves prefer glide to tension, early care emphasizes sliders and interface de-loading—dosed by irritability and governed by the 24-hour rule—followed by graded motor control and functional loading as tolerance improves. This low-risk, mechanism-aware approach turns a common subset of “non-specific” CLBP into a tractable problem with measurable within-session change and meaningful patient-reported gains [1–3,11–18,20–21,22–24,27–28,30–31,33–37,39–42,46–53,58–59,60–63,67–72,74–76,78–80,84–87,88–92,93–99].

2.2 Defining Subclinical Neurodynamic Restrictions

Subclinical neurodynamic restrictions (SNR) are mild impairments in neural mobility and load-sharing that generate familiar, position- or movement-linked symptoms without overt neurological deficits (no dermatomal sensory loss, no myotomal weakness, no pathologic reflex changes) [1–3,7–9,11,17,33–37]. The construct is clinically important because many individuals with chronic low back pain (CLBP) present with pain that is clearly load-modifiable during neurodynamic testing yet have normal imaging and routine neurological screens [4–6,55–57,77]. In these patients, the nervous system appears intact from a conduction standpoint but sensitive from a mechanobiologic standpoint—entering a strain-dominant regime too early in everyday postures due to reduced longitudinal sliding, inadequate transverse excursion, or heightened intraneural mechanosensitivity (see §2.1) [13–18,27,28,38,45]. Below, we (1) sharpen the definitional boundaries of SNR; (2) delineate etiologic contributors and interfaces; (3) specify operational clinical criteria; (4) map common phenotypes and differentials; and (5) propose measurement anchors and a pragmatic research agenda.

2.2.1 Core definition and boundary conditions

At its core, SNR is defined by three elements:

1.Mechanically provoked familiar symptoms at reproducible ranges during biased postures or tests (e.g., SLR, Slump, PKB) that modulate predictably with sensitizers (e.g., ankle dorsiflexion/plantarflexion, cervical flexion/extension, hip extension/flexion) [1–3,7–9,11,33–37,39–42]. Predictable modulation means symptoms worsen when overall neural load is increased (e.g., ankle DF or cervical flexion in SLR/Slump) and ease when neural load is reduced (e.g., ankle PF or cervical extension).

2.Absence of overt neurological deficit on examination: normal light touch/pinprick across dermatomes; normal myotomal strength; physiologic reflexes. If objective deficit is present, the presentation should be classified as radiculopathy rather than SNR and managed along those lines [5,6,77].

3.Compatibility with interface findings (often, not always): palpatory tenderness or stiffness at known neural corridors (deep gluteal region, posterior iliac crest for cluneal branches, iliopsoas/inguinal for femoral) that changes immediately after targeted interface techniques or slider blocks [20,21,31,39,40,88–92].

SNR is subclinical in the narrow neurological sense (no frank deficit) but clinically meaningful in that it shapes the person’s pain behavior, movement choices, and treatment response. Conceptually, it describes a left-shift of the neural stress–strain landscape (early entry into strain-dominant terrain) with ordinary postures [1–3,13–18,27,28,33–37,38,45].

2.2.2 Etiologic contributors: where restriction comes from

SNR rarely stems from a single cause. Rather, multiple converging contributors combine to reduce glide and heighten intraneural sensitivity:

– Minor fibrosis or scarring. Repetitive microtrauma or incomplete remodeling in perineural tissues (e.g., after a strain, repetitive end-range sitting, or localized compression) can tether nerves and limit sliding [18,27,28,38,45]. Even small adhesions can alter how load is distributed, particularly in long-lever regions (sciatic corridor). Prolonged sitting, for instance, may create microtrauma in the lumbopelvic fascia, hindering sciatic excursion and promoting earlier symptom onset during SLR [19,33–36].

– Fascial adhesions and interface stiffness. Chronic tension in the lumbopelvic fascia—due to sustained postures, high-load training without adequate recovery, or altered motor patterns—can restrict transverse excursion and create focal pinch points [19–21,31]. Maigne's description of cluneal nerve entrapment illustrates how thickened fascia over the posterior iliac crest can mechanically irritate cutaneous branches; a similar interface picture may clinically mimic entrapment even without frank neuropathy [21,88–92].

– Chronic low-grade inflammation. Persistent inflammatory mediators alter the viscoelastic properties of neural and perineural tissues—reducing shear, increasing stiffness, and lowering mechanosensitive thresholds [17,18,20,27,28,38,45]. Schmid and colleagues demonstrate how inflammation increases neural mechanosensitivity, meaning smaller deformations produce larger afferent output [18,38,45]. Importantly, these changes do not require large structural lesions; subtle biochemical shifts can change how the tissue behaves under ordinary load.

– Mechanical tension from surrounding structures. Tight or tonically active muscles (piriformis, hamstrings, iliopsoas), joint malalignment, and postural biases can compress or tether neural pathways, reducing sliding during movement [20,21,31,88–92]. Example: deep gluteal hypertonicity in a sitter with CLBP may mechanically tether the sciatic nerve so that hip flexion in SLR produces earlier familiar symptoms; slider dosing and interface de-loading reduce onset intensity and shift angles favorably [31,33–36,39–41,51–53,71,72,74–76].

– Neurovascular coupling shifts. Intraneural perfusion is pressure-sensitive. When sliding is limited and strain increases locally, endoneurial pressure can rise, challenging microcirculation and further sensitizing afferents [15–18,27,28,41,46]. This sets up a mechanics–perfusion feedback loop that sustains sensitivity even after the initial trigger abates.

These influences accumulate in regions with long levers and complex interfaces—sciatic, femoral, and cluneal pathways in CLBP being prime examples [20,21,31,33–37,39–41,88–92].

2.2.3 Vulnerable pathways in CLBP

– Sciatic nerve (posterior chain). Passing through the pelvis and deep gluteal region, then along the posterior thigh, the sciatic nerve must negotiate large hip and knee excursions. Hypertonic gluteals or hamstrings, or posterior fascial stiffness, reduce longitudinal sliding and transverse freedom, biasing the system toward early strain during SLR/Slump [20,21,31,33–37,39–41,46].

– Femoral nerve (anterior pathway). Traversing iliopsoas and the inguinal region, the femoral nerve is biased by hip extension and knee flexion (PKB). Iliopsoas stiffness, anterior pelvic tilt, and prolonged sitting can predispose to early load sensitivity along this pathway [32,37,39–41].

– Superior/inferior cluneal nerves. Crossing the posterior iliac crest under firm fascial bands, these cutaneous branches can be sensitized by local tissue thickening or persistent postures. Patients often report posterior pelvic tenderness and activity-linked discomfort (e.g., with prolonged standing) that responds to transverse gliding techniques and positional off-loading [21,40,88–92].

2.2.4 Clinical phenomenology of SNR

SNR presents with load-coupled symptom behavior:

– Symptom qualities. Deep ache, pulling, or a familiar line of discomfort provoked by biased postures. Often non-dermatomal and localized, but with clear mechanical linkage.

– Reproducibility. Consistent onset at a similar range during SLR/Slump/PKB within and across sessions, with directionally predictable changes under sensitizers [33–37].

– Rapid reversibility. Symptoms ease quickly when load is reduced (e.g., ankle PF, cervical extension), distinguishing neural load from purely contractile or joint capsular end-feel [33–37,39–41].

– Interface concordance. Palpation over suspected corridors produces local tenderness or familiar ache; targeted interface techniques (brief soft tissue or positional glides) produce within-session improvement in test behavior [20,21,31,39,40,88–92].

– Normal routine neurology. Strength/reflexes/sensation normal; straight dermatomal mapping unhelpful (by definition) [5,6,11,77].

2.2.5 Operational clinical criteria (proposed)

To standardize SNR identification and improve reliability, we propose a four-criterion operational definition suitable for research and practice:
Criterion A (Signature Modulation): During at least one neurodynamic test (SLR, Slump, PKB), the patient's familiar symptom is reproduced and shows predictable modulation with sensitizers—worsens with load-increasing maneuvers (e.g., DF, cervical flexion) and eases with load-reducing maneuvers (e.g., PF, cervical extension) [1–3,7–9,11,33–37,39–42].
Criterion B (Symptom-First Endpoint): Testing is terminated at the first familiar symptom (not maximal stretch), aligning the mechanical stress with the patient's complaint and reducing false positives [33–37].
Criterion C (No Overt Neurological Deficit): Routine neurological screen is normal (sensation, strength, reflexes). If deficits exist, classify as radiculopathy rather than SNR [5,6,11,77].
Criterion D (Interface Corroboration): At least one of: (i) palpatory tenderness/stiffness along the biased corridor; (ii) within-session improvement in onset angle or intensity after a short block (≤2 minutes) of interface-focused manual work or sliders [20,21,31,39,40,88–92].

Supportive features (non-mandatory): position-linked daily aggravators (prolonged sitting/standing); asymmetrical SLR/PKB/Slump ranges; improved test behavior after a week of slider practice; and stable "green" 24-hour responses (no next-day flare with correct dosing) [1–3,19,33–37,39–41,51–53,71,72,74–76].

2.2.6 Grading irritability to guide dosing

SNR spans a spectrum of irritability—the ease with which symptoms are provoked and their persistence after testing or daily tasks:

– High irritability: Symptoms arise early with small arcs; after provocation they linger (>2–3 hours) or flare next day. Management: micro-sliders (8–10 reps, tiny arcs), strict 24-hour rule, short interface work; no tensioners [1–3,39–41,51–53,71,72,74–76].

– Moderate irritability: Symptoms arise at mid-range; post-test soreness is mild and short-lived (<24 hours). Management: expand slider arcs or reps; consider very low-dose tension only after consecutive green 24-hour responses; begin simple motor control in non-provocative ranges [1–3,30,39–42,50,67–70].

– Low irritability: Symptoms arise at end-range only; recovery is rapid. Management: integrate functional loading (hinge, squat, gait drills), proprioception, and occasional

tensioners to consolidate glide gains and normalize load sharing [22–24,30,41,42,50,67–70].

Irritability grading is integral to the SNR definition because it determines safe exposure. Two patients may both be SNR-positive by Criterion A–D but require very different starting doses.

2.2.7 Distinguishing SNR from look-alikes

Myofascial length limitation (true muscle shortness). End-range "stretch" that does not modulate with neural sensitizers suggests muscular or capsular limitation. Here, end-range static stretching is appropriate. In SNR, sensitizers change symptoms and sliders outperform long-hold stretches initially [33–36,39,40,51–53,71,72,74–76].
Facet/SIJ-dominant pain. Localized extension- or rotation-provoked lumbar pain with negative or non-modulating neurodynamic tests argues for a segmental or ring-dominant source. Treat segmental impairments first; re-screen neurodynamic behavior after irritability falls [4–6,30,41,42,55–57,77].
Radiculopathy. Dermatomal pain with objective neuro deficits (sensation/strength/reflex) is not SNR; it may still benefit from some neurodynamic principles, but classification and primary management shift to radicular pathways [5,6,11,77].
Nociplastic/centralized pain. Widespread tenderness, sleep disturbance, and poor load–symptom coupling suggest a nociplastic tilt [25,26,47–49,60–63,78,79]. Gentle, predictable sliders can be retained as graded exposure tools, but education, sleep, pacing, and cognitive strategies become co-primary [25,26,64–66,78–80,100].
Local entrapment neuropathy. Focal neurological signs (e.g., Tinel's, numbness in a cutaneous distribution) and persistent allodynia at one site favor true entrapment. SNR can mimic aspects (mechanical linkage) but lacks objective deficit; interface exam and response to sliders help differentiate [20,21,88–92].

2.2.8 Measurement anchors and documentation

To make SNR actionable, pair patient-important outcomes with mechanism-linked anchors:
Patient-important: NPRS (average/worst), ODI, PSFS (patient-selected tasks) [93–99].
Mechanism-linked:

– Angle at first familiar symptom in SLR/PKB.

– Slump intensity at a matched knee angle (0–10).

– Presence/direction of sensitizer modulation (worse/better/none).

– Palpatory interface tolerance (0–10) at gluteal/hamstring, posterior iliac crest, or iliopsoas corridors [33–37,39,40,88–92].

Within-session change after a 60–120 second slider or interface block is a powerful construct check. Between-session patterns over 2–6 weeks—e.g., +10–15° later SLR onset, ≥2/10 reduction in Slump intensity at the same knee angle, and clean 24-hour responses—indicate meaningful mobility gains [3,39–41,51–53,71,72,74–76,93–99].

2.2.9 Practical scripting to standardize SNR assessment

Testing script (symptom-first):

1.SLR: “Tell me when you first feel your usual symptom.” Note angle/intensity.

2.Add ankle DF (worse?) then PF (better?). Add cervical flexion (worse?) then extension (better?) [33–37].

3.Slump: spine and neck flexed; extend knee to first familiar symptom; reverse neck to see if symptoms ease; then modulate ankle [33–37].

4.PKB: prone; flex knee to first familiar symptom; modulate hip extension and neck [32,37].

5.Classification rule: SNR is likely if familiar symptoms are reproduced and consistently modulate in the predicted directions under sensitizers and routine neurology is normal; interface findings further support the call [5,6,11,20,21,31,33–37,39,40,77].

2.2.10 Case vignettes (pattern recognition)

Posterior-chain SNR (sciatic-biased).

A 50-year-old teacher with CLBP experiences a deep familiar ache at ~60° SLR. Ankle dorsiflexion worsens; plantarflexion eases; cervical flexion worsens; extension eases. Palpation reveals deep gluteal tenderness. After 90 seconds of micro-sliders and brief interface work, SLR onset shifts to ~70° and intensity drops by 2 points. Neurology is normal. Interpretation: SNR-positive (Criteria A–D). Plan: high-irritability dose initially; progress based on 24-hour responses [31,33–36,39–41,51–53,71,72,74–76].

Anterior pathway SNR (femoral-biased).
A 38-year-old office worker reports pain rising from sitting; PKB provokes anterior thigh discomfort early; slight hip extension worsens; cervical extension eases marginally. Iliopsoas corridor is tender. After femoral sliders and interface work, symptoms reduce within session. Neurology is normal. Interpretation: SNR-positive along anterior pathway. Plan: micro-sliders + positional off-loading; integrate pelvic alignment drills as irritability falls [32,37,39–41,51–53,71,72,74–76].

Cluneal-focused SNR.
A 42-year-old nurse with posterior pelvic pain on long standing shows localized tenderness over the posterior iliac crest. Slump is modestly provocative and modulates with neck/ankle position; targeted transverse gliding over the crest, plus sliders, yields immediate symptom reduction. Neurology is normal. Interpretation: SNR with cluneal interface contribution [21,40,88–92].

2.2.11 Patient education: making SNR intuitive

– “Living cables need glide.” “Your nerves are living cables. When they don’t slide well, they feel load earlier. Changing ankle or neck position reroutes that load—watch how symptoms change as we do it.”

– “Glide first, tension later.” “We’ll start with smooth, small gliding motions. If you’re sorer tomorrow, we made the arc too big—next time we’ll shrink it. As it settles, we’ll add a little more stretch.”

– “Proof within minutes.” “We’ll test, do one minute of glides, then re-test so you can feel the difference.”

Education reduces threat, builds adherence, and turns modulation into a self-check the patient can use at home [25,26,33–37,39–41,51–53,64–66,71,72,74–76,78–80,95,96,100].

2.2.12 Program building for SNR (clinic-ready)

Start with sliders at an irritability-appropriate dose and brief interface de-loading (soft tissue/manual or self-release). Layer in motor control (neutral pelvis, hip hinge) as symptoms stabilize; add proprioception (single-leg balance, gentle dynamic stability) once daily function improves [22–24,30,31,39–42,50,58,59,67–70,80–83]. Reinforce movement ecology: micro-releases every 60–90 minutes when sitting; gradual exposure to hip extension in gait; hinge form in daily bends [19,30,33–36,39–42,67–70]. The goal is to shift daily mechanics back into glide-dominant behavior and maintain it.

2.2.13 Special populations and contexts

Adolescents. Rapid growth temporarily reduces slack; SNR-like behavior may appear during spurts. Use conservative arcs, frequent breaks from sitting, and monitor 24-hour responses carefully [33–36].
Pregnancy/postpartum. Hormonal changes and postural shifts alter interface compliance. Keep tests symptom-first with positional supports; emphasize sliders and off-loading; avoid high-tension maneuvers; collaborate with obstetric guidance as needed [1–3,39–41,51–53,71,72,74–76].
Older adults. Reduced tissue hydration and vascular elasticity increase pressure

sensitivity. Tests retain value when cadence is slow, sensitizers are added gently, and progression respects longer recovery windows [15–18,27,28,39–41,51–53,71,72,74–76].
Athletic populations. Repetitive end-range tasks (distance running, deep hip flexion/extension sports) can accumulate interface stiffness. Early integration of sport-specific hinges and graded stride work consolidates gains [30,31,37,39–42,50,67–70,80–83].

2.2.14 Why SNR matters in CLBP

The Lancet series underscores CLBP heterogeneity and the limited dominance of any single treatment [4–6,55–57,77]. SNR offers a mechanism-aligned lens for a prevalent subgroup: people with normal imaging and normal neurology whose pain is reliably load-modifiable [5,6,11,47–49,60–63,78,79]. Treating SNR is low-risk (gliders, interface work, graded exposure), scalable for home programs, and produces within-session wins that improve confidence and adherence [1–3,39–42,51–53,64–66,71,72,74–76]. By reducing mechanosensitive input and protective tone, SNR-informed care can dovetail with education, pacing, strengthening, and proprioception to produce cumulative benefit [1–3,22–26,30,31,39–42,50,58,59,67–70,80–87,93–99,100].

2.2.15 Research and quality improvement agenda

To mature SNR from promising construct to standard phenotype, we recommend:

1.Protocol harmonization for testing: scripted sequences (apply → observe → reverse), symptom-first endpoints, and a simple modulation taxonomy (worse/better/none), enabling pooled reliability and outcome analyses across sites [33–37].

2.Phenotype-stratified pragmatic trials: CLBP cohorts randomized to slider-first multimodal care vs usual multimodal care, stratified by SNR (criteria A–D). Co-primary outcomes: NPRS/ODI/PSFS plus mechanism-linked markers (SLR onset angle; Slump intensity at matched knee angle; interface tolerance). Hypothesis: SNR-positive patients derive greater incremental benefit from slider-first programs [3,8,39–41,43,51–53,71,72,74–76,93–99].

3.Feasible objective adjuncts: in sub-studies, ultrasound excursion pre/post a short slider set as a construct check; not required in routine care but useful for mechanistic linkage [41,46,74–76].

4.Dosing algorithms based on irritability (high/moderate/low) embedded into clinical pathways to prevent flare-biased failures and to mirror real-world constraints [1–3,39–41,51–53,71,72,74–76].

5.Longitudinal registries tracking SNR status, irritability, dosing, and outcomes across diverse clinics to identify responders, non-responders, and optimal progression rules [93–99].

2.2.16 Common pitfalls and practical solutions

– Pitfall: Treating all end-range posterior thigh discomfort as “tight hamstrings.”
Solution: Test modulation with sensitizers first. If symptoms change predictably, classify as SNR and start sliders + interface work; defer long static holds until irritability drops [33–36,39,40,51–53,71,72,74–76].

– Pitfall: Advancing to tensioners too early.
Solution: Enforce the 24-hour rule; expand arc or add minimal tension only after multiple green responses [1–3,39–41,51–53,71,72,74–76].

– Pitfall: Relying on angles alone.
Solution: Record first familiar symptom angle and directional changes with sensitizers; these behaviors are more diagnostic than raw range [33–37].

– Pitfall: Ignoring movement ecology.
Solution: Install micro-releases in sedentary blocks; coach hinge mechanics; align home environment (chair height, break cadence) with glide-dominant behavior [19,30,33–36,39–42,67–70].

– Pitfall: Overlooking psychosocial context.
Solution: Pair SNR interventions with pain education, pacing, and sleep hygiene; reassure with within-session proof to reduce threat and catastrophizing [25,26,47–49,60–63,78,79,95,96,100].

2.2.17 A concise case definition (for manuscripts and protocols)

Subclinical neurodynamic restriction (SNR) is present when:

(A) the patient’s familiar symptom is reproduced during a neurodynamic test (SLR, Slump, or PKB) and modulates predictably with sensitizers (worse with load-increase; better with load-decrease) [1–3,7–9,11,33–37,39–42];

(B) testing uses a symptom-first endpoint [33–37];

(C) routine neurological examination shows no overt deficit (sensation, strength, reflexes) [5,6,11,77]; and

(D) at least one interface sign is present (tender corridor or within-session improvement after sliders/interface work) [20,21,31,39,40,88–92].

Irritability (high/moderate/low) should be graded to guide dosing and progression [1–3,39–41,51–53,71,72,74–76].

2.2.18 Summary

SNR defines the gray zone between normal neural mechanics and overt neuropathy: the nervous system conducts normally but feels load earlier because sliding and transverse freedom are constrained or mechanosensitivity is heightened. In CLBP—particularly among patients with normal imaging and "non-specific" labels—SNR is common and clinically tractable [4–6,11,47–49,55–57,60–63,77–79]. Its signature is predictable modulation of familiar symptoms during neurodynamic tests, without neurological deficit, often accompanied by interface tenderness and within-session responsiveness to sliders or interface techniques [1–3,7–9,11,13–21,27–28,31,33–37,39–42,46,51–53,71,72,74–76,88–92]. An operational, irritability-informed definition enables consistent identification, safer dosing (glide first, tension later), and meaningful outcome tracking [1–3,33–37,39–42,51–53,71,72,74–76,93–99]. Integrating SNR care with education, motor control, and movement ecology provides a low-risk, mechanism-aligned pathway that can yield immediate, demonstrable changes and sustained functional gains [1–3,5–6,13–21,22–26,30–31,33–42,46,47–49,50,55–57,58–59,60–63,67–70,71–72,74–76,77–80,84–87,88–92,93–99,100].

2.3 Mechanisms of Neural Restriction

The mechanisms underlying subclinical neurodynamic restrictions (SNR) are complex, layered, and mutually reinforcing. They involve interactions among peripheral mechanotransduction, neuroimmune chemistry, interface biomechanics, microvascular/perfusion dynamics, and sensorimotor control. While none of these processes alone must be catastrophic to drive symptoms, their convergence can shift the neural system toward early load sensitivity—so that ordinary postures or movements produce a familiar ache, pull, or sting, even though routine neurological examination remains normal [4–6,11,17,18,27,28,33–37,38,41,45,55–57,60–63,77–79]. This section details the peripheral and central pathways by which minor restrictions in neural mobility alter sensation and motor behavior, perpetuate symptoms, and shape clinical responses in chronic low back pain (CLBP).

2.3.1 Peripheral mechanotransduction: how small deformations become sensations

Peripheral nerves contain mechanosensitive afferents—including A-delta and C-fiber nociceptors as well as proprioceptive receptors—capable of transducing mechanical deformation (tension, compression, shear) into afferent discharge [17,18,27,28,38,45]. In healthy conditions, the system tolerates modest deformation because slack is first absorbed through longitudinal and transverse sliding (see §2.1). However, when sliding is constrained—by interface stiffness, perineural adhesions, or sustained posture—the same limb or trunk position can enforce greater intraneural deformation at a given range. Afferent endings in the epineurial and perineurial environment then

fire at lower thresholds, broadcasting a signal of mechanical threat long before frank neural injury is at stake [17,18,27,28,38,45]. Bove and Light's experimental work supports this principle: even minor restrictions can alter sensory processing so that modest mechanical inputs become salient and sometimes painful, despite the absence of an overt neuropathy [17,28]. Complementary studies from Dilley and colleagues demonstrate that repeated cycles of loading change intraneural behavior over time, consistent with viscoelastic and rate-dependent responses in neural tissues [27,41,46]. Practically, this means a patient may not react to a single movement in isolation but becomes symptomatic with repetition, speed, or sustained end-range—patterns clinicians routinely observe [1–3,33–37,39–42,51–53,71,72,74–76].

2.3.2 The sliding–strain trade-off: entering the steep part of the curve too early

In a well-behaving system, the first portion of motion is glide dominant: neural tissues translate longitudinally and transversely, keeping tensile strain modest [13–16,27,41,46]. When glide is limited, the system becomes strain dominant earlier, and a given posture crosses the stress inflection where small additional motion produces a disproportionate rise in intraneural stress. The patient experiences this as a sudden onset of familiar discomfort at an angle that seems "too soon." Importantly, this discomfort modulates predictably with distal or proximal sensitizers (e.g., ankle dorsiflexion or cervical flexion), confirming that the neural continuum—not a single muscle—is bearing the extra load [1–3,33–37,39–42]. This left-shift of the stress–strain relationship defines SNR's mechanical signature and explains why sliders (which restore glide) reduce symptoms more reliably than aggressive end-range stretching in early care [1–3,33–37,39–42,51–53,71,72,74–76].

2.3.3 Interface biomechanics: where restrictions arise

Nerves live within interfaces—gluteal and hamstring tissues for the sciatic, iliopsoas/inguinal corridor for the femoral, and dense fascia over the posterior iliac crest for the cluneal branches. Restrictions typically reflect three overlapping interface phenomena:

1.Perineural adhesions and minor fibrosis. Repetitive microtrauma or incomplete tissue remodeling yields tethering that resists longitudinal or transverse excursion. Schmid and colleagues show that low-grade inflammation stiffens perineural tissues and lowers mechanosensitive thresholds, compounding the mechanical problem [18,38,45]. Even small adhesions can be clinically potent when they sit at curves or tunnels where excursion demand is high [15–18,27,28,41,46].

2.Fascial thickening and tone-maintained stiffness. Chronic tension in lumbopelvic fascia—common with prolonged sitting, repetitive flexion, or bracing patterns—reduces shear between layers, so that sliding occurs less freely [19–21,31]. Over the posterior iliac crest, Maigne et al. describe cluneal nerve entrapment under taut fascia; a spectrum likely exists where subclinical compression mimics entrapment features but without frank neuropathy [21,88–92].

3.Muscle hypertonicity as interface load. Hyperactive or shortened gluteals/hamstrings (sciatic) or iliopsoas (femoral) can act as dynamic tethers, pulling the nerve against surrounding structures during motion. In SLR, for instance, deep gluteal tone limits posterior sliding, so the nerve enters strain dominance earlier; sliders and interface de-loading shift symptom onset later and reduce intensity [20,21,31,33–37,39–41,51–53,71,72,74–76,88–92].

2.3.4 Microvascular coupling: perfusion under pressure

Neural function depends on intraneural perfusion via the vasa nervorum. Mechanical tension narrows microvascular lumens; compression raises endoneurial pressure; both challenge oxygen and nutrient delivery [15–18,27,28,41,46]. When sliding is limited, strain accumulates locally, increasing endoneurial pressure and further sensitizing mechanoreceptors. This mechanics–perfusion loop is self-reinforcing: stiff tissues raise pressure sooner; reduced perfusion heightens sensitivity; heightened sensitivity promotes protective tone, which then tightens interfaces further [17,18,22–24,30,31,45]. Clinically, patients report “burning” or “sharp pull” that eases rapidly when load is removed (e.g., ankle plantarflexion in SLR), consistent with perfusion-sensitive nociceptor behavior rather than inertia of a dense connective-tissue stretch [33–37].

2.3.5 Neuroimmune chemistry: the low-grade inflammatory amplifier

Low-grade inflammation—whether triggered by microtrauma, posture-related stress, or local irritation—modifies both mechanical and electrophysiological properties. Schmid et al. demonstrate that inflammatory mediators can increase neural mechanosensitivity, making modest deformation feel threatening [18,38,45]. In perineural tissues, inflammatory chemistry promotes stiffness and adhesion formation, reducing shear planes that normally facilitate sliding [18,20]. Over time, this adds a chemical amplifier to a mechanical bottleneck: the threshold for nociceptor firing falls, and the cost (in symptoms) of a given degree of motion rises. At the broader pain-system level, such sensitization aligns with concepts of central sensitization and nociplastic pain, where augmented nociceptive processing persists despite limited structural pathology [47–49,60–63,78,79]. That the inflammatory drive is subclinical (no gross swelling or redness) does not diminish its clinical impact; SNR thrives in precisely such gray zones.

2.3.6 Sensorimotor consequences: why protective tone persists

Input from mechanosensitive afferents feeds into spinal and supraspinal circuits, shaping motor output. When neural tissues signal load too readily, the central nervous system selects protective strategies: earlier hamstring recruitment, gluteal co-contraction, and trunk bracing to limit further neural deformation [22–24,30,31,41,42,50,58–59,80–83]. Hodges and colleagues have shown how pain and perceived threat reconfigure trunk muscle recruitment, often yielding stiffness at the

expense of movement variability [24,50,83]. Brumagne et al. report proprioceptive deficits in CLBP—altered use of sensory inputs that impairs postural control—likely exacerbated when neural tissues are load sensitive and movement is curtailed [22,30,84,85]. The result is a loop: neural load sensitivity → protective tone → interface stiffness → reduced sliding → earlier load sensitivity. Breaking this loop requires peripheral (glide restoration) and central (motor control and confidence) strategies [30,41,42,58–59,67–70,80–87].

2.3.7 Central modulation: nociception, meaning, and behavior

At the supraspinal level, pain is constructed from nociceptive input plus context, beliefs, and prior experience. Moseley's work highlights how meaning and expectation alter pain perception and motor planning [25]. Vlaeyen and Linton describe the fear-avoidance model, in which threat interpretations lead to avoidance and deconditioning, creating more opportunities for tissues to become stiff and load-sensitive [26,100]. In SNR, predictable within-session modulation (e.g., symptoms ease with ankle plantarflexion) becomes a powerful educational lever: it demonstrates reversibility and control, countering threat and facilitating graded exposure [25,26,33–37,39–41,64–66,71,72,74–76,95,96]. This sits comfortably within contemporary frameworks for nociplastic pain and precision rehabilitation, which emphasize targeted education, exposure, and self-management [47–49,60–63,78–80].

2.3.8 The role of rate, repetition, and duration: viscoelastic time constants

Neural and perineural tissues are viscoelastic. Two practical corollaries follow:
– Rate matters. Rapid "yanks" produce high stress peaks and spike intraneural pressure; slow, even arcs reduce peak stress and afferent barrage [15–18,27,28,41,46].
– Time under load matters. Prolonged holds invite creep (time-dependent deformation) in surrounding structures but may exceed tolerable perfusion limits in the nerve if performed early in irritable states. This is why sliders—short, smooth excursions that share motion—are better tolerated initially than long static end-range holds [1–3,33–37,39–41,51–53,71,72,74–76].

Dilley's experimental work supports the importance of loading history (hysteresis) in neural tissues: what you just did influences what happens next [27,41,46]. Clinically, adhering to the 24-hour rule (down-dose if soreness >3/10 or persists to the next day) respects these time constants and keeps patients in a constructive adaptation window [1–3,39–41,51–53,71,72,74–76].

2.3.9 Posture and movement ecology as mechanistic drivers

Daily life delivers thousands of micro-loads. Prolonged sitting places the posterior chain in a shortened or compressed configuration; small changes in chair height, hip angle, and break cadence alter the cumulative load experienced by the sciatic and cluneal pathways [19–21,31]. Repeated forward flexion without hip hinge increases demand on posterior sliding; repeated standing without dynamic gluteal activity may stiffen posterior iliac crest fascia over time [20,21,30,31,41,42]. When posture and

movement ecology favor stiffness, the nervous system gets fewer chances to glide and more chances to strain. Installing micro-releases (brief sliders or ankle plantarflexion cycles every 60–90 minutes) is therefore a mechanistic intervention, not a generic ergonomic tip [19,30,33–37,39–42,67–70].

2.3.10 Why ordinary neuroimaging can look normal

SNR often evades detection on conventional imaging: there may be no large disc protrusion, no frank nerve root compression, and no signal changes that meet pathologic thresholds. The problem lies in function—reduced sliding, altered perineural viscosity, and perfusion-sensitive nociception—not in gross structure visible on routine MRI or CT [4–6,55–57,60–63,77–79]. Emerging methods (e.g., ultrasound measures of excursion) can sometimes objectify glide differences before and after a brief slider block, but such tools are not required for clinical reasoning; behavioral modulation during SLR/Slump/PKB remains the most practical, sensitive probe of neurodynamic function [33–37,39–41,41,46,74–76].

2.3.11 The hamstring “tightness” illusion

A frequent clinical puzzle is the patient with “tight hamstrings.” If sensitizers change the symptom angle/intensity in SLR—worse with ankle dorsiflexion or cervical flexion, better with plantarflexion or cervical extension—the limitation is unlikely to be pure muscle shortness. Instead, the system is entering strain dominance early because sliding is restricted [1–3,13–18,27,28,33–37]. Aggressive static hamstring stretching in this context often flares irritability; sliders and interface de-loading usually improve tolerance first. Only after glide is restored should longer holds be layered in, if needed [33–37,39–41,51–53,71,72,74–76].

2.3.12 Sympathetic tone and mechanosensitivity

Elevated sympathetic arousal increases muscle tone, alters local perfusion, and can heighten the gain on mechanosensitive afferents. This state makes patients more vulnerable to rate and amplitude errors during testing or exercise [17,18,27,28,60–63,78,79]. Simple strategies—slow cadence, breathing with exhalation during glides, and predictability in dosing—modestly lower sympathetic tone and thus reduce the chance of flares. This is one reason the education piece (explaining modulation and control) is mechanistically helpful: it reduces perceived threat and sympathetic drive, indirectly improving glide tolerance [25,26,33–37,39–41,64–66,95,96,100].

2.3.13 From peripheral mechanism to clinical signature

The clinical signature of SNR arises directly from these mechanisms:

– Early symptom onset in biased tests because the system reaches the steep portion of the stress–strain curve too soon.

– Predictable modulation with sensitizers because distal and proximal joints alter how load is shared across the neural continuum.

– Rapid easing when load is reduced, consistent with perfusion-sensitive behavior rather than slow viscoelastic recoil.

– Interface tenderness at bottlenecks—deep gluteal region, posterior iliac crest, or iliopsoas corridor—consistent with local mechanical contributors.

– Normal neuro exam because conduction is intact; the issue is mechanobiologic sensitivity, not axonal failure [5,6,11,20,21,31,33–37,39–41,88–92].

2.3.14 Central reinforcement: planning, prediction, and practice

Once peripheral signals are repeatedly labeled "threat," supraspinal systems adapt. Motor planning prioritizes protective patterns; attention is drawn toward the anticipated pull or sting; avoidance reduces exposure to healthy glide and increases exposure to static stiffness. Moseley emphasizes that reconceptualizing pain and demonstrating controllability can reverse these trends; the within-session test → intervene → re-test loop is therefore both diagnostic and therapeutic, recalibrating predictions through lived experience [25]. Over time, graded exposure that starts with glide and layers function re-teaches the nervous system that movement can be safe, further lowering mechanosensitive gain [1–3,25,26,33–37,39–41,51–53,64–66,71,72,74–76,80–83,95,96,100].

2.3.15 Why sliders first (and how they work)

Sliders emphasize movement sharing across joints and segments, using small, smooth arcs that promote longitudinal and transverse excursion without sustained end-range strain. Mechanistically, they:

– Restore shear at perineural planes, improving sliding economy;

– Reduce peak intraneural pressure, supporting perfusion;

– Provide graded afferent exposure that recalibrates thresholds without flaring;

– Demonstrate controllability through predictable symptom changes.

Only when irritability is repeatedly green under the 24-hour rule should tensioners be trialed, and then at low dose, because they increase the proportion of true elongation within the load mix [1–3,33–37,39–41,51–53,71,72,74–76].

2.3.16 The protective-tone paradox

Protective tone helps today (it reduces felt neural deformation), but it harms tomorrow (it stiffens interfaces that enable sliding). This paradox explains why short-term rest or rigidity feels relieving yet predicts longer-term sensitivity. A mobility-savvy plan therefore respects today's irritability (no yanking) while introducing tiny, smooth glides that keep the system from collapsing into the tone–stiffness loop. Integrating motor control (hip hinge, neutral pelvis) redistributes load over larger kinematic fields, reducing recurrent neural hotspots [22–24,30,31,39–42,50,67–70,80–83].

2.3.17 A mechanistic look at common tests

– SLR (sciatic bias). Hip flexion increases posterior chain load. If deep gluteal/hamstring interfaces are stiff, longitudinal sliding is reduced; ankle dorsiflexion and cervical flexion further increase neural load, worsening familiar symptoms; plantarflexion or neck extension eases them [1–3,20,21,31,33–37,39–41].

– Slump (dural bias). Spinal and cervical flexion tighten the cranio-caudal envelope; knee extension then expresses load along the posterior chain. Reversing cervical flexion should ease symptoms if neural load is the driver [1–3,33–37,39–41].

– PKB (femoral bias). Knee flexion (often with slight hip extension) loads the anterior pathway; iliopsoas stiffness reduces transverse space. Symptom change with hip or cervical adjustments confirms a neural component [32,37,39–41].

The behavior—onset angle, intensity at a matched angle, and direction of modulation—matters more than raw flexibility [33–37,39–41].

2.3.18 The role of proprioception and balance

Brumagne and colleagues highlight how CLBP involves altered proprioceptive integration [22,30,84,85]. If nerves are load-sensitive, the nervous system receives noisy afferent input from positions that should be neutral; in response it increases co-contraction and reduces movement variability, which further reduces glide exposure and sustains the problem [22–24,30,31,50,80–83]. Incorporating proprioceptive retraining (e.g., single-leg balance, gentle dynamic stabilization) alongside sliders provides cleaner sensory input and encourages the motor system to release protective bracing that otherwise acts as a mechanical tether [30,31,39–42,50,58–59,67–70,80–87].

2.3.19 “Normal imaging, real problem”: reconciling the paradox for patients

Patients often struggle with the “normal MRI” message: “If nothing’s wrong, why does it hurt?” SNR offers a clear answer: “Your nerves move as well as conduct. Right now, they’re not sliding well, so they feel load too soon. That’s why small changes at the ankle or neck change your symptoms—because they change how the load is shared.” This explanation ties mechanism to experience, turning modulation into a proof-of-concept that builds buy-in for home dosing [25,26,33–37,39–41,55–57,60–63,77–79,95,96].

2.3.20 Measuring what matters: mechanism-linked anchors

Because SNR is fundamentally behavioral, measurement should reflect the mechanism:

– Angle at first familiar symptom in SLR/PKB;

– Slump intensity (0–10) at a matched knee angle;

– Direction of sensitizer modulation (worse/better/none);

– Interface tenderness/tolerance (0–10) [33–37,39–41,88–92].

Between-session changes consistent with improved sliding include later onset angles (+10–15° common in responders), lower symptom intensity at matched angles (≥2/10 reduction), and stable 24-hour responses—all in parallel with patient-important changes (NPRS, ODI, PSFS) [3,39–41,51–53,71,72,74–76,93–99].

2.3.21 Boundaries and differentials (mechanistic nuance)

Mechanisms help with differentials:

– Radiculopathy with objective neuro deficit indicates axonal compromise at the root; neurodynamic strategies can be adjunctive for comfort but are not the primary lever [5,6,11,41,77].

– Facet/SIJ-dominant presentations show poor neurodynamic modulation and clearer segmental mechanical signatures; address segmental issues first [4–6,30,41,42,55–57,77].

– Pure myofascial shortness presents as end-range stretch without neurodynamic modulation; static stretching can be primary once SNR is ruled out [33–37,39–41,51–53,71,72,74–76].

– Nociplastic dominance shows widespread tenderness and poor load–symptom coupling; maintain gentle, predictable glides as graded exposure but emphasize education, pacing, sleep, and cognitive strategies [25,26,47–49,60–63,78,79,95,96,100].

2.3.22 Practical implications drawn from mechanism

1.Glide before tension. Start with sliders to lower peak stress and support perfusion; add tension only after multiple green 24-hour responses [1–3,33–37,39–41,51–53,71,72,74–76].

2.Treat interfaces. Brief manual or self-release at deep gluteal/hamstring, posterior iliac crest, or iliopsoas corridors often yields immediate test improvements—evidence of restored transverse and longitudinal motion [20,21,31,39,40,88–92].

3.Control rate and rhythm. Slow, smooth arcs; pair with exhalation to reduce sympathetic tone and mechanosensitive gain [15–18,27,28,41,46].

4.Shape movement ecology. Install micro-releases in sedentary blocks; coach hip hinge to distribute load; encourage gentle hip extension within tolerance during gait [19–21,30,31,39–42,67–70].

5.Educate with proof. Use the test → intervene → re-test loop to show reversibility and build adherence; link improvements in onset angle/intensity to daily strategy [25,26,33–37,39–41,64–66,95,96,100].

2.3.23 Two extended clinical vignettes (mechanism in action)

Vignette A — Posterior chain with high irritability.
A 55-year-old warehouse worker reports CLBP and posterior thigh ache worsened by sitting and lifting. SLR reproduces the familiar ache at 50°; ankle dorsiflexion worsens, plantarflexion eases; cervical flexion worsens, extension eases. Deep gluteal palpation is tender. Neurology is normal. Mechanism: limited longitudinal/transverse sliding in the deep gluteal corridor; left-shifted stress–strain; perfusion-sensitive nociceptor gain [20,21,31,33–37,39–41,88–92]. Intervention: high-irritability micro-sliders (8–10 tiny reps), slow cadence with exhalation, brief gluteal interface work, seated micro-releases every 60–90 minutes. Result: within session, onset to 60° and −2/10 intensity; 24-hour response clean. Week 2 adds gentle hip-hinge drills and proprioception. Week 4: SLR onset 70–75°, improved PSFS; patient demonstrates confidence adjusting ankle/neck to manage flare [3,22–24,30,39–42,50,58–59,67–70,93–99].

Vignette B — Anterior pathway with moderate irritability.
A 38-year-old office worker with CLBP struggles to stand upright after sitting. PKB

provokes proximal anterior thigh discomfort at modest angles; slight hip extension worsens; cervical extension eases; iliopsoas corridor is tender. Neurology is normal. Mechanism: femoral-pathway sensitivity with iliopsoas interface stiffness limiting transverse motion; perfusion-sensitive nociception in anterior corridor [20,21,31,32,37,39–41,88–92]. Intervention: femoral sliders in side-lying, short-arc knee flexion with hip near neutral, graded iliopsoas off-loading and desk posture tweaks (hip slightly open). Result: immediate PKB intensity drop; 24-hour response green. Weeks 2–3: add pelvic control and short bouts of gait emphasizing small hip extension; by week 4: better stand-to-walk transitions and reduced daily symptoms [3,22–24,30,39–42,50,58–59,67–70,93–99].

2.3.24 A concise mechanistic model for SNR

1.Trigger/load bias (posture, repetition, microtrauma) →

2.Interface change (tone, fascial thickening, minor adhesions) →

3.Reduced sliding / early strain dominance →

4.Microvascular challenge + mechanosensitive gain (per Schmid, Bove/Light) [17,18,27,28,38,45] →

5.Protective motor strategies (Hodges; Brumagne) with proprioceptive drift [22–24,30,31,50,58–59,80–87] →

6,Further interface stiffness and movement avoidance (fear-avoidance, Moseley; Vlaeyen & Linton) [25,26,47–49,60–63,78,79,100] →

7.Persistent load-sensitive pain despite normal imaging/neurology [4–6,55–57,60–63,77–79].

Interventions that restore glide, improve interface compliance, and rebalance motor control interrupt this loop and shift the system back toward glide-dominant function [1–3,33–37,39–42,51–53,67–70,71,72,74–76,93–99].

2.3.25 Summary

SNR does not require a large lesion or overt neuropathy. It emerges from a convergence of small mechanical and chemical shifts: restricted sliding, early strain dominance, microvascular stress, neuroimmune sensitization, and protective motor strategies. Bove and Light show that minor restrictions can amplify afferent signaling [17,28]; Schmid et al. detail how inflammation stiffens perineural tissues and lowers thresholds [18,38,45]; Dilley's work illustrates rate and repetition effects in neural mechanics [27,41,46]. Brumagne and colleagues link altered proprioception to motor control changes in CLBP [22,30,84,85], while Hodges describes protective trunk strategies that become maladaptive [24,50,83]. Within this mechanistic frame, SNR's clinical signature—predictable modulation of familiar symptoms in SLR/Slump/PKB

with a normal neurological exam—makes sense, as do the observed benefits of sliders, interface de-loading, irritability-based dosing, and education that demonstrates controllability [1–3,5–6,11,13–21,27–28,31,33–42,46,51–53,64–66,71,72,74–76,77–79,88–92,93–99,100]. Because daily posture and movement ecology continually shape neural mechanics, installing micro-releases and hinge-based task form is not ancillary; it is core mechanism-aligned care [19–21,30,31,39–42,67–70]. In short, SNR is the mechanobiologic expression of a nervous system that still conducts well but moves poorly; restoring movement—carefully, predictably, and progressively—is therefore the most rational way to change its behavior.

2.4 Clinical Relevance in CLBP

In clinical practice, subclinical neurodynamic restrictions (SNR) present as a coherent cluster of findings that implicate impaired neural mobility—reduced longitudinal sliding, diminished transverse excursion, and early transition to strain-dominant loading—in the genesis and persistence of chronic low back pain (CLBP). Clinicians typically encounter the following manifestations:
-Diffuse or localized pain: Patients report persistent low back or lumbopelvic pain that worsens with specific movements or postures—classically forward bending, prolonged sitting, or start-up after inactivity [4–6,30,33]. The quality is often “deep” or “aching,” without a strict dermatomal distribution [22,30,34].
-Muscle tightness: Chronic hypertonicity in paraspinals, gluteals, or hamstrings can reflect protective motor responses to perceived neural tension, with guarding that limits hip flexion and other ranges [22–24,26,30–31,50,83].
-Movement asymmetries: Subtle but consistent limitations in hip or lumbar mobility during functional tasks (squatting, walking, sit-to-stand) indicate restricted neural excursion and compensatory strategies, such as spine-dominant bending [22,30,38,50,58–59].
-Hypersensitivity: Localized tenderness along plausible neural pathways—sciatic in the deep gluteal corridor, femoral in the iliopsoas corridor—suggests mechanosensitivity unaccompanied by overt neuropathic signs [18,34,38,45,60–63].

These features often persist despite otherwise rational programs aimed at muscle strength, joint mobility, or psychosocial contributors, implying that neural mechanics may be an overlooked driver in a subset of CLBP presentations [4–6,43,55–57,77]. Addressing subclinical restriction provides a testable, low-risk lever to interrupt pain–protection cycles.

2.4.1 Phenotypic presentation and load coupling

SNR is characterized by load-coupled symptoms that appear reliably under specific mechanical contexts. Patients frequently describe a familiar ache or pulling that escalates during prolonged sitting, forward flexion, or the first steps after rising, and diminishes with brief positional changes or short bouts of ambulation. Unlike radiculopathy, symptoms are non-dermatomal and typically activity-contingent; routine neurological examination is normal [4–6,33–36,55–57,77]. The clinical hallmark is predictable modulation in neurodynamic testing when sensitizers are

applied in a standardized sequence. In a posterior-chain presentation, for example, ankle dorsiflexion and cervical flexion aggravate, while plantarflexion and cervical extension ease [33–36]. Directional predictability indicates that neural load sharing, rather than isolated myofascial shortness or articular restriction, is materially involved [1–3,33–37,39–41].
A consistent temporal pattern is also common: mornings are tolerable; cumulative sitting or repeated forward-reaching increases discomfort; periodic positional "resets" reduce symptoms. Patients adapt by shortening stride, avoiding terminal hip extension, or substituting lumbar flexion for a hip hinge. Such adaptations accord with the mechanistic account in §§2.1–2.3: limited sliding/excursion shifts ordinary tasks prematurely into strain-dominant loading, prompting protective tone and movement simplification [13–18,22–24,30–31,50,58–59,80–83].

2.4.2 Functional consequences and movement ecology

Functionally, SNR narrows the available movement envelope. In sitting, posterior-chain tension accumulates with time, heightening mechanosensitivity. During forward bending, individuals favor spine-dominant patterns that curtail demands on sciatic excursion but increase segmental strain [22–24,30–31,50,80–83]. Gait changes—shortened stride and avoidance of terminal hip extension—reduce neural demand acutely yet perpetuate stiffness around the iliopsoas corridor, reinforcing femoral-biased complaints [31–32,37–41]. Over time, this movement ecology produces deconditioning of efficient strategies (e.g., hip hinge), persistent co-contraction, and recurrent symptom provocation. It also explains why strengthening or mobility programs that do not normalize neural excursion may deliver suboptimal outcomes despite apparent biomechanical logic [24,30,41–43,50,67–70].

2.4.3 Differential diagnosis and decision boundaries

A structured differential prevents misclassification:

-Radiculopathy: Dermatomal pain with neurological deficits (myotomal, sensory, reflex) suggests root involvement; neurodynamic tests may be positive, but deficits re-prioritize radicular pathways. SNR methods can be adjunctive only if deficits are absent or resolving [5–6,41,77].

-Facet or sacroiliac joint–dominant pain: Segmental provocation (extension/rotation) and local tenderness, with weak or inconsistent change to neurodynamic sensitizers, support an articular primary [4–6,30,41–42,77].

-Myofascial shortness without neural contribution: Firm end-feel limitations that do not change predictably with ankle or cervical sensitizers argue against a neural mechanism; here, end-range stretching becomes more appropriate once SNR is excluded [33–37,39–41,51–53,71,72,74–76].

-Nociplastic tilt: Widespread tenderness, poor sleep, and weak load–symptom coupling suggest central augmentation; graded exposure, education, and sleep measures lead, with neural excursion serving as controlled exposure rather than as a principal lever [25–26,47–49,60–63,78–79,95–96,100].

Where symptom-first endpoints on neurodynamic testing show predictable sensitizer modulation and routine neurology is normal, an SNR-focused plan is justified [11,33–37,39–41].

2.4.4 Structured assessment and documentation

Standardization enhances reliability and interpretability:

1.Routine neurology (sensation, myotomes, reflexes) is expected to be normal in SNR [5–6,77].

2.Neurodynamic tests with symptom-first endpoints:

3.SLR (sciatic bias): Record the angle at first familiar symptom; apply ankle dorsiflexion/plantarflexion and cervical flexion/extension; note direction and magnitude of change [33–36].

4.Slump (dural bias): With spinal/cervical flexion pre-loaded, use knee extension as the driver; document intensity at a fixed knee angle and modulation with ankle/cervical adjustments [33–36].

5.PKB (femoral bias): Record knee-flexion angle at onset; examine effects of slight hip extension (often aggravating) and cervical extension (often easing) [32,37].

6.Interface palpation: Focal tenderness in deep gluteal (sciatic), posterior iliac crest (superior cluneal), or iliopsoas corridor (femoral) often reproduces familiar symptoms [20–21,31,40,88–92].

7.Functional sampling: Hip hinge, sit-to-stand, step-downs, and gait observation reveal compensations (spine-dominant bending, reduced hip extension, guarded transitions) [22,30,38,50,58–59,80–83].

8.Test–intervention–retest loop: Deliver a brief, low-amplitude neural excursion in the implicated bias and/or limited interface de-loading; immediately repeat the index test, recording the shift in onset angle and intensity at a matched angle. Within-session change corroborates mechanism and guides initial dosing [1–3,39–41,51–53,71,72,74–76].

Documentation should prioritize behavioral markers—onset angle, matched-angle intensity, sensitizer directionality—over absolute end-range. These markers are sensitive to early improvement and connect intervention to mechanism [33–37,39–41].

2.4.5 Lumbopelvic interfaces as practical levers

Although SNR is a property of neural tissues, interface structures (osseofascial tunnels, myofascial planes) frequently provide clinical leverage:

-Deep gluteal corridor (sciatic bias): Hypertonic deep rotators or fibrotic bands near the greater sciatic notch constrain transverse glide. Indicators: early SLR onset with posterior-chain quality, predictable sensitizer effects, deep gluteal tenderness, shortened stride [20–21,31,33–36,40,88–92].

-Posterior iliac crest (superior cluneal interface): Fascial tunnels over the crest can tether cluneal branches. Indicators: posterior pelvic ache aggravated by standing, focal tenderness just superior-lateral to the PSIS, Slump positivity with expected modulation [21,40,88–92].

-Iliopsoas corridor (femoral bias): The femoral nerve's course across iliopsoas and beneath the inguinal ligament is vulnerable to pressure and stiffness. Indicators: PKB-provoked anterior thigh/lumbopelvic ache, worsening with hip extension, easing with cervical extension, corridor tenderness, start-up discomfort [32,37,39–41].

Targeted, symptom-limited interface de-loading combined with bias-appropriate neural excursion frequently yields within-session changes in neurodynamic behavior, supporting a reversible mechanical component [39–41,51–53,71,72,74–76].

2.4.6 Dosing principles governed by irritability

Dose progression is governed by irritability and the 12–24 h response:

-High irritability: Small-amplitude, slow excursion (8–10 repetitions, 1–2 sets) within non-provocative arcs; very brief interface de-loading; re-test after 60–120 seconds; progress only after repeated green next-day responses [1–3,39–41,51–53,71,72,74–76].

-Moderate irritability: Modest increases in arc or repetitions; add motor control (partial-range hip hinge, pelvic control) within non-provocative zones; introduce minimal tension components only after consistent tolerance.

-Low irritability: Functional integration (hip-hinge substitution in daily tasks; graded hip extension in gait), proprioceptive challenges, and rare, symptom-limited tensioners to consolidate tolerance rather than chase flexibility [30,41–42,67–70].

This sequence respects intraneural perfusion, viscoelastic constraints, and interface contributions discussed in §2.3 [1–3,15–18,27–28,39–41,51–53,71,72,74–76].

2.4.7 Outcomes: pairing patient-important and mechanism-linked indices

A dual-track strategy aligns clinical relevance with mechanistic coherence:

1.Patient-important: NPRS, ODI, and PSFS items aligned with lived roles (sitting 45–60 minutes, reaching tasks, walking 20–30 minutes) [93–99].

2.Mechanism-linked: shifts in onset angle (SLR/PKB), reduced intensity at matched angle (Slump), direction/magnitude of sensitizer effects, and interface tenderness [33–37,39–41,88–92].

Within 2–6 weeks, typical progress includes a later symptom onset by ~10–15°, a ≥2/10 drop in Slump intensity at matched angles, durable green next-day responses, and improved PSFS items. Divergence between tracks prompts targeted audits (technique, dosage, movement ecology) [3,39–41,93–99].

2.4.8 Communication and education aligned to mechanism

Education is concise and mechanism-congruent: nerves must slide and share load with interfaces; predictable changes with ankle and cervical positions indicate controllability; dosing is governed by next-day tolerance, not by maximal range [25–26,33–36,39–41,64–66,95–96,100]. Framing motor control as task substitution—hip hinge for forward flexion in everyday actions—improves adherence while directly reducing unnecessary neural strain [24,30,41–42,50,67–70,80–83].

2.4.9 Contextual modifiers and special populations

-Adolescents/young adults: Rapid growth reduces slack and exaggerates early symptom onset in SLR/Slump without pathology. Emphasize low-amplitude excursion and frequent posture variation during study [33–36].

-Pregnancy/postpartum: Hormonal and postural shifts alter interface compliance and venous return. Symptom-first endpoints remain valid; progressions are slower; use positional supports and avoid high-tension strategies early [1–3,39–41].

-Older adults/metabolic comorbidity: Glycation-related stiffness and microvascular changes increase pressure sensitivity; extend observation windows and progress amplitude cautiously; integrate balance demands gradually [15–18,27–28,30,41–42].

-Athletes/manual workers: Repetitive end-range exposure accumulates interface stiffness; pair neural excursion with task-specific motor control (hip-hinge for lifts; drills restoring hip extension in gait) to reduce recurrence [30–31,39–42,67–70].

2.4.10 Implementation in time-limited or hybrid care

SNR care fits brief-visit formats using short test–intervention–retest loops. A minimal sequence—identify the most positive neurodynamic finding (symptom-first endpoint), deliver 60–120 seconds of bias-appropriate excursion or interface de-loading, re-test—produces actionable feedback. Electronic record templates capturing onset angle, matched-angle intensity, sensitizer effects, interface findings, and 24-hour response improve reproducibility and enable audit [33–41,51–53,71,72,74–76]. Remote or hybrid care can safely incorporate self-applied sensitizers and explicit abort criteria (e.g., stop with lancinating pain; stop if symptoms persist after releasing sensitizers) with careful monitoring of next-day behavior prior to progression [33–37].

2.4.11 Methodological considerations for reliable practice

Reliability depends on several elements:

-Endpoint definition: Use the first familiar symptom rather than end-range to reduce examiner dependence and clarify sensitizer effects [33–37].

-Sensitizer sequencing: Pre-specify order (e.g., ankle, then cervical) and record direction/magnitude to minimize order effects [33–36].

-Rater calibration: Brief, periodic checks of patient position, goniometer alignment, and cueing mitigate rater drift.

-Observation windows: In high irritability or comorbidity, prioritize 24-hour behavior as the safety governor [15–18,27–28,39–41].

-Co-intervention control: Re-test after the most mechanistically central component to preserve causal attribution when multiple modalities are employed.

-Transparent reporting: Pair NPRS/ODI/PSFS with mechanism-linked anchors so the theory of change remains visible across sessions [3,33–41,93–99].

2.4.12 Illustrative vignettes

Posterior-chain SNR with sedentary provocation.
A 46-year-old accountant develops deep posterior thigh–low back ache after ~25 minutes of sitting, relieved by standing. Neurology is normal. SLR elicits familiar ache at 56°; dorsiflexion and cervical flexion aggravate; plantarflexion and cervical extension ease. Deep gluteal palpation reproduces familiar symptoms. After 90 seconds of low-amplitude posterior-chain excursion and short deep-gluteal de-loading, SLR onset improves to 67°, and Slump intensity at a matched knee angle falls. A home plan prescribing brief excursions during prolonged sitting and hip-hinge substitution for forward reaches yields progressive onset shifts and PSFS improvement over 3–4 weeks [31,33–36,39–41,51–53,67–70,71,72,74–76,93–99].
Femoral-biased SNR with start-up pain.
A 39-year-old software engineer reports anterior thigh/lumbopelvic discomfort on rising and during brisk walking. PKB provokes a familiar ache at modest knee flexion; slight hip extension aggravates; cervical extension eases. Iliopsoas corridor is tender. Side-lying femoral-biased excursion and corridor de-loading reduce PKB intensity within session; graded hip-extension drills during walking normalize start-up within 4 weeks with parallel improvements in PSFS and PKB behavior [32,37,39–41,51–53,67–70,71,72,74–76,93–99].
Posterior iliac crest interface with standing intolerance.
A 34-year-old clinician experiences posterior pelvic ache during long standing. Palpation at the posterior iliac crest reproduces familiar symptoms; Slump is positive with expected modulation. Local crest de-loading and posterior-chain excursion reduce Slump intensity acutely; standing tolerance extends to 45–60 minutes over 3 weeks, with reduced corridor tenderness [21,40,88–92].

2.4.13 Service-level, economic, and policy implications

SNR-oriented care is low-risk and equipment-light, consistent with guideline-concordant pathways for non-specific CLBP when imaging is unrevealing and routine neurology is intact [4–6,55–57,77]. Because it demonstrates within-session changes in mechanism-linked markers, it can reduce demand for repeat imaging and support stepped-care models. Multidisciplinary programs assign roles by mechanistic leverage: manual therapists target interface de-loading when indicated; movement professionals operationalize hip-hinge substitution and graded hip extension; education reinforces controllability and dose governance [20–21,24,30,39–43]. At the system level, documenting mechanism-linked progress (later symptom onset, reduced matched-angle intensity) alongside PSFS gains supports return-to-work planning that is function-centric and graded [3,39–41,93–99].

2.4.14 Limitations and boundary conditions

SNR constitutes one contributor among many in CLBP. Where segmental signs dominate, radicular deficits emerge, or nociplastic features predominate, SNR strategies may be adjunctive rather than central [4–6,25–26,47–49,60–63,78–79,77]. While advanced imaging and ultrasound can quantify sliding or excursion, behavioral surrogates—onset angle, matched-angle intensity, sensitizer directionality—remain the most feasible anchors for decision-making in routine practice. High-tension

strategies warrant caution; build excursion tolerance first under explicit 24-hour safety rules [33–37,39–41,46,51–53,71,72,74–76].

2.4.15 Patient selection, escalation triggers, and safety governance

Selection is grounded in three anchors: (a) symptom-first endpoints with predictable sensitizer modulation; (b) normal routine neurology; (c) interface concordance where palpation over a plausible corridor reproduces familiar symptoms or reveals focal tenderness [20–21,31,33–41,88–92]. If all are present, prioritize SNR-directed care. If only (a) is present, integrate SNR within multimodal management; if (a) is absent or neurology is abnormal, re-appraise for alternative primaries [5–6,77].
Escalation triggers include new neurological deficit, constitutional red flags, or intolerable exacerbation despite conservative dosing (e.g., next-day severity >6/10 persisting >48 h). If mechanism-linked markers fail to improve after 3–4 well-dosed sessions, institute a diagnostic pause: review test execution fidelity, dosing adherence, and daily movement ecology; consider adjunct evaluation as indicated [3,33–41]. Informed consent should specify expected sensations during testing, clear abort criteria, and the primacy of next-day behavior in governing progression [1–3,25–26,39–41].

2.4.16 Competency, audit, and continuous improvement

Minimum competencies include reliable administration of SLR/Slump/PKB with symptom-first endpoints; consistent capture of onset angles and matched-angle intensities; anatomically literate palpation of deep gluteal, posterior iliac crest, and iliopsoas corridors; and construction of brief test–intervention–retest loops [20–21,31–37,39–41,88–92]. Regular calibration (peer review of consecutive cases) mitigates rater drift. Audit dashboards track early-phase markers (≥10–15° delay in onset, ≥2/10 matched-angle intensity reduction, PSFS improvement) and consolidation markers (maintenance/progression with functional integration and sustained green next-day responses). Deviations trigger focused review before resource-intensive escalation [3,33–41,93–99].

2.4.17 Psychometrics and measurement precision in clinic

Although formal reliability estimates (e.g., ICC, SEM, MDC) are typically derived from dedicated studies, clinicians can approximate precision operationally by attending to three elements that are tractable in practice: (i) standardized patient positioning (pelvic neutral for SLR; consistent slump depth), (ii) device consistency (same goniometer/inclinometer and side-table height), and (iii) cue fidelity (identical instructions for “first familiar symptom”). Repeated within-session measures at matched angles (e.g., Slump intensity at 30° knee extension) serve as internal checks: reductions ≥2/10 coupled with stable neurology and green next-day responses are considered beyond noise and actionable [33–37,93–99]. Where services have the capacity, small blinded repeatability drills (two raters, same patient, 10-minute interval) can be embedded quarterly to monitor drift and reinforce technique.

2.4.18 Common pitfalls and how to avoid them

Five recurrent errors undermine fidelity:

1.Chasing end-range flexibility. End-range endpoints increase irritability and obscure mechanism. Use symptom-first endpoints and matched-angle intensities to guide care [33–37,39–41].

2.Unsequenced sensitizers. Random order produces ambiguous responses. Pre-specify ankle then cervical (or vice versa) and document direction/magnitude [33–36].

3.Over-reliance on manual techniques. Interface de-loading without neural excursion and task substitution yields transient relief but weak transfer. Pair interventions within the same session [39–41,51–53,71,72,74–76].

4.Ignoring the 24-hour governor. Progression absent next-day tolerance increases flares. Dose changes should be small, and progression should be contingent on green responses [1–3,39–41,51–53,71,72,74–76].

5.Under-documenting mechanism. Without onset angle and matched-angle intensity, improvement narratives drift to global impressions. Mechanism-linked markers keep the causal chain explicit [33–37,39–41,93–99].

2.4.19 Comorbidity and medication considerations

Vascular/metabolic comorbidities (diabetes, dyslipidemia) and medications that influence tissue compliance or pain processing can alter irritability. Where microvascular compromise is suspected, lower starting amplitudes, longer inter-set intervals, and conservative progressions are prudent [15–18,27–28,30,41–42,60–63,78–79]. Sedating agents may blunt symptom perception; in such cases, preference is given to objective behavioral markers (onset angle; matched-angle intensity) and functional anchors (PSFS), rather than subjective comfort alone [93–99]. Sleep disturbance, regardless of cause, is addressed early because it interacts with mechanosensitivity and central amplification [25,26,48,49,60–63,78–79,95–96,100].

2.4.20 Documentation template for mechanism-linked practice

A concise template supports reproducibility:

-Index test: SLR R/L onset ___°; Slump intensity at ___° knee extension ___/10; PKB onset ___°.

-Sensitizer sequence/results: Ankle dorsiflexion ↑/↓ by ___; cervical flexion/extension ↑/↓ by .

-Interface findings: Deep gluteal / posterior iliac crest / iliopsoas corridor tenderness Y/N; reproduction of familiar symptoms Y/N.

-Intervention (dose): Bias-appropriate excursion ___ reps × ___ sets; interface de-loading ___ s; motor task substitution specified.

-Retest: SLR onset shift +°; Slump matched-angle intensity −/10; PKB onset shift +°.

-24-h response: Green/amber/red; action taken (progress/hold/regress).

-Patient-important: NPRS, ODI, PSFS items with targets and dates [93–99].

2.4.21 Integration with existing guideline-concordant care

SNR-aligned practice complements rather than replaces guideline-concordant CLBP care [4–6,55–57,77]. Education (threat reduction, graded exposure), general activity resumption, and targeted strengthening proceed in parallel, with the neurodynamic thread ensuring that exercises do not inadvertently load neural tissues prematurely. For example, hip-dominant hinge drills substitute for spine-dominant reach tasks in early phases; walking cadence and step length are adjusted to avoid terminal hip extension while excursion tolerance is developed; lumbopelvic motor control is layered in once symptom-first endpoints improve and matched-angle intensities fall [24,30,39–43,50,67–70].

2.4.22 Practical algorithm (text description)

1.Screen: Red flags; routine neurology (if abnormal → alternate pathway) [5–6,77].

2.Test: SLR/Slump/PKB with symptom-first endpoints and standardized sensitizers; document direction/magnitude [33–37,39–41].

3.Localize: Interface palpation for concordant tenderness (deep gluteal, posterior crest, iliopsoas corridor) [20–21,31,40,88–92].

4.Decide: If symptom-first + predictable modulation + normal neurology ± interface concordance → SNR-primary; else multimodal with SNR as adjunct [11,20–21,31,33–41].

5.Treat: Low-amplitude bias-appropriate excursion + targeted interface de-loading; specify home dosage; task substitution (hip hinge; graded hip extension) [30,39–42,51–53,67–70,71,72,74–76].

6.Re-test: Onset angle, matched-angle intensity; record change [33–37,39–41,93–99].

7.Govern: Progress contingent on green 24-h responses; diagnostic pause at 3–4 sessions if mechanism markers stall; escalate on neurological change [3,5–6,33–41,77].

Clinical implication. When neurodynamic testing reveals symptom-first endpoints with predictable sensitizer effects, routine neurology is normal, and focal interface tenderness is present, SNR represents a treatable contributor to CLBP. Operationalizing care through standardized assessment, irritability-guided neural excursion, targeted interface de-loading, and task-level movement substitution enables safe, scalable implementation while maintaining fidelity to the neurodynamic construct [4–6,11,20–21,31,33–41,43,55–57,77].

2.5 Case Example: Subclinical Sciatic Nerve Restriction

Presenting complaint and background.
A 50-year-old secondary-school teacher presented with a 3-year history of non-specific chronic low back pain (CLBP). Symptoms began insidiously during a period of increased administrative workload requiring prolonged sitting (≥6 hours/day) and frequent forward-flexed postures while marking papers [4–6,30,33,55–57,77]. The pain was described as a deep, dull ache localized to the right lumbogluteal region with intermittent spread into the upper posterior thigh but without dermatomal distribution. There were no paresthesias, no perceived weakness, and no red-flag features (no constitutional symptoms, no history of malignancy, trauma, infection, bowel/bladder disturbance) [4–6,55–57,77]. Pain intensity fluctuated between 3/10 and 7/10 on the NPRS, with typical exacerbations after prolonged sitting (>30–40 minutes), forward bending to reach low shelves, and the first few steps after rising. Walking on level ground for 10–15 minutes reduced symptoms. Sleep was mildly fragmented by stiffness when turning. Analgesic use was intermittent (paracetamol as needed). The patient reported psychosocial stress related to deadlines but denied significant fear-avoidance; ODI baseline was 28% (moderate) [25–26,93–99].

Functional consequences.
The patient avoided long drives and limited household tasks requiring forward reach (laundry baskets, low cabinets). At work, he unconsciously adopted shorter stride length and occasionally leaned on desks to offload the right side. He had reduced tolerance for standing assemblies (>20–25 minutes), often shifting weight to the left. He reported reduced physical activity overall and deconditioning, consistent with adaptations commonly observed in CLBP with altered motor control and movement ecology [22–24,30–31,38,50,58–59,80–83].

Medical and social history.
No prior lumbar surgery. BMI 27. Non-smoker. Borderline dyslipidemia. No diabetes. No peripheral neuropathy risk factors. No lower-limb injuries in the past 5 years. No medications affecting connective tissue compliance. Work demands remained high; typical day alternated between teaching blocks (standing/walking) and seated marking.

Examination—screening and observation.
General observation showed guarded forward-bending strategy with early lumbar flexion and a reduced hip-hinge pattern. Gait revealed shortened stride and avoidance of terminal right-hip extension. Posture in sitting involved posterior pelvic tilt with a slumped thoracolumbar posture. No scoliosis or gross asymmetry. Neurological screen (myotomes, dermatomes, reflexes) was normal bilaterally [5–6,77]. Heel and toe walking were intact; no Trendelenburg sign.

Palpation and interface findings.
Palpation of the right deep gluteal corridor elicited familiar, non-radiating ache over the piriformis/deep rotator region with mild reproduction into the upper posterior thigh. Posterior iliac crest palpation was non-provocative. Iliopsoas corridor (anterior) was non-tender. These findings are compatible with interface-related contributions along the sciatic pathway rather than cluneal or femoral bias [20–21,31,39–41,88–92].

Neurodynamic testing (symptom-first endpoints, standardized sensitizers).

-Straight Leg Raise (SLR, right): Onset of familiar posterior thigh–gluteal ache at 60° hip flexion with knee extended and neutral ankle. Ankle dorsiflexion increased the ache; plantarflexion reduced it. Cervical flexion aggravated; cervical extension eased. Left SLR onset at 78° with only hamstring stretch, no familiar pain.

-Slump test: In seated slump with cervical flexion and knee extension, the right-sided familiar ache appeared at 30° of knee extension; easing cervical flexion reduced symptoms. Left side provoked only distal hamstring stretch without familiar symptoms.
-Prone knee bend (PKB): No anterior thigh pain; only quadriceps stretch at end range.

The pattern suggested a posterior-chain neural bias with directionally predictable sensitizer effects and normal neurology, consistent with a subclinical sciatic-biased neurodynamic restriction (SNR) rather than radiculopathy [1–3,5–6,11,13–18,27–28,31,33–37,39–41,46,51–53,71,72,74–76].

Lumbar motion and segmental tests.
Active lumbar flexion was limited by discomfort at mid-range with early lumbar contribution and reduced hip hinge. Extension was mildly restricted but less symptomatic. Passive accessory intervertebral motion tests were within expected limits for age with no segmental provocation. Sacroiliac joint provocation tests were negative, making segmental and SIJ-dominant pain less likely as primary drivers [4–6,30,41–43,77].

Imaging.
Lumbar MRI (obtained 6 months earlier for reassurance) showed age-expected degenerative changes without disc herniation, spinal canal stenosis, or nerve-root compression. No inflammatory findings. These results supported the clinical impression that structural compromise was not driving symptoms, in line with the frequent imaging–symptom discordance described in CLBP [4–6,55–57,77,80].

Differential diagnosis and clinical reasoning.
The non-dermatomal distribution, predictable modulation to ankle and cervical sensitizers, normal neurology, and focal deep-gluteal tenderness pointed toward SNR as an active contributor [1–3,11,20–21,31,33–41,51–54,71,72,74–76]. Radiculopathy was unlikely (no deficits, imaging unremarkable) [5–6]. Facet-dominant patterns were inconsistent with the patient's flexion-provoked symptoms and lack of segmental pain [4–6,30,41–43]. Pure myofascial shortness was insufficient to explain the consistent changes with sensitizers and asymmetry between sides [33–37,39–41]. A nociplastic tilt was not prominent given clear load–symptom coupling and good sleep aside from positional stiffness [25–26,47–49,60–63,78–79,95–96,100].

Hypothesized mechanism.
Restricted longitudinal sliding and transverse excursion of the right sciatic pathway—likely at the deep gluteal corridor—were thought to shift ordinary movements into strain-dominant loading earlier than expected, amplifying mechanosensitivity of intraneural nociceptors and prompting protective hamstring and gluteal co-contraction [13–18,20–21,27–28,31,41,46,50]. Prolonged sitting increased time under posterior-chain tension, perpetuating sensitivity and interface stiffness [19,30,33–36]. The clinical objective was to restore excursion tolerance first (sliders), reduce local interface loading (brief, symptom-limited de-loading), and re-educate daily movement (hip hinge; graded hip extension in gait), all under 24-hour response governance [1–3,22–24,30–32,39–43,51–54,67–70,71,72,74–76]. Mirroring changes reported in trials of neural mobilization for CLBP [3,39,51–54,71,72,74–76].

Management plan
Goals (12 weeks).
Reduce average NPRS from 5/10 to ≤2/10; 2) improve ODI from 28% to ≤14%; 3) increase right SLR onset from 60° to ≥80° with reduced symptom intensity at matched angles; 4) restore sitting tolerance to ≥60 minutes without flare; 5) normalize hip-hinge pattern in forward-reach tasks; 6) restore comfortable walking for ≥30 minutes with symmetrical stride [3,22–24,30,39–43,50,58–59,67–70,80–83,93–99].

Outcome metrics.

-Patient-important: NPRS, ODI, PSFS (1: sit and grade papers 60 min; 2: lift laundry with hip hinge; 3: walk 30 min) [93–99].

-Mechanism-linked: right SLR onset angle; Slump intensity at a matched knee angle (30°); direction/magnitude of sensitizer effects; deep-gluteal corridor tenderness (0–10) [33–37,39–41].

-Safety: 24-hour response coded green (settled/neutral), amber (elevated but settles <24 h), red (worsened >24–48 h) [1–3,39–41,51–53,71,72,74–76].

Phase 1 (Weeks 0–2): Establish excursion tolerance; reduce interface load
Clinic (2 sessions/week initially).

-Posterior-chain sliders (right): Supine, hip flexion/knee extension seesaw within symptom-first endpoint, avoiding sustained end-range. 8–10 reps × 2 sets, slow tempo, 60–90 s rest [1–3,33–37,39–41,51–53,71,72,74–76].

-Ankle/cervical sensitizer titration: Begin neutral; progress to gentle ankle dorsiflexion/plantarflexion coupling only if green next-day responses.

-Deep-gluteal de-loading: 60–90 s of low-amplitude soft-tissue work and positional unloading over the deep rotator region, symptom-limited, immediately followed by sliders (test–intervention–retest loop) [20–21,31,39–41,88–92].

-Motor control (hip hinge drill): Dowel-assisted hinge pattern from high blocks; range constrained to non-provocative arc; 2–3 sets of 6–8 quality reps [22–24,30,41–43,50,67–70,80–83].

-Education: Mechanism-congruent explanation; 24-hour rule; abort criteria (stop with lancinating pain; stop if symptoms persist after releasing sensitizers) [25–26,33–37,39–41,93–99].

Home (daily):

-Posterior-chain sliders: 8–10 reps × 2 sets, once daily; add a micro-set (5 reps) before and after prolonged sitting blocks [1–3,33–37,39–41,51–53,71,72,74–76].

-Sitting hygiene: Timer at 25–30 minutes for brief stand/walk; lumbar support to reduce sustained flexion [4–6,19,30,33–36,55–57,77].

-Walking: 10–12 minutes at comfortable pace once daily to utilize movement-induced analgesia and low-load excursion [22–24,30–31,39–43,67–70].

Expected early changes.
Increase in right SLR onset by ~5–10°; reduction of Slump matched-angle intensity by ≥1/10; improved comfort during first steps after sitting; green or mild amber 24-hour responses [3,33–37,39–41,51–53,71,72,74–76,93–99].

Phase 2 (Weeks 3–6): Consolidate excursion; integrate function
Clinic (weekly).

-Progress sliders: Expand arc modestly if green responses persist; continue symptom-first endpoints [1–3,33–37,39–41,51–53,71,72,74–76].

-Introduce minimal tension components (e.g., small end-range holds of ≤2 s at the limit of comfort) only if matched-angle intensities have fallen and SLR onset has improved by ≥10° [1–3,33–37,39–41,51–53,71,72,74–76].

-Interface work: Brief, symptom-limited treatment of deep gluteal corridor as needed [20–21,31,39–41,88–92].

-Task substitution: Hip-hinge integrated into real tasks (lifting light loads from mid-thigh height; reaching low shelves with hinge first/lumbar flexion second) [22–24,30,41–43,50,67–70,80–83].

-Gait drills: Gentle emphasis on symmetrical step length and graded restoration of terminal hip extension without symptom escalation [30–32,39–43,67–70].

-Proprioception: Single-leg balance 3 × 20–30 s/bilateral, ensuring non-provocative loading [22,30,41–42,50,84–87].

Home:
-Sliders on alternate days; hinge practice with household tasks; walking 15–20 minutes 4–5×/week; continue sitting breaks [22–24,30,39–43,67–70].

Progress criteria.
Right SLR onset ≥75°; Slump matched-angle intensity reduced by ≥2/10; PSFS gains (sitting 45–60 minutes; lifting light household items) [3,33–37,39–41,93–99].

Phase 3 (Weeks 7–12): Functional loading; self-management
Clinic (biweekly or discharge planning).

-Functional integration: Add light hip-dominant strengthening (e.g., elevated hip hinge with kettlebell 6–8 kg, 2–3 × 8) if green responses; introduce low-amplitude posterior-chain tensioners sparingly (≤5 reps) only in low-irritability states [1–3,22–24,30,39–43,50,67–70,71,72,74–76].

-Conditioning: Brisk walking 25–30 minutes; optional cycling with neutral lumbopelvic posture [22–24,30–31,39–43,67–70].

-Relapse prevention: Clear rules for dose adjustments during heavier weeks (marking periods), early re-initiation of slider micro-sets, and recognition of amber/red responses [25–26,33–37,39–41,93–99].

Home:
-Maintenance sliders 3–4×/week; hip-hinge patterns embedded in all forward-reach tasks; walking 30 minutes most days [22–24,30,39–43,67–70].

Test–intervention–retest data (selected)
Initial (Week 0):

-Right SLR onset 60° (neutral ankle, neutral cervical); dorsiflexion/cervical flexion ↑ symptoms; plantarflexion/cervical extension ↓ [33–37].

-Slump (right bias): familiar ache at 30° knee extension; intensity 5/10; easing cervical extension ↓ to 3–4/10 [33–37].

-Deep gluteal tenderness 6/10 [20–21,31,39–41,88–92].
-NPRS average 5/10; ODI 28%; PSFS (sit 60 min = 3/10; lift laundry with hinge = 4/10; walk 30 min = 5/10) [93–99].

After first session (immediate):

-Right SLR onset 66° following 90 s sliders + de-loading.

-Slump matched-angle intensity −1/10.

-Deep gluteal tenderness 5/10.

-24-h response green [1–3,33–37,39–41,51–53,71,72,74–76].

Week 3:

-Right SLR onset 75°; Slump matched-angle intensity 2–3/10; deep gluteal tenderness 3–4/10.

-NPRS average 3–4/10; PSFS sitting 45 min 6/10; gait more symmetrical.

-No adverse events [3,22–24,30,39–43,50,67–70,93–99].

Week 6:

-Right SLR onset 82°; Slump matched-angle intensity 1–2/10; deep gluteal tenderness 2/10.

-NPRS 2–3/10; ODI 18%; PSFS sitting 60 min 7/10, lifting 7/10, walking 30 min 8/10.

-24-h responses consistently green [3,22–24,30,39–43,50,67–70,93–99].

Week 12 (discharge):

-Right SLR onset 88–90° with minimal posterior-thigh stretch and no familiar ache; sensitizer effects attenuated and symmetrical to the left [33–37].

-Slump matched-angle intensity 0–1/10; deep gluteal tenderness 0–1/10.

-NPRS 1–2/10; ODI 12%; PSFS: sitting 60 min 8–9/10, hinge lifting 8/10, walking 30 min 9/10.

-Patient self-manages with maintenance sliders and task-level strategies [3,22–24,30,39–43,50,67–70,93–99].

Mechanistic interpretation

The within-session improvement in SLR onset and matched-angle reduction in Slump intensity following low-amplitude sliders plus interface de-loading supports the inference that reversible mechanical factors—reduced sciatic excursion and increased mechanosensitivity—were active contributors [1–3,13–18,20–21,27–28,31,33–37,39–41,46,51–53,71,72,74–76,88–92]. The attenuation of sensitizer effects over time suggests improved load sharing between neural tissues and adjacent interfaces, consistent with proposed viscoelastic and perfusion mechanisms (restored intraneural sliding reduces pressure spikes and ischemic sensitivity) [13–18,27–28,41,46]. Functional translation—restored hip hinge and graded hip extension—likely prevented recurrence by lowering repeated end-range loading of the implicated corridor during daily tasks [22–24,30–32,39–43,50,67–70,80–83].

Safety, adherence, and adverse events

No red-flag evolution occurred. The patient experienced two amber days early in Phase 2 after prolonged sitting without scheduled breaks; symptoms settled within 12–18 hours after temporarily reducing slider amplitude and increasing micro-sets. No red responses were recorded. Adherence was high (>85% by self-report), facilitated by pairing sliders with routine activities (before/after marking blocks) and by demonstrating immediate, measurable change during sessions (test–retest) [25–26,33–37,39–41,93–99].

Discussion

This case illustrates a sciatic-biased subclinical neurodynamic restriction in CLBP with: (i) non-dermatomal, load-coupled symptoms; (ii) predictable neurodynamic sensitizer modulation; (iii) normal neurology; and (iv) deep-gluteal interface tenderness. The management sequence prioritized symptom-first endpoints, low-

amplitude sliders, and task-level substitution before any tension-dominant techniques, under 24-hour safety governance [1–3,11,20–21,30–31,33–41,51–54,67–70,71,72,74–76,93–99]. The trajectory—early shifts in SLR onset and Slump matched-angle intensity, followed by PSFS and ODI gains—aligns with a mechanistic pathway in which restoring excursion tolerance reduces protective tone and permits re-normalization of efficient movement patterns [13–18,22–24,27–28,30–32,39–43,50,58–59,67–70,80–83]. While this is a single case and cannot establish causality at a population level, the coherence among assessment, mechanistic markers, and outcomes strengthens internal validity. It also exemplifies how an SNR-oriented approach can be integrated into guideline-concordant care for non-specific CLBP (education, activity resumption, graded strengthening) without increasing risk or resource burden [4–6,55–57,71,72,74–77].

Boundary conditions. If neurological deficits had emerged or if mechanism-linked markers failed to improve after 3–4 well-dosed sessions, a diagnostic pause with re-appraisal and potential referral would have been indicated [3,5–6,33–41,77]. Likewise, in patients with high irritability or metabolic comorbidities, longer observation windows and smaller progression steps would be necessary [15–18,27–28,30,41–42,60–63,78–79].

Practical template derived from the case

1. Identify: Load-coupled, non-dermatomal symptoms; normal neurology; positive neurodynamic tests with predictable sensitizer modulation [1–3,5–6,11,33–37,39–41].

2. Localize: Interface tenderness in a plausible corridor (here, deep gluteal) [20–21,31,39–41,88–92].

3. Intervene: Low-amplitude sliders → brief interface de-loading → immediate retest [1–3,33–37,39–41,51–53,71,72,74–76].

4. Educate: Mechanism-congruent explanation; 24-hour rule; abort criteria [25–26,33–37,39–41,93–99].

5. Integrate: Task substitution (hip hinge), graded restoration of hip extension in gait [22–24,30–32,39–43,50,67–70,80–83].

6. Govern: Progression contingent on green responses; hold/regress on amber; re-appraise on red [1–3,33–37,39–41,51–53,71,72,74–76].

7.Document: Onset angles, matched-angle intensities, sensitizer effects, PSFS; track ODI/NPRS [33–37,39–41,93–99].

Conclusion.
In this teacher with CLBP, a structured, mechanism-linked program targeting subclinical sciatic neurodynamic restriction produced meaningful improvements in pain, function, and mechanistic markers over 12 weeks. The case underscores the value of symptom-first endpoints, standardized sensitizer sequencing, interface-aware

interventions, and rigorous 24-hour safety governance in operationalizing neurodynamic care within everyday practice [1–3,4–6,11,13–21,27–28,30–32,33–43,50,58–59,67–70,71,72,74–77,80–83,88–92,93–99].

2.6 Case Example: Subclinical Cluneal Nerve Restriction

Presenting complaint and background.
A 42-year-old staff nurse presented with a 2-year history of focal posterior pelvic pain localised to the superolateral sacral crest, aggravated during long ward shifts that required static standing for medication rounds and patient observations. Pain was described as a deep, pinpoint ache with occasional "scratchy" sensitivity along the posterior iliac crest (PIC) radiating 3–5 cm laterally, without gluteal or thigh radiation. She denied paresthesia, dermatomal spread, or weakness. Symptoms intensified predictably after 20–30 minutes of quiet standing and eased with short walking bouts or sitting with lumbar support. Sleep was intact except for end-of-shift stiffness when turning in bed. No constitutional symptoms or bowel/bladder changes were reported [4–6,33–36,55–57,77].

Functional consequences.
Standing tolerance during 12-hour shifts had fallen from ~60–75 minutes to ~20–30 minutes. She increasingly leaned on the bedframe or shifted weight, and she avoided tasks that required sustained upright posture (e.g., IV preparation). Leisure walking was preserved but with shorter stride and reduced arm swing during symptomatic days, consistent with protective motor adaptations and altered movement ecology in CLBP [22–24,30–31,38,41–43,50,58–59,80–83].

Medical and social history.
No history of lumbar surgery. Two uncomplicated pregnancies (last 10 years ago). BMI 24. Non-smoker. No diabetes. Mild hypermobility traits in adolescence but no current instability symptoms. No prior pelvic trauma. Routine labs normal. She wore flat nursing shoes with soft heel counters.

Examination

Observation and movement sampling.
Static standing showed subtle pelvic rotation to the left with intermittent co-contraction of ipsilateral gluteus maximus during task focus [50]. Forward bending displayed an early lumbar-dominant strategy with limited hip hinge. During gait, stride length was mildly shortened and terminal hip extension attenuated; foot progression angle neutral; no Trendelenburg sign [22–24,30–31,38,41–43,50].

Neurological screen.
Sensation, myotomes, and reflexes were normal bilaterally; heel/toe walking intact; no neural deficits elicited [5–6,11].

Palpation—interface findings.
Point tenderness was elicited 6–8 cm lateral to the midline over the posterior iliac crest, slightly superior to the PSIS, reproducing the patient's familiar ache ("that spot") with a small zone of allodynia along 2–3 cm of the crest. Deep gluteal palpation provoked non-familiar pressure only. Iliopsoas corridor was non-tender. These

findings matched the course of the superior cluneal nerves (SCN) as they traverse fibro-osseous tunnels across the posterior iliac crest [20–21,31,40,88–92].

Neurodynamic testing (symptom-first endpoints; standardized sensitizers).
-Slump (dural bias): With thoracolumbar/cervical flexion and knee extension, the patient reported the familiar posterior-crest ache at relatively low knee extension (approx. 20–30°), despite no distal neurogenic pain. Cervical extension reduced the ache; ankle plantarflexion marginally eased it; cervical flexion restored symptoms.
-SLR (posterior chain bias): Right SLR onset of non-familiar hamstring stretch at 78°; left 82°. No reproduction of the posterior-crest ache.
-PKB (femoral bias): Negative for anterior thigh symptoms.

The paradox—posterior-crest pain reproduced in Slump without distal neural features—aligned with load sharing across lumbodorsal fascia/dural system provoking symptoms at a cluneal interface rather than root-level pathology [11,20–21,31,33–37,40].

Segmental tests and provocation.
Lumbar segmental springing was non-provocative; facet-dominant patterns (extension/rotation) were negative. SIJ provocation battery (distraction, compression, thigh thrust, Gaenslen, sacral thrust) was negative. Hip ROM was full with mild posterior-chain stiffness on the symptomatic side. No red-flag findings [4–6,30,41–43,55–57,77].

Imaging.
Lumbar and pelvic imaging (plain films 1 year earlier; no MRI requested in current episode) were unremarkable. Given the focal, mechanically coupled presentation, additional imaging was not pursued, consistent with guideline recommendations for non-specific CLBP when serious pathology is not suspected [4–6,55–57,77].

Differential diagnosis and reasoning

-Radiculopathy: Unlikely—no dermatomal distribution, neurological deficits, or imaging correlates; neurodynamic tests modulated local crest pain without distal symptoms [5–6,11].

-Facet/SIJ-dominant pain: Provocation tests were negative; symptom modulation with Slump sensitizers argued against a segmental articular primary [4–6,30,41–43].

-Myofascial trigger/myalgia: While peri-crest myofascial tenderness was present, the predictable change with cervical/ankle sensitizers pointed to a neural/meningeal component [33–37,39–41].

-Superior cluneal nerve entrapment (interface problem): Supported by point tenderness over the PIC at the known tunnel, mechanical coupling to prolonged

standing, and symptom reproduction with Slump that eased with neural unloading [20–21,40,88–92].

Working diagnosis: Subclinical cluneal neurodynamic restriction (SNR–cluneal bias) with mechanical interface sensitivity at the posterior iliac crest fibro-osseous tunnel, coexisting with maladaptive movement strategies (lumbar-dominant bending, guarded hip extension) [11,20–21,30–31,33–37,39–41,88–92].

Mechanistic hypothesis.
Tethering or stiffness at the cluneal tunnel reduces transverse excursion of the SCN branches. During sustained standing, posterior fascial tension and low-amplitude trunk movements increase perineural pressure, heightening mechanosensitivity. Slump testing pre-loads the dural–fascial system; small changes in cervical position unload/reload it, producing directionally predictable symptom modulation. Local hypertonicity of gluteus maximus and lumbodorsal fascia amplifies interface load [13–18,20–21,27–28,30–31,40,41,46,88–92]. Similar changes have been reported in CLBP cohorts where neural mobilization or interface-directed approaches produced improvements in pain and function [3,39,51–54,71,72,74–76].

Management

Goals (8–10 weeks).
Extend comfortable quiet-standing tolerance from ~25 minutes to ≥60 minutes; 2) reduce NPRS from 5/10 (peak) to ≤2/10 during shifts; 3) reduce PIC tenderness from 6/10 to ≤2/10; 4) normalise Slump behaviour (reduced intensity at matched knee angle; preserved sensitizer directionality with lower gain); 5) restore hip-hinge pattern in forward-reach tasks [3,22–24,30,33–37,39–41,50,67–70,93–99].

Outcome architecture.

-Patient-important: NPRS during standing tasks; PSFS items—(i) stand during med rounds 60 min, (ii) prepare IVs 20 min without leaning, (iii) complete handover standing 15 min [93–99].

-Mechanism-linked: Slump intensity at a matched knee angle (e.g., 30°) with/without cervical flexion; palpated PIC corridor tenderness (0–10); presence/magnitude of sensitizer effects; focal allodynia area (cm) [33–37,39–41,88–92].
-Safety: 24-hour response (green/amber/red) governing dose progression [1–3,39–41,51–54,71,72,74–76].

Phase 1 (Weeks 0–2): Establish control; reduce interface load

Clinic (weekly ×2).

-Cluneal interface de-loading: Gentle transverse gliding over the PIC corridor (1–2 minutes), progressing to small-amplitude oscillatory mobilization of overlying fascia;

strict symptom ceiling, no sustained pressure spikes [20–21,31,39–41,88–92].

-Neurodynamic sliders (posterior chain, low amplitude): Side-lying or seated variations to move the lumbodorsal–dural continuum without high tension (8–10 reps ×2 sets). Cervical extension bias was added early given immediate easing during assessment [1–3,33–37,39–41].

-Motor control: Dowel-guided hip hinge from a tall support to re-distribute flexion demand away from the lumbar segments and PIC corridor (2–3 × 6–8 reps in non-provocative range) [22–24,30–31,41–43,50,67–70,80–83].

-Standing ecology: Foot stance width just outside shoulder width; micro-weight shifts every 90–120 seconds; permission to alternate brief step-ups (one foot on low stool) during prolonged tasks [19,30,33–36,41–43].

Home (daily).

-Self-glide (PIC corridor): Patient-applied gentle cross-fiber skin/fascia glide (30–45 s) with a soft pad, once/twice daily, below a 3/10 ceiling [20–21,40,88–92].

-Seated posterior-chain sliders with cervical assist: 8–10 reps × 1–2 sets; stop if symptoms do not settle promptly after easing sensitizers [1–3,33–37,39–41,51–54,71,72,74–76].

-Standing breaks: Clock-based prompts every 20–25 min for brief ambulation (30–60 s) [19,30,33–36,41–43].

Expected early changes.
Reduced Slump intensity at matched angle by ≥1/10; smaller allodynia zone; improved tolerance to standing by ~5–10 minutes; green next-day responses [3,33–37,39–41,51–54,71,72,74–76,93–99].

Phase 2 (Weeks 3–6): Consolidate excursion; integrate task specificity

Clinic (weekly).

-Progress sliders by slightly expanding arc if green responses persist; introduce brief holds ≤2 s at symptom-first endpoint only if matched-angle intensity has fallen [1–3,33–37,39–41,51–54,71,72,74–76].

-Interface work remains short and symptom-limited; aim to reduce tenderness gain rather than chase “release” [20–21,31,39–41,88–92].

-Task substitution: Embed hip hinge into real tasks—trolley-height reaches, bed-level adjustments [22–24,30–31,41–43,50,67–70].

-Proprioception: Single-leg balance 3 × 20–30 s bilaterally; eyes-open only; ensure non-provocation of PIC corridor [22,30,41–42,50,84–87].

-Gait tuning: Encourage symmetrical stride with graded terminal hip extension within tolerance [30–32,41–43,67–70].

Home.

-Sliders on alternate days; self-glide daily; integrate hinge during domestic tasks (dishwasher/laundry).

-Standing interval extension to 35–45 minutes with scheduled micro-shifts [19,30,33–36,41–43].

Progress criteria.
PIC tenderness ≤3–4/10; Slump matched-angle intensity ↓ by ≥2/10; PSFS standing ≥45 minutes [3,33–37,39–41,93–99].

Phase 3 (Weeks 7–10): Functional robustness; discharge planning

Clinic (biweekly → discharge).

-Light posterior-chain loading (hip-dominant patterns) if green responses: elevated hinge with 6–8 kg load, 2–3 × 8, ensuring no PIC provocation [22–24,30–31,39–43,50,67–70,80–83].

-Minimal tensioners (rare): Only in low-irritability states and symptom-limited (≤5 reps), to consolidate tolerance [1–3,33–37,39–41,51–54,71,72,74–76].
-Relapse plan: Clear rules for heavier shift weeks—pre-shift sliders (1 micro-set), scheduled micro-shifts, and abort criteria [25–26,33–37,39–41,93–99].

Home.
-Maintenance sliders 3×/week; continue hinge patterns; progress standing intervals toward ≥60 minutes [22–24,30–31,39–43,50,67–70].

Test–intervention–retest data (selected)

Baseline (Week 0):
-Slump: Familiar PIC ache at 30° knee extension; intensity 5/10 with cervical flexion; reduced to 3–4/10 with cervical extension.

-SLR: 78° (right), 82° (left)—non-familiar distal stretch only.

-PIC tenderness: 6/10; allodynia strip ~3 cm.

-NPRS during quiet standing: 5/10 at 25–30 min.

-PSFS: med rounds 60 min 3/10; IV prep 20 min 4/10; handover 15 min 5/10 [93–99].

After first session (immediate):

-Slump matched-angle intensity −1/10 (now 4/10 with cervical flexion; 2–3/10 with cervical extension).

-PIC tenderness 5/10; allodynia strip ~2 cm.

-24-h response green [1–3,33–37,39–41,51–54,71,72,74–76].

Week 4:

-Slump matched-angle intensity 2–3/10 (flexion), 1–2/10 (extension).

-PIC tenderness 3–4/10; allodynia confined to ~1 cm.

-NPRS in standing 3/10 at 40–45 min.

-PSFS: med rounds 6/10; IV prep 6/10; handover 7/10.
-Patient reports ~50% reduction in peak pain and improved shift endurance [30–31,39–41,40,93–99].

Week 8–10 (discharge):

-Slump matched-angle intensity 1/10 (flexion) and 0–1/10 (extension); sensitizer effects persist but with low gain and no spontaneous PIC pain.

-PIC tenderness 1–2/10; no allodynia.

-NPRS in standing 1–2/10 at 60–70 min with planned micro-shifts.

-PSFS: med rounds 8–9/10; IV prep 8/10; handover 9/10.

-No adverse events [3,22–24,30–31,39–43,93–99].

Mechanistic interpretation
Two features strengthen the SNR–cluneal inference:

1. Predictable sensitizer modulation in Slump (worse with cervical flexion, easier with extension) affecting local PIC symptoms rather than distal radicular pain, indicating involvement of the dural–fascial continuum and perineural excursion at the cluneal tunnel [11,20–21,31,33–37,40,88–92].

2. Within-session and sustained reductions in matched-angle Slump intensity and PIC tenderness following short, symptom-limited interface de-loading plus low-amplitude sliders, consistent with reversible mechanical/mechanosensitive contributions at the interface [3,20–21,31,33–37,39–41,51–54,71,72,74–76,88–92].

3.As excursion tolerance improved and interface load reduced, the gain on sensitizers fell, suggesting better load sharing across the neural–myofascial complex. Task-level redistribution (hip hinge; scheduled micro-shifts) lowered repeated interface stress during the most provocative context—quiet standing—supporting durability of change [22–24,30–32,39–43,50,67–70].

Safety, adherence, and adverse events
One amber day occurred (Week 2) after a double shift without scheduled micro-shifts; symptoms settled within 24 hours after temporarily reducing slider amplitude and increasing walking breaks. No red responses or neurological changes occurred. Adherence was high (~85–90%) due to concise home dosing and clear standing-ecology rules [25–26,33–37,39–41,93–99].

Discussion
This case demonstrates a superior cluneal nerve–biased subclinical neurodynamic restriction as a treatable contributor to CLBP-spectrum posterior pelvic pain, mirroring changes reported in trials of neural mobilization for CLBP [3,39,51–54,71,72,74–76]. Key discriminators included: (i) focal PIC tenderness at the known fibro-osseous tunnel; (ii) load-coupled aggravation during sustained standing; (iii) predictable Slump sensitizer effects on local symptoms; (iv) normal routine neurology; and (v) unremarkable imaging [4–6,11,20–21,31,33–41,40,55–57,77,88–92]. Management aligned with neurodynamic principles emphasising symptom-first endpoints, low-amplitude excursion, short interface de-loading, and task substitution, overseen by a 24-hour response rule [1–3,33–37,39–41,51–54,71,72,74–76].

Measurable mechanistic shifts (matched-angle Slump intensity ↓, PIC tenderness ↓) paralleled clinically meaningful improvements (longer standing tolerance, lower NPRS, higher PSFS), supporting internal coherence between theory and outcome [3,22–24,30–32,33–41,50,67–70,93–99].

Boundary conditions.
In cases with widespread tenderness, poor sleep, or weak mechanical coupling, a nociplastic tilt may dominate and neurodynamic work should be framed as graded exposure rather than as the primary lever [25–26,47–49,60–63,78–79,95–96,100]. Emergence of neurological deficits or failure of mechanism-linked markers to improve after 3–4 well-dosed sessions should prompt a diagnostic pause and re-appraisal [3,5–6,33–41,77].

Practical template (cluneal-biased SNR)
1.Identify: Focal PIC tenderness over the cluneal tunnel; symptoms coupled to quiet standing; Slump reproduces local ache with predictable sensitizer modulation; normal neurology [5–6,11,20–21,31,33–37,39–41,40,88–92].

3. Intervene: Short, symptom-limited PIC interface de-loading → low-amplitude sliders with cervical-extension assist → immediate retest (matched-angle Slump) [1–3,20–21,31,33–37,39–41,51–54,71,72,74–76,88–92].

4. Integrate: Standing ecology (micro-shifts, occasional foot support), hip-hinge substitution for forward tasks, graded restoration of hip extension in gait [19,22–24,30–32,39–43,50,67–70].

5. Govern dose: Progress only on green 24-h responses; hold/regress on amber; re-appraise on red [1–3,33–37,39–41,51–54,71,72,74–76].

5.Document: Slump matched-angle intensity, sensitizer direction/magnitude, PIC tenderness and allodynia, PSFS for standing tasks [33–37,39–41,88–92,93–99].

Conclusion.
A structured, mechanism-linked approach targeting subclinical cluneal neurodynamic restriction yielded clinically meaningful gains in a nurse with posterior pelvic pain aggravated by standing. The case underscores the utility of predictable neurodynamic modulation, interface-aware intervention, and task-level redistribution under explicit safety rules to operationalize cluneal-biased care within guideline-concordant management of non-specific CLBP [1–6,11,20–21,30–32,33–41,40,51–54,55–57,71,72,74–77,88–92,93–99].

3. Neurodynamic Restrictions and Muscle Tone

3.1 Protective Motor Responses

When neural mobility is subtly impaired, the motor system often reorganizes to keep tensile and compressive loads on the affected neural tissue within a tolerable window. These protective motor responses arise from the interaction of segmental spinal circuits, brainstem centers, and supraspinal networks that continuously reconcile afferent input (mechanical and nociceptive) with ongoing task demands. In chronic low back pain (CLBP), this reorganization can stabilize symptoms in the short term but, if persistent, it tends to degrade movement efficiency, narrow the available movement envelope, and perpetuate pain and disability [22–26,30–32,58–59,80–83].

3.1.1 Control objectives and the logic of protection

The nervous system must solve a constrained optimization problem: accomplish a task (standing, bending, gait) while keeping multiple biologic states within safe bounds—perfusion within intraneural microvessels, strain within the viscoelastic limits of neural sheaths, and pressure across perineural interfaces [15–18,27–28]. If sliding or transverse excursion of a nerve is reduced (e.g., by perineural adhesions, fascial tunnel stiffness, or heightened mechanosensitivity), the controller achieves protection by reshaping motor output. That reshaping typically includes: (i) raising baseline tone in muscles that can mechanically shield the sensitized pathway; (ii) altering intermuscular timing to reduce end-position demands on the threatened interface; and (iii) simplifying movement (co-contraction, reduced degrees of freedom) to minimize variability in load [22–24,30–31,50,58–59,80–83].
This "protect first" priority is adaptive in acute phases but becomes maladaptive when it persists beyond the irritative driver, because it increases segmental compression, raises metabolic cost, impairs proprioception, and repeatedly exposes the same tissues to suboptimal loading patterns [22–26,30–32,58–59,80–83]. Hodges and colleagues showed that such protective strategies can be detected as altered deep-to-superficial muscle recruitment, increased trunk stiffness, and delayed or exaggerated feedforward responses during limb movement [24,50,58–59,80–83]. These adaptations reduce kinematic error at the price of excess stiffness and reduced capacity to absorb perturbations.

3.1.2 Mechanistic substrates: reflex arcs and supraspinal gain

Segmental reflexes. Mechanically sensitive afferents from the perineurium/epineurium and interfacing fascia (including small-diameter fibers) can increase dorsal horn excitability when repeatedly activated by strain or pressure [15–18,27–28]. In turn, gamma motor drive to muscle spindles rises, increasing intrafusal sensitivity and biasing the system toward muscle shortening within the threatened corridor. The result is a higher resting tone in muscles that reduce excursion demand on the irritated neural tissue. For example, in a posterior-chain bias, hamstring and deep gluteal tone may increase to limit hip flexion range during daily tasks, thus cutting down on sciatic longitudinal sliding requirements [31,33–36]. In an anterior corridor bias, iliopsoas tone may increase to limit hip extension and femoral nerve excursion [32,37].

Presynaptic inhibition and reciprocal modulation. Increased nociceptive and mechanosensitive input can alter presynaptic inhibition of Ia afferents, changing the

balance between agonist facilitation and antagonist inhibition. The outcome is co-contraction around the lumbar–pelvic axis: abdominal and paraspinal groups activate together, increasing trunk stiffness and reducing the variability of spinal segment motion [24,30,50,58–59,80–83]. While co-contraction stabilizes threatened tissues, it reduces movement efficiency and increases compressive forces that, over time, may aggravate interface load and perpetuate symptoms [24,30,50,58–59,80–83].

Supraspinal modulation. Brainstem centers (reticulospinal pathways) and cortical regions concerned with body schema and error prediction (e.g., supplementary motor areas and parietal cortex) modulate gain based on perceived threat and prediction error [23,25,58–59,60–63,80–83]. With persistent peripheral input, protective set points shift: anticipatory postural adjustments become earlier and larger, and movement planning favors shorter arcs and slowed accelerations to keep neural load predictable—patterns consistent with observed changes in trunk motor control, cortical representation of deep trunk muscles, and reduced movement variability in CLBP [24,50,58–59,80–83]. The subjective correlate is a sense of tightness or guardedness even when formal flexibility is not grossly limited.

3.1.3 Canonical protective patterns in CLBP

(a) Increased baseline tone.
Patients with CLBP frequently present with elevated tone in the lumbar extensors, deep hip rotators, gluteus maximus, or hamstrings. In a sciatic-biased presentation, increased hamstring tone reduces hip flexion and slackens longitudinal neural demand during everyday bending. In a femoral-biased pattern, increased iliopsoas tone limits hip extension during gait, decreasing femoral nerve excursion [31–32,37]. These patterns often co-exist with localized interface tenderness (deep gluteal corridor, posterior iliac crest, or iliopsoas corridor) and with neurodynamic tests that show symptom-first endpoints and predictable sensitizer effects [22–24,30–32,33–37,39–41,50,58–59,80–83].

(b) Altered recruitment timing and organization.
Protective responses reorder recruitment: superficial global muscles (e.g., erector spinae) dominate early, while deep segmental stabilizers (e.g., multifidus) are delayed or reduced in contribution, consistent with observations by Hodges et al. of altered feedforward postural adjustments in low back pain [24,50,58–59,80–83]. Functionally, people bend more with the lumbar spine than the hips (spine-dominant bending), step with shorter stride, and avoid terminal hip extension during gait—strategies that lower neural excursion demand but increase segmental strain and energy cost [22–24,30–32,37–41,50,58–59,80–83].

(c) Segmental guarding and co-contraction.
Co-contraction stiffens the trunk, narrows movement options, and decreases perturbation resilience. It also loads the passive elements of the motion segment and compresses perineural interfaces, potentially sustaining mechanosensitivity. Guarding is commonly observed during end-range or sustained positions (prolonged sitting/standing), where fine oscillations in posture normally distribute load but, under protection, become damped and monotonic, raising time-under-load for the interface [22–24,30,50,58–59,80–83].

3.1.4 Consequences for sensorimotor control and proprioception

Brumagne and colleagues have shown proprioceptive deficits in individuals with CLBP, including altered lumbar position sense and increased reliance on distal proprioceptive sources [22,30,84–87]. Protective patterns that reduce joint excursion and increase co-contraction likely contribute to poorer afferent richness (fewer varied spindle inputs across ranges), while sustained tone increases background spindle firing and noise, reinforcing a high-gain, low-variability control strategy [50,58,59,80–85]. Over time, the system shifts toward stiffness control rather than precision control, which suffices for low-variability tasks but degrades performance in activities requiring adaptable, multi-planar coordination (e.g., lifting with turn, uneven-ground walking) [58,59,80–85]. Clinically, this appears as movement simplification: fewer strategies, smaller arcs, lower peak velocities, and reduced ability to pivot quickly between strategies without symptom flare [58,59,80–85].

3.1.5 Task ecology: why ordinary situations provoke protection

-Prolonged sitting. Posterior chain under low-grade tension increases intraneural pressure and reduces perineural perfusion; subtle "fidgets" that would normally distribute load are suppressed under co-contraction, raising exposure time. Symptom onset is then more about duration than angle, consistent with a time-under-load threshold [22,30,50,84,85].

-Forward reach/bending. Spine-dominant bending reduces hip flexion and therefore sciatic excursion but increases lumbar segment shear and compressive loads. The system accepts higher joint stress to keep neural load low [24,30,50,68–70,80–83].

-Quiet standing. Micro-oscillations at the pelvis normally distribute fascial tension; under protection, individuals adopt quieter strategies with reduced sway and episodic co-contractions, increasing static load on cluneal or gluteal interfaces (posterior iliac crest tunnels, deep gluteal corridor) [21–22,40,50,84,85].

-Gait. Shortened stride and avoidance of terminal hip extension protect the femoral corridor but increase energy cost and reduce elastic contributions of the anterior chain. Over time, this can yield deconditioning and further reliance on stiffness for control [31–32,37–41,50].

3.1.6 Measurement signatures in clinic

Protective motor responses are inferred from converging behavioral and mechanism-linked markers:

– Neurodynamic tests (SLR, Slump, PKB) show symptom-first endpoints and predictable sensitizer directionality (worse with added neural load; easier with unloading) without neurological deficits [33–37,72–77].

– Functional sampling reveals spine-dominant bending, shortened stride, and guarded transitions (sit-to-stand), consistent with the low-variability, stiffened movement patterns described in CLBP [31,38,58,59,80–85].

– Palpation elicits focal interface tenderness over plausible corridors (deep gluteal, posterior iliac crest, iliopsoas), often aligning with known entrapment zones for sciatic, femoral, and cluneal nerves [17–19,21,40,88–92].

– Within-session retest after low-amplitude neural excursion or brief interface de-loading demonstrates improved onset angle or reduced matched-angle intensity—evidence that the protective set point is modifiable and that neurodynamic dosing is targeting a mechanosensitive subsystem [39–41,72–77].

– Twenty-four-hour behavior tracks irritability: appropriate dosing yields "green" responses (no delayed exacerbation), while overloading manifests as next-day amplification—an indicator to regress amplitude or frequency, mirroring graded exposure principles and guideline-concordant titration of load in CLBP [57,67–70,95,100].

3.1.7 Interaction with central sensitization and cognitive–affective factors

Protective motor responses are not purely spinal. Pain-related cognitions (catastrophizing, fear of movement) and sleep disturbance can elevate supraspinal gain, thereby lowering the threshold for protective activation and sustaining co-contraction even when peripheral drivers are waning [25,26,48,49,60–63,78–80,95–100]. Moseley's work on pain neuroscience emphasizes that threat appraisal shapes motor plans; when the system "expects" danger at certain arcs, it pre-activates protective patterns, narrowing movement exploration and reinforcing the learned association between a task and pain [25]. In practice, concise mechanistic education combined with graded, evidence-based disconfirmation—via test–intervention–retest and progressive exposure—helps recalibrate expectations and reduce the need for high-gain protection, in line with fear-avoidance and cognitive functional approaches to CLBP [57,67–70,95–100].

3.1.8 Metabolic and structural costs of persistent protection

Sustained co-contraction increases oxygen demand and metabolic burden without proportional mechanical work, fostering early fatigue, especially during static or low-load tasks [15–18,27,28,50]. Elevated resting tone compresses small vessels in muscle and perineural tissues, risking relative ischemia and amplifying mechanosensitivity in already sensitised interfaces [15–19,27,28,60–63]. Structurally, excessive trunk stiffness steers load through passive restraints rather than shared segmental motion,

potentially aggravating articular and fascial tissues and creating a self-maintaining loop—protection → interface load → afferent drive → more protection—that mirrors experimentally observed low-variability, stiff control strategies in CLBP [31,38,50,58,59,80–85].

3.1.9 Clinical leverage: de-threatening neural load while preserving control

Because protection serves a real constraint (neural load), simply suppressing tone or forcing range is counterproductive. Effective change requires de-threatening the neural interface while maintaining a sense of control:

1. Excursion before tension. Low-amplitude sliders in the implicated bias (posterior chain for sciatic, femoral bias for anterior corridor) move neural tissue relative to its bed without high end-range loading, often reducing matched-angle intensity on Slump/PKB within minutes [1–3,33–37,39–41,72–77]. This preserves control while beginning to normalise interface viscosity and mechanosensitivity.

2. Interface de-loading. Short, symptom-limited work over the tender corridor (deep gluteal, posterior iliac crest, iliopsoas) reduces local pressure and improves transverse glide, lowering the gain of sensitizers in known entrapment zones for sciatic, femoral, and cluneal nerves [17–21,40,88–92]. The aim is not aggressive soft-tissue work but gentle, targeted de-loading that immediately changes mechanosensitive responses.

3. Task substitution. Replacing spine-dominant bending with a hip hinge redistributes flexion demand away from the neural corridor while retaining function; graded restoration of terminal hip extension in gait progressively re-exposes the femoral pathway within tolerance [30–32,37–41,58,59,67–70,80–85]. This reflects guideline-concordant advice to maintain activity while modifying load and is consistent with motor-control findings of altered trunk strategies in CLBP.

4. Irritability-governed dosing. Progression is contingent on “green” next-day behavior; amber/red responses cue regression of arc, repetitions, or frequency. This maintains safety and builds a predictable exposure history, which is itself analgesic and de-sensitizing [33–37,39–41,57,67–70,95,100]. In effect, neurodynamic dosing becomes a structured form of graded exposure linked to mechanosensitive signs.

1. Concise mechanism education. Explaining that nerves must slide and share load—then demonstrating controllability by altering ankle/cervical positions during testing—links the patient's experience to a modifiable mechanism, diminishing the need for high-gain protection [25,26,33–36,57,95–100]. This integrates pain neuroscience education with behavioural experiments, aligning with contemporary fear-avoidance and cognitive functional models of CLBP.
2.

3.1.10 Illustrative protective constellations

Posterior-chain bias (sciatic corridor).

Findings: early symptom-first endpoint on SLR or Slump with posterior-chain quality; hamstring and deep gluteal hypertonicity; spine-dominant bending; shortened stride [31,33–37,58,59,80–85]. Mechanism: reduce hip flexion excursion to lower sciatic sliding and peak strain; increase lumbopelvic stiffness to stabilise the neural bed and limit variability in load [15–19,27,28,31,38,50,58,59,80–85]. Management: posterior-chain sliders, deep gluteal interface de-loading, and hinge substitution to redistribute flexion demand away from the sciatic corridor, with graded restoration of hip flexion and trailing-limb extension as irritability falls; outcome anchors—later onset angle, reduced matched-angle intensity, and longer tolerated sitting/forward reach [31,33–41,58,59,67–70,72–77].Anterior corridor bias (femoral nerve).

Findings: start-up pain on rising; PKB symptom-first endpoint; avoidance of terminal hip extension during gait; iliopsoas corridor tenderness [31,32,37–41,58,59,80–85]. Mechanism: increase iliopsoas tone to limit femoral excursion and peak strain; reduce stride length to keep neural load predictable [15–19,27,28,31,38,50,58,59,80–85]. Management: femoral-biased sliders in side-lying/prone, corridor de-loading, graded restoration of hip extension; outcome anchors—PKB onset shift, gait extension tolerance, reduced corridor tenderness [32,37–41,58,59,67–70,72–77].

Posterior iliac crest interface (superior cluneal).
Findings: focal PIC tenderness; standing-provoked ache; Slump reproduces local crest pain with predictable cervical/ankle modulation; normal neurology [21,31,38,40,58,59,80–85,88–92]. Mechanism: tunnel stiffness tethering cluneal branches with dural–fascial coupling [17–21,40,88–92]. Management: brief PIC de-loading + low-amplitude sliders; standing ecology (micro-shifts); outcome anchors—reduced matched-angle Slump intensity, decreased PIC tenderness, longer standing tolerance [21,31,38,40,58,59,67–70,72–77,88–92].

3.1.11 From protection to restoration: sequencing change
A practical sequence respects the original protective logic:

1.Stabilize irritability (excursion-first dosing, avoid high tension) [33–37,39–41,72–77].

2.Restore strategy diversity (reintroduce hip hinge; graded terminal hip extension; small increments in task complexity) [31,38,58,59,67–70,80–85].

3.Recalibrate gain (show repeatable improvements on mechanism-linked tests; pair with patient-important tasks) [33–37,39–41,57,93–99].

4.Consolidate robustness (light functional loading; infrequent, symptom-limited tensioners only in low irritability to broaden tolerance) [57,67–70,72–77].

5.Plan for variability (written rules for high-demand days; pre-emptive micro-sets of sliders; explicit abort criteria) [57,67–70,95–100].

This trajectory preserves the benefits of protection (control, predictability) while releasing its costs (excess stiffness, narrowed repertoire) [31,38,58,59,80–85,95–100].

3.1.12 Boundary conditions and when protection is not primary

Not all CLBP protection is neurally driven. Segmental articular pain (facet, SIJ) may dominate when extension/rotation provokes pain with local tenderness and neurodynamic sensitizers have little impact [4–6,31,38,57]. Conversely, in presentations with weak mechanical coupling and widespread sensitivity, a nociplastic tilt may predominate; here, neural excursion becomes a graded exposure tool rather than the primary mechanism [5,6,25,26,48,49,60–63,78,79,95–100]. Emergence of neurological deficit or absence of improvement in mechanism-linked markers after several well-dosed sessions should prompt re-appraisal of diagnosis and plan [33–37,39–41,57].

3.1.13 Quantifying protection: objective indices and clinic-friendly proxies

While protective motor responses are often recognized qualitatively, several **quantitative indices** can anchor interpretation and progression:

-Electromyography (EMG) patterns. Surface EMG can demonstrate elevated resting activity in lumbar extensors and hip musculature, earlier onset of superficial trunk muscles during anticipatory tasks, and reduced differential activation between deep and superficial layers (e.g., multifidus vs. erector spinae) in CLBP cohorts [24,50,58,59,80–85]. In practice, few clinics deploy EMG routinely; however, clinical proxies—palpable resting tone, difficulty “letting go” of paraspinals in prone, and visible co-contraction during limb movements—convey similar information when recorded systematically [50,58,59,80–85].

-Kinematic signatures. Two-dimensional video or inclinometry can objectify spine-dominant bending (greater lumbar than hip excursion in the first half of forward flexion) and gait truncation (reduced terminal hip extension). A simple strategy is to record lumbar and pelvic angles at 30°, 60°, and 90° of forward reach, and stride length/step time during preferred walking. Serial improvement (larger hip share of flexion; modest restoration of terminal extension) often parallels reductions in neurodynamic test gain [30–32,37–41,58,59,67–70,80–85].

Stiffness surrogates. Hand-held dynamometry during low-load trunk perturbations (e.g., seated or semi-squat) provides a coarse estimate of apparent trunk stiffness. Although not specific to neural protection, decreasing stiffness under comparable task conditions, along with preserved task accuracy, suggests safe unwinding of co-contraction [31,38,50,58,59,80–85].

-Mechanism-linked anchors. The most pragmatic indices remain neurodynamic markers—onset angle, intensity at matched angle, and sensitizer directionality—paired with interface tenderness scores. These change early, are reproducible with standardized execution, and map closely to the protective logic described above [33–37,39–41,72–77].

Together, these indices create a triangulated picture: if neurodynamic gain falls but stiffness surrogates do not, emphasis should shift to motor pattern retraining; if motor patterns normalize but neurodynamic gain remains high, interface de-loading and excursion dosing likely need refinement [31,38,50,58,59,67–70,72–77,80–85].

3.1.14 Biomechanical modeling perspective: why co-contraction “works” (and then fails)

From a modeling standpoint, the trunk–pelvis–hip complex is a redundant system with many muscle combinations capable of producing similar net moments. When the controller is asked to reduce variability in a threatened tissue, the optimal solution shifts toward co-contraction, which increases joint impedance and reduces the variance of end-range demand on the neural bed [31,38,50,58,59,80–85]. Initially, this strategy improves predictability and reduces afferent noise [31,38,50,58,59,80–85]. Over time, however, co-contraction:

1.Raises compressive load at motion segments and interfaces,

2.Increases metabolic cost for the same external work, and

3.Suppresses exploration of alternative, potentially less provocative solutions [31,38,50,58,59,80–85].

The result is local plateaus—short-term symptom containment with long-term performance loss. Clinical reversal therefore requires not only reducing neural gain (excursion + interface work) but also re-introducing variability in a controlled way (graded hinge, terminal hip extension, perturbation-lite balance work) once irritability permits [24,30–32,37–41,58,59,67–70,80–85].

3.1.15 A brief applied vignette: detecting protection in five minutes.

A 35-year-old physiotherapist with intermittent lumbopelvic pain reports flares after extended charting. In a single visit:

1.Slump reproduces a local posterior pelvic ache at a modest knee extension, worsened by cervical flexion and eased by extension (symptom-first endpoint) [33–37].

2.Forward bend shows early lumbar dominance with visible paraspinal bracing [31,38,58,59,80–85].

3.Deep gluteal palpation is tender and familiar [17–19,21,40,88–92].

4.After 90 seconds of low-amplitude sliders (posterior chain) and brief interface de-loading, Slump intensity at a matched angle drops by 2/10; forward bend shows a small but visible increase in hip share [33–37,39–41,72–77].

This sequence—test → brief intervention → retest—documents that protection is contingent on modifiable neural load, not fixed “tightness,” and justifies an excursion-first plan with hinge substitution. It also provides a patient-specific benchmark for subsequent sessions [31,33–41,58,59,67–70,72–77,80–85].

3.1.16 Integrating protection logic into rehabilitation dosing

A dosing framework that respects protection:

-Anchor to the most irritable component. If matched-angle intensity in Slump drops readily but forward-bend pattern does not change, maintain excursion emphasis and postpone loading of hinge drills until next-day responses are reliably green [33–37,39–41,57,67–70,72–77].

-Progress one dial at a time. Increase amplitude of sliders before frequency, and frequency before tension; add hinge range before external load; add terminal hip extension in gait before speed work [31,33–41,58,59,67–70,72–77,80–85].

-Use sentinel tasks. Select one repeated, meaningful task (e.g., 25-minute quiet standing for a retail worker) and revisit it after clinic dosing. If the sentinel task shows earlier symptom onset after progression, roll back progression even if formal tests improved [57,67–70,93–99].

-Codify “abort rules.” Stop home sets if the familiar symptom does not settle within seconds after releasing sensitizers; revert to the last green dose for 48 hours before re-progressing. Clear rules reduce uncertainty, which itself lowers supraspinal gain [25,26,57,95–100].

-Consolidate with low-threat loading. In low irritability states, light hip-dominant loading and simple balance challenges (eyes open, stable surface) help shift control from stiffness to skill, making protection unnecessary for routine tasks [31,38,58,59,67–70,80–85,95–100].

3.1.17 Common pitfalls and how to avoid them

-Chasing end-range flexibility. Forcing range at high tension tends to inflate protective gain and risks next-day flares. Prioritize excursion at symptom-first endpoints; delay tensioners until low irritability and only for consolidation [15–19,27,28,33–37,39–41,72–77].

-Misreading “tight hamstrings.” If ankle/cervical sensitizers modulate symptoms during SLR/Slump, the constraint is more likely neural than purely myofascial. Focusing solely on hamstring stretching can aggravate neural load [1–3,33–37,39–41,72–77].

-Ignoring 24-hour behavior. Inconsistent green responses indicate dose mismatch or poor sentinel-task hygiene (e.g., long static standing without micro-shifts). Adjust the plan before adding complexity [33–37,39–41,57,67–70,95,100].

-Overlooking interface corridors. Failure to examine deep gluteal, posterior iliac crest, and iliopsoas corridors misses efficient leverage points for reducing protection. Even brief, symptom-limited de-loading can convert a non-responsive session into a responsive one [17–21,40,88–92].

3.1.18 Special populations revisited: protection through a pragmatic lens

-Adolescents/rapid growers. Transient slack reductions amplify neural gain; brief sliders and frequent positional variation during study hours prevent over-reliance on protective co-contraction [33–36,50,58,59,80–85].

-Pregnancy/postpartum. Hormonal laxity and venous changes alter interface mechanics; dose sliders conservatively, rely on positional supports, and emphasize hinge mechanics for caregiving tasks [1–3,39–41,57,67–70].

-Older adults/metabolic comorbidity. Glycation-related stiffness and microvascular fragility make high tension counterproductive. Use shorter arcs, longer observation windows, and gradual balance integration to shift from stiffness to confidence-based control [15–18,27,28,30,31,38,41,42,58,59,67–70,80–85].

-Athletes/manual workers. High exposure to end-range/loading creates "learned" protection. Program task-specific hinge retraining (e.g., hip-dominant lift sequencing) and paced restoration of terminal hip extension for runners; monitor next-day performance and sentinel tasks to prevent overshooting [31,32,37–41,58,59,67–70,72–77,80–85].

3.1.19 Implications for research and reporting

To clarify the role of protection in SNR-positive CLBP, studies should:

1.Define the phenotype a priori: normal neurology; symptom-first endpoints with predictable sensitizer modulation; at least one concordant interface finding [33–37,39–41,48,49,60–63,78,79].

2.Report co-primary outcomes: a patient-important measure (ODI or PSFS) and a mechanism-linked anchor (e.g., change in matched-angle Slump intensity) [93–99].

3.Stratify by irritability: analyze trajectories for high vs. low irritability; dosing effects differ meaningfully between strata [57,67–70,95,100].

4.Track fidelity: document sensitizer sequencing, endpoint definitions, and 24-hour governance to ensure that "neurodynamic care" is not an uncontrolled mixture of stretching and general exercise [3,33–41,72–77].

Such designs will help disentangle whether observed gains reflect true reductions in protective gain secondary to improved neural excursion/interface mechanics, rather than nonspecific exercise effects [15–19,27,28,33–41,57,72–77,93–99].

3.1.20 Consolidated clinical algorithm (protection-aware)

1.Screen for red flags and neurological deficit (if present, manage accordingly) [5–6].

2.Elicit symptom-first endpoints on SLR/Slump/PKB; record sensitizer effects and interface tenderness [33–37,40].

3.Intervene briefly: low-amplitude sliders in the implicated bias + short, symptom-limited interface de-loading [17–21,33–37,39–41,72–77,88–92].

4.Retest: confirm change in onset angle or matched-angle intensity; if absent, adjust technique/dose rather than escalate tension [33–37,39–41,72–77].

5.Prescribe a home micro-dose (excursion first) with explicit abort criteria; add task substitution (hip hinge; micro-shifts in standing) [31–33,37–41,57,67–70,72–77,95,100].

6.Progress only after repeated green 24-hour responses; add motor pattern complexity before loading; consider minimal tensioners only in low irritability and for consolidation [31,38,58,59,67–70,72–77,95,100].

7.Audit weekly: pair patient-important outcomes with mechanism-linked anchors; if either stalls, diagnostic pause, review fidelity, reconsider primary driver [15–19,27,28,33–41,57,67–70,72–77,93–99].

Clinical take-home. Protective motor responses in SNR-positive CLBP are purposeful, load-governing solutions that keep neural tissues within tolerable limits at the expense of efficiency and adaptability. Because these behaviors are contingent on modifiable neural mechanics and interface load, they can be reversed safely with an excursion-first, interface-aware, irritability-governed program that reintroduces movement variability only when the system's need for protection has eased. This preserves the protective intent while dismantling its costs, aligning mechanistic change with patient-important recovery [1–3,20–21,24–26,30–41,43,57,58,59,67–70,72–77,80–85,93–100].

In summary, protective motor responses in the context of subclinical neurodynamic restriction represent coherent adaptations—higher baseline tone, altered recruitment and timing, segmental guarding, and co-contraction—aimed at keeping neural load tolerable. They are mediated by segmental reflex modulation and supraspinal gain changes, persist when afferent threat is recurrent, and manifest as characteristic changes in bending, gait, and postural control [22–26,30–32,50,58,59,60–63,80–85]. Because these responses solve a real constraint, intervention must de-threaten the neural interface (excursion before tension; targeted interface de-loading), redistribute task mechanics (hip hinge; graded hip extension), and govern dose by 24-hour behavior, while demonstrating controllability through test–intervention–retest procedures [1–3,20–21,33–41,57,67–70,72–77,93–100]. Done in this order, protection can be safely unwound, movement efficiency restored, and patient-important

outcomes improved without provoking the very neural loading that the system was designed to avoid.

3.2 Sensorimotor Implications

Restricted neural mobility alters afferent input to the spinal cord and brain, disrupting proprioceptive signaling and motor control [27,28,30,60–63]. Brumagne and colleagues have shown that individuals with CLBP exhibit proprioceptive deficits, including altered lumbar position sense and increased reliance on distal proprioceptive sources, which may be exacerbated by neural restrictions [22,30,84–87]. These deficits can lead to:

-Persistent muscle co-contraction: chronic activation of antagonistic muscle groups (e.g., lumbar extensors and abdominals) that stabilises the spine but reduces flexibility and increases stiffness, metabolic cost, and reduced functional mobility [24,30,31,38,50,58,59,80–85].

-Impaired postural control: reduced or distorted proprioceptive input driving compensatory movement patterns, such as excessive lumbar flexion during functional tasks, thereby increasing mechanical stress and perpetuating pain [22,30,58,59,84–87].

-Altered gait mechanics: restrictions in sciatic nerve mobility shortening stride length or reducing hip extension, increasing lumbopelvic stress; for example, a patient with restricted sciatic mobility may adopt a limping or trunk-stiffening gait as a compensation [31,32,38,58,59,80–85].

Moseley's work on pain neuroscience suggests that such sensorimotor changes, when interpreted as threatening, can amplify pain perception even in the absence of major structural pathology [25,26]. Restricted neural mobility may also increase mechanosensitivity, leading to heightened pain during movement, which reinforces protective motor responses and perpetuates dysfunction [15–19,27,28,60–63].

3.2.1 Rationale and scope

This section expands the sensorimotor implications of restricted neural mobility into a coherent, testable framework for a CLBP subgroup whose clinical presentation features (i) movement-evoked symptoms during neurodynamic loading, (ii) tonic paraspinal activity and bracing, (iii) proprioceptive uncertainty during lumbopelvic tasks, and (iv) gait adaptations consistent with reduced sciatic excursion [1–3,15–19,22,24,27,28,30–32,38,58,59,80–85]. The argument proceeds from first principles of neural tissue mechanics and afferent physiology, through observable changes in posture and movement, to practical assessment and intervention strategies [15–19,27,28,30–32,38,58,59,80–85]. Throughout, the emphasis is on mechanistic plausibility and clinical falsifiability: each link in the chain generates predictions that can be evaluated with standardized tests and outcome measures [33–37,39–41,57,67–70,72–77,93–99].

3.2.2 Neural excursion, afferent fidelity, and sensorimotor control

Neural tissues must slide, elongate, and accommodate multi-segmental movement. The lumbosacral roots, dura, and peripheral pathways (notably the sciatic and femoral nerves) traverse osteoligamentous and myofascial tunnels that impose direction-dependent constraints [15–19,27,28,30]. During hip flexion, for instance, the sciatic pathway undergoes a characteristic combination of elongation and sliding relative to surrounding tissues. In a healthy system, this excursion is noiseless from the standpoint of the central nervous system (CNS): mechanoreceptor discharge scales predictably with joint displacement and load, contributing to accurate state estimation [15–19,27,28,30,60–63].

When excursion is restricted—by adhesions, altered perineural gliding surfaces, fascial thickening, or sustained bracing that reduces relative motion—three consequences for afferent fidelity follow:

1.Distorted mechanotransduction. Localized strain concentrations at "sticking points" can disproportionately activate low-threshold mechanoreceptors. The same external movement now yields a larger, less linear intraneural mechanical signal. Over time, thresholds may fall and receptive fields broaden, a substrate for mechanosensitivity [15–19,27,28,30,60–63].

2.Perfusion–tension interplay. Elevated resting tension or repeated end-range strain can intermittently impair microvascular flow within the nerve. Transient hypoperfusion and metabolite accumulation alter membrane excitability, biasing discharge toward nociception during movements that were previously neutral [15–19,27,28,30,60–63].

3.Noise in the proprioceptive stream. Proprioception is an integration problem: signals from spindles, Golgi organs, joint and cutaneous receptors, and neural sheaths must be combined into a coherent estimate of body state. Distorted neural afference lowers the signal-to-noise ratio, forcing the motor system to adopt more conservative control policies [22,30,58,59,84–87].

From a control-systems perspective, the CNS can respond to noisy afference by increasing joint/segmental stiffness—effectively "tightening the controller" to reduce reliance on unreliable feedback. This is adaptive for stability, but costly for efficiency and adaptability [31,38,50,58,59,80–85].

3.2.3 Proprioceptive deficits in CLBP: clinical significance

Across multiple paradigms, people with CLBP show impaired lumbopelvic proprioception—for example, increased repositioning error and reduced confidence in trunk orientation tasks [22,30,84–87]. These deficits are not mere epiphenomena of pain intensity: they can persist during low-pain states and predict functional limitations [22,30,58,59,84–87]. Restricted neural mobility provides a plausible peripheral contributor to such deficits by degrading the fidelity of afferent input during trunk and hip motion [15–19,27,28,30,60–63].

Clinically, this presents as uncertain movement: patients hesitate at mid-range, "search" for safe positions, and prefer slow, braced patterns over fluid transitions [31,38,58,59,80–85]. They frequently report that certain stretches (e.g., hamstring tensioners) feel like "nerve pulling" rather than muscle stretch, and they avoid lunge-like tasks that load hip extension [31,32,37–41,58,59,80–85,95–100]. Over time, the repertoire of practiced movements contracts, reinforcing the sensory deficit by disuse of the neural excursion envelope [31,38,58,59,80–85,95–100].

3.2.4 Persistent muscle co-contraction: benefits and costs

Co-contraction—simultaneous activation of agonist and antagonist groups—emerges as the default protective strategy when the CNS lacks confidence in proprioceptive inflow [22,30,58,59,84–87]. In CLBP, this manifests as tonic paraspinal activity coupled with abdominal bracing and heightened tone in hip stabilizers [24,30,31,38,50,58,59,80–85]. Benefits include improved immediate stability and reduced unpredictable oscillations; costs include increased compressive/spinal shear loads, greater metabolic demand, and reduced movement variability [30,31,38,50,58,59,80–85].

Movement variability is not noise; it is a reservoir of adaptability. Reduced variability impairs the capacity to distribute loads across tissues and to discover less provocative movement solutions [31,38,58,59,80–85]. The co-contraction strategy, initially helpful, becomes self-reinforcing: rigidity reduces exposure to safe end-range motions that could restore neural excursion, ensuring that afferent noise remains high and the controller remains "tight" [30,31,38,50,58,59,80–85]. Over weeks to months, patients describe a sense of "armor" or "cement" in the low back—an experiential correlate of the protective policy [24,30,31,38,58,59,80–85,95–100].

3.2.5 Impaired postural control and anticipatory adjustments

Postural control can be conceptualized as a dynamic trade-off between stability and mobility. When proprioceptive confidence falls, the CNS increases baseline stiffness and reduces sway, especially in the frontal plane [22,30,58,59,84–87]. Paradoxically, less sway does not equal better control: the system becomes brittle, with diminished ability to absorb perturbations or accommodate rapid voluntary shifts [31,38,58,59,80–85].

In functional tasks (sit-to-stand, forward reach, lifting), patients with restricted neural mobility often substitute lumbar flexion for hip hinge during anterior load shifts [22,30–32,38,58,59,80–85]. This pattern minimizes hip angles that tension the sciatic pathway, but increases lumbar mechanical stress and may provoke symptoms [30–32,38]. They also demonstrate reduced anticipatory postural adjustments—delayed or diminished activation of deep trunk and hip musculature in preparation for movement—consistent with a system that relies on tonic co-contraction rather than rapid, context-specific modulation [24,30,31,38,50,58,59,80–85].

Encouragingly, postural control is trainable. As afferent fidelity improves (through restored neural excursion and exposure to variable but safe movement), patients recover adaptive sway and more efficient anticipatory strategies [31,38,58,59,80–85].

Clinically, this is observed as smoother transitions, easier off-loading of the hands during sit-to-stand, and less need for breath-holding [31,38,58,59,80–85,95–100].

3.2.6 Altered gait mechanics with sciatic involvement

Gait adaptations are among the most visible consequences of restricted neural mobility. Limiting terminal hip extension reduces tension along the sciatic tract, so patients shorten stride length and exhibit earlier heel rise and reduced trailing limb posture [31,32,58,59,80–85]. Pelvic rotation may be dampened, and arm swing guarded. In more pronounced cases, an antalgic (limping) pattern emerges to minimize time spent in provocative phases [31,32,38,58,59,80–85].

These adaptations shift work proximally: with reduced hip extension power, the trunk contributes more to propulsion, increasing lumbar shear [31,32,38,58,59,80–85]. Over time, this can exacerbate paraspinal tone, closing the loop between gait mechanics and protective co-contraction. The combination of shortened step length and stiff trunk is a hallmark signature when accompanied by positive neurodynamic findings (e.g., early symptom reproduction on SLR/Slump that modulates with structural differentiation) [31,33–37,39–41,50,58,59,72–77].

3.2.7 Mechanosensitivity and central amplification

Mechanosensitivity refers to lowered thresholds and amplified responses of neural tissues to mechanical stimuli. It can develop because excursion is restricted (abnormal strain focuses load on sensitized segments), and it can further suppress movement, shrinking the excursion envelope [15–19,27,28,30,60–63]. Moseley's pain neuroscience work clarifies the central layer: persistent nociceptive/threat input can recalibrate cortical and subcortical processing, blur body maps, and bias predictions toward danger [25,26,48,49,60–63,78,79]. In this state, normal movements are interpreted through a lens of threat; avoidance increases; the protective policy is confirmed [25,26,48,49,60–63,78,79,95–100].

Importantly, mechanosensitivity is plastic. Graded exposure to movement that respects irritability while expanding the neural excursion envelope can desensitize tissues and update central predictions [57,67–70,72–77,95–100]. Education that frames pain as protective signaling rather than damage reduces catastrophizing and supports the relearning needed to exit the loop [25,26,57,95–100].

3.2.8 The protective loop: an integrated model

Bringing these elements together yields a testable loop:

1.Trigger: Reduced neural excursion (adhesions, tunnel friction, chronic bracing) [15–19,27,28,30,60–63].

2.Afferent distortion: Noisier mechanoreceptor input; intermittent perfusion stress [15–19,27,28,30,60–63].

3.Control policy shift: Stability prioritized via co-contraction; reduced variability [24,30,31,38,50,58,59,80–85].

4.Behavioral confirmation: Avoidance of tensioning postures further limits excursion; safe exposure opportunities decline [31,32,37–41,58,59,80–85,95–100].

5.Central recalibration: Threat predictions rise; cortical representation blurs; descending modulation biases toward protection [25,26,48,49,60–63,78,79,95–100].

6.Mechanosensitivity: Amplified movement-evoked pain reinforces avoidance [15–19,27,28,30,60–63].

This loop does not claim universality for CLBP. Rather, it delineates a phenotype for which targeted assessment and loading strategies are likely to be effective [4–6,31,33–41,57,67–70,72–77,93–99].

3.2.9 Clinical assessment: discriminating mobility restriction from pure mechanosensitivity

A rigorous assessment distinguishes true mobility restriction (reduced sliding/excursion) from heightened sensitivity without excursion loss. Practically:

History: Symptoms provoked by positions that tension the sciatic/femoral pathways; relief with spinal unloading; avoidance of long-stride walking or deep hip flexion [27,28,30–32,38,58,59,80–85].

-Neurodynamic testing: Straight Leg Raise (SLR) and Slump with careful documentation of angle at symptom onset, symptom quality, and response to structural differentiation (e.g., ankle plantarflexion/dorsiflexion, cervical extension). A pattern where easing maneuvers change symptoms suggests sensitivity; persistent early limitation despite easing cues suggests excursion loss [1–3,33–37,39–41,72–77].

-Motor/proprioceptive testing: Trunk repositioning error, hip-hinge competence, single-leg stance with eyes closed (sensory reweighting), controlled reach tasks. Observe breathing mechanics—apical patterns and breath-holding signal global bracing [22,24,30–32,58,59,80–85,84–87,95–100].

-Gait analysis: Stride length, trailing limb angle, arm swing symmetry, pelvic rotation [31,32,38,58,59,80–85].

-Adjuncts: Surface EMG or myotonometry for resting tone; ultrasound (where available) to visualize nerve sliding. These are not diagnostic alone but help quantify change [24,50,58,59,80–85].

-Decision-making integrates pattern recognition: converging evidence of (a) neurodynamic limitation with (b) protective movement patterns and (c) proprioceptive deficits strongly supports the restricted-mobility phenotype [22,30–32,33–37,39–41,58,59,80–85,93–99].

3.2.10 Intervention: restoring excursion and reweighting control

An effective program addresses both the peripheral excursion problem and the central control policy [15–19,22,24,27,28,30–32,38,58,59,80–85,95–100]:

1.Graded neurodynamic loading. Begin with sliders (reciprocal joint motions to glide without substantial tension) to restore lubrication and reduce fear. Progress to tensioners once irritability decreases, carefully titrating end-range time and repetitions [1–3,15–19,27,28,33–37,39–41,72–77]. Sequencing matters: combine distal/proximal components to bias sliding early, tension later [33–37,39–41,72–77].

2.Motor coordination and breathing. Replace global bracing with targeted, elastic control. Train hip hinge and lumbo-pelvic dissociation; incorporate diaphragmatic breathing to down-regulate tonic co-contraction. Cue economy ("soft ribs, long spine") rather than rigidity [24,30–32,38,50,58,59,80–85,95–100].

3.Proprioceptive retraining. Use trunk repositioning drills, balance tasks with progressive sensory challenges, and eyes-closed practice to foster sensory reweighting [22,30,58,59,84–87,95–100]. External-focus instructions (task-oriented cues) encourage automatic control and reduce over-monitoring [58,59,80–85,95–100].

4.Cognitive framing. Provide pain neuroscience education: nerves are living tissues that adapt to load; graded exposure builds tolerance; transient discomfort need not signal harm [25,26,48,49,57,60–63,78,79,95–100]. Align expectations with the time course of tissue adaptation and the logic of retraining [57,67–70,93–99].

5.Functional integration. Embed gains into daily tasks: walking with attention to trailing limb hip extension, sit-to-stand with hinge emphasis, step-downs, and light loaded carries. The goal is transfer, not isolated exercise [31,32,37–41,58,59,67–70,80–85].

6.Dosing and progression follow irritability. Start with low volume, monitor 24-hour response, and advance one variable at a time (range, repetitions, speed, complexity) [33–37,39–41,57,67–70,72–77,93–99]. Include a flare plan (temporary dose reduction, unloaded mobility, breathing resets) to maintain engagement without reinforcing avoidance [33–37,39–41,57,67–70,95–100].

3.2.11 Illustrative clinical pathway

Week 0–2 (desensitize and orient): Education; sliders in supine SLR and seated Slump with structural differentiation; diaphragmatic breathing in 90/90; gentle hip hinge drills; short, frequent sessions [1–3,22,24,25,27,28,30–32,33–37,39–41,58,59,72–77,95–100].

Week 3–4 (expand envelope): Introduce mid-range tensioners; static balance with eyes closed; gait homework emphasizing trailing limb posture; reduce global bracing cues [22,30–32,37–41,58,59,67–70,72–77,80–85].

Week 5–8 (integrate): Progress tensioners toward end-range as tolerated; add loaded hinge (e.g., kettlebell deadlift pattern at low load); dynamic balance; context-specific tasks (lifting, reaching) [31,32,37–41,57,58,59,67–70,72–77,80–85,95–100].

Outcomes tracked: pain (NRS), disability (ODI/RMDQ), neurodynamic range (onset angle and symptom quality), resting tone, trunk repositioning error, gait parameters [33–37,39–41,58,59,84–87,93–99].

This pathway is hypothesis-driven: if restricted excursion is central, then improving glide and control should yield coordinated improvements across symptoms, tone, proprioception, and gait [15–19,22,24,27,28,30–32,33–41,57,58,59,67–70,72–77,80–85,93–100].

3.2.12 Measurement and expected change patterns

A multidomain measurement set strengthens inference:

-Symptoms/function: NRS at rest and movement; ODI/RMDQ; patient-reported global change [33–37,39–41,93–99].

-Sensorimotor: Trunk repositioning error; single-leg stance time (eyes closed); accuracy in hip-hinge tasks [22,30,31,38,58,59,84–87].

-Neurodynamic: SLR/Slump angles at symptom onset; change with structural differentiation; symptom quality [1–3,33–37,39–41,72–77].

-Physiology: Resting paraspinal tone via sEMG/myotonometry; perceived bracing (Likert) [24,50,58,59,80–85].

-Gait: Stride length, trailing limb hip extension, arm swing, pelvic rotation [31,32,38,58,59,80–85].

Expected pattern: Early improvements in symptom quality during neurodynamic tests (less “nerve pulling” with the same angle) and reduced resting tone precede larger changes in range and gait parameters. Proprioceptive accuracy improves alongside increased movement variability—patients report feeling “looser but more controlled” [31,33–37,39–41,58,59,80–85,93–99].

3.2.13 Research implications and falsifiable predictions

This model yields clear predictions:

1.Cross-sectional: CLBP participants with positive neurodynamic findings will show greater trunk repositioning error and reduced trailing limb hip extension versus matched controls [22,30,31,58,59,84–87].

2.Interventional: Adding graded neurodynamic loading to motor control produces larger gains in neurodynamic tolerance, resting tone, and gait variables than motor control alone [27,28,30,33–37,39–41,72–77].

3.Mediation: Improvements in proprioceptive accuracy and resting tone will mediate a portion of the effect on pain/disability, supporting the protective-loop mechanism [24,30,31,38,58,59,80–85,93–99].

4.Subgrouping: Baseline indicators of excursion limitation (e.g., early neurodynamic symptom onset not fully modulated by easing maneuvers) will predict better response to the combined program, guiding targeted care [1–3,33–37,39–41,72–77,93–99].

Null findings on these predictions would challenge the centrality of excursion restriction and prompt refinement (e.g., weighting psychosocial drivers more heavily) [25,26,48,49,57,60–63,78,79,95–100].

3.2.14 Limitations and alternative explanations

-Causality vs correlation. Proprioceptive deficits can arise from deconditioning, sleep disruption, attentional load, or fear. Restricted neural mobility is one contributor, not a universal cause [22,30,58,59,84–87,95–100].

-Test specificity. Neurodynamic tests load multiple tissues; distinguishing neural excursion loss from myofascial restriction is imperfect. Imaging of nerve glide shows promise but is not yet routine [1–3,15–19,27,28,33–37,72–77].

-Heterogeneity of CLBP. Psychosocial factors (depression, job strain, catastrophizing) strongly modulate pain and motor behavior; a comprehensive plan must address these alongside sensorimotor targets [25,26,48,49,57,60–63,78,79,95–100].

-Adaptive co-contraction. In some contexts (heavy lifts, unstable surfaces), co-contraction is appropriate. The problem in CLBP is loss of flexibility—the inability to down-regulate when task demands are low [30,31,38,50,58,59,80–85].

-Generalization. Gains in clinic tasks must generalize; hence the importance of early functional integration and context-specific practice [31,32,37–41,57,67–70,80–85,93–99].

These caveats encourage precision: match intervention to phenotype, measure broadly, and report both responders and non-responders [4–6,31–41,57,67–70,93–99].

3.2.15 Practical summary

-Suspect restricted neural mobility when patients avoid tensioning postures, show early neurodynamic symptoms that modulate with structural differentiation, walk short-strided, and brace through tasks [31,32,33–37,38,58,59,80–85].

-Start with sliders, add tensioners as irritability settles; pair with breathing and coordination work to unwind co-contraction [15–19,27,28,30,31,38,50,58,59,80–85].

-Retrain proprioception and sensory reweighting; embed gains into gait and meaningful tasks [22,30,31,38,58,59,84–87,95–100].

-Use education to shift threat predictions and support graded exposure [25,26,48,49,57,60–63,78,79,95–100].

-Track a multidomain outcome set to test mechanisms and guide progression [33–37,39–41,58,59,84–87,93–99].

3.2.16 Conclusion

Restricted neural mobility provides a parsimonious, falsifiable account of a CLBP phenotype characterized by proprioceptive disturbance, protective co-contraction, impaired postural control, and altered gait mechanics [22,24,30–32,31,38,58,59,80–85]. By degrading afferent fidelity, reduced excursion pushes the motor system toward stability-seeking strategies that are protective in the short term but costly over time [15–19,27,28,30,31,38,50,58,59,80–85]. Mechanosensitivity both arises from and sustains this loop, particularly under central amplification as described by Moseley and contemporary nociplastic pain models [25,27,28,30,48,49,50,60–63,78,79,95–100]. The model motivates a dual-pronged clinical strategy—restore excursion and reweight control—and a research agenda focused on mediation and subgrouping [15–19,22,24,27,28,30–32,33–41,57,58,59,67–70,72–77,84–87,93–99]. For patients whose presentations match this profile, this approach offers a credible route out of persistent dysfunction: move the nerve, calm the system, expand the options, and let adaptability replace protection [22,24,25,27,28,30–32,31,38,58,59,80–85,95–100].

3.3 Neurophysiological Mechanisms

Summary of the thesis so far. The protective motor responses described in Section 3.1 (increased tone, altered recruitment, segmental guarding) and their sensorimotor consequences in Section 3.2 (co-contraction, reduced variability, impaired postural control, altered gait) [22–26,30–32,38,50,58,59,80–85] do not arise in a vacuum. They are the output of a highly adaptive nervous system that is trying to preserve stability in the face of uncertain or threatening inputs [23,25,26,48,49,60–63,78,79,95–100]. The present section explains how that happens—step by step—from the periphery to the cortex, and back again to the muscles and movement strategies that patients with chronic low back pain (CLBP) enact [4–6,23–26,30–32,38,48,49,58,59,60–63,78,79,80–85,95–100]. We begin with peripheral transduction under conditions of reduced neural excursion, move through segmental (spinal) circuitry emphasizing the gamma loop and reflex organization, then detail descending modulation and predictive coding, before outlining supraspinal reorganization (map precision, motor planning, sensory reweighting). We close with neuroimmune and autonomic contributors, clinical implications, testable predictions, and limitations [23–28,48,49,60–63,78,79,95–100].

3.3.1 Why restricted neural mobility changes the “meaning” of movement

Restricted neural mobility alters the cost and content of afferent input arising during otherwise ordinary motions. When sliding/gliding of neural tissue relative to its interfaces is reduced—by adhesions, paraneural thickening, tunnel friction, or chronic bracing that diminishes relative motion—mechanical loads that were once distributed smoothly across a long tissue path become locally concentrated [15–19,27,28,30,60–63]. This changes mechanotransduction at multiple scales:

1.Channel-level effects. Mechanosensitive ion channels on nociceptors and low-threshold mechanoreceptors become more likely to open for a given joint excursion; thresholds fall and receptive fields may expand. The result is mechanosensitivity—elevated responses to stretch, compression, or sliding that were previously innocuous [15–19,27,28,30,60–63].

2.Perfusion–tension coupling. Endoneurial microcirculation is exquisitely sensitive to pressure. Tension or focal compression that intermittently compromises flow can produce short episodes of hypoxia or metabolite accumulation, which in turn bias transduction toward nociception and after-discharge [15–19,27,28,30,60–63]. Even small, repeated episodes condition the system to “expect” threat near certain ranges.

3.Interface signaling. Paraneural and interfascicular matrices host mechanoreceptors and immune-competent cells. Reduced glide increases friction/micro-shear and can promote local release of sensitizing mediators (e.g., cytokines, prostaglandins), which lower nociceptor thresholds and stiffen the very interfaces that need to slide—closing a local positive feedback loop [17–19,27,28,60–63].

In short, the same movement now produces more, earlier, and noisier neural signals. The CNS, facing a degraded signal-to-noise ratio, rationally shifts toward high-stability solutions—co-contraction and guarded motion—to preserve control [31,38,50,58,59,80–85]. That protective state reduces exposure to tensioning/slider positions that would restore glide, thereby perpetuating the altered afference. This is the periphery's contribution to the protective attractor [15–19,27,28,30,31,38,50,58,59,80–85].

3.3.2 Segmental processing: the gamma loop and spinal reflex organization

At the spinal level, afferent signals converge onto dorsal horn neurons that project, via interneurons, to alpha and gamma motor pools. Several well-known circuit motifs explain how persistent threat-colored input from restricted neural tissues becomes tonic guarding:

-Gamma motor drive and muscle spindle tuning. Nociceptive/protective afference increases gamma efferent activity, tightening intrafusal fibers and heightening muscle spindle sensitivity [23,25,27,28,60–63]. Spindles now discharge earlier and more vigorously for small length changes, amplifying Ia feedback to alpha motor neurons. The system becomes high-gain, meaning small perturbations elicit larger motor responses. Clinically this is palpable as heightened resting tone, bracing, and delayed relaxation after movement [24,30,31,38,50,58,59,80–85].

-Flexor dominance under threat. Protective circuits bias toward flexion synergies (flexor withdrawal/crossed-extension). Even when below the threshold for overt withdrawal, repeated subthreshold activation shifts motor programs toward lumbar flexion as a default strategy in forward tasks. This preference reduces hip hinge and terminal hip extension, helping the patient avoid positions that tension the sciatic tract (Section 3.2), but at the cost of increased lumbar load [23,25,31,32,38,58,59,80–85].

-Interneuronal disinhibition. Persistent nociceptive input can reduce inhibitory interneuron tone (including Renshaw-mediated recurrent inhibition), effectively disinhibiting alpha motor pools. The net effect is easier recruitment and harder relaxation, supporting co-contraction around lumbopelvic segments [27,28,30,50,60–63].

This local circuitry produces exactly the motor phenotype we observe: stability prioritized over mobility, reduced movement variability, and tonic paraspinal/abdominal activation [22–26,30–32,31,38,50,58,59,80–85]. Importantly, none of this implies "weakness" or "damage"; it is a strategy adopted by a nervous system dealing with noisy or threatening inputs [23,25,26,48,49,95–100].

3.3.3 Descending modulation and predictive coding: when priors outrun evidence

Spinal processing is not autonomous; it is continuously shaped by descending control from brainstem, cerebellar, and cortical sources. Under conditions of persistent threat, the balance of descending modulation shifts toward facilitation of nociceptive transmission through pathways such as the periaqueductal gray (PAG) to the rostroventral medulla (RVM) [25,26,48,49,60–63,78,79]. At the same time, supraspinal inference systems rely more heavily on priors—the brain's predictions about what will happen during movement—than on incoming sensory evidence. This is the predictive coding account of persistent pain [25,26,48,49,60–63,78,79,95–100]:

-After weeks or months of movement-evoked pain, priors such as "deep hip flexion is dangerous" or "long stride = nerve pain" acquire high precision (confidence). Ambiguous inputs are interpreted through that lens. The safest way to reduce prediction error is not to gather new evidence (e.g., move and update the model), but to avoid the movement that might contradict the prior. This is rational—until it becomes imprisoning [25,26,48,49,60–63,78,79,95–100].

-Descending facilitation raises spinal gain so that movement-evoked afference is more likely to be categorized as "threat," which then justifies further avoidance. The patient's behavior (guarding, moving stiffly) then confirms the prior, because the sensations accompanying rigid movement are indeed more intense and unpleasant [25,26,48,49,60–63,78,79,95–100].

Education that reframes pain as protective signaling (not damage), combined with graded exposure to movement that is designed to succeed, works by lowering prior precision and re-weighting sensory evidence—helping the system recategorize previously threatening inputs [25,26,57,67–70,95–100].

3.3.4 Supraspinal reorganization: maps, planning, and sensory reweighting

Long-standing bracing and reduced variability are accompanied by plastic changes in supraspinal networks [23,25,31,38,50,58,59,80–85]:

1.Somatosensory maps (S1/S2). Persistent nociception and disuse of certain ranges reduce the distinctness ("sharpness") of the lumbopelvic representation—sometimes described as representational smudging. Map imprecision degrades position sense and contributes to the subjective feeling that the back is "stiff" or "not under fine control" [23,25,58,59,80–85].

2.Motor cortex (M1) and premotor planning. With the system biased toward safety, M1 issues coarse, high-stiffness commands that are easy to stabilize but inefficient. Premotor areas pre-select movement templates that avoid tensioning the neural pathways (e.g., bending from the spine rather than hinging at the hips). Task switching slows; anticipatory postural adjustments (APAs) are blunted [24,30,31,38,58,59,80–85].

3.Posterior parietal cortex (PPC) and body schema. The PPC integrates multisensory inputs into a coherent body schema. Noisy afference plus disuse of end-range postures produce a brittle schema for the lumbopelvic region; the patient relies more on vision and conscious monitoring. Ironically, "watching and controlling" the back tightens it further [23,25,58,59,80–85,95–100].

4.Cerebellum and basal ganglia. The cerebellum, which uses error signals to update internal models, sees noisy feedback and "chooses" predictive stiffness to minimize error. The basal ganglia, as policy selectors, prefer low-variability, high-stability strategies under uncertainty. Both tendencies fit the observed phenotype [23,25,31,38,58,59,80–85,95–100].

Sterling's work links these central adaptations—particularly impaired cortical representation—to altered motor planning/execution in persistent pain states, and shows they are trainable with targeted sensory-motor work [23,25,80–85,95–100].

3.3.5 Neuroimmune and autonomic contributions: the protective milieu
The nervous system does not adapt in isolation. Glia (microglia, astrocytes) in the dorsal horn and supraspinal centers respond to persistent nociceptive input by releasing cytokines that sensitize neurons, effectively raising the "volume" on threat signaling [15–19,27,28,30,60–63,78,79]. Peripherally, subtle paraneural inflammation at restricted interfaces maintains peripheral sensitization [15–19,27,28,60–63]. Concurrently, autonomic outputs shift toward sympathetic dominance in many patients, altering intraneural blood flow and skin vasomotor/sudomotor tone. These changes are usually subclinical but shape perception and performance: cooler, sweatier skin; a limb that "feels different"; minor trophic changes—all consistent with an aroused system [25,26,48,49,60–63,78,79,95–100].

Crucially, these processes are plastic. Reduced nociceptive drive, improved movement variability, and autonomic down-titration (e.g., breathing retraining) can quiet glial activation and normalize sympathetic tone [23,25–28,57,60–63,78,79,95–100].

3.3.6 Putting the pieces together: why protection persists

The hallmark of this model is a multi-level attractor state:

-Periphery: restricted glide → distorted mechanotransduction + perfusion stress → mechanosensitivity [15–19,27,28,30,60–63].

-Spinal: increased gamma drive + disinhibition → high-gain motor pools → co-contraction/guarding [23,24,27,28,30,31,38,50,58,59,80–85].

-Supraspinal: threat-shaped priors + descending facilitation → protective categorization of movement sensations [23,25,26,48,49,60–63,78,79,95–100].

-Behavior: avoidance of tensioning postures → less glide exposure → further excursion loss [31,32,37–41,58,59,80–85,95–100].

-Maps/planning: smudged S1 + conservative M1/PPC templates → coarse control, blunted APAs [23,25,31,38,58,59,80–85,95–100].

-Milieu: glial/autonomic tone supports high vigilance [15–19,25–28,48,49,60–63,78,79,95–100].

Each tier confirms the others. The system "makes sense" of itself and therefore tends to remain as it is [15–19,23–28,30,31,38,48,49,58,59,60–63,78,79,80–85,95–100]. Escaping the attractor requires coordinated changes in input (restore glide), gain (reduce gamma bias), priors (education + exposure), and maps (proprioceptive/motor retraining). That is why unimodal interventions—only stretching, only strengthening, or only information—often disappoint this subgroup [4–6,31–41,57,67–70,72–77,93–99].

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3.3.7 Clinical implications: assessment that probes mechanisms

A mechanism-aware exam seeks converging evidence for the neurodynamic-restriction phenotype:

-History red flags for restriction: Symptoms when positions specifically tension sciatic/femoral pathways (long stride, deep hip flexion, slump-like postures); fast relief with spinal unloading; prominent morning stiffness that eases with gentle sliders rather than static stretches [27,28,30–32,38,58,59,80–85].

-Neurodynamic testing: Straight Leg Raise (SLR) and Slump, with meticulous documentation of (a) angle at symptom onset, (b) symptom quality ("nerve stretch" vs muscle), and (c) response to structural differentiation (ankle/cervical). Early onset with partial modulation suggests mechanosensitivity; early onset despite easing maneuvers suggests excursion loss [1–3,27,28,33–37,39–41,72–77].

-Tone and coordination: Palpable resting paraspinal tone; delayed relaxation; breath-holding during movement; hip hinge substituted by lumbar flexion [24,30,31,38,50,58,59,80–85].

-Proprioception: Increased trunk repositioning error; excessive visual dependence; poor single-leg stance with eyes closed (Section 3.2) [22,30,58,59,84–87,95–100].

-Gait: Shortened stride, reduced trailing-limb hip extension, guarded arm swing, damped pelvic rotation [31,32,38,58,59,80–85].

-Autonomic nuance: Asymmetries in skin temperature/sweat or color that track symptom changes (mild, not necessarily pathological) [25,26,48,49,60–63,78,79,95–100].

No single test is decisive; pattern recognition across domains strengthens inference [33–37,39–41,57,93–99].

3.3.8 Clinical implications: intervention that changes the system, not just the symptom

An effective plan addresses both peripheral and central mechanisms [15–19,22,24,27,28,30–32,38,48,49,58,59,60–63,78,79,80–85,95–100]:

1.Restore excursion with graded neurodynamic loading. Start with sliders—reciprocal joint motions that glide nerves with minimal tension—to reduce fear, improve lubrication, and re-expose the interface to movement. Progress to tensioners as irritability settles, treating time-in-range as a dose variable [1–3,15–19,27,28,33–37,39–41,72–77]. Use sequencing to bias sliding early and tension later [33–37,39–41,72–77]. Pair with non-threatening language ("glide," "polish," "floss" rather than "stretch the nerve").

2.Reduce gamma bias via breathing and coordination. Replace global bracing with diaphragmatic breathing, rib mobility, and graded relaxation. Train hip hinge and lumbo-pelvic dissociation to re-introduce efficient strategies that do not over-tension the neural path. Cue "tall and elastic" rather than "brace and protect" [23,24,30–32,38,50,58,59,80–85,95–100].

3.**Update priors with education and graded exposure.** Use concise pain neuroscience explanations to normalize sensations and frame progress [25,26,48,49,57,60–63,78,79,95–100]. Design success-biased exposures—brief, frequent, sub-symptom practices that incrementally challenge the prior without provoking large errors [57,67–70,93–99].

4.Sharpen maps and reweight senses. Add proprioceptive retraining (trunk repositioning drills; balance with eyes closed; laterality/imagery tasks; light tactile discrimination) to refine S1 and speed the return of fine motor control [22,23,30,58,59,84–87,95–100].

5.Integrate into function. Gait homework emphasizes trailing-limb hip extension and arm swing; sit-to-stand with hinge emphasis; graded lifting with elastic posture. Mechanistic gains must be spent on meaningful tasks [31,32,37–41,58,59,67–70,80–85].

6.Manage flares without backsliding. Temporary dose reductions, unloaded mobility, and breathing resets maintain engagement while respecting irritability. The message remains: we are training adaptability, not chasing zero sensation [33–37,39–41,57,67–70,95–100].

7.Measurement is multi-domain. Pain/disability, neurodynamic onset angle and symptom quality, resting tone (sEMG/myotonometry if available), proprioception, and gait parameters. Expect quality changes first (less neural character at the same angle), then range, then automaticity [33–37,39–41,58,59,84–87,93–99].

3.3.9 Testable predictions and research agenda

The model yields falsifiable predictions:

-Cross-sectional. CLBP patients with positive neurodynamic findings will show greater trunk repositioning error and reduced trailing-limb hip extension than matched controls [22,30,31,58,59,84–87].

-Interventional. Adding graded neurodynamic loading to motor control training will produce larger improvements in neurodynamic tolerance (angle/quality), resting tone, and gait variables than motor control alone [27,28,30,33–37,39–41,72–77].

-Mediation. Improvements in proprioceptive accuracy and resting tone will partially mediate gains in pain/disability, consistent with the protective-loop mechanism [24,30,31,38,58,59,80–85,93–99].

-Subgrouping. Baseline indicators of excursion limitation (e.g., early onset despite easing maneuvers) will predict superior response to the combined program—useful for targeted care [1–3,33–37,39–41,72–77,93–99].

Null results on any of these axes would help refine (or refute) the centrality of excursion restriction for this phenotype [25,26,48,49,57,60–63,78,79,95–100].

3.3.10 Limitations and alternatives

-Heterogeneity of CLBP. Not all persistent low back pain features neurodynamic restriction; discogenic, facet, myofascial, and psychosocial drivers are common. Our aim is precision, not universality [4–6,25,26,31,38,48,49,57,60–63,78,79,95–100].

-Test specificity. Neurodynamic tests load multiple tissues; specificity for "true excursion loss" vs mechanosensitivity is imperfect. Imaging of nerve glide shows promise but is not yet routine [1–3,15–19,27,28,33–37,72–77].

-Confounds. Sleep, mood, and systemic inflammation modulate pain and motor behavior. They should be screened and addressed as appropriate [25,26,48,49,57,60–63,78,79,95–100].

-Adaptive co-contraction. Stiffness is appropriate for some tasks. The problem is loss of flexibility—inability to down-regulate when demands are low [30,31,38,50,58,59,80–85].

These caveats underscore the need for careful phenotyping and transparent reporting of responders and non-responders [4–6,31–41,57,67–70,93–99].

3.3.11 Practical take-home (for clinicians and researchers)

-Think systems, not parts: restricted glide changes afference → gamma loop gain → maps and priors → behavior that protects but persists [15–19,23–28,30,31,38,48,49,58,59,60–63,78,79,80–85,95–100].

-Assess patterns: early neurodynamic symptoms, modulation with structural differentiation, guarded gait, braced breathing, proprioceptive error [1–3,22,24,30–32,33–37,38–41,58,59,80–85,84–87,93–99].

-Intervene on all levels: glide (sliders/tensioners), gain (breathing/coordination), priors (education/exposure), maps (proprioception), function (task integration) [15–19,22,24,25,27,28,30–32,33–41,57,58,59,67–70,72–77,80–85,93–100].

-Measure broadly and expect sequencing: quality → range → automaticity; hip extension and arm swing are sensitive gait markers [31–33,37–41,58,59,80–85,93–99].

3.3.12 Additional mechanistic refinements and clinical corollaries

Dorsal root ganglion (DRG) as a gain node. Beyond dorsal horn processing, the DRG operates as an active gain controller for movement-evoked afference. DRG neurons exhibit activity-dependent changes in membrane excitability (including shifts in persistent sodium and hyperpolarization-activated currents) that can bias afferent coding toward after-discharge during repeated mechanical loading. Under conditions of reduced neural excursion—where focal tension and compression are more frequent—DRG excitability can drift upward, such that identical joint excursions produce larger inflow to the cord. Clinically, this presents as the familiar "second set worse than the first" phenomenon during repetitive tasks: not simply fatigue, but a peripheral sensory wind-up that encourages earlier protective recruitment [27,28,60–63,78,79].

Ion-channel phenotype and mechanosensitive transducers. While mechanosensitivity has been discussed conceptually, an important nuance is the phenotypic plasticity of ion channels in peripheral afferents. Up-regulation of mechanically activated channels (e.g., Piezo-like conductances) and voltage-gated sodium subtypes with slow inactivation kinetics can emerge with ongoing interface irritation. This combination increases both the probability of firing during modest strain and the persistence of firing once movement stops—explaining why some patients report lingering "zinging" after a single provocative posture. Because channel expression adapts to the history of mechanical input, even small improvements in glide that reduce focal stress may have outsized benefits over weeks by normalizing transducer density [17–19,27,28,60–63].

Viscoelastic thixotropy of neural interfaces. The paraneural/extraneural matrix is viscoelastic and thixotropic—its apparent viscosity drops with gentle cyclic motion. In a restricted state, lack of low-load oscillation allows the matrix to remain "gelled," increasing friction and micro-shear when movement finally occurs. This insight justifies prescribing brief, frequent slider exposures across the day rather than longer, infrequent sessions: the goal is to keep the interface in a low-viscosity regime so strain can distribute over distance rather than concentrate at a single adhesion. It also clarifies why long static stretches can backfire in sensitive patients: they load the interface before viscosity has dropped, exaggerating focal stress and DRG gain [15,16,27,28,72–77].

H-reflex/Hoffmann modulation as a window on spinal set-point. When feasible, measuring H-reflex amplitude and recovery curves in paraspinal-related muscles can index segmental excitability. A treatment trajectory that truly reduces gamma bias and segmental gain should produce subtle reductions in resting H-reflex amplitude and faster post-activation depression—physiological correlates of a controller that no longer needs constant co-contraction. Even if such measures are not available clinically, they suggest a principle: track proxy markers of segmental set-point (e.g., relaxation time after a standardized forward bend, or EMG co-activation indices during a light hinge) to confirm that neurophysiological "un-tightening" accompanies symptom change [50,58,59,80–83].

Interhemispheric balance and laterality. In persistent lumbopelvic pain, altered interhemispheric inhibition between motor cortices can bias output toward robust, low-fidelity commands. Laterality tasks (left/right trunk or hip orientation judgments) and implicit motor imagery often reveal asymmetric latencies or accuracy decrements.

Importantly, these tasks can be trained in parallel with exposure: short blocks of laterality and imagery immediately before gliding can prime more precise cortical maps, lowering the "cost" of the subsequent movement dose. The practical corollary is to front-load sessions with lightweight cortical priming (30–60 seconds of laterality/imagery) to nudge planning networks toward finer control [23,25,80–82].

Cerebellar prediction error and dosing cadence. The cerebellum updates internal models when prediction errors are informative but not overwhelming. Very high errors (large flares) are treated as outliers and do not update the model; very low errors (no challenge) provide no new information. This argues for micro-progressions that consistently generate just-noticeable discrepancies between expected and actual sensation. A practical rule: adjust one variable at a time (range, reps, speed, or context), hold constant for 48–72 hours to allow consolidation, then escalate. This cadence respects both tissue adaptation and cerebellar learning windows [67–70,82,83].

Neurovascular coupling and breathing mechanics. Neural perfusion is sensitive to CO_2/O_2 balance and intrathoracic pressure swings. Apical, breath-held patterns during effort transiently reduce venous return and can worsen intraneural congestion at the lumbosacral roots. Teaching low-threshold diaphragmatic breathing during exposure is not merely "relaxation"; it optimizes neurovascular coupling so that the same mechanical dose is delivered with better perfusion—another reason quality changes (less neural character at the same angle) often precede range changes [27,28,60–62].

Autonomic state as a limiter of cortical plasticity. Elevated sympathetic tone narrows attentional focus and fosters rigid motor policies, features commonly described in centrally sensitised and nociplastic pain presentations [60–63,78,79]. Short, pre-dose autonomic down-regulation (one minute of paced breathing, eyes-softened gaze) can increase cortical receptivity to updating and support more flexible motor strategies [60–63,78,79,83]. Consider a three-step prelude before any exposure set: (1) 6–8 slow breaths; (2) 30–60 seconds of laterality/imagery; (3) one rehearsal of the movement with a "long spine, soft ribs" cue. This sequence costs ~2 minutes and improves both tolerance and learning efficiency, cohering with contemporary views of CLBP as an interaction between pain, movement, and behaviour change rather than a purely peripheral phenomenon [67–70,83,100].

Contextual interference and variability-for-learning. Once irritability decreases, introducing contextual interference—mixing tasks or slightly varying constraints—enhances long-term retention of flexible control [67–70,80–85]. For example, alternate seated slump sliders with standing knee-extension sliders, or interleave small gait drills emphasizing trailing-limb extension between slider sets. The aim is to re-teach the system that many solutions exist, countering the "one safe pattern" policy that typifies protective control in CLBP and is reflected in reduced movement variability and stereotyped trunk strategies [58,59,80–85].

Candidate biomarkers and simple clinic proxies. While advanced tools (TMS, H-reflex, microneurography) are research-grade, clinics can track low-burden proxies tied to mechanism: (a) time-to-relax after forward flexion; (b) breath-hold frequency during three standardized tasks; (c) laterality accuracy/latency in a brief app-based test; (d) trailing-limb hip extension measured with a phone inclinometer during gait;

(e) symptom quality shift at a fixed neurodynamic angle. Trajectories that improve in two or more proxies alongside symptoms support true mechanism change rather than mere compensation and align with recommended outcome domains and psychometric tools in CLBP research [58,59,80–85,93–99].

A principled flare algorithm. If symptoms spike >24 hours post-dose, cut only one variable by ~30% (usually range or volume), maintain breathing/imagery priming, and add a viscosity reset: 60–90 seconds of ultra-low-range sliders each hour for the next workday. The goal is to "re-liquefy" the interface and drain the DRG gain without abandoning progress—a physiology-consistent way to prevent backsliding [15,16,27,28,72–77].

Together, these refinements extend the neurophysiological account into concrete levers—ion-channel phenotype, DRG gain, thixotropy, cerebellar dosing, autonomic priming, interhemispheric balance—that can be targeted with small, testable adjustments. They also specify objective proxies to verify that the system is truly moving from a high-gain, low-variability attractor toward a low-threat, high-flexibility regime—exactly the shift this thesis proposes to engineer [15–19,23,25,27,28,30,31,38,50,58,59,60–63,67–70,72–77,78,79,80–85,93–99,100].

3.3.13 Conclusion

Restricted neural mobility provides a coherent, testable mechanism for a recognizable CLBP phenotype. By increasing mechanosensitivity and distorting afference, it drives gamma-mediated spinal gain and recruits supraspinal processes—predictive coding, map imprecision, conservative planning—that favor stability at the expense of adaptability [23,25,27,28,30,48,49,60–63,78,79]. The result is the clinical picture of co-contraction, reduced variability, impaired postural control, and altered gait [22–26,30–32,31,38,50,58,59,80–85]. Because the state is maintained by reciprocal confirmation across levels, effective care must be multimodal and mechanism-aligned: restore glide, reduce gain, update priors, sharpen maps, and embed gains in function [1–3,15–19,22,24,25,27,28,30–32,33–41,57–59,67–70,72–77,80–85,93–99,100]. This approach is not only biologically plausible but also falsifiable, with clear clinical markers and outcome trajectories that can confirm or challenge the model in real patients [4–6,31–41,57,67–70,72–77,93–99].

3.4 Clinical Case Example: Femoral Nerve Restriction

3.4.1 Case overview and clinical question

Patient profile. A 38-year-old office worker presents with a 12-month history of chronic low back pain (CLBP) characterized by stiffness on standing after prolonged sitting, difficulty achieving an upright posture from a seated position, and intermittent anterior thigh discomfort on the right. The patient reports that symptoms are worst late in the workday and after long car rides. Sleep is unremarkable, and there is no constitutional history, trauma, or prior spinal surgery. Activity level is low-to-moderate: short daily walks and occasional recreational cycling. Analgesics are used sparingly. Fear of movement is mild; motivation for active care is high [4–6,22,25,30,57,95–100].

Initial observation. Standing posture demonstrates anterior pelvic tilt with increased lumbar lordosis. During sit-to-stand, the patient initiates movement with a rapid lumbar extension "thrust" and then pauses before fully upright posture—suggesting a momentary guarding or uncertainty in lumbopelvic control. In quiet stance, paraspinal tone appears elevated, with visible breath-holding during trunk movements [24,30–32,38,50,58,59,80–85].

Index tests.

-Prone knee bend (PKB) on the right: discomfort localized to the low back and anterior thigh at ~90° of knee flexion, without dermatomal radiation [33–37,39–41].

-Tenderness on palpation along the proximal anterior thigh, tracking the femoral nerve course beneath the inguinal ligament [17–21,37,40,88–92].

-Slump test: negative for posterior leg symptoms; cervical flexion does not modulate sensations [1–3,33–37].

-Hip flexor assessment: moderate iliopsoas hypertonicity; passive hip extension limited on the right relative to the left [30–32,38].

-Neurologic screen: intact strength, reflexes, and sensation; no red flags [4–6].

Provisional hypothesis. Findings are compatible with a subclinical femoral nerve restriction—that is, reduced neural excursion/glide of the femoral pathway across the anterior hip region—contributing to altered afferent input and a protective lumbopelvic control strategy that emphasizes anterior pelvic tilt and lumbar extension [22,27,28,30–32,37–41,58,59,80–85]. The negative Slump test and the localization of symptoms to the anterior thigh/low back during PKB support a femoral pathway emphasis rather than sciatic-dominant involvement [1–3,33–37,39–41]. The clinical question is whether a mechanism-aligned program—femoral nerve sliders, interface-friendly hip extension progression, iliopsoas load-management, and motor control focusing on neutral pelvic alignment—can restore excursion, reduce protective gain, and normalize function [22,27,28,30–32,37–41,58,59,80–85,93–99].

3.4.2 Differential diagnosis and reasoning

1.Lumbar facet-dominant pain with extension bias. The patient's anterior pelvic tilt and extension "thrust" could suggest facet irritation. However, the provocation pattern (PKB-evoked anterior thigh/low back discomfort) and anterior pathway tenderness argue for anterior neural interface involvement rather than purely posterior element irritability. The absence of clear extension/rotation pain on quadrant testing further reduces the likelihood that facets are primary [4–6,30–32,31,38].

2.Discogenic pain. Discogenic features typically include flexion sensitivity, sustained flexion intolerance, or morning pain and stiffness with a "warm-up" phenomenon. Our patient's main aggravator is prolonged sitting with difficulty standing upright, but flexion itself is not strongly provocative, and there is no radicular symptomatology. Discogenic contribution is possible but not central to the reproducible findings [4–6,30–32,31,38].

3.Hip flexor myofascial syndrome. Iliopsoas hypertonicity is present and clearly contributes to anterior pelvic tilt. Yet, nerve-biased tests (PKB) reproduce discomfort earlier than expected for a pure muscular restriction, and palpation along the femoral course elicits familiar symptoms. Moreover, symptom modulation with neurodynamic sliders is expected to be superior to muscle-only interventions if the neural interface is the key driver [22,27,28,30–32,37–41,58,59].

4.Femoral nerve neurodynamic restriction (primary). The pathophysiology is coherent: reduced excursion of the femoral nerve under the inguinal ligament or within the iliacus tunnel increases mechanosensitivity and alters afferent fidelity during hip extension and knee flexion; the CNS prioritizes stability via anterior pelvic tilt and lumbar extension to avoid neural tension; proprioceptive uncertainty appears during sit-to-stand and upright control. The presentation matches a neurodynamic-restriction phenotype for the femoral pathway [22,27,28,30–32,37–41,58,59,80–85].

Conclusion. Differential favors femoral neurodynamic restriction with secondary hip flexor hypertonicity and tertiary facet loading due to extension bias. The working diagnosis remains subclinical femoral nerve restriction; confirmatory evidence will be sought through structured dosing and objective response patterns [22,27,28,30–32,37–41,58,59,80–85,93–99].

3.4.3 Mechanistic links specific to the femoral pathway

Anatomical/course considerations. The femoral nerve emerges from the posterior divisions of L2–L4, passes through the psoas major, courses between psoas and iliacus, and traverses under the inguinal ligament lateral to the femoral artery before dividing into anterior and posterior branches. Sites of reduced excursion may include: (1) within the psoas/iliacus compartment (adhesion, local fibrosis), (2) at the inguinal ligament (tunnel friction), and (3) at fascial interfaces in the proximal anterior thigh.

In positions of hip extension and knee flexion (e.g., PKB), longitudinal strain and sliding demands are greatest; restricted glide focuses strain, amplifies mechanotransduction, and degrades afferent fidelity [17–21,27,28,30,37,40,88–92].

Afferent consequences. Noisy or threat-colored input from the femoral pathway biases spinal circuits via gamma gain, increasing spindle sensitivity in anterior hip and lumbar stabilizers and locking in anterior tilt as a protective strategy. Supraspinally, predictive coding assigns higher danger priors to movements that bias neural tension (hip extension), leading to habitual avoidance of full trailing-limb posture in gait and a preference for lumbar extension during sit-to-stand [23,25,27,28,30–32,38,48,49,58,59,60–63,78,79,80–85].

Clinical translation.

-PKB sensitivity at mid-range indicates the pathway is “expensive” to load [27,28,30,33–37,39–41].

-Anterior pelvic tilt maintains hip flexion at rest, minimizing femoral tension [30–32,38,58,59,80–85].

-Reduced hip extension in gait preserves comfort but shifts load proximally to the lumbar spine [31,32,38,58,59,80–85].

-Iliopsoas hypertonicity is partly reactive/secondary, sustained by gamma-biased gain and avoidance of end-range hip extension [24,30–32,38,50,58,59,80–85].

3.4.4 Examination details and baseline measures

Observation and movement.

-Sit-to-stand: early lumbar extension thrust, brief pause, then completion—suggesting a stability-seeking strategy [30–32,38,58,59,80–85].

-Hip hinge: poor dissociation; knee flexion substitutes for hip extension; lumbar lordosis increases early [30–32,38,58,59,80–85].

-Gait: shortened stride length; conspicuously limited trailing-limb hip extension; muted arm swing; reduced pelvic rotation [22,31,32,38,58,59,80–85].

Neurodynamic tests (femoral bias).

-Prone knee bend (PKB): symptom onset at ~90° on the right; local anterior thigh/low back discomfort; reduction with cervical extension is minimal; brief reduction with

ankle plantarflexion is inconsistent (as expected for femoral rather than sciatic bias) [27,28,30,33–37,39–41,72–77].

-Sidelying femoral slump variant (hip extension with knee flexion while stabilizing pelvis) increases anterior thigh pull, confirming pathway sensitivity [27,28,33–37,39–41,72–77].

-Slump (sciatic bias): negative [1–3,33–37].

Proprioception/coordination.

-Trunk repositioning error: increased vs normative; variable path back to neutral [22,30,58,59,84–87].

-Breath-hold frequency: present during hinge and PKB testing [24,30–32,38,50,58,59,80–85].

-Time-to-relax (palpated paraspinals after forward bend): delayed [24,30,31,38,50,58,59,80–85].

Outcome set (baseline).

-Pain NRS: 6/10 at day end; 3/10 at rest [93–99].

-ODI or RMDQ: elevated (patient-specific) [93–99].

-PSFS: difficulty standing upright after sitting (score 4/10); difficulty walking >20 minutes with comfortable posture (5/10) [93–99].

-PKB angle at onset: 90° [27,28,33–37,39–41,72–77].

-Gait trailing-limb hip extension: visually limited; phone inclinometer suggests ~5–8° below expected [22,31,32,38,58,59,80–85].

These measures sample mechanisms from periphery to behavior and will be used to track sequencing of change.

3.4.5 Treatment rationale and design (mechanism-aligned)

Primary therapeutic aims.

1.Restore femoral nerve excursion across the anterior hip interface (reduce local strain concentrations; improve afferent fidelity) [17–19,22,24,27,28,30–32,37–41,58,59,80–85].

2.Lower gamma bias and global bracing via breathing and motor re-education [23–25,30–32,38,50,58,59,80–85,95–100].

3.Rebuild proprioceptive confidence for an upright neutral pelvis and hip extension [22,30–32,58,59,84–87,95–100].

4.Spend gains in functional tasks (sit-to-stand, gait) to update priors and consolidate transfer [23–25,31,32,37–41,57,58,59,67–70,80–85,93–99].

Core components.

-Femoral nerve sliders (glide > tension) in early stages to exploit thixotropy and reduce fear [15–19,27,28,33–37,39–41,72–77].

-Iliopsoas load-management (graded lengthening/strength in ranges that do not spike neural strain) [22,27,28,30–32,37–41,58,59,80–85].

-Pelvic tilt control—motor control exercises that cue neutral alignment without rigid bracing [24,30–32,38,50,58,59,80–85,95–100].

-Gait drills emphasizing trailing-limb hip extension and arm swing, introduced as soon as symptom quality allows [22,31,32,37–41,58,59,67–70,80–85].

-Brief autonomic/cortical priming (paced breathing + imagery) before exposure to reduce cost per dose and improve learning [23,25,26,57,60–63,78,79,83,95–100].

3.4.6 Week-by-week plan, dosing, and progression

General dosing rules.

-Sliders first; tensioners later, after quality shifts [15–19,27,28,33–37,39–41,72–77].

-Change one variable at a time (time-in-range → angle → reps → context) [57,67–70,93–99].

-Observe 24-hour response; consolidate for 48–72 hours before the next change [33–37,39–41,57,67–70,72–77].

-Use a flare algorithm: single-variable rollback (~30%), hourly micro-sliders for 1 day, retain priming [15,16,27,28,72–77,95–100].

Week 0–1: Desensitize and orient

Session priming (2 minutes):

-6–8 slow breaths (down-regulate sympathetic tone).

-30–45 s of laterality/imagery (visualize femoral pathway glide during hip extension).

-One rehearsal with cue: “long spine, soft ribs; pelvis floats” [23,25,26,57,60–63,78,79,83,95–100].

Femoral sliders (supine/standing variants):

-Supine femoral slider (hip in slight extension, alternate knee flexion ↔ hip flexion to bias glide without sustained tension).

-Standing step-back slider (small step-back with posterior pelvic cue; knee flex/extend gently as the pelvis returns to neutral).
Dose: 2–3 sets × 8–10 smooth reps per position, no breath-holds [15–19,27,28,33–37,39–41,72–77].

Iliopsoas lengthening (interface-friendly):

-Short-lever lunge hold (rear knee on cushion; pelvis gently posterior; 20–30 s × 2–3), guarding against lumbar extension [22,27,28,30–32,37–41,58,59,80–85].

-90/90 diaphragmatic breathing (2 minutes), linking exhale with paraspinal softening [23–25,30–32,38,50,58,59,80–85,95–100].

Pelvic motor control:

-Pelvic tilts in crook-lying (3 × 8–10) with external focus (“tilt the bowl to neutral”) rather than internal bracing cues.

-Hinge pattern (dowel feedback) 2 × 3 reps, emphasizing dissociation [24,30–32,38,50,58,59,80–85].

Gait homework:

-Three 60–90 s bouts/day focusing on trailing-limb hip extension and relaxed arm swing; cadence natural; avoid over-striding [22,31,32,37–41,58,59,67–70,80–85].

Expected response: reduced "neural quality" at the same PKB angle, fewer breath-holds, slight increase in trailing hip extension confidence. If irritability remains high, keep all variables constant and emphasize priming [27,28,30–32,33–37,39–41,58,59,72–77,95–100].

Week 2: Expand the excursion envelope

Progression decision point: If symptom quality at PKB's onset angle improves (e.g., from sharp nerve-like to dull stretch), introduce mid-range tensioners [27,28,30,33–37,39–41,72–77].

-Femoral tensioners: from slider end-position, add 5–8 s of sustained knee flexion with hip stabilized, then release back to glide.
Dose: 2–3 sets × 3–4 exposures, separated by sliders [27,28,33–37,39–41,72–77].

Hip extension in context:

-Wall-assisted split stance: small range hip extension with posterior pelvic cue, exhale-soften at end-range; 2 × 5.

-Hinge drill progresses to 2 × 4 reps; pelvic tilts maintained.

-Gait homework: unchanged volume; add external focus cue (e.g., "leave the floor behind you") [31,32,37–41,58,59,67–70,80–85,95–100].

Expected response: PKB onset angle increases by ~5–10° or quality shifts; time-to-relax improves; ODI/PSFS small gains [33–37,39–41,58,59,93–99].

Week 3–4: Consolidate and begin transfer

-Tensioner time-in-range increases (8–12 s holds), then add a slight angle increase while keeping holds steady [27,28,33–37,39–41,72–77].

-Add context: sidelying femoral bias slider (pelvis stabilized) to refine interface glide [27,28,33–37,39–41,72–77].

-Strength/coordination: introduce light loaded hinge (6–8 kg) for 2 × 5 reps with an elastic posture cue (no Valsalva) [30–32,38,58,59,67–70,80–85].

-Proprioceptive drill: brief trunk repositioning practice (eyes closed → open) between sets [22,30,58,59,84–87,95–100].

-Gait: interleave gait bouts between slider sets (contextual interference) to push transfer [67–70,80–85,95–100].

Expected response: clearer arm swing; trailing-limb hip extension improves by another 3–5°; PKB onset angle now ~100–105°. Breath-hold frequency decreases [31,32,37–41,58,59,80–85,93–99].

Week 5–6: Generalize and challenge

-Tensioners move closer to end-range with short holds (5–8 s) to prevent flare while sampling new ranges [27,28,33–37,39–41,72–77].

-Split-squat pattern introduced with minimal depth, emphasizing neutral pelvis and femoral glide comfort [30–32,38,58,59,67–70,80–85].

-Functional tasks: graded sit-to-stand without lumbar thrust (use hinge and exhale cue) [30–32,38,58,59,80–85].

-Gait: add uneven surface or tempo changes; keep success high [31,32,37–41,58,59,67–70,80–85].

Expected response: PSFS items improve by ≥2 points; ODI meaningful trend downward; PKB ~110° with stretch-dominant quality; patient reports less “jammed” feeling on rising from sitting [22,27,28,30–32,33–41,58,59,72–77,93–99].

3.4.7 Objective trajectories and early signs of true mechanism change

1.Neurodynamic behavior.

-Quality shift at a fixed angle precedes angle increases: the same PKB angle feels less neural and more “stretchy” or neutral [27,28,33–37,39–41,72–77].

-Modulation with structural differentiation becomes less necessary as excursion improves—consistent with reduced mechanosensitivity [27,28,30,33–37,39–41,72–77].

2.Resting tone/coordination.

-Time-to-relax after forward bend shortens by ~25–50%; fewer palpable paraspinal “holds” on exhale [23,24,30,31,38,50,58,59,80–85].

-Breath-hold frequency during testing/exercise drops toward zero [23,24,30–32,38,50,58,59,80–85,95–100].

3.Proprioception/maps.

-Trunk repositioning error decreases; movement paths to neutral are smoother. Brief laterality/imagery latencies improve—a proxy for map sharpening [22,23,30,58,59,84–87,95–100].

4.Gait.

-Trailing-limb hip extension increases and is maintained without cue saturation; arm swing and pelvic rotation normalize in parallel—evidence of transfer [22,31,32,38,58,59,80–85].

When these domains improve together, the case supports a genuine change in excursion + gain + priors, rather than compensation [22–24,27,28,30–32,33–41,58,59,67–70,72–77,80–85,93–99].

3.4.8 The patient's home program (written script)

1.Before each set (2 minutes): 6–8 slow breaths → 30–45 s imagery ("smooth glide under the inguinal ligament") → one rehearsal ("long spine, soft ribs") [23,25,26,57,60–63,78,79,83,95–100].

2.Femoral sliders: 2–3 sets of 8–10 reps (supine and standing), smooth tempo, no breath-holds [15–19,27,28,33–37,39–41,72–77].

3.Femoral tensioners: only if advised; 3–4 exposures of 5–10 s, separated by sliders [27,28,30,33–37,39–41,72–77].

4.Hip extension in context: wall-assisted split stance, 2 × 5, exhale at end-range [22,27,28,30–32,37–41,58,59,80–85].

5.Pelvic control: pelvic tilts 3 × 10; hinge 2 × 4 [24,30–32,38,50,58,59,80–85].

6.Gait: 3 × 90 s/day, focus on trailing-limb extension and relaxed arms [22,31,32,37–41,58,59,67–70,80–85].

7.Flare plan: if symptoms spike the next day, reduce one variable by ~30% (time, angle, or reps), add hourly 45–60 s micro-sliders for a day, keep the 2-minute priming [15,16,27,28,57,72–77,95–100].

This script reduces cognitive load and supports adherence [23,25–28,30–32,33–41,57,58,59,67–70,72–77,80–85,93–99].

3.4.9 Outcomes at 4 weeks and interpretation

Subjective/functional.

-Pain NRS reduces by ~60% at its worst (e.g., from 6/10 to ~2–3/10 at day end).

-PSFS: standing upright after sitting improves from 4/10 to 7/10; walking 30 minutes improves from 5/10 to 8/10.

-Perceived control: patient reports feeling “more elastic” and less apprehensive when rising.

Objective.

-PKB right: knee flexion at onset increases from 90° to ~110°, with symptoms described as “tight stretch” rather than nerve-like discomfort.

-Gait: trailing-limb hip extension increases by measured degrees (e.g., +6–8°) with a visible return of arm swing symmetry.

-Time-to-relax: reduces by ~40%; breath-holds now absent in testing.

Mechanistic inference. The sequence—quality change first, then range; tone/relaxation shifts early; gait improving later—matches the expected trajectory for restored femoral excursion and reduced gamma bias, with updated priors enabling transfer to function [23,25,27,28,31,33–37,39–41,58,59,80–85,93–99].

3.4.10 Why this case matters: specificity to femoral involvement

This case highlights a less discussed contributor to CLBP: anterior neural pathway restriction. Many CLBP protocols focus on the posterior chain and sciatic bias. Here, selective testing (PKB/sidelying femoral bias), pathway-specific gliding, and hip extension in context were crucial [22,27,28,30–32,37–41,58,59,80–85]. Two practical lessons emerge:

1.If PKB provokes anterior thigh/low back discomfort at mid-range without dermatomal radiation, consider the femoral pathway—even if Slump is negative [22,27,28,30–32,37–41].

2.Anterior pelvic tilt may be a protective policy to avoid femoral tension, not just a habitual "postural fault." Correcting it requires restoring glide first, then coaching neutral alignment [24,30–32,38,50,58,59,80–85].

3.4.11 Alternative scenarios and how to pivot

Scenario A: No change in PKB quality after 2–3 sessions.
Revisit dosing (too much angle, not enough time-in-range?); add more frequent micro-sliders to exploit thixotropy; emphasize breathing priming; consider local interface mobilization if irritability is low [15–19,23,27,28,33–37,39–41,57,60–63,72–77,83,88–92].

Scenario B: PKB improves, gait does not.
This is a transfer problem. Interleave gait bouts between slider sets; apply external focus cues; add temporal constraints (metronome) to invite trailing hip extension and normalize arm swing and pelvic rotation [22,31,32,37–41,58,59,67–70,80–85,95–100].

Scenario C: High irritability / flare with tensioners.
Regress to sliders; halve time-in-range; increase session frequency with very small volumes; keep functional tasks but in reduced doses; use the flare algorithm consistently to respect tissue irritability while maintaining exposure [15–19,27,28,33–37,39–41,57,72–77,95–100].

Scenario D: Psychosocial overlay emerges (fear spikes).
Increase education time and frame each exposure as safe success; scale back novelty; introduce graded activity blocks (e.g., timed walking) to build agency; consider co-management if distress persists [23,25,26,48,49,57,60–63,78,79,93–100].

3.4.12 Limitations and caveats

-Multitissue load. PKB and femoral bias tests stress more than nerve (muscle, fascia, joint). Specificity is imperfect; repeated patterned response to neurodynamic dosing strengthens inference [1–3,15–19,27,28,30,33–37,39–41,72–77].

-Heterogeneity. Not all anterior thigh symptoms are femoral pathway problems; hip joint pathology, lateral femoral cutaneous neuritis, or psoas tendinopathy can mimic [4–6,22,27,28,30–32,37–41,58,59,80–85].

-Measurement noise. Phone inclinometers and palpation-based proxies are operator-dependent; use consistent methods and trends rather than single values [33–37,39–41,58,59,84–87,93–99].

-Generalizability. A motivated office worker without major comorbidity may respond more readily than complex, multi-site pain patients; expectations should be calibrated accordingly [4–6,57,93–99].

3.4.13 Clinician checklist (per visit)

1.Priming done (breaths + imagery)?

2.Sliders delivered without breath-holds?

3.If tensioners used: time-in-range progressed before angle?

4.PKB quality at fixed angle noted?

5.Time-to-relax measured? Hinge score updated?

6.Gait bout completed with trailing-limb focus?

7.24-hour plan: progress, consolidate, or flare protocol? [15–19,22,24,25,27,28,30–32,33–41,57–59,67–70,72–77,80–85,93–100]

3.4.14 Patient one-minute brief (at home)

“First, breathe slowly and picture the nerve gliding under the belt-line crease. Then glide—polish, don’t provoke. If it feels okay for a day, hold a few seconds at the edge. Keep your ribs soft as you move. Spend what you gained by taking a short walk where the back leg trails a little more. If it’s cranky tomorrow, just roll back one step and do tiny glides every hour.” [23,25,27,28,31,57,67–70,95–100]

3.4.15 Synthesis and conclusion

This 38-year-old’s CLBP with anterior pelvic tilt and PKB-provoked anterior thigh/low back discomfort exemplifies a femoral neurodynamic restriction phenotype. A mechanism-aligned plan—femoral sliders progressing to carefully dosed tensioners, interface-friendly hip extension, iliopsoas load-management, and motor control toward neutral pelvis—produced clinically meaningful improvements at four weeks:

pain reduced by ~60%, PKB onset shifted from 90° to ~110° with non-neural quality, trailing-limb hip extension increased, and functional confidence improved, consistent with reported effects of neural mobilization in CLBP trials [39,51–54].

The distinguishing feature of this case is anterior pathway specificity. Rather than treating anterior pelvic tilt as a static postural flaw, we interpreted it as a protective control policy that makes sense when femoral excursion is costly. By lowering the cost—via restoring glide and re-weighting control—the policy could soften, allowing neutral alignment without coercive bracing. The measurable sequence—quality → range → tone/proprioception → gait/automaticity—accords with the broader model linking peripheral excursion to spinal gain and supraspinal priors [23,25,27,28,30,31,33–37,39–41,58,59,80–85,93–99].

As with all neurodynamic work, falsifiability is essential. If quality never shifts, if tone/proprioception remain unchanged, or if gait fails to improve despite better test angles, the hypothesis should be revised and the plan redirected. In this case, convergence across domains supports the femoral restriction diagnosis and the efficacy of targeted neurodynamic interventions for a meaningful subgroup of CLBP patients [22,24,27,28,30–32,37–41,51–54,58,59,80–85,93–99].

3.5 Case Example: Combined Sciatic and Cluneal Restrictions

3.5.1 Case overview and clinical question

Patient profile. A 55-year-old warehouse worker presents with a 4-year history of chronic low back pain (CLBP). Symptoms include a constant, dull ache across the lower lumbar region with intermittent posterior pelvic discomfort—sharper, localized pain just superior to the posterior iliac crest on the right, especially after long shifts that involve standing, walking, and frequent lifting of 10–20 kg boxes. Pain is aggravated by prolonged standing and by lifting, particularly when bending to pick items from floor-level pallets. Sitting is tolerable for short periods; rising from sitting is stiff but not sharply painful. Morning stiffness improves after 30–45 minutes of movement. Sleep is fair. He denies red flags (no fevers, weight loss, night pain, or neurological changes). He reports no prior lumbar surgery. He is motivated to remain at work and is open to active care [4–6,30,31,38,57,93–99].

Initial observation. Posture in quiet stance shows a mild anterior pelvic tilt with increased tone of the right gluteal region. During forward bending, the patient performs a spine-dominant flexion with limited hip hinge and a visible co-contraction around the lumbopelvic segment (guarded control) [24,30–32,38,50,58,59,80–85]. Returning to upright, there is a brief hesitation in mid-range followed by a quick extension thrust—typical of a high-gain controller attempting to stabilize through bracing [22–26,30–32,31,38,50,58,59,80–85].

Index tests.

-Straight Leg Raise (SLR) on the right: limited to ~50° with posterior thigh tightness and ipsilateral low back discomfort (no radicular radiation below the knee) [1–3,27,28,30,33–37,39–41,72–77].

-Slump test: provokes right-sided low back pain and posterior pelvic discomfort; symptoms modulate slightly with ankle plantarflexion and cervical extension (i.e., structural differentiation) [1–3,27,28,33–37,39–41,72–77].

-Prone knee bend (PKB): negative bilaterally (no anterior thigh symptoms).

-Palpation over posterior iliac crest: discrete tenderness approximately 7–8 cm lateral to midline along the iliac crest where cluneal nerves traverse fascial tunnels—suggestive of cluneal involvement [17–21,37,40,88–92].

-Hamstring/gluteal tone: increased on the right; delayed relaxation after stretch [24,30–32,38,50,58,59,80–85].

-Gait: shortened stride on the right, reduced contralateral arm swing, and damped pelvic rotation [31,32,38,58,59,80–85].

-Imaging: non-specific age-appropriate changes; no structural pathology explaining symptoms [4–6,30,31,38,57].

Provisional hypothesis. The combined findings suggest dual neural contributions: (1) sciatic pathway sensitivity/restricted excursion manifesting as early SLR limitation and slump-provoked low back/posterior thigh symptoms that modulate with differentiation; and (2) superior (± middle) cluneal nerve irritation at the posterior iliac crest—accounting for focal tenderness and the characteristic "hot spot" of posterior pelvic pain [17–21,27,28,30–32,37,39–41,58,59,80–85,88–92]. The central clinical question is whether a comprehensive, mechanism-aligned neurodynamic approach—addressing both the long sciatic track and the short, cutaneous cluneal branches—can reduce protective gain, restore movement variability, and improve work tolerance (lifting/standing) meaningfully [22,24,27,28,30–32,33–41,57–59,67–70,80–85,93–99].

3.5.2 Differential diagnosis and reasoning

1.Lumbar facet-dominant pain. Extension and prolonged standing aggravate symptoms in many facet-dominant cases. However, this patient's hallmark provocation includes neurodynamic loading (SLR/Slump), and palpation identifies a focal tenderness at the posterior iliac crest consistent with cluneal nerve entrapment sites, not purely facet referral. Quadrant testing does not reproduce familiar pain specifically in extension–rotation. Facet involvement is plausible but not primary [4–6,27,28,30–32,37,38,40,88–92].

2.Discogenic pain. Discogenic features typically include sustained flexion intolerance, morning pain that "warms up," and sometimes leg-dominant referral. Our patient displays flexion tolerance during sitting and no clear radicular pattern. Imaging lacks decisive disc pathology. Discogenic contribution remains possible but insufficient to explain the crest tenderness pattern [4–6,30–32,38,57,93–99].

3.Sacroiliac (SI) joint pain. Posterior pelvic pain with lifting can implicate the SI joint. Yet the reproducible crest point (~7–8 cm lateral to midline) and symptom modulation with Slump differentiation point toward cluneal/sciatic neural components rather than pure SI ligamentous nociception. SI provocation tests are non-diagnostic here [4–6,17–21,30–32,37,38,40,88–92].

4.Myofascial hamstring/gluteal syndrome. Hamstring/gluteal hypertonicity is evident, but the early SLR limit with neural quality and Slump modulation suggests that muscle tone is reactive to a neural driver, not the sole cause [22,24,27,28,30–32,33–37,39–41,58,59,80–85].

5.Combined neural restriction: sciatic track + cluneal nerve irritation (primary). The convergence of early SLR limitation with neural quality, Slump responsiveness, and crest tenderness strongly supports dual neural contributions. The posterior iliac crest is a well-described entrapment zone for superior cluneal nerves (dorsal rami branches), while the sciatic track is commonly sensitive in CLBP subgroups with posterior chain restrictions. This dual involvement plausibly explains both diffuse low back ache (sciatic/Slump contribution) and focal posterior pelvic pain (cluneal crest point) [17–21,27,28,30–32,37,39–41,58,59,80–85,88–92].

Conclusion. Working diagnosis: combined sciatic and cluneal neural restrictions, with secondary hamstring/gluteal hypertonicity and protective co-contraction causing reduced variability and braced lifting patterns [22–26,30–32,31,38,50,58,59,80–85]. The mechanism-aligned pathway is indicated.

3.5.3 Mechanistic links specific to sciatic and cluneal involvement

Sciatic pathway mechanics. The sciatic nerve (L4–S3) experiences significant sliding and elongation demands during hip flexion and ankle dorsiflexion. Restricted excursion increases focal strain and mechanosensitivity, driving early SLR limitation and slump-provoked discomfort that modulates with structural differentiation (cervical/ankle) [1–3,27,28,30,33–37,39–41,72–77]. Afferent noise biases spinal circuits, elevating gamma drive, which sustains co-contraction (hamstrings/gluteals) and reduces movement variability—particularly at the hip [22–26,30–32,31,38,50,58,59,80–85].

Cluneal nerve mechanics. The superior cluneal nerves are cutaneous branches of the dorsal rami (T12–L5) that cross the iliac crest through osteofibrous tunnels; middle cluneal nerves originate from S1–S3 dorsal rami and cross near the sacrum. These short, superficial nerves are susceptible to tunnel friction and local entrapment, generating focal crest tenderness and posterior pelvic pain, especially with trunk movements that tension fascial planes [17–21,30,37,40,88–92]. While sensory-dominant, cluneal irritation can amplify protective bracing via nociceptive input that increases segmental gain; in turn, guarding reduces normal glide of superficial fascial

layers, perpetuating the irritation—a local loop nested within the broader sciatic-driven protective set [22–26,30–32,31,38,50,58,59,80–85].

System behavior. With two neural contributors, the nervous system "chooses" high-stability, low-variability patterns for lifting and standing: shortened stride (to reduce sciatic tension), increased lumbar co-contraction (to stabilize against unpredictable inputs), and protective avoidance of trunk/hip ranges that shear the iliac crest fascia (to reduce cluneal provocation) [22–26,30–32,31,38,50,58,59,80–85]. The result is a compound protective attractor that burdens work tasks.

3.5.4 Baseline assessment: what we measured and why

Symptoms and function. Worst pain NRS 7/10 at end of shifts; resting NRS 3–4/10. Disability (ODI/RMDQ) elevated. PSFS targets: (1) stand comfortably for 60 minutes, (2) lift and carry 10–15 kg without next-day flare, (3) walk 30 minutes with normal stride [93–99].

Neurodynamic tests.

-SLR right: onset at 50° with neural quality; ankle plantarflexion reduces symptoms modestly; cervical extension reduces Slump discomfort [1–3,27,28,30,33–37,39–41,72–77].

-Slump: right-sided low back/posterior pelvic discomfort; structural differentiation changes symptom intensity [1–3,27,28,33–37,39–41,72–77].

-PKB: negative bilaterally (rules against femoral bias).

Palpation/crest sign. Focal tenderness ~7–8 cm lateral to midline on right iliac crest; symptom reproduction with local pressure aligns with superior cluneal tunnel [17–21,37,40,88–92].

Tone/coordination proxies. Elevated paraspinal tone; time-to-relax delayed after forward bending; multiple breath-holds during SLR/Slump. Hip hinge substituted by lumbar flexion; hinge score 0–1/2 [24,30–32,38,50,58,59,80–85].

Proprioception. Trunk repositioning error increased; reliance on vision for balance (single-leg stance eyes-closed <10 s) [22,30,58,59,84–87,95–100].

Gait. Shortened stride; trailing-limb hip extension reduced; arm swing damped; pelvic rotation limited [22,31,32,38,58,59,80–85].

Why this set? It samples mechanism from periphery (neural glide) to behavior (gait, lifting) and allows us to verify mechanism-consistent sequencing of change (quality → range; tone → proprioception; then gait/work tolerance) [22,24,27,28,30–32,31,38,58,59,80–85,93–99].

3.5.5 Treatment design: integrating sciatic and cluneal strategies

Targets.

1.Restore sciatic excursion with graded sliders → tensioners, dosed to respect irritability [1–3,27,28,30,33–37,39–41,72–77].

2.Desensitize cluneal crest via gentle superficial nerve sliders, local interface hygiene (myofascial glide of posterior crest fascia), and load sharing [17–21,30,37,40,88–92].

3.Reduce gamma bias (breathing + coordination) to break co-contraction [23–25,30–32,38,50,58,59,80–85,95–100].

4.Rebuild proprioception (repositioning drills, balance) and transfer gains into standing, walking, and lifting [22,30–32,31,38,58,59,84–87,93–99].

Key tools.

-Sciatic sliders/tensioners: Supine SLR slider with ankle/cervical sequencing; seated Slump slider; later, mid-range tensioners (short holds) [1–3,27,28,30,33–37,39–41,72–77].

-Cluneal sliders/glide hygiene: Small-amplitude skin/fascial glides over the crest in pain-free range (no aggressive compression), gentle trunk side-glide and short arc hip movements biasing superficial posterior fascia; education on avoiding belt or hard-edge pressure over the crest [17–21,30,37,40,88–92].

-Myofascial release: Low-load, slow-tempo work on gluteal/posterior pelvic fascia to facilitate glide, not to "break adhesions" [22,24,30–32,37,40,58,59,80–85].

-Coordination/breathing: Diaphragmatic breathing (down-titrates autonomic tone and gamma gain), hip-hinge re-patterning, micro-relaxation on exhale [23–25,30–32,38,50,58,59,80–85,95–100].

-Proprioceptive drills: Trunk repositioning practice; single-leg stance (eyes-closed as tolerated) [22,30,58,59,84–87,95–100].

-Work-specific integration: Hinge-based lifting with external focus cues; load management and task sequencing to protect consolidation [31,32,37–41,58,59,67–70,80–85,93–99].

Priming ritual (2 minutes at each session/home dose).

-6–8 slow breaths.

-30–45 seconds of brief imagery/laterality (visualize smooth sciatic glide; soften crest tissues).

-One rehearsal cue: “long spine, soft ribs; easy jaw; pelvis floats” [23,25,26,57,60–63,78,79,83,95–100].

3.5.6 Week-by-week plan, dosing, and progression

Weeks 0–1: Desensitize, establish glide, protect the crest

Sciatic sliders.

-Supine SLR slider: gentle hip flexion with ankle plantarflexion ↔ slight hip extension with ankle dorsiflexion (reciprocal), synchronized with breath (exhale on the more “loaded” half).

-Seated Slump slider: thoracic/hip flexion ↔ cervical extension and knee extension ↔ cervical flexion and knee flexion (small arcs first).
Dose: 2–3 sets × 8–10 reps/position, slow tempo, stop before neural quality spikes [1–3,27,28,30,33–37,39–41,72–77].

Cluneal glide hygiene.

-Skin/fascial glide over crest: therapist-taught self-technique—two fingers apply tiny amplitude, slow, comfortable glides perpendicular to the crest line for 30–45 s, 1–2 bouts.

-Trunk side-glide in standing (short arc): shift pelvis 1–2 cm away/toward the crest side while maintaining soft ribs; 2 × 6–8 reps; aim is non-provocative superficial motion [17–21,30,37,40,88–92].

Myofascial release (therapist).

-Gluteal/posterior pelvis: low load, sustained holds; avoid direct compression on the crest tunnel; follow with brief skin glide [22,24,30–32,37,40,58,59,80–85].

Coordination/breathing.

-Diaphragmatic breathing (90/90) 2 minutes; pair exhale with paraspinal softening.

-Hip hinge drill (dowel or wall touch) 2 × 3 reps—quality first [23–25,30–32,38,50,58,59,80–85,95–100].

Proprioception.

-Trunk repositioning (eyes closed → open); single-leg stance eyes-closed as tolerated (up to 30 s; 2–3 sets) [22,30,58,59,84–87].

Work modifications (light).

-Alternate tasks to avoid >20 minutes of continuous floor-level lifting; use a step or short platform to reduce deep trunk flexion early on; encourage breathing out on the effort.

Expected response. Slump/SLR quality at fixed angle begins to soften (less "nerve pull," more neutral stretch), crest tenderness slightly less irritable, fewer breath-holds. If crest is touchy, reduce frequency and amplitude of local glide but keep the priming and sciatic sliders [27,28,30–32,33–37,39–41,58,59,72–77].

Weeks 2–3: Expand excursion; begin transfer; protect consolidation

Introduce sciatic mid-range tensioners if quality changed at fixed angles:

-From the end of a slider arc, hold 5–8 seconds at mid-range while maintaining breath; back out to slider.
Dose: 2–3 exposures per set, 2 sets; time-in-range first (5 → 8 → 10 s), angle later [27,28,33–37,39–41,72–77].

Cluneal program.

-Maintain skin/fascial glide (brief and comfortable); add short-arc trunk rotation in prone on elbows (if tolerated) to gently bias posterior fascial shearing without crest compression (2 × 6).

-Educate: avoid tight belts or tool belts crossing the crest; add padding if belt is compulsory [17–21,30,37,40,88–92].

-Myofascial release. Continue low-load, slow holds; follow with brief skin glide to encourage superficial sliding [22,24,30–32,37,40,58,59,80–85].

Coordination/breathing.

-Diaphragmatic practice progresses to standing hinge (couple exhale with the "hip-back" phase).

-Hip hinge 2 × 4 reps; external focus cues ("touch wall with hips") reduce over-monitoring [23–25,30–32,38,50,58,59,80–85].

Proprioception.

-Trunk repositioning with a target; single-leg balance with micro-perturbations (therapist "taps").

-Start eyes-closed increments (5 → 10 → 15 s) as tolerated [22,30,58,59,84–87].

Functional integration.

-Gait homework 2–3 × 90 s/day focusing on trailing-limb hip extension and relaxed arm swing; avoid over-striding.

-Lifting pattern (empty box): hinge to mid-shin, exhale on lift, no breath-hold; 2 × 3 reps [31,32,37–41,58,59,67–70,80–85].

-Expected response. SLR onset angle improves by ~5–10° or the neural quality diminishes at the same angle; crest tenderness gradually reduces; time-to-relax improves; gait shows small gains in trailing hip extension and arm swing [27,28,30–32,33–37,39–41,58,59,80–85,93–99].

Weeks 4–5: Consolidate; introduce context variability; address asymmetries

-Sciatic tensioners: increase time-in-range to 10–12 s, still mid-range; then add a few degrees of angle while keeping holds short (5–8 s) to avoid flares [27,28,33–37,39–41,72–77].

Cluneal program.

-Continue hygiene; add gentle hip "figure-8" in quadruped (small arcs) to distribute superficial load; 2 × 6–8 reps.

-If crest is quiet, trial light lateral flexion arcs in standing (non-provocative range) [17–21,30,37,40,88–92].

-Myofascial release. Maintain low-load approach; never provoke "sharp" crest pain during manual work [22,24,30–32,37,40,58,59,80–85].

Coordination/strength.

-Hinge with light load (6–8 kg) 2 × 5 reps; cue elastic posture (long spine, soft ribs).

-Add anti-rotation holds (short sets) to improve trunk control without bracing [30–32,38,58,59,67–70,80–85].

Proprioception.

-Increase eyes-closed single-leg stance to target 20–30 s as tolerated; brief laterality/imagery blocks before exposure [22,23,30,58,59,84–87,95–100].

Functional integration.

-Lift with load (5–8 kg) 2 × 3; insert gait bouts between slider sets (contextual interference) to foster transfer; begin task sequencing mirroring work (two light lifts → short walk → shelf place) [31,32,37–41,58,59,67–70,80–85].

-Expected response. Arm swing symmetry returns; pelvic rotation less damped; SLR improves to ~60–65°; Slump discomfort further modulates; crest point sensitive only with firm pressure [22,24,27,28,30–32,37–41,58,59,80–85,93–99].

Week 6: Generalize; protect return to heavier tasks

-Sciatic work: brief forays toward end-range tensioners with short holds (3–5 s), bracketed by sliders [27,28,33–37,39–41,72–77].

-Cluneal: continue hygiene; reduce frequency if calm; keep brief check-ins to prevent recurrence [17–21,30,37,40,88–92].

Strength/integration.

-Lifting 8–10 kg 2 × 3 with hinge and exhale cue; step-off and carry 10–15 m as tolerated.

-Standing endurance practice with micro-shifts (2–3 minutes) trains variability without bracing [31,32,37–41,58,59,67–70,80–85].

-Expected outcomes at 6 weeks (illustrative). Pain NRS reduced by ~65% at shift end; SLR right to ~70°; Slump discomfort mild and highly modifiable; tolerance for repetitive moderate lifting improved; crest tenderness minimal [27,28,30–32,37–41,40,58,59,80–85,93–99].

3.5.7 Measurement trajectories and sequencing of true mechanism change

Neurodynamic behavior (sciatic).

-Quality at fixed angle improves first (less neural character) before angle expands—signature of reduced mechanosensitivity [27,28,30,33–37,39–41,72–77].

-Structural differentiation dependence declines as excursion and tissue tolerance improve (Slump less sensitive to ankle/cervical shifts) [27,28,30,33–37,39–41,72–77].

Cluneal behavior.

-Crest tenderness decreases with skin/fascial glide hygiene and reduced guarding; superficial sliding tolerance improves (short arcs without “zinging”) [17–21,30,37,40,88–92].

-Patient learns to pad/avoid belt pressure and to keep short, frequent micro-glide exposures [17–21,30,37,40,88–92].

Tone/coordination.

-Time-to-relax shortens by 25–50%; breath-holds drop toward zero [23,24,30–32,38,50,58,59,80–85].

-Hinge score improves (2/2) as dissociation returns, reducing spinal shear during tasks [23–25,30–32,38,50,58,59,80–85].

Proprioception/maps.

-Trunk repositioning error decreases; movement back to neutral is smoother (less “searching”) [22,30,58,59,84–87,95–100].

-Balance (eyes-closed) extends toward 20–30 s; brief imagery/laterality latencies fall—proxies for map sharpening [22,23,25,30,58,59,84–87,95–100].

Gait/function.

-Trailing-limb hip extension increases by 5–8°; arm swing and pelvic rotation normalize; standing tolerance extends without bracing; lifting tasks are possible without next-day flares [22,31,32,37–41,58,59,67–70,80–85,93–99].

-Coherent, cross-domain improvement supports restored excursion + reduced gain + updated priors, rather than mere compensation [22–25,27,28,30–32,33–41,57–59,67–70,72–77,80–85,93–99].

3.5.8 The home program (combined sciatic + cluneal script)

Priming (2 minutes): slow breaths (6–8/min) → 30–45 s imagery (smooth sciatic glide; crest tissue sliding) → one rehearsal cue ("long spine, soft ribs").

1.Sciatic sliders: 2–3 sets × 8–10 reps (supine SLR and seated slump), smooth tempo; no breath-holds.

2.Sciatic tensioners (when advised): 3–4 exposures of 5–10 s mid-range, always bracketed by sliders.

3.Cluneal hygiene: 30–45 s gentle skin/fascial glides at crest (no pressing on a sharp point), 1–2 bouts; short side-glide arcs (2 × 6).

4.Hinge practice: 2 × 4 reps with light dowel/wall cue.

5.Proprioception/balance: trunk repositioning (eyes closed → open), single-leg stance (eyes-closed increments).

6.Gait: 2–3 × 90 s/day with trailing-limb focus and relaxed arms.

7.Flare plan: If next day is cranky, reduce one variable by ~30% (time, range, or reps), keep the priming, add hourly micro-sliders (45–60 s) for a day, and avoid belt pressure over the crest [15–21,22–28,30–32,33–41,57–59,60–63,67–70,72–77,80–85,88–92,93–100].

3.5.9 Work-specific coaching and load management

Lifting strategy.

-External focus: “send hips back to touch the wall,” “exhale as the box leaves the floor,” “eyes on a mid-level target.”

-Dose: short sets (2–3 lifts), longer rest (20–30 s), then integrate into walking/carry with attention to arm swing and stride length [22,24,27,28,30–32,33–41,57–59,67–70,80–85,93–99].

Standing strategy.

-Avoid prolonged stillness; use micro-sways and heel-to-toe rocking every few minutes to keep the interface in a low-viscosity regime.

-If a work belt is mandatory, pad the crest tunnel and periodically re-position the belt [15–19,22,27,28,30,37,40,57,72–77,88–92].

Shift structure.

-Front-load lighter tasks post-warm-up; schedule brief movement snacks (1–2 minutes) each hour (two slider reps + two micro-sways + one hinge rehearsal).

-Goal: maintain glide all day, not just during therapy—preventing the build-up of focal strain and DRG gain [15–19,22,24,25,27,28,30–32,33–41,57–59,67–70,72–77,80–85,93–99].

3.5.10 Alternative courses and how to pivot

Scenario A: Crest remains highly irritable.

-Reduce frequency of direct crest glide; use more proximal/distal superficial movements (short arcs of trunk and hip) that bias the crest interface without local compression.

-Check for external irritants (tool belt edge); ensure padding/position change [17–21,30,37,40,72–77,88–92].

Scenario B: SLR improves, slump unchanged.

-Slump loads multiple regions (thoracic dura, lumbosacral roots). Add thoracic mobility elements (seated extension over towel) and sequencing (cervical extension early), then re-dose [1–3,27,28,30,33–37,39–41,72–77].

Scenario C: Transfer failure—test angles better, work still provocative.

-Increase contextual interference: interleave two gait bouts between slider sets, add task constraints (metronome for pace, target reach distance) to invite trailing hip extension and reduce guarding.

-Re-teach breathing during lifts (no Valsalva) [22,24,27,28,30–32,37–41,57–59,67–70,80–85,93–99].

Scenario D: Flare with tensioners.

-Regress to sliders; shorten holds; increase frequency with smaller per-dose volume; keep function light (carry only) while re-establishing tolerance [15–19,27,28,33–37,39–41,72–77].

3.5.11 Safety and boundary conditions

Avoid end-range tensioners in high-irritability states; prioritize sliders and superficial cluneal hygiene [27,28,30,33–37,39–41,72–77].

Stop and reassess with any new neurologic changes (weakness, sensory loss below knee, progressive symptoms) [4–6,30–32,38,57,93–99].

Consider co-management if psychosocial load (catastrophizing, sleep disruption) prevents graded exposure; dose expectations accordingly [23,25,26,48,49,57,93–99].

For osteoporosis or fracture risk, use shallow arcs and slow tempos; protect against aggressive trunk flexion under load [4–6,30–32,38,57,93–99].

3.5.12 Outcomes at 6 weeks and interpretation

Subjective/function.

-Pain reduction ~65% at end of shifts (e.g., worst NRS 7 → ~2–3/10).

-PSFS: standing 60 min improved from 4/10 → 8/10; lifting 10–15 kg without next-day flare from 3/10 → 7/10; walking 30 min with normal stride from 5/10 → 8/10.

-Confidence: the patient reports feeling “looser, less guarded,” and more able to pace heavy tasks.

Objective.

-SLR right: increased from 50° → ~70°, with symptom quality “stretchy” rather than neural.

-Slump: still mildly provocative but highly modifiable by structural differentiation; overall intensity reduced.

-Crest tenderness: minimal with light palpation; only firm pressure reproduces mild local discomfort.

-Gait: trailing-limb hip extension improved by measured degrees; arm swing and pelvic rotation more symmetrical; standing endurance improved with minimal bracing [39,40].

Mechanistic inference.
The sequencing—quality change first, then angle; early tone/relaxation gains; later gait/functional gains—mirrors the expected pattern for restored excursion (sciatic + superficial cluneal glide), reduced gamma bias, and updated priors that permit transfer to work tasks [23,25,27,28,30,31,33–37,39–41,58,59,80–85,93–99].

3.5.13 Limitations, caveats, and clinical pearls

Multitissue reality. SLR/Slump load nerves, muscles, fascia, joints. Specificity is never perfect; we seek converging patterns across tests, palpation, and response to dosing. The cluneal crest sign adds a local neural clue that pure posterior chain myofascial syndromes lack [1–3,15–19,27,28,30,33–37,39–41,72–77,88–92].

Short vs long nerve behavior. Short, superficial nerves (cluneal) respond best to brief, gentle, high-frequency glides and avoidance of compressive irritants. Long nerves

(sciatic) can tolerate graded tension when quality has shifted and thixotropy is established. Do not treat them identically [15–21,27,28,30,37,40,72–77,88–92].

Transfer or it didn't happen. If lifting and standing tolerance don't improve, revisit context and external focus cues. Gains confined to the plinth are clinically inadequate [22,24,27,28,30–32,31,38,58,59,67–70,80–85,93–99].

Priming is not optional. The 2-minute breathing + imagery prelude reduces autonomic arousal, optimizes neurovascular conditions, and increases the learning rate for exposure [23,25,26,57,60–63,78,79,83,95–100].

One variable rule. Change time-in-range before angle, and angle before speed/complexity. Consolidate for 48–72 hours after a successful change [57,67–70,72–77,93–99].

Flare is feedback. Use the viscosity reset (hourly micro-sliders) plus single-variable rollback to maintain progress. Do not abandon the plan; adjust the dose [15,16,27,28,72–77,95–100].

3.5.14 Clinician checklist (per visit)

1.Priming performed (breaths + imagery)?

2.Sciatic sliders delivered without breath-holds?

3.If tensioners used: time-in-range progressed before angle?

4.Cluneal hygiene: brief, comfortable; no crest compression?

5.Quality at fixed SLR/Slump angles recorded?

6.Time-to-relax and hinge score updated?

7.Gait and lift bouts completed with external focus cues?

8.24-hour plan: progress, consolidate, or flare protocol? [15–21,22–28,30–32,33–41,57–59,67–70,72–77,80–85,88–92,93–100]

3.5.15 Patient brief (one-minute script)

"First, breathe slow and picture the sciatic track sliding; keep the belt-line area comfortable and gliding, not pressed. Then do small glides—polish, don't provoke. If that feels okay tomorrow, hold only a few seconds at the edge. Keep your ribs soft and jaw easy. Spend the win by walking with a natural arm swing and letting your back leg trail a little more. If it's cranky tomorrow, roll back one notch and do tiny

glides each hour. We're training your system to move smoothly and safely again." [23,25,27,28,30,31,57,67–70,95–100]

3.5.16 Conclusion

This case illustrates the complexity of CLBP when multiple neural restrictions coexist. The sciatic pathway contributed an early SLR limit and Slump-provoked symptoms that responded to structural differentiation and graded gliding. The cluneal nerves, traversing the posterior iliac crest, added a focal posterior pelvic pain generator with local tenderness at an entrapment-prone tunnel, mirroring changes reported in trials of neural mobilization for CLBP [51–54]. The patient's global presentation—guarded lifting strategy, reduced gait variability, and standing intolerance—reflected a compound protective attractor: high spinal gain, reduced excursion, and priors that coded common workplace movements as unsafe [23,25,27,28,30,31,39,40].

A comprehensive neurodynamic approach—sciatic sliders progressing to brief tensioners, superficial cluneal glide hygiene, low-load myofascial techniques to facilitate sliding rather than compression, breathing-driven reduction of gamma bias, proprioceptive reweighting, and immediate functional transfer—yielded meaningful improvements by six weeks: ~65% pain reduction, SLR gains from 50° to ~70°, reduced Slump reactivity, crest tenderness largely resolved, and better tolerance for standing and lifting. The sequence of change (quality → range; tone → proprioception; gait/standing capacity → lifting) matched the mechanistic predictions that underlie this thesis and accords with outcome trajectories reported in neural mobilization trials for CLBP [23,25,27,28,30,31,39,40,51–54].

Importantly, this case underscores that short, superficial nerves (cluneal) and long, mixed nerves (sciatic) require different gliding strategies and dosing, even as they converge on shared neurophysiological pathways (afferent distortion → gamma bias → protective control). Precision in phenotyping, disciplined dosing, and relentless focus on transfer are the levers that convert a theoretical model into practical, verifiable clinical change. For the many CLBP patients in manual-handling occupations whose pain is sustained by combined neural restrictions, this integrated pathway offers a rational, testable route away from protection and back toward adaptable, efficient movement [23,25,27,28,30,31,39,40].

4.Clinical Implications

4.1 Assessment Strategies

Identifying subclinical neurodynamic restrictions requires a deliberate, mechanism-aligned approach that goes well beyond a conventional neurological screen. Traditional exams (myotomes, dermatomes, reflexes) are indispensable for ruling out frank neuropathy, but they have low sensitivity for the subclinical phenotype described throughout this thesis—patients who display movement-evoked neural symptoms without overt neurological loss, whose pain is sustained by reduced nerve

excursion, mechanosensitivity, and a protective motor set. The purpose of this section is to specify how to examine these patients so that your findings are reliable, interpretable, and actionable. We will (1) define test constructs; (2) standardize procedures; (3) operationalize interpretation using quality-at-fixed-angle, structural differentiation, and irritability; (4) include adjunct assessments (palpation, gait, functional tasks); (5) cover documentation, error management, and safety; and (6) give a practical decision algorithm that turns disparate observations into a clear clinical impression [1–3,22,24,27,28,30,33–37,39–41,58,59,93–99].

4.1.1 Construct overview: what each test actually measures

Slump Test (seated neurodynamic sequence).
Primary construct: sciatic/dural system mechanosensitivity and excursion under combined spinal flexion, hip flexion, knee extension, and ankle dorsiflexion. It is a multitissue test: neural tissue load increases in parallel with posterior chain muscle tension and spinal/postural load. Structural differentiation (e.g., changing ankle or cervical position) helps attribute symptoms to neural tissue by altering neural load without equivalently loading adjacent tissues. In subclinical restriction, you may see localized low back or posterior thigh discomfort at end-range without dermatomal radiation; intensity that modulates with ankle or neck movement suggests a neural driver [1–3,27,28,33–37,39–41].

Straight Leg Raise (SLR).
Primary construct: longitudinal excursion and mechanosensitivity of the sciatic nerve and lumbosacral nerve roots under passive hip flexion, with ankle and cervical adjustments for differentiation. Subclinical restriction typically presents as early onset of posterior thigh tension (e.g., 50–60°), protective pelvic motion, or stiffening behaviors without neurological deficits. Tracking angle at onset and symptom quality is crucial [1–3,27,28,30,33–37,39–41].

Prone Knee Bend (PKB).
Primary construct: femoral nerve and anterior thigh neural tissue excursion and sensitivity with knee flexion loading the anterior pathway; lumbar extension can add posterior element load. Subclinical femoral restriction often appears as localized anterior thigh pull or low back discomfort at ~90° of flexion without dermatomal radiation or quadriceps weakness [31,32,37].

Adjunct femoral-bias tests (sidelying femoral slump variants).
With pelvis stabilized, combining hip extension with knee flexion increases anterior pathway load while minimizing lumbar extension confounding. Useful when PKB is equivocal but history suggests anterior pathway involvement [31,32,37].

Cluneal provocation (posterior iliac crest).
Superior cluneal nerves traverse osteofibrous tunnels ~7–8 cm lateral to midline over

the crest. Point tenderness and symptom reproduction with gentle local palpation or short-arc fascial shearing suggest superficial neural irritation. Movement tests that bias posterior fascial glide (short arcs of trunk side-glide or rotation) can serve as a superficial slider screen [17–21,37,40,88–92].

Palpation and movement observation.
Palpating along neural courses (sciatic in gluteal region, femoral in anterior thigh, cluneal over posterior crest) for tenderness of neural character, paired with movement quality (guarding, breath-holding, reduced variability, compensations such as lumbar flexion during squatting), adds convergent evidence [24,30–32,38,50,58,59,80–85]. Observation of functional tasks (sit-to-stand, gait, reaching/lifting) often reveals asymmetries that the supine/seated tests miss [31,32,38,58,59,80–85].

Contextual signs.
Hypertonicity in synergists (hamstrings, gluteals, iliopsoas) and reduced joint mobility (e.g., hip extension loss) frequently accompany neural restrictions; they are secondary in the mechanism chain but primary in everyday function [22,24–26,30–32,38,50].

These tools must be used bilaterally and interpreted against the patient's history, goals, and irritability. A positive subclinical pattern is a pattern, not a single test [22,24,27,28,30–32,33–37,39–41,93–99].

4.1.2 Testing principles for the subclinical phenotype

1.Quality precedes angle.
First document what the patient feels (neural pull, stretch, pressure, pinch, burn) at a standardized angle, then track whether quality changes at that angle after interventions. A shift from "nerve-like" to neutral/stretch at the same angle is an early, sensitive marker of change [27,28,30,33–37,39–41].

2.Use structural differentiation.
In Slump, add or remove ankle dorsiflexion or cervical flexion/extension; in SLR, adjust ankle and neck; in femoral tests, stabilize the pelvis and slightly alter head/ankle as tolerated. If symptom intensity tracks with neural load changes (without equivalent musculoskeletal load change), neural attribution strengthens [33,36,37].

3.Dose by irritability.
In high-irritability states, minimize range and rely on sliders (reciprocal movements) rather than prolonged holds. In low/moderate irritability, small end-range doses are permissible if quality remains acceptable post-test [27,28,33–37,39–41].

4.One variable at a time.
When repeating a test intra-session to evaluate an intervention's effect, modify only one of: range, sequence, or structural differentiation. This protects interpretability and helps link observed changes to a specific mechanistic lever [27,28,33–37,39–41].

5.Stop before overdrive.
If breath-holds, grimacing, or guarding escalate, terminate the test. Overdriving mechanosensitive tissue obscures useful information and risks flares by pushing mechanosensitive and nociceptive fibers into high-gain behavior [23–25,27,28,30,33–37].

6.Standardize your language.
Avoid nocebo-laden cues ("pinched nerve," "damage"). Prefer neutral, accurate phrases ("tension on the nerve," "glide is a bit limited; we'll explore that gently"). This supports threat reduction and aligns with contemporary pain neuroscience education principles [23,25,26,48,49,57,93–99].

4.1.3 Slump Test: standardized procedure and interpretation

Set-up. Patient seated at the table edge, thighs supported, knees flexed ~90°, feet flat. Hands behind back or across chest to prevent support. Examiner stands lateral to the limb tested [1–3,27,28,33–37,39–41].

Sequence (baseline):

1.Thoracic and lumbar flexion ("slump"). Confirm neutral breathing (no breath-hold).

2.Cervical flexion (chin to chest). Ask for symptom report.

3.Knee extension on the test side to patient tolerance. Note angle at first symptom and quality.

4.Ankle dorsiflexion (if tolerated).

5.Structural differentiation: extend the cervical spine (return head to neutral/slight extension) without changing knee/ankle; record change in symptom intensity/quality and range.

6.Return to start [1–3,27,28,33–37,39–41].

Documentation:

-Angle (or distance) at first symptom.

-Quality (neural vs muscular stretch) and location.

-Modulation with structural differentiation (cervical/ankle): better, worse, no change.

-Post-test after-effect at 2–5 minutes (better, same, worse) [27,28,33–37,39–41].

Interpretation (subclinical restriction):

Positive pattern: localized posterior thigh or low back discomfort at end-range that improves with cervical extension or ankle plantarflexion (i.e., neural load reduction), without dermatomal symptoms [33,36,37].

Likely mechanosensitivity > excursion loss: strong modulation with structural differentiation and palpable guarding; quality highly dependent on ankle/cervical position [27,28,33–37,39–41].

Likely excursion loss > mechanosensitivity: early symptom onset that does not fully modulate with easing maneuvers; end-feel "tight" even when neural load is reduced [27,28,33–37,39–41].

Common errors:

-Adding multiple differentiators at once (neck and ankle simultaneously).

-Allowing trunk to "un-slump" during knee extension.

-Interpreting hamstring stretch as neural without checking modulation [27,28,33–37,39–41].

Safety. In high irritability, use micro-arcs (small knee extensions), prioritize breathing, and stop on rising neural quality [27,28,33–37,39–41,93–99].

4.1.4 Straight Leg Raise (SLR): standardized procedure and interpretation

Set-up. Supine; pelvis aligned; opposite leg straight and secured if possible (strap or clinician hand) to reduce pelvic rocking and lumbar contribution [1–3,27,28,30,33–37,39–41].

Sequence (baseline):

1.Maintain neutral ankle and neck. Slowly flex the hip with knee extended.

2.Record angle at first symptom and quality (posterior thigh tightness, back pull, neural tingle).

Structural differentiation:

1.Ankle dorsiflexion/plantarflexion at the same hip angle.

2.Cervical flexion/extension (if tolerated).

3.Optionally, modest hip adduction/medial rotation to bias peroneal/tibial components (advanced; use sparingly) [1–3,27,28,30,33–37,39–41].

Documentation:

-Angle at symptom onset (e.g., 50–60°).

-Quality/location.

-Modulation with ankle and neck.

-Observed pelvic motion (compensatory tilt/rotation).

Interpretation (subclinical restriction):

Positive pattern: limited range with posterior thigh tension or low back discomfort without radicular symptoms; modulation with ankle or neck supports neural attribution [33,36,37].

Mechanosensitivity-dominant: large quality/intensity changes with ankle/neck at fixed angle (clear neural character when loaded, softer when eased) [27,28,33–37,39–41].

Excursion-dominant: early limitation with "tight" end-feel that barely modulates; quality may be less overtly "neural" but range remains restricted even after easing maneuvers [27,28,30,33–37,39–41].

Common errors:

-Lifting too fast (spindle-dominant responses confound interpretation).

-Failing to stabilize the pelvis (apparent hip range is exaggerated).

-Over-interpreting hamstring stretch as neural without differentiation [27,28,33–37,39–41].

Safety. In high irritability, favor sliders (reciprocal ankle movement at sub-provocative hip angles) and small arcs; do not chase angle [27,28,33–37,39–41,93–99].

4.1.5 Prone Knee Bend (PKB) and femoral-bias variants

PKB set-up. Prone; pelvis neutral, ASIS supported if needed to limit lumbar extension [31,32,37].

Sequence (baseline PKB):

1.Slowly flex the knee toward the buttock while monitoring lumbar extension (keep minimal).

2.Note angle at first symptom (e.g., discomfort at ~90°).

3.Differentiate by gently stabilizing the pelvis or slightly flexing the lumbar spine (pillow under abdomen); record any change in quality/intensity.

Sidelying femoral-bias (when PKB equivocal):

1.Patient sidelying, test limb uppermost. Stabilize pelvis in slight posterior tilt.

2.Combine hip extension with knee flexion in small arcs.

3.Observe anterior thigh pull or low back discomfort; if symptoms are neural-like and sensitive to minimal changes in hip extension, the anterior pathway is implicated [31,32,37].

Documentation:

-Angle at first symptom; quality (anterior thigh pull vs neural sting vs back pinch).

-Response to pelvic stabilization or lumbar flexion.

-After-effect at 2–5 minutes.

Interpretation (subclinical femoral restriction):

Positive pattern: anterior thigh/low back discomfort at mid-range, improved by reducing anterior pathway load (pelvic stabilization, slight lumbar flexion), without quadriceps weakness or dermatomal symptoms [31,32,37].

Mechanosensitivity vs excursion logic mirrors sciatic tests: marked quality modulation with easing maneuvers suggests sensitivity-dominant; persistent early limit despite easing suggests excursion-dominant [27,28,30–32,37].

Common errors:

-Allowing brisk lumbar extension to "steal" motion (turning the test into a facet provocation rather than a neural bias).

-Forcing end-range knee flexion in high irritability (risking flares and obscuring useful information) [27,28,30–32,37,93–99].

4.1.6 Cluneal nerve screening over the posterior iliac crest

Anatomical cue. Superior cluneal branches traverse through small osteofibrous tunnels ~7–8 cm lateral to midline along the posterior iliac crest [17–21,37,40,88–92].

Screen:

-Gentle point palpation over the crest corridor to identify focal tenderness and familiar pain.

-Short-arc superficial sliders: in standing or prone on elbows, invite tiny trunk side-glide or rotation arcs while maintaining a soft abdomen and breath. Record whether superficial motion is comfortable vs "zingy."

-Load checks: note responses to belts/tool belts or hard edges over the crest [17–21,37,40,88–92].

Interpretation. Local tenderness that reduces with very light superficial glides and avoidance of compressive edges supports a superficial neural irritation model. Do not press aggressively; the goal is to determine whether skin/fascial sliding changes symptoms [17–21,30,37,40,72–77,88–92].

4.1.7 Palpation and movement observation

Palpation along neural courses.

-Sciatic: deep gluteal region (midway between ischial tuberosity and greater trochanter).

-Femoral: under inguinal ligament in femoral triangle (caution with depth and patient comfort).

-Cluneal: crest corridor (light pressure).

Findings: tenderness of neural character (sharp, electric, “zing”) vs dull myofascial tenderness; reproduction of familiar pain strengthens inference [24,30–32,38,50,58,59,80–85].

Movement observation (quality > quantity).

-Breath-holding during transitions.

-Guarding (co-contraction) on forward bend/return.

-Hip hinge competence vs lumbar substitution in squats and lifts.

-Gait: shortened stride, reduced trailing-limb hip extension, damped arm swing, stiff trunk [22,24,30–32,38,50,58,59,80–85].

These observations contextualize your neurodynamic findings and often explain the functional complaints better than range numbers alone [22,24,25,27,28,30–32,38,50].

4.1.8 Interpreting results: a practical schema

A. Categorize irritability.

-High: symptoms with small arcs; prolonged after-effects; strong guarding.

-Moderate: symptoms at mid-range; after-effects < 24 h; some guarding.

-Low: symptoms only at end-range; minimal after-effects [22,24,27,28,30,33–37,39–41].

B. Mechanosensitivity vs excursion.

-Mechanosensitivity-dominant: pronounced structural differentiation effects; quality changes rapidly with ankle/neck (or pelvic stabilization).

-Excursion-dominant: early limitation with little modulation by easing maneuvers; “tight” end-feel persists [27,28,30,33–37,39–41].

C. Provisional phenotype call.

-Sciatic, femoral, cluneal, or combined (e.g., sciatic + cluneal).

-Note secondary contributors: hamstring/gluteal or iliopsoas hypertonicity; reduced hip extension; coordination and breath-holds [22,24–26,30–32,38,50].

D. Link to function.

-Which functional tasks reproduce a similar load (e.g., long stride for sciatic; upright hip extension for femoral; belt pressure/crest shearing for cluneal)?

-This link is vital for setting transfer goals and for later mediation analyses in research [22–25,27,28,30–32,33–37,39–41,58,59,93–99].

4.1.9 Standardized documentation template (suggested)

-History anchor: chief complaint; aggravators (standing, lifting, sitting, walking), easers; duration; prior care.

-Irritability: high / moderate / low (with brief justification).

-Slump: angle at first symptom; quality; modulation with cervical/ankle (better/worse/none); after-effect.

-SLR: angle at first symptom; quality; modulation with ankle/neck; pelvic motion notes.

-PKB/femoral: angle at first symptom; quality; change with pelvic stabilization/lumbar flexion; sidelying variant if used.

-Cluneal: crest tenderness (Y/N); superficial slider comfort (Y/N); belt tolerance (Y/N).

-Palpation: neural-like tenderness sites; myofascial findings.

-Movement: breath-holds (count across 3 tasks), hinge score (0–2), gait notes (stride, trailing hip extension, arm swing).

-Proprioception: trunk repositioning error (simple method), single-leg stance eyes-closed (s).

-Provisional phenotype: pathway(s), sensitivity vs excursion bias.

-Functional link: task(s) to target for transfer (sit-to-stand, gait, lifting).

-Red flags: screened/none or listed.

This structure supports consistent reassessment, facilitates communication between clinicians, and creates data that can later be used to test mediation and subgroup hypotheses in research [22–25,27,28,30–32,33–37,39–41,58,59,93–99].

4.1.10 Reliability, validity, and minimizing error

Reliability realities. Neurodynamic tests are multitissue and operator-dependent. Intra-rater reliability improves when you:

-Use consistent landmarks (e.g., goniometer or inclinometer for SLR/PKB angles).

-Standardize tempo (slow, even speed).

-Fix one differentiator at a time.

-Record quality using the patient's own descriptors.

-Note after-effects at 2–5 minutes (short form).

These steps improve repeatability enough to make within-subject comparisons (pre/post) clinically meaningful in CLBP cohorts [27,28,30,33–37,39–41,58,59].

Validity and inference. No single test proves "neural restriction." Valid inference emerges from convergence: early onset + modulation with structural differentiation + mechanistic palpation + functional behavior that stresses the same pathway [33,36–38]. A change in quality at fixed angle after a dose-controlled intervention is a particularly persuasive within-subject validation signal, especially when mirrored by improvements in protective movement patterns [22,24,27,28,30,33–37,39–41].

Measurement error controls.

-*Angle:* use the same side of the table, same examiner, same device (phone inclinometer acceptable if consistent).

-*Quality:* anchor with a 5-point semantic scale (e.g., muscle stretch ↔ neutral ↔ neural pull ↔ sting ↔ burn).

-*Breath-holding:* score as binary per repetition (Y/N) to quantify guarding.

-*Gait:* measure trailing-limb hip extension at mid-stance using a phone app; accept change ≥5° as clinically meaningful within your service norms [31].

Such controls do not eliminate error but narrow it enough that patterns over time (especially across domains) become trustworthy for clinical and research use [22,24,27,28,30–32,33–37,39–41,58,59,93–99].

4.1.11 Special populations and modifications

High irritability/widespread pain. Lower range; use micro-sliders and omit tension components; prioritize breathing and education. Consider examining in supported positions (e.g., reclined Slump) to reduce non-neural loads and avoid overwhelming a sensitized system [48,49].

Older adults/osteoporosis risk. Avoid end-range spinal flexion/rotation under load; emphasize ankle and hip differentiators in mid-range; use slower tempos and smaller arcs [4–6,30–32,38,93–99].

Post-operative spine. Require medical clearance; avoid aggressive dural loading early; prefer gentle sliders, functional observation, and low-threat exposure until healing is more mature [4–6,27,28,30,93–99].

Hip arthropathy. SLR/PKB angles may be limited by joint pain; consider sidelying and standing variants with tiny arcs; interpret neurodynamic findings within joint constraints [30–32,38].

Athletes/heavy laborers. These patients often tolerate larger arcs and loads but may mask protective bracing as "strength." Standardize tempo and breathing; monitor for over-bracing (stiff, non-elastic strategies) and dose progression carefully [22,24,30–32,38,50].

4.1.12 Safety, red flags, and boundaries

Stop testing and refer (or co-manage) if new neurological deficits arise (progressive weakness, saddle anesthesia, bladder/bowel changes). Persistent night pain, fever, unexplained weight loss, or history of malignancy should be managed along standard red-flag pathways [4–6,93–99].

For neurodynamic testing specifically, avoid end-range force in acute radicular pain or where severe allodynia is present. Subclinical assessment favors small arcs and

structural differentiation over end-range provocation, with particular care in high-irritability or medically complex presentations [27,28,30,33–37,39–41,93–99].

4.1.13 From findings to a decision: a simple algorithm

1.Screen red flags and clear misfits for the neurodynamic-restriction model (per Section 3.4 logic and standard guidelines) [4–6,30,93–99].

2.Collect bilateral Slump, SLR, and PKB/femoral-bias tests (as indicated) with standardized documentation.

3.Add a cluneal screen if posterior crest pain is present.

4.Observe movement: breath-holds, hinge score, gait (stride, trailing-limb hip extension, arm swing).

5.Classify irritability (high / moderate / low).

6.Assign bias: mechanosensitivity-dominant vs excursion-dominant for each implicated pathway.

7.Link pathway to function: identify one task that loads that pathway (e.g., long stride = sciatic; rising upright/hip extension = femoral; belt/crest shearing = cluneal).

8.Trial a micro-dose intervention (e.g., 1–2 sets of sliders with breathing), then re-test one marker (quality at fixed angle).

9.If quality improves without flare: confirm phenotype, plan graded exposure and functional transfer. If no change, adjust dose or reconsider attribution; if domains are discordant, broaden the differential.

This algorithm emphasizes falsifiability: the exam is designed to set up a near-term test of your hypothesis within the same visit [22–25,27,28,30–32,33–37,39–41,58,59,93–99].

4.1.14 Worked micro-examples (subclinical phenotype)

Example A: Slump-positive, SLR-borderline (sciatic sensitivity).

-History: worse with prolonged sitting; diffuse low back ache; no radicular symptoms.

-Slump: right-sided low back discomfort at end-range; improves when neck extends.

-SLR: onset ~60°, quality ambiguous; improves with ankle plantarflexion.

-Interpretation: sciatic mechanosensitivity, subclinical restriction [33,36].

-Next step: sciatic sliders + breathing; track quality at 60° later that day.

Example B: PKB-positive with anterior thigh pull at 90° (femoral).

-History: difficulty standing upright from sitting; anterior pelvic tilt.

-PKB: low back/anterior thigh discomfort; improves with pelvis stabilized or slight lumbar flexion.

Slump: negative.

-Interpretation: femoral subclinical restriction [31,32,37].

-Next step: femoral sliders; check quality at 90° after a micro-dose.

Example C: Crest tenderness, Slump-positive (cluneal + sciatic).

-History: posterior iliac crest "hot spot"; standing/lifting aggravate.

-Slump: modulates with cervical/ankle; SLR early onset at ~50°.

-Crest: point tenderness; light superficial glides comfortable; belts/tool belts aggravate.

-Interpretation: combined superficial cluneal irritation + sciatic restriction [37,38,40].

-Next step: brief superficial glides + sciatic sliders; verify Slump quality and crest tenderness reduction post-dose.

These micro-examples show how pattern recognition across tests and tasks leads to a pragmatic phenotype call and an immediate, testable intervention [22–25,27,28,30–32,33–37,39–41].

4.1.15 Building the "minimum viable battery" (clinic-ready)

Time is limited in busy clinics. A 10–12 minute battery is realistic and sufficient to characterize the subclinical neurodynamic phenotype:

1.Slump (both sides) with one structural differentiator; document angle/quality/modulation.

2.SLR (both sides) with ankle differentiator at fixed angle.

3.PKB (if anterior symptoms/history suggest); otherwise omit to save time.

4.Crest screen if posterior iliac pain is present.

5.Movement observation: hinge (3 reps), breath-hold count, quick gait look (trailing hip extension/arm swing).

6.One micro-dose (sliders + breathing), **one re-test** (quality at fixed angle).

7.Phenotype statement and task link: one line each in the note (e.g., “Right sciatic, mechanosensitivity-dominant; linked to prolonged sitting”).

This compact battery yields enough information to start specific, mechanism-aligned care while keeping the diagnostic hypothesis explicitly falsifiable and ready for refinement at follow-up [22–25,27,28,30–32,33–37,39–41,58,59,93–99].

4.1.16 Common pitfalls and how to avoid them

-Chasing angles, ignoring quality. Remedy: always record quality at fixed angle; treat a quality shift as success, in line with contemporary neurodynamic dosing principles that emphasize symptom behavior over raw range [1–3,27,28,29,33,72–76].

-Stacking differentiators. Remedy: change one thing at a time (ankle or neck), consistent with recommendations for interpretability of structural differentiation in neurodynamic testing [1–3,29,33,36,73].

-Provoking flares during testing. Remedy: shorter arcs, slower tempo, end early; favor sliders in high irritability, reflecting evidence that sliders are better tolerated in sensitive states and that excessive end-range loading can exacerbate mechanosensitivity [27,28,33,72–76].

-Interpreting every stretch as neural. Remedy: insist on structural differentiation evidence (symptom change with ankle/cervical modification) before attributing symptoms to neural tissue, as outlined in neurodynamic assessment literature [1–3,29,33,36,73].

-No functional linkage. Remedy: always identify a task analog for the implicated pathway (e.g., long stride ↔ sciatic), in keeping with best-practice guidelines that emphasize functionally relevant assessment and treatment targets [55–57,77].

-Inconsistent documentation. Remedy: adopt the template; it halves cognitive load and increases reliability, aligning with recommendations for standardized outcome capture in CLBP research and practice [93–99].

4.1.17 Why bilateral testing and history integration matter

Subclinical restrictions often present asymmetrically—but not always where

symptoms are loudest. Bilateral testing prevents a false-normal when the symptomatic side is compared only to population norms. History integration (e.g., “pain during prolonged standing”) helps you select the most relevant differentiator and the right retest: a patient whose symptoms worsen in long stride deserves gait observation even if SLR looks “near normal” in supine [33,36,38]. This approach mirrors contemporary views that mechanistic findings should be interpreted within a biopsychosocial and task-specific context [25,26,55–57,77].

4.1.18 From assessment to plan: closing the loop

A high-quality assessment should immediately inform dosing. If your findings suggest mechanosensitivity-dominant sciatic involvement, your first intervention is sliders + breathing, not heavy tensioners, consistent with trials and reviews showing that gentle neurodynamic loading can reduce symptoms without provoking flares [27,28,33,72–76]. If excursion-dominant femoral restriction is suspected, early mid-range holds (5–8 seconds) may be appropriate after the interface is “warmed” by sliders [27,28,37]. If cluneal irritation is present, you will prescribe superficial glides and reduce crest compression before loading deeper tissues, as suggested in anatomical and clinical work on cluneal neuralgia [20,21,88–92]. The re-test—quality at fixed angle or crest tenderness—confirms whether you are on the right track the very same day. When it shifts, you have evidence that your mechanistic hypothesis is viable; when it doesn’t, you adjust promptly, in line with mechanism-based and cognitive-functional approaches to CLBP [23,25,26,64–66].

4.1.19 Summary (clinician-ready)

-Use Slump, SLR, PKB (as indicated), a cluneal screen, and movement observation to triangulate neural excursion and sensitivity, following established neurodynamic and motor-control frameworks [1–3,29,30,33,36–38,80–83].

-Document angle at first symptom, quality, structural differentiation effects, and a 2–5 minute after-effect, using standardized outcome and documentation principles recommended for CLBP [33,36–38,93–99].

-Classify irritability and mechanosensitivity vs excursion bias for each implicated pathway, as per contemporary models of central/peripheral sensitization and motor adaptation [23,25–28,60–63,80–83].

-Always link test findings to a functional task (gait, sit-to-stand, lifting), echoing guideline recommendations for function-oriented, mechanism-informed care [55–57,64–66,77].

-Re-test one marker after a micro-dose intervention within the same session to keep your hypothesis falsifiable, consistent with best practice in mechanism-based clinical reasoning and research design [3,33,39,51–54,72–76,97–99].

In short, assessment for subclinical neurodynamic restrictions is not about a single positive test; it is about generating a coherent pattern that explains the patient’s experience, guides precise dosing, and produces verifiable change when you

intervene—exactly the standard this thesis proposes for mechanism-aligned care [1–3,25–27,33,36–38,55–57,72–77].

4.2 Therapeutic Interventions

Purpose. This section details a comprehensive, mechanism-aligned program for treating subclinical neurodynamic restrictions—interventions that restore neural excursion, reduce mechanosensitivity, and re-establish efficient motor control. We present (I) principles that govern safe and effective dosing; (II) a complete toolkit of techniques across the continuum from neurodynamic mobilizations to manual therapy, movement re-education, and proprioceptive retraining; (III) progression rules, flare management, and decision points; (IV) integration with conventional exercise therapy (strength, flexibility, conditioning); (V) home programming and adherence strategies; and (VI) outcome measurement to verify true mechanism change rather than mere compensation [1–3,24,29,39–41,51–57,67–70,72–77].

4.2.1 First principles for mechanism-aligned care

1.Glide before you load. Neural interfaces are viscoelastic and thixotropic; gentle, reciprocal motion lowers apparent viscosity and improves sliding (“glide”) before end-range tension is safe. Early treatment emphasizes sliders, not long holds [15,16,27,28,39,40,72–76].

2.Quality precedes range. A shift from “neural pull/sting” to neutral or stretch at the same test angle (e.g., SLR, slump, PKB) is the earliest, most reliable sign of improvement. Only then should you chase angle/range [1–3,11–14,29,33,36,39,51–54,72–76].

3.Dose for irritability. High-irritability patients need micro-doses and higher frequency; low-to-moderate irritability can tolerate modest end-range exposure. Match the cost of a dose to the patient’s capacity and 24-hour response profile [27,28,33,37,39,51–54,72–77].

4.Change one variable at a time. To avoid flares and protect learning, adjust only one of: time-in-range, angle, repetitions, speed/tempo, context (position/load), or complexity (multi-segment sequencing). Consolidate for 48–72 hours before the next change, consistent with tissue adaptation and motor learning data [27,28,39,51–54,67–70,72–76].

5.Priming lowers cost and increases learning. Two minutes of paced breathing and brief motor imagery/laterality reduce sympathetic tone and prime cortical planning networks so the same mechanical dose produces more adaptation [23,25,60–63,71,80–83,100].

6.Spend gains in function immediately. Improvements in glide must transfer into meaningful tasks (gait, lifting, sit-to-stand). Embed short functional bouts between

mobilization sets to consolidate learning and update priors, in line with motor control and cognitive functional approaches for CLBP [24,30,31,55–57,64–70,77,80–83].

4.2.2 Neurodynamic mobilizations (sliders and tensioners)

Concept. The goal is to restore relative movement between the nerve and its mechanical interfaces and to normalize afferent signaling—first by gentle sliding with low tensile load (sliders), then by carefully dosed tensioners when quality improves [1–3,11–14,27,28,33,36,39,40,51–54,72–76].

A) Sciatic nerve mobilizations

Supine SLR slider (foundational).
-Set-up: Supine; non-test leg stabilized.

-Motion: Slow reciprocal cycle: (1) hip flexion with ankle plantarflexion → (2) slight hip extension with ankle dorsiflexion. Keep pelvis quiet.

-Dose (start): 2–3 sets × 8–12 cycles; smooth 2–3 s per half-cycle; 1–2×/day.

-Cues: “Glide, don’t reach; breathe out on the more loaded half.”

-Progressions: increase reps, then time-in-range (brief 3–5 s micro-holds at the end of the more loaded half), then a few degrees of hip angle.

-Indicators to advance: neural quality softens at a fixed angle; after-effects ≤ 12–24 h [27,28,33,39,51–54,72–76].

Seated slump slider (complementary).
-Set-up: Seated; thoracolumbar slump with neutral breathing.

-Motion: Cycle cervical extension with knee extension (less neural load) ↔ cervical flexion with knee flexion; small arcs first.

-Dose: 2 sets × 8–10 cycles; 1–2×/day.

-Differentiation: adjust ankle or neck alone if needed [1–3,33,36].

-Progressions: larger arcs; mid-range holds of 5–8 s after quality improves [27,28,33,39,51–54,72–76].

Sciatic tensioners (introduced later).
-Pre-condition: quality shift at fixed angle and stable after-effects.

-Motion: Move to a comfortable end of a slider arc, then add a short hold (5–10 s) with breathable effort; bracket with sliders.

-Dose: 2–3 exposures per set, 1–2 sets; 48–72 h consolidation.

-Rule: increase time-in-range before angle.

-Stop criteria: return of sharp neural quality, breath-holding, or next-day flares [27,28,39,51–54,72–76].

B) Femoral nerve mobilizations

Prone knee bend (PKB) slider.
-Set-up: Prone; slight abdominal support to minimize lumbar extension.

-Motion: Small arcs of knee flexion ↔ extension synchronized with smooth breathing; pelvis stable.

-Dose: 2 sets × 8–10 cycles; 1–2×/day.

-Progressions: add tiny hip extension bias once quality softens [27,28,37,39].

Sidelying femoral slider (pelvis stabilized).
-Set-up: Sidelying, test limb uppermost; pelvis in gentle posterior tilt.

-Motion: Hip extension (small) with knee flexion ↔ return; short arcs.

-Dose: 2 sets × 8–10 cycles; low irritability only.

-Tensioners: 5–8 s holds at mid-range after clear quality shift; always bracketed by sliders [27,28,33,37,39,51–54,72–76].

C) Cluneal nerve mobilizations (superficial)

Superficial transverse gliding at posterior iliac crest.
-Set-up: Prone on elbows or standing.

-Motion: Very gentle skin/fascial glides perpendicular to crest (tiny amplitude, slow).

-Dose: 30–45 s × 1–2 bouts; 1–3×/day.

-Rule: zero “zing”—comfort only.

-Adjunct sliders: short-arc trunk side-glide or rotation while maintaining a soft abdomen and easy breath.

-Rationale: brief, frequent superficial motion biases the osteofibrous tunnel to reduce friction without compression, consistent with anatomical and clinical descriptions of cluneal nerve entrapment and management [20,21,38,40,88–92].

4.2.3 Manual therapy to facilitate glide and reduce interface load

Intent.

Manual techniques are adjuncts that (i) improve interface compliance, (ii) reduce paraneural tone, and (iii) make active mobilizations more effective—not stand-alone cures. [2,3,29,39,40,72–76,77]

A) Myofascial release (MFR) and soft tissue mobilization (STM)

-Gluteal/posterior pelvis (sciatic/cluneal contexts). Low-load, sustained MFR to gluteus maximus/medius and deep fascial planes; avoid direct compression on suspected cluneal tunnels. Follow with brief skin glides to encourage superficial slide. [20,21,38–40,72–76,88–92]

-Hamstrings (sciatic context). Slow, graded MFR to reduce co-contraction; pair with sliders to prevent "stiffening back up." [24,27,28,30,39,50,72–76]

-Iliopsoas/inguinal region (femoral context). Gentle STM to iliacus and proximal anterior hip fascia with patient consent and comfort; avoid aggressive pressure at neurovascular structures. [27,28,30,37,39]

Dosing: 2–6 minutes per regional target; no provocation of neural pain during treatment; re-test a marker (quality at fixed angle) immediately after. [29,33,36,39,72–76]

B) Joint techniques (when motion loss impedes glide)

-Thoracic mobilization (to reduce dural tension in slump). [1–3,11–14,39,41,72–76]

-Hip joint mobilization (posterior/anterior glides) if joint stiffness restricts hip hinge/extension and thus neural excursion. [27,28,30,39–41,67–70,72–76]

-Lumbopelvic manipulation (select patients): used judiciously to reduce segmental guarding; immediate re-test is mandatory to confirm relevance. [24,39–41,77]

-Guardrails: Manipulative thrusts are not indicated in high-irritability neural states. The contribution of manipulation is indirect—through tone reduction and improved movement options. [24,30,39–41,60–63,77]

4.2.4 Movement re-education: reducing gamma bias, restoring patterns

Intent.

Replace protective bracing with elastic control and restore dissociation between lumbar spine and hips so daily movement does not re-provoke neural load. [23,24,30,50,67–70,80–83]

A) Breathing and coordination (non-negotiable prelude)

-Paced diaphragmatic breathing (90/90 or hook-lying).

-6–8 breaths/min × 1–2 minutes, cueing “soft ribs,” “long spine,” and exhalation-linked paraspinal softening. [23,25,60–63,71,80–83,100]

-Carryover: maintain the same breath during sliders and functional tasks.

-Micro-relaxation drill. Simple cue: “On exhale, melt the paraspinals.” Measure time-to-soften after a standardized forward bend; use as a feedback KPI. [23,24,30,50,58–59,80–83]

B) Hip hinge and lumbopelvic dissociation

-Dowel or wall-touch hinge. 2–3 sets × 3–5 reps; external focus (“touch the wall with your hips”) reduces over-monitoring. [24,30,67–70,80–83]

-Sit-to-stand with hinge cue. 2 sets × 4–6; exhale on effort; no breath-hold. [24,30,67–70]

-Progressions: light load (6–8 kg) after quality is stable; integrate anti-rotation holds for trunk control without bracing (short 10–15 s bouts). [24,30,67–70,80–83]

C) Flexion/extension coordination (cat-cow, pelvic tilts, bird-dog)

-Pelvic tilts (3 × 10) to explore neutral zone control.

-Cat-cow with breathing focus (1–2 minutes).

-Bird-dog (3 × 6 slow reps each side), emphasizing reaching long rather than lifting high; stop short of lumbar hinge. [24,30,67–70,80–83]

-Rule: movement remains quiet in the symptomatic neural range while glide is being restored; expansion comes later. [27,28,39,72–76]

4.2.5 Proprioceptive retraining and sensory re-weighting

Intent.

Improve map precision and reduce reliance on protective co-contraction by upgrading position sense and balance. [22,30,50,58–59,80–87]

Trunk repositioning drills.

-Laser or inclinometer feedback: find neutral, move away, return with eyes closed, then open to check accuracy.

-2–3 minutes, 1–2×/day; small errors are corrected gently, not punished. [22,58–59,80–87]

Balance training (single-leg stance).

-Start eyes open; progress to eyes closed in 5–10 s increments up to ~30 s if safe; 3 sets each side.

-Add micro-perturbations (therapist taps) when appropriate. [22,30,58–59,80,84–87]

Dynamic stabilization (low-threshold).

-Short planks or side-planks (3 × 15–20 s), focusing on breath continuity; no valsalva.

-Wobble board or compliant surface for small arcs; maintain quiet breathing and soft ribs. [24,30,67–70,80–83]

Laterality/imagery.

-30–60 s blocks before exposure: left/right trunk/hip orientation judgments or brief imagery of smooth gliding to prime cortical networks. [23,25,71,80–83,100]

Progression logic: increase complexity only after accuracy improves. The goal is confidence and precision, not fatigue. [22,58–59,80–87]

4.2.6 Making the pieces work together: a standard session arc

1.Priming (2 minutes). Diaphragmatic breathing → laterality/imagery → one rehearsal of the intended movement with soft ribs and long spine. [23,25,60–63,71,80–83,100]

2.Neurodynamic mobilization (8–12 minutes). Choose one or two positions (e.g., supine SLR slider + seated slump slider). Deliver sliders first; if criteria met, add short tensioner holds; bracket with sliders. [1–3,11–14,27,28,33,36,39,40,51–54,72–76]

3.Movement re-education (5–8 minutes). Hinge practice, pelvic control, micro-relaxation, and a low-threshold dynamic task (bird-dog, short plank). [24,30,50,67–70,80–83]

4.Proprioception (2–3 minutes). Trunk repositioning or balance (brief, successful exposures). [22,58–59,80–87]

5.Functional transfer (2–4 minutes). Gait bout with trailing-limb hip extension and arm swing; or sit-to-stand/lift drills with exhalation cue. [24,30,31,55–57,64–70,77]

6.Re-test a marker (1 minute). Quality at fixed angle (e.g., SLR at 60°) or time-to-soften after forward bend. Log change. [29,33,36,39,41,93–99]

This arc fits in a 30–40 minute session and scales to a 10–15 minute home dose. [55–57,64–70,77]

4.2.7 Dosing, progression, and consolidation

Starting doses (typical).

-Sliders: 2–3 sets × 8–12 cycles per position, slow tempo, 1–2×/day.

-Tensioners: 2–3 exposures of 5–10 s once per day only after quality shift; always bracket with sliders.

-Breathing/coordination: 2 minutes per session (clinic + home).

-Proprioception: 2–3 minutes/day (brief, frequent beats).

-Functional bouts: 2–3 × 60–90 s gait with trailing-limb focus; or 2–3 sets of 3–5 sit-to-stands with hinge cue. [27,28,33,36,39,51–54,67–70,72–76]

Progression rules (one change at a time):

1.Increase time-in-range (5 → 8 → 10–12 s).

2.Then increase angle (a few degrees).

3.Then increase reps or add a second position.

4.Then increase context (e.g., from supported to standing).

5.Finally add complexity (multi-segment sequencing, light load). [27,28,39,51–54,67–70,72–76]

Consolidation: hold a successful step for 48–72 hours before advancing. [27,28,39,67–70,72–76]

4.2.8 Flare management: viscosity reset, not retreat

Three-step algorithm.

1.Single-variable rollback: Cut one of range/time/reps by ~30–40% for 48 hours; keep all other elements (breathing, imagery, light function) intact.

2.Viscosity reset: Add micro-sliders (45–60 s) hourly while awake for 1 day to maintain low interface viscosity and reduce dorsal root gain. [15,16,27,28,60–63,72–76]

3.Language and expectations: “This is feedback, not failure. We’re optimizing dose to keep adapting.” [25,26,48–49,60–63,100]

Do not abandon functional tasks entirely. Instead, scale intensity and use more external focus cues to protect confidence. [55–57,64–70,77,100]

4.2.9 Decision points and tailoring to phenotype

Mechanosensitivity-dominant (large differentiation effects).

-Stay longer with sliders; keep breathing central; add very short tensioners only after stable quality shifts; avoid stacking progressions. [27,28,33,36,39,51–54,72–76]

Excursion-dominant (stubborn early limit with little modulation).

-Introduce mid-range tensioners earlier, but with very short holds; pair with interface-oriented manual therapy and precise hinge/hip extension drills. [27,28,33,37,39–41,51–54,67–70,72–76]

Cluneal irritation present.

-Prioritize superficial glides and avoid compression (belts, hard edges); micro-dose frequently; integrate gentle posterior fascial shearing arcs; keep deep tissue work low-load and non-provocative. [20,21,38,40,88–92]

Fragile stability (frequent breath-holds).

-Expand breathing/coordination time; reduce per-set reps and increase session frequency; emphasize “quiet quality” over range. [23,24,30,50,60–63,80–83]

Psychosocial overlay (fear/vigilance).

-Brief education every session; hard-code success experiences; use graded activity blocks (timed walking) to build self-efficacy; consider co-management if high distress persists. [25,26,48–49,55–57,60–63,64–66,100]

4.2.10 Integration with conventional rehabilitation

Strength training.

-Begin with low-threshold patterns (hinge, split-stance, carries) emphasizing breath continuity and elastic posture.

-Progress to moderate loads only after glide and quality stabilize.

-Favor compound movements that honor dissociation (hip/knee) and avoid reflex bracing. [24,30,39–43,67–70,77,80–83]

Flexibility.

-Use short, frequent exposures; avoid long static holds at end range in early stages (increase interface friction).

-Once neural quality has softened, integrate active mobility (leg swings, controlled articular rotations) to maintain excursion. [15,16,27,28,39,72–76]

Conditioning.

-Low-intensity walking or cycling with posture and arm-swing focus (for walking); time-based progression; maintain nasal or diaphragmatic breathing. [24,30,31,55–57,64–70,77,80–83]

Education and pacing.

-Micro-lessons: “nerves like to glide,” “quality before range,” “change one thing at a time,” “spend your gains.”

-Activity scheduling to spread mechanical load and reduce end-of-day spikes. [55–57,60–63,64–66,77,100]

Synergy model. Neurodynamic work re-enables motion options; strength and conditioning stabilize those options in real life. [24,30,39–43,55–57,64–70,77]

4.2.11 Home program architecture and adherence

Structure each home dose (8–12 minutes):

-Priming (2 min): breathing + brief imagery/laterality. [23,25,60–63,71,80–83,100]

-Sliders (4–6 min): one or two positions; smooth tempo; zero breath-holds. [1–3,27,28,33,36,39,51–54,72–76]

-(When indicated) Tensioners (1–2 min): short holds; bracket with sliders.

-Movement/proprioception (2–3 min): hinge or pelvic tilts + 30–60 s balance or trunk repositioning. [22,24,30,58–59,67–70,80–87]

-Functional minute (1–2 min): gait with trailing-limb cue or sit-to-stand set. [24,30,31,55–57,64–70,77]

Adherence levers.

-Micro-bouts across the day vs one long session.

-Habit stacking (e.g., sliders after brushing teeth).

-Visible trackers (calendar check-offs).

-One-line script taped to mirror: “Breathe slow, glide gently, spend the win.” [55–57,64–70,77,100]

4.2.12 Measurement: verifying true mechanism change
Track a minimum dataset every 1–2 weeks:

-Neurodynamic: angle at first symptom and quality at fixed angle (SLR/slump/PKB); modulation with structural differentiation. [1–3,11–14,27,28,29,33,36,37,39,51–54,72–76]

-Tone/coordination: time-to-soften after forward bend; breath-hold count across three tasks; hinge score (0–2). [23,24,30,50,58–59,80–83]

-Proprioception: trunk repositioning error; single-leg stance (eyes-closed) up to 30 s. [22,58–59,80–87]

-Gait/function: trailing-limb hip extension (phone inclinometer acceptable), arm-swing symmetry; PSFS items tied to work/life roles. [31,55–57,93–99]

-Symptoms/disability: NRS (rest/movement), ODI/RMDQ. [41,55–57,93–99]

Expected sequencing:

1.Quality improves at a fixed angle →

2.Range increases →

3.Tone/relaxation improves →

4.Proprioception sharpens →

5.Gait and function consolidate. [22,24,30,31,33,36–37,39,41,50,58–59,80–87,93–99]

If test ranges improve without functional or proprioceptive change, suspect compensation and adjust plan. [24,30,55–57,64–70,77,100]

4.2.13 Safety, contraindications, and boundaries

-Defer tensioners in high-irritability or acute radicular pain; use sliders and breathing only. [27,28,33,36,39,72–76,77]

-Stop and refer for red flags (new weakness, saddle anesthesia, bladder/bowel changes, night pain, systemic symptoms). [5–7,55–57,77]

-Respect co-morbidities (osteoporosis, anticoagulation, severe peripheral neuropathy); favor small arcs, slow tempo, and superficial techniques. [5–7,24,39–41,60–63,77]

-Avoid direct compression over suspected cluneal tunnels; use padding if belts must be worn. [20,21,38,40,88–92]

4.2.14 Representative treatment pathways (condensed)

A) Sciatic-dominant, mechanosensitivity-biased.

-Weeks 0–2: sliders (supine SLR + seated slump), breathing/hinge, gait bouts; no tensioners.

-Weeks 2–4: add mid-range tensioners (5–8 s), maintain sliders; progress hinge; add light carries; keep function between sets.

-Milestones: quality shift at fixed angle by week 2; range +10–15° by week 4; improved trailing-limb hip extension. [1–3,27,28,33,36,39,51–54,67–70,72–76]

B) Femoral-dominant, excursion-biased.

-Weeks 0–1: PKB sliders with pelvic stabilization; iliacus/inguinal STM; pelvic tilt control; short-lever hip extension context.

-Weeks 1–3: mid-range tensioners (5–8 s) bracketed by sliders; add wall-assisted split stance; hinge drills; early gait spend.

-Milestones: PKB quality shift, then angle to ~100–110°; upright tolerance improved. [27,28,33,37,39,51–54,67–70,72–76]

C) Combined sciatic + cluneal.

-Weeks 0–2: sciatic sliders; superficial crest glides (brief, comfortable); avoid crest compression; breathing/hinge; short gait bouts.

-Weeks 2–6: add short sciatic tensioners; posterior fascial short arcs; progress balance and hinge; integrate lifting with external focus.

-Milestones: SLR quality/angle improvements; crest tenderness minimal; work tolerance up. [20,21,27,28,30,31,38–40,51–54,72–76,88–92]

4.2.15 Common pitfalls (and fixes)

-Over-chasing angle early. Fix: time-in-range first; end range last.

-Stacking progressions. Fix: change one variable; consolidate 48–72 h.

-Ignoring breath. Fix: 2-minute priming is mandatory; abort a set if breath-holds appear.

-Provoking cluneal tunnels. Fix: superficial glides only; avoid pressure; pad belts.

-No functional transfer. Fix: insert gait/lift bouts between slider sets with external focus cues.

-Abandoning after flares. Fix: viscosity reset + single-variable rollback; keep priming and light function. [1–3,15,16,24,27,28,29,33,36–39,41,50,60–63,67–70,72–76,77,80–83,88–92,100]

4.2.16 Clinician and patient micro-checklists

Clinician (per visit):

1.Priming delivered?

2.Sliders done without breath-holds?

3.If tensioners used: time before angle?

4.Manual therapy non-provocative and followed by sliders?

5.Functional spend performed?

6.Re-test (quality at fixed angle or time-to-soften) recorded?

7.24-hour plan: progress, consolidate, or flare protocol? [1–3,24,27,28,29,33,36–41,50,55–57,67–70,72–77,80–83,93–99,100]

Patient (at home, 20-second script):
“Breathe slow. Picture the nerve gliding. Do gentle sliders—polish, don’t provoke. If it’s easy tomorrow, hold a few seconds at the edge. Keep ribs soft. Spend the win—walk with relaxed arms and let the back leg trail. If cranky tomorrow, step back one notch and do tiny glides each hour.” [25,26,55–57,60–63,64–66,100]

4.2.17 Conclusion

Therapeutic management of subclinical neurodynamic restrictions is most effective when it aligns with the system’s biology: glide precedes load; quality precedes range; small, frequent exposures reshape viscous interfaces and recalibrate afferent gain; breathing and coordination reduce gamma-driven bracing; proprioception and functional practice sharpen maps and update priors. [1–3,15,16,23–25,27,28,30,39–43,58–59,60–63,67–70,72–76,80–83] The integrated program laid out here—neurodynamic mobilizations (sliders → tensioners), manual therapy to facilitate slide and reduce interface load, movement re-education for elastic control, and proprioceptive retraining to improve map precision—provides a falsifiable, clinically feasible pathway. It must be taken in consideration that in Basson’s work it shows that neurodynamic mobilizations show modest improvements in pain and function. [51] Progress is verified with multidomain markers (quality at fixed angle, tone/relaxation, proprioception, gait/function). When the expected sequence of change emerges—quality → range → tone → proprioception → functional automaticity—we can infer genuine mechanism modification rather than compensation. [1–3,22,24,30,31,33,36–37,39–43,51–54,58–59,67–70,72–76,80–87,93–99] If that sequence does not appear, the model invites recalibration: adjust dose, re-phenotype, or pivot pathways. In this way, therapeutic interventions become both scientific and client-centered, restoring adaptable, confident movement while remaining anchored to measurable physiology. [5–7,24,30,39–43,55–57,64–70,72–77,93–99,100]

4.3 Case Study: Integrated Approach in an Athlete

4.3.1 Case overview and clinical question

Athlete profile. A 30-year-old competitive marathon runner presents with a 6-month history of chronic low back pain (CLBP). Pain emerges during long runs (beyond ~15–18 km) and after training blocks that include hill repeats or tempo sessions, and it is aggravated by forward bending after runs (e.g., to untie shoes). The athlete reports a diffuse, band-like ache across the lumbosacral region with a focal sense of tightness in the right gluteal area. No radicular symptoms, paresthesia, or weakness are reported. Morning stiffness is brief (<15 minutes). Sleep is intact. Past medical history is unremarkable; no prior lumbar surgery or significant injuries besides mild plantar fasciopathy three years earlier. Current weekly volume fluctuates between 60–80 km depending on race cycle. [4–7,55–57]
Initial observation. Standing posture is symmetric with mild anterior pelvic tilt. In dynamic tasks (hip hinge, forward bend, single-leg squat), the athlete demonstrates breath-holding at movement initiation and a subtle co-contraction across the lumbopelvic segment, most evident when asked to reach toward the floor. [50,58,59,80–83] During a quick in-clinic running appraisal (treadmill at easy pace), there is reduced right trailing-limb hip extension, damped arm swing on the left, and slightly reduced pelvic rotation. [31,58,59,80–83]
Index tests.

-Straight Leg Raise (SLR), right: symptom onset (posterior thigh tightness + ipsilateral low back ache) at ~55°; quality is described as “nerve-y stretch.”

-Slump test: reproduces right-sided low back pain late in the sequence; symptoms modulate with ankle plantarflexion and cervical extension (structural differentiation).

-Palpation: tenderness along the sciatic nerve midway between the ischial tuberosity and greater trochanter on the right; gluteus maximus and deep rotators show increased tone with delayed relaxation. [1–3,11–14,27,28,29,33,36,39]

-Prone knee bend (PKB): negative bilaterally (no anterior thigh symptoms). [37]

-Gait analysis (treadmill): shortened right stride, diminished right hip extension, slightly increased vertical oscillation during fatigue simulation (3–5 minutes at marathon pace + 5%). [31,58,59,80–83]

-Imaging: MRI unremarkable (no discs/facets explaining symptoms). [4–7,55–57]

Provisional hypothesis. Findings are consistent with a sciatic neural excursion/sensitivity problem on the right that is provoking protective gamma-biased co-contraction of gluteal and paraspinal musculature. [1–3,23,24,27,28,30,39–41,50] The athlete’s motor system has likely adopted stability-dominant patterns in running (shortened stride, reduced trailing hip extension, damped pelvic rotation) to avoid end-range neural tension. [31,50,58,59,80–83] The clinical question: can a mechanism-aligned, integrated program—sciatic sliders progressing to carefully dosed tensioners, interface-friendly manual therapy, movement re-education with breathing/hinge work, proprioceptive retraining, and running-specific transfer—

reduce pain, improve SLR from 55° toward normative ranges, normalize gait, and safely return the athlete to full training? [1–3,27,28,33,36,39–43,50,51–54,58,59,67–70]

4.3.2 Sport-specific context and risk factors in endurance runners

Distance runners experience repetitive loading cycles—tens of thousands of steps per week—primarily in the sagittal plane. Over time, the combination of high mileage, limited variability, and end-range repetition in the hip/trunk complex can reduce the relative sliding of neural tissues within their interfaces. [31,39–41,58,59,80–83] When stride shortens and trailing hip extension declines (whether from fatigue, prior niggles, or cautious motor policies), the runner spends more of each cycle in mid-range where neural tissues are repeatedly tensioned but never fully glided across their excursions. Add hill work (which increases hip flexion/extension demands) or plyometric drills performed with braced breathing, and you have a recipe for interface "stickiness" and afferent noise. [1–3,15,16,27,28,39,50,58,59]
In these athletes, subclinical neurodynamic restriction rarely appears as frank sciatica; rather, it manifests as stiffness, guarding, and vague posterior chain discomfort that worsens with volume or intensity. Because cardiorespiratory fitness is high, athletes can "run through" early warning signs, deepening protective patterns. The unremarkable MRI in this case is compatible with such systems-level contributions rather than discrete structural lesions. [4–7,55–57,60–63,80–83]

4.3.3 Differential diagnosis and reasoning

1.Facet-dominant extension pain. Long-run discomfort and anterior pelvic tilt might suggest facet loading, but passive extension/rotation testing does not reproduce familiar symptoms, and the neural character on SLR and slump modulation point toward neurodynamic contributions rather than pure posterior element nociception. [4–7,24,33,36,39,50]

2.Discogenic pain. Forward bending after runs is uncomfortable, but there is no flexion-dominant symptom pattern, no radicular pain, and MRI is unremarkable. Discogenic pain is a less likely primary driver. [4–7,55–57]

3.Deep gluteal syndrome / piriformis-dominant myofascial pain. Palpation elicits gluteal tenderness; however, structural differentiation during slump shifts symptoms, and SLR is early with neural quality. The gluteal tone is interpreted as reactive to a neural driver. [1–3,11–14,24,27,28,33,36,39,50]

4.Proximal hamstring tendinopathy. The athlete denies ischial tuberosity point pain; running uphill is not uniquely provocative. Palpation/functional provocation specific to the tendon is negative.

5.Sciatic neural excursion + mechanosensitivity (primary). Early SLR limitation with neural quality, slump provocation that modulates with ankle/neck, focal sciatic course tenderness, gait with reduced trailing hip extension—all converge on a sciatic pathway problem. [1–3,11–14,27,28,33,36,39,50] The working diagnosis is subclinical sciatic restriction with secondary protective bracing and altered running mechanics. [24,30,31,39–41,50,58,59,80–83]

4.3.4 Baseline assessment and metrics (mechanism-aligned)

Symptoms/function: worst NRS 6/10 during latter half of long runs; 3–4/10 after hill workouts; 1–2/10 at rest. Athlete-specific goals: complete 90-minute long run pain ≤2/10; resume weekly tempo (20–30 min at threshold) pain ≤2/10; tolerate post-run forward bend to shoes with minimal stiffness. [55–57,93–99]
Neurodynamic:

-SLR (R): onset at 55°, neural quality; modulates with ankle plantarflexion (less) and cervical extension (less).

-Slump (R): low back ache that decreases with cervical extension; ankle plantarflexion helps modestly.

-After-effect: 5-minute reassessment shows persistent stiffness post-test if range is chased.

Tone/coordination: palpable paraspinal tone; time-to-relax after forward bend prolonged (subjectively "takes a while" to soften); breath-holds present during SLR/slump. [23,24,30,50,58,59,80–83]

Proprioception: trunk repositioning error mildly elevated; single-leg stance eyes-closed 20–25 s (left) vs 12–15 s (right) before toe tap. [22,58,59,80–87]

Gait (treadmill): right trailing-limb hip extension reduced by an estimated 5–7° vs left; arm-swing asymmetry; pelvic rotation damped. [31,58,59,80–83]

Performance context: weekly mileage 70 km; long run ~22–25 km; intensity sessions 1–2/week. The athlete seeks to maintain conditioning while reducing pain—an important constraint for programming. [55–57]

These metrics provide mechanism-level anchors for change: quality at fixed angle on SLR/slump, time-to-relax, balance asymmetry, and trailing-hip extension during gait. [1–3,22–24,27,28,30,31,33,36–39,50,58,59,80–87,93–99]

4.3.5 Treatment rationale and design

Objectives. (1) Restore sciatic excursion and reduce mechanosensitivity with graded neurodynamic loading; (2) lower gamma bias and breath-hold behavior via diaphragmatic control and coordination; (3) sharpen proprioception and re-open movement options (hip hinge, pelvic rotation); (4) spend gains immediately in running-relevant tasks; (5) maintain aerobic capacity with low-threat conditioning; (6) implement a structured return-to-run (RTR) progression. [1–3,23–25,27,28,30,39–43,50,58,59,67–70,80–83]
Core components.

-Neurodynamic mobilization (sliders → tensioners): supine SLR slider, seated slump slider, then mid-range tensioners after quality shifts. [1–3,11–14,27,28,29,33,36,39,51–54,72–76]

-Manual therapy (adjunct): interface-friendly gluteal/posterior fascial techniques, not deep compression; follow with sliders to reinforce glide. [20,21,27,28,30,38–40,72–76,88–92]

-Movement re-education: diaphragmatic breathing, hip hinge, pelvic control, short-arc cat-cow; micro-relaxation on exhale. [23–25,30,41,50,58,59,67–70,80–83,100]

-Proprioceptive retraining: trunk repositioning drills; single-leg balance progressions; brief laterality/imagery blocks. [22,58,59,80–87]

-Gait/running-specific transfer: trailing-hip extension drills, arm-swing restoration, cadence and external-focus cues; RTR stages tightly coupled to symptoms and neurodynamic markers. [31,55–57,58,59,64–70,80–83]

-Load management: reduce session density and hill intensity early; preserve frequency and easy mileage as tolerated. [55–57,64–70,77,100]

Priming (2 minutes each session/home): 6–8 slow diaphragmatic breaths → 30–45 s motor imagery of smooth sciatic glide → one rehearsal with cue: “long spine, soft ribs, easy jaw.” [23,25,60–63,71,80–83,100]

4.3.6 Neurodynamic mobilizations: technique and dosing

Supine SLR slider (primary).

-Motion: hip flexion with ankle plantarflexion ↔ slight hip extension with ankle dorsiflexion; pelvis quiet, slow 2–3 s halves.

-Dose (start): 2–3 sets × 8–12 cycles, 1–2×/day.

-Cues: avoid reaching for angle; “polish, don’t provoke.”

-Progression: reps → tiny time-in-range holds (3–5 s) at the comfortable end → a few degrees of angle after quality shifts. [1–3,11–14,27,28,33,36,39,51–54,72–76]

Seated slump slider (secondary).

-Motion: small arcs pairing cervical extension with knee extension (reduced neural load) ↔ cervical flexion with knee flexion; ankle adjustments as needed.

-Dose: 2 sets × 8–10 cycles, 1×/day initially.

-Progression: larger arcs; short mid-range holds when tolerated. [1–3,11–14,27,28,33,36,39,51–54,72–76]

Mid-range tensioners (introduced after 1–2 weeks if quality improves).

=Motion: from the end of a slider arc, hold 5–8 s with quiet breathing; then back out to sliders.

-Dose: 2–3 exposures per set, always bracketed by sliders; consolidate 48–72 hours before angle increases.

Stop rules: return of sharp neural quality, breath-holding, or next-day flare beyond 24 hours triggers single-variable rollback and viscosity reset (hourly micro-sliders for one day). [27,28,29,33,36,39,51–54,72–76]

4.3.7 Manual therapy: facilitating slide, not forcing length

Gluteal/posterior pelvis (interface focus).

-Technique: low-load myofascial release (2–5 minutes) to gluteus maximus/medius and deep posterior fascial planes; avoid direct, sustained compression over the sciatic corridor.

-Sequence: manual work → brief skin glides → immediate SLR sliders to capitalize on reduced friction.

-Re-test: quality at fixed SLR angle post-intervention.

Rationale: manual therapy here decreases paraneural tone and eases superficial sliding, making active neurodynamic work cheaper (less symptomatic cost). [20,21,27,28,30,38–40,72–76,88–92]

4.3.8 Movement re-education and coordination

Breathing (non-negotiable). 90/90 diaphragmatic breathing, 1–2 minutes per session; explicit cue to melt paraspinals on exhale; abort sets where breath-holding appears. [23,25,60–63,71,80–83,100]

Hip hinge and pelvic control.

-Wall-touch hinge with external focus: 2–3 × 3–5 reps; neutral pelvis with soft ribs; no lumbar thrust when returning upright.

-Pelvic tilts (3 × 10) to find and own a comfortable neutral.

-Cat-cow (short arc) synced to breath: 1–2 minutes, never into symptoms. [24,30,50,67–70,80–83]

Micro-relaxation KPI: measure seconds to palpable paraspinal softening after a standardized forward bend; trend each week (aim 25–50% reduction over 4–6 weeks). [23,24,30,50,58,59,80–83]

4.3.9 Proprioceptive retraining and sensory re-weighting

-Trunk repositioning drills: eyes closed → open; laser/inclinometer feedback; 2–3 minutes/day.

-Single-leg balance: start eyes open; progress to eyes closed in 5–10 s increments to ≤30 s; 3 sets per side.

-Laterality/imagery (pre-dose): 30–45 s blocks to prime cortical planning networks before sliders.

-Dynamic stabilization (low threshold): short planks (3 × 15–20 s) with breathing continuity; no Valsalva.

Aim: improve map precision so the CNS can release protective co-contraction without feeling “unsafe.” [22,23,25,30,50,58,59,71,80–87,100]

4.3.10 Running-specific transfer and re-patterning

Gait levers (in-clinic and home).

-Trailing-limb hip extension practice: 60–90 s bouts at easy pace with cue “leave the floor behind you” (external focus). Avoid over-striding.

-Arm-swing restoration: cue “loose elbows, pockets forward” to re-animate contralateral arm swing and unlock pelvic rotation.

-Cadence check: if cadence is low (<165–170 at easy pace), encourage a ~5% increase at easy runs to reduce over-striding without forcing hip extension.

-Drills: A-march with soft ribs; ankling; short strides emphasizing symmetry rather than reach. [31,55–57,58,59,64–70,80–83]

Return-to-run (RTR) framework.

-Stage 0 (Week 0–1): maintain frequency; cap long run at pain-free duration (e.g., 60 min); no hills/tempos; strides optional if neutral.

-Stage 1 (Week 1–2): add 10–15% time to long run only if SLR quality at fixed angle improves and post-run pain ≤2/10.

-Stage 2 (Week 2–3): introduce short cruise intervals (e.g., 4 × 5 min at steady) if daily markers remain positive; maintain gait cues.

-Stage 3 (Week 3–4): progress long run toward target; re-introduce tempo (15–20 min) with immediate post-session sliders and breathing resets.

-Stage 4 (Week 5–6): add gentle hills if tolerated; preserve one easy day after any quality session. [55–57,64–70,77,100]

“Spend the win” rule: insert a 90-s gait bout with trailing-hip focus between slider sets during home sessions to consolidate transfer. [31,55–57,58,59,64–70,80–83]

4.3.11 Week-by-week plan and progression

Weeks 0–1: Desensitize and establish glide

-Priming: 2 minutes breathing + imagery.

-Neurodynamic: SLR sliders (R): 2–3 × 8–12; slump sliders: 2 × 8–10.

-Manual: low-load gluteal/posterior fascial MFR (2–4 min) → brief skin glides → repeat 1 set SLR sliders.

-Movement: wall-touch hinge 2 × 3–4; pelvic tilts 3 × 10; cat-cow 1–2 min.

-Proprioception: trunk repositioning 2–3 min; balance (eyes open) 3 × 30 s/side.

-Running: frequency intact; longest run limited to pain-free duration (≤60–70 min); no hills/tempos.

-Re-test: quality at 55° SLR; “time-to-relax” post-bend.

Expected: quality softens at same SLR angle; fewer breath-holds; post-run stiffness less intense. [1–3,22–24,27,28,30,31,33,36–39,50,58,59,67–70,80–83]

Weeks 2–3: Expand excursion; begin tensioners; add steady running

-Neurodynamic: introduce mid-range tensioners (5–8 s) bracketed by sliders; keep progressions to one variable (time-in-range first).

-Manual: as needed; never provocative.

-Movement: hinge 2 × 5; add short plank 3 × 15–20 s with breathing continuity.

-Proprioception: balance progresses to eyes closed (10–15 s holds).

-Running: long run +10–15%; cruise intervals (e.g., 4 × 5 min steady) if post-run pain ≤2/10 and markers stable.

-Re-test: SLR now 60–65° or quality change at 55°; time-to-relax improved ~25%. [1–3,22–24,27,28,30,31,33,36–39,50,51–54,58,59,67–70,72–76]

Weeks 4–5: Consolidate; introduce tempo; emphasize transfer

-Neurodynamic: tensioners 8–10 s; slight angle increase if after-effects <24 h.

-Movement: hinge with light load (6–8 kg) 2 × 5; anti-rotation holds (10–15 s).

-Proprioception: balance eyes closed 20–25 s; laterality/imagery pre-dose daily.

-Running: tempo 15–20 min (split if needed); long run approaches pre-injury volume but capped by pain ≤2/10; insert 90-s gait bouts with trailing-hip focus between slider sets on home days.

-Re-test: SLR 70–75° with neutral/stretch quality; improved arm swing and pelvic rotation on treadmill. [24,30,31,39–41,50,55–57,58,59,64–70,80–83]

Week 6: Generalize and stress inoculate

-Neurodynamic: short forays toward near end-range with brief holds (3–5 s); always bracket by sliders.

-Movement: hinge load optional to 10–12 kg if perfect breathing; maintain soft ribs.

-Running: add gentle hills; resume normal tempo; consider low-dose strides at session end; ensure one easy day buffer.

-Outcomes: pain reduction ~70%; SLR ~80°; normalized gait symmetry; athlete resumes full training block. [31,39–41,50,55–57,58,59,64–70,80–83]

4.3.12 Measurement trajectory and interpretation

Expected sequencing:

1.Quality at fixed SLR angle improves (neural → neutral/stretch).

2.Range increases (55° → 60–65° by week 2–3; to ~80° by week 6).

3.Tone coordination: time-to-relax halves; breath-holds extinguish.

4.Proprioception: right single-leg eyes-closed approaches left (20–30 s).

5.Gait: right trailing-hip extension normalizes; arm swing symmetry returns; pelvic rotation unlocks.

6.Performance: long run and tempo re-introduced without spikes.

If ranges improve but gait does not, emphasize transfer (gait bouts between slider sets, external focus cues). If tempo triggers flare, roll back one variable (duration or speed), preserve frequency, and run the viscosity reset algorithm. [1–3,22–24,27,28,30,31,33,36–39,41,50,51–54,58,59,67–70,72–76,80–87,93–99]

4.3.13 Load management and cross-training

To maintain aerobic capacity while respecting neural irritability:

-Replace one quality session in weeks 0–2 with low-threat conditioning (elliptical or cycling, nasal breathing emphasis).

-Keep total weekly frequency (e.g., 6 runs) but lower density (no back-to-back hard days).

-Distribute movement snacks (1–2 minutes of micro-sliders + hinge rehearsal) every 2–3 hours on heavy workdays.

This preserves fitness and reduces the “cost per dose” of neurodynamic work. [55–57,60–63,64–70,77,100]

4.3.14 Flare management in athletes

Athletes often test limits. The 3-step algorithm applies:

1.Single-variable rollback (e.g., reduce tensioner time-in-range from 10 s to 5 s for 48 h or shorten the tempo set by 20–30%).

2.Viscosity reset: hourly micro-sliders (45–60 s) on the affected side for one day; maintain breathing priming.

3.Language: frame the flare as training feedback, not failure; re-establish a success-bias within 24–48 h.

Do not detrain completely; keep easy runs if pain ≤2/10 and daily markers stable. [15,16,24–28,29,33,36,39,51–54,55–57,60–63,67–70,72–76,77,100]

4.3.15 Decision points and alternative pivots

-If mechanosensitivity dominates (large differentiation effects, quick quality changes): prolong slider-only phase; keep tensioners very brief and well-bracketed; expand breathing/imagery time. [27,28,33,36,39,51–54,60–63,72–76]

-If excursion limitation dominates (stubborn early SLR limit with little modulation): introduce mid-range tensioners earlier, but keep holds short and follow with sliders; consider more interface-oriented manual therapy; add hip joint mobilization if hinge remains restricted. [1–3,11–14,20,21,27,28,30,33,36–41,51–54,67–70,72–76,88–92]

-If tempo training uniquely provokes symptoms: check cadence and over-stride; cue external focus; shorten continuous tempo into cruise intervals with easy float between; schedule post-session sliders + breathing. [31,55–57,58,59,64–70,80–83,100]

-If hills provoke: limit steep grades initially; emphasize technique (shorter steps, cadence uptick, torso quiet); post-hill recovery sliders are mandatory. [31,55–57,58,59,64–70]

4.3.16 Athlete education: scripts and cues

Short script (pre-run):
“Breathe low and slow for a minute. Picture the nerve gliding. Keep ribs soft. During the run, think ‘leave the floor behind’ and ‘loose elbows.’ If the back tightens, shorten the stride a touch, take 3 relaxed breaths, and let the pelvis roll.” [25,26,55–57,60–63,64–66,100]

Short script (post-run):
“Two minutes: breathing + gentle sliders. Then hinge to take off shoes—no lumbar thrust. If stiff tomorrow, do tiny glides each hour. We’re training your system to move smoothly under load.” [1–3,24–28,29,33,36,39,51–54,55–57,60–63,67–70,72–76,77,100]

These scripts reduce cognitive load and promote adherence. [55–57,60–63,64–66,100]

4.3.17 Outcomes at 6 weeks and interpretation

Subjective/function.

-Pain reduced ~70% at peak efforts (NRS 6 → ~2/10 late in long runs; ≤2/10 post-tempo).

-Perceived gluteal tightness minimal; forward bend after runs is comfortable with soft cues.

-Confidence restored; athlete resumes structured training without fear of relapse.

Objective.

-SLR (R): from 55° → ~80° with neutral/stretch quality at previous angles.

-Slump: right-sided low back ache minimal and highly modifiable by structural differentiation.

-Tone/coordination: time-to-relax after forward bend halved; breath-holds absent in testing and drills.

-Proprioception: single-leg stance eyes-closed right 25–30 s, matching left.

-Gait: trailing-hip extension symmetric; arm swing restored; pelvic rotation normalized on video at easy and steady paces.

-Performance: long run 24 km pain ≤2/10; tempo 20 min continuous, pain ≤2/10; strides added without next-day flare. [1–3,22–24,27,28,30,31,33,36–39,41,50,51–54,58,59,67–70,72–76,80–87,93–99]

Mechanistic inference. The expected sequence of change (quality → range → tone/relaxation → proprioception → gait/automaticity → performance) emerged, supporting the model that restored neural excursion and reduced gamma-biased protection enabled efficient running mechanics. [23–25,30,31,39–41,50,58,59,67–70,80–83,93–99]

4.3.18 Limitations, caveats, and generalizability

-Multi tissue reality. SLR/slump are multi tissue; specificity is improved by structural differentiation and consistent quality-at-angle tracking, but not absolute. [1–3,11–14,27,28,29,33,36–39]

-High-level athletes may suppress symptom reporting; insist on objective markers (SLR angle/quality, time-to-relax, balance parity, gait symmetry). [22–24,30,31,50,58,59,80–87,93–99]

-Programming conflicts. Race timelines can pressure premature progression; protect consolidation windows (48–72 h after a successful step). [55–57,64–70,77,100]

-Alternative drivers. If no gains despite dose matching, broaden differential (hip joint pathology, bone stress, SI drivers, metabolic factors). [4–7,55–57,60–63]

4.3.19 Clinician checklist (per visit)

1.Priming (breathing + imagery) completed?

2.Sliders delivered without breath-holds?

3.If tensioners used: time-in-range increased before angle?

4.Manual therapy non-provocative and followed by sliders?

5.Functional transfer performed (gait bout with trailing-hip cue)?

6.Re-test captured (quality at fixed SLR angle; time-to-relax)?

7.RTR stage confirmed and adjusted based on markers and symptom rule (≤2/10)? [1–3,22–24,27,28,29,33,36–41,50,51–54,55–57,58,59,67–70,72–76,77,80–83,93–99,100]

4.3.20 Practical summary for the athlete

"Your nerves are living tissues that need to glide. Right now the sliding on the right isn't great, so your body wisely braces and shortens your stride to stay safe. We'll restore glide with small, frequent movements, retrain your breathing and hinge so the system relaxes while moving, and then spend those gains in your running form. We'll change one thing at a time, watch the day-after response, and build from quality to range to automaticity. The goal isn't just less pain—it's easier, more efficient running." [23–26,30,31,39–41,50,55–57,58,59,64–70,80–83,93–99,100]

4.3.21 Conclusion

This case demonstrates that a mechanism-aligned, integrated approach can produce meaningful, measurable improvements in an athletic population with subclinical sciatic neurodynamic restriction. By prioritizing glide before load, tracking quality at fixed angles, and embedding gains in running-specific tasks, the program reduced pain ~70%, increased SLR from 55° to ~80°, normalized gait (trailing-hip extension and arm swing), and enabled a safe return to structured training within six weeks. [1–3,27,28,31,33,36,39–41,50,51–54,58,59,67–70,72–76] The athlete's improvements followed the predicted physiological sequence: afferent quality normalized, spinal gamma bias decreased, proprioceptive maps sharpened, and motor policies shifted from high-stability bracing to elastic, efficient control. [23–25,30,31,39–41,50,58,59,67–70,80–83,93–99]
Equally important, the plan remained falsifiable at every step: if quality had not improved at a fixed SLR angle after sliders, or if gait had not changed despite better ranges, the model would have been revised. [29,33,36,41,55–57,93–99,100] This disciplined pairing of mechanistic rationale with behavioral transfer is what makes the approach robust for athletes whose performance depends on both capacity and coordination under repetitive load. [4–7,24,30,39–43,50,55–57,64–70,77,80–83,93–99,100]

4.4 Case Study: Chronic Sedentary Presentation

4.4.1 Case overview and clinical question

Patient profile. A 45-year-old accountant presents with a five-year history of chronic low back pain (CLBP) that is worsened by prolonged sitting and relieved by walking. Pain is described as a diffuse, band-like ache spanning L3–S1 with intermittent, focal

posterior pelvic discomfort just superior to the posterior iliac crest—more noticeable on the left. Secondary complaints include tight hamstrings bilaterally, stiffness when rising from a chair, and a tendency to brace the trunk before bending. No leg paresthesia, dermatomal radiation, night pain, or constitutional symptoms. Sleep is acceptable but not restorative during high-stress weeks. Physical activity is low: a brief dog walk most evenings; otherwise largely sedentary with long hours at a computer, limited breaks, and frequent end-of-quarter overtime. [4–7,55–57,60–63,78–80]

Initial observation. In quiet stance the patient shows mild anterior pelvic tilt with increased lumbar lordosis and visible paraspinal tone. During sit-to-stand there is a small lumbar extension thrust followed by a pause before fully upright. Forward bending is spine-dominant with limited hip hinge. Gait over 20 meters is symmetrical but slightly shortened in stride with damped arm swing; a three-minute walk reduces back discomfort—consistent with movement-responsive symptoms. [22–24,30,31,50,58,59,80–83]

Index tests and findings.

-Straight Leg Raise (SLR) left: ~50° with posterior thigh tightness and ipsilateral low back discomfort; no distal symptoms. Right SLR ~60° with similar but milder quality.

-Slump (seated): left-sided low back pain late in the sequence; symptoms reduce with cervical extension and/or ankle plantarflexion (i.e., structural differentiation). [1–3,11–14,27–29,33,36,39]

-Prone Knee Bend (PKB): negative bilaterally (no anterior thigh symptoms), arguing against femoral bias. [36,37]

-Posterior iliac crest palpation: discrete tenderness ~7–8 cm from midline over the iliac crest, consistent with superior cluneal nerve tunnel sensitivity. [20,21,88–92]

-Palpation of gluteals/hamstrings: increased tone and delayed relaxation bilaterally, left > right. [23,24,30,50]

-Neurological screen: strength, reflexes, and sensation intact. [4–7,47]

-Imaging: prior lumbar X-ray two years ago reported as “age-appropriate changes.” No MRI on file. [4–7,55–57]

Provisional hypothesis. Findings fit a combined neural phenotype: (1) sciatic pathway mechanosensitivity/reduced excursion (early SLR with neural-like quality, slump provocation that modulates with differentiators), and (2) superficial cluneal irritation at the posterior iliac crest (crest tenderness, posterior pelvic hot-spot). [1–3,11–14,20,21,27–29,33,36,38–40,88–92] Secondary consequences include tonic co-contraction (paraspinals, gluteals, hamstrings), reduced lumbopelvic dissociation, and substitution of lumbar flexion for hip hinge. [22–24,30,31,50,58,59,80–83] The clinical question: can a comprehensive neurodynamic approach—sciatic and cluneal sliders, interface-friendly manual therapy, and motor-control re-education—produce meaningful symptom reduction, restore SLR toward ≥75°, and increase sitting

tolerance without provoking mechanosensitivity? [1–3,27–29,33,36,38–41,39,40,50,51–54,67–70,72–76]

4.4.2 Sedentary context and mechanism fit

Sustained sitting places the posterior chain in relatively fixed positions with modest, repetitive neural tension and minimal glide. Over months to years, this can increase interface "stickiness" (thixotropic behavior of fascial/neural beds), impairing relative movement between nerve and surrounding tissues. [1,2,15–18,27,28,36,39,74] Simultaneously, psychosocial load (time pressure, deadlines, rumination) can bias autonomic tone upward, increasing gamma gain and paraspinal co-contraction as a default stability policy. [23–26,50,55–57,60–63,78,100] In such contexts, patients often present with:

-Early SLR limitation that modulates with ankle/neck changes (neural signature). [1–3,11–14,27–29,33,36,39]

-Vague posterior pelvic discomfort that maps to cluneal tunnels along the iliac crest. [20,21,88–92]

-A "tight hamstring" story that is partly protective co-contraction rather than sarcomeric shortness. [23,24,30,31,50]

-Relief with walking (small, frequent excursion cycles reduce viscosity and normalize afference). [31,39–41,58,59,67–70,80–83]

-This pattern matches the patient's history and exam and supports the dual-pathway hypothesis (sciatic + cluneal) as the primary driver of symptoms, with muscle "tightness" as a secondary adaptation. [1–3,20,21,27–29,33,36,38–41,50,88–92]

4.4.3 Differential diagnosis and reasoning

1.Facet-dominant pain. Extension-provoked, standing-worse presentations are common in facet syndromes. Here, pain is sitting-worse, walking-better; extension/rotation quadrant testing does not reproduce familiar pain; neural tests are more congruent with the complaint. Facet contribution may be secondary but not primary. [4–7,24,30,39,55–57]

2.Discogenic pain. Flexion-dominant or morning-worse pain, often with centralization behaviors, would raise this likelihood. The patient's symptoms improve with walking and do not centralize with repeated movements. Slump and SLR modulation point toward neurodynamic rather than nucleus-driven sensitivity. [4–7,10,11,33,36,39,55–57,60–63]

3.SI joint drivers. Posterior pelvic pain can stem from SI ligaments. However, the crest corridor tenderness and superficial slider comfort favor cluneal rather than ligamentous drivers. SI provocation tests are non-diagnostic. [20,21,88–92]

4.Myofascial syndrome (hamstrings/gluteals). Tonicity is evident but tracks with neural load and breath-holding, suggesting it is a reactive stability strategy rather than an isolated myofascial disorder. [23,24,30,31,50,58,59,80–83]

Conclusion. The most coherent diagnosis is combined sciatic mechanosensitivity/excursion loss plus superior cluneal irritation with secondary tonic bracing and limited hip hinge. [1–3,20,21,27–29,33,36,38–41,50,88–92] The plan should be mechanism-aligned and falsifiable at each step. [29,33,36,41,55–57,93–99,100]

4.4.4 Baseline assessment: minimum dataset and anchors

To capture change across the mechanism chain:

Symptoms & function.

-NRS worst with sitting: 6/10 after ≥45–60 minutes; 2–3/10 with walking.

-Sitting tolerance: 30–40 min without postural change; goal ≥90 min with micro-breaks.

-Patient-Specific Functional Scale (PSFS): (1) sit for 60–90 min (current 4/10), (2) bend to floor without bracing (4/10), (3) sleep through deadline weeks with pain ≤2/10 (5/10). [55–57,93–99]

Neurodynamic.

-SLR L: onset 50°, neural/stretch quality; improves with ankle plantarflexion and cervical extension. SLR R: onset 60°.

-Slump: left low back ache late in sequence; modulates with cervical extension/ankle plantarflexion.

-After-effect: mild stiffness for ~10 minutes if range is chased. [1–3,11–14,27–29,33,36,39,51–54,72–76]

Tone/coordination.

-Palpable paraspinal tone; time-to-relax after forward bend prolonged (subjectively “takes ~10–12 s”).

-Breath-holding during SLR/slump and sit-to-stand.

-Hinge score 0/2 (lumbar substitution). [23,24,30,31,41,50,58,59,80–83]

Proprioception.

-Trunk repositioning error elevated; hesitant path back to neutral.

-Single-leg stance eyes-closed: 8–10 s left; 12–15 s right. [22,58,59,80–87]

Gait.

-Shortened stride; reduced trailing-limb hip extension; muted arm swing. [31,58,59,80–83]

Posterior crest.

-Point tenderness ~7–8 cm from midline (left); superficial glides tolerated if amplitude is tiny. [20,21,88–92]

These anchors allow us to track quality at fixed angles, tone, maps, and transfer into function. [1–3,22–24,27–29,30,31,33,36–41,50,58,59,80–87,93–99]

4.4.5 Treatment rationale: integrated and staged

Objectives.

1.Restore neural excursion (sciatic sliders → tensioners when appropriate).

2.Reduce superficial cluneal irritation with tiny-amplitude glides and avoidance of compressive inputs.

3.Lower gamma-driven bracing via breathing/coordination and hip hinge retraining.

4.Enhance proprioception so the system trusts movement without co-contraction.

5.Translate gains to sitting tolerance via ergonomic strategies + micro-dosed mobility.

6.Keep the plan falsifiable by re-testing “quality at fixed angle” and crest tenderness every session.[1–3,20,21,23–25,27–29,30,31,33,36,38–41,39,40,50,58,59,67–70,72–76,80–87,93–99]

Core components.

Neurodynamic mobilizations:

-Sciatic sliders (supine SLR slider; seated slump slider).

-Progress to short-hold tensioners only after quality shifts and stable after-effects. [1–3,11–14,27–29,33,36,39,51–54,72–76]

-Cluneal superficial glides (posterior crest) 30–45 s, 1–2 bouts; no aggressive pressure. [20,21,38,40,88–92]

-Manual therapy (adjunct): low-load myofascial techniques to gluteals/posterior pelvis; follow with sliders to reinforce glide; never compress the crest tunnels. [20,21,27,28,30,38–40,72–76,88–92]

-Movement re-education: diaphragmatic breathing, hip hinge, pelvic tilts, short-arc cat-cow; micro-relaxation on exhale (measure time-to-soften). [23–25,30,31,41,50,58,59,67–70,80–83,100]

-Proprioceptive retraining: trunk repositioning; balance progressions; brief laterality/imagery. [22,58,59,80–87]

-Ergonomics & micro-breaks: chair setup, 90–120 min cycle with mobility "snacks," belt/waistband padding or relief for crest. [55–57,60–63,77,100]

-Home structure: 8–12 minutes, 2×/day; "spend the win" with a one-minute functional bout after mobilization. [24–26,31,39–41,55–57,64–70,93–99]

Priming (mandatory, 2 min).

-Paced breathing (6–8 breaths/min) + brief imagery (smooth sciatic glide; easy crest slide) + one rehearsal cue ("long spine, soft ribs, easy jaw.") [23–25,60–63,71,80–83,100]

4.4.6 Neurodynamic mobilizations: techniques and dosing

A) Sciatic sliders (foundation)

Supine SLR slider.

-Motion: hip flexion with plantarflexion ↔ slight hip extension with dorsiflexion; pelvis quiet; slow 2–3 s halves.

-Dose (start): 2–3 sets × 8–12 cycles, 1–2×/day.

-Cues: "glide, don't reach," "exhale on the loaded half."

-Progression: reps → micro-holds 3–5 s at the comfortable end → small angle increase after quality softens at fixed angle. [1–3,11–14,27–29,33,36,39,51–54,72–76]

Seated slump slider.

-Motion: small arcs pairing cervical extension + knee extension (reduced neural load) ↔ cervical flexion + knee flexion; optional ankle adjustment.

-Dose: 2 sets × 8–10 cycles, 1×/day.

-Progression: longer arcs only when symptom quality remains neutral at prior arcs. [1–3,11–14,27–29,33,36,39,51–54,72–76]

B) Sciatic tensioners (introduced later)

-Prerequisite: clear quality shift at a fixed SLR/slump angle and ≤24 h after-effects.

-Motion: from the end of a slider arc, add 5–8 s hold with quiet breathing, then back out to slider.

-Dose: 2–3 exposures per set, 1–2 sets; consolidate 48–72 h before increasing time-in-range or angle.

-Stop: sharp neural quality, breath-holds, or next-day flare—trigger single-variable rollback and viscosity reset (hourly micro-sliders for one day). [27–29,33,36,39,51–54,72–76]

C) Cluneal superficial glides (crest corridor)

-Motion: tiny-amplitude skin/fascial glides perpendicular to the crest; 30–45 s × 1–2 bouts; 1–3×/day; strictly non-provocative.

-Adjunct: short-arc trunk side-glide or small rotations with soft ribs; no compression from belts or hard chair edges; consider padding at waistband.

-Rationale: brief, frequent superficial motion reduces tunnel friction without sensitizing the nerve. [20,21,38,40,88–92]

4.4.7 Manual therapy (adjunct to facilitate glide)

-Gluteals/posterior pelvis: low-load myofascial holds (2–5 min total) to decrease paraneural tone; immediately follow with sliders to encode glide.

-Hamstrings: slow, graded techniques to reduce co-contraction; never chase “length” aggressively; pair with SLR sliders.

-Crest corridor: avoid direct compression; use only feather-light skin glides.

Manual therapy is supportive, not curative; its role is to lower dose cost and enable active neurodynamic work. [20,21,27,28,30,38–40,72–76,88–92]

4.4.8 Movement re-education and coordination

Breathing first.

-90/90 diaphragmatic breathing 1–2 min; cue paraspinal softening on exhale.

-Abort any set where breath-holding emerges; breathing continuity is non-negotiable. [23–25,60–63,71,80–83,100]

Hip hinge & pelvic control.

-Wall-touch hinge (external focus): 2–3 × 3–5 reps; hinge on exhale; no lumbar thrust on return.

-Pelvic tilts (3 × 10) to explore a comfortable neutral zone.

-Cat-cow (short arc) synced with breathing (1–2 min) within non-provocative ranges. [24,30,31,41,50,67–70,80–83]

Micro-relaxation KPI.

-After a standard forward bend, measure seconds to paraspinal softening; aim for 25–50% reduction over 6–8 weeks. [23,24,30,31,41,50,58,59,80–83]

4.4.9 Proprioceptive retraining and sensorimotor re-weighting

-Trunk repositioning drills: eyes closed → open; laser or inclinometer feedback; 2–3 min/day; focus on quality, not speed.

-Balance progressions: single-leg stance eyes open → eyes closed (increments of 5–10 s up to ~30 s), 3 sets per side; add micro-perturbations when stable.

-Laterality/imagery: 30–45 s before sliders to prime maps and reduce protective gain.

-Dynamic stabilization: short planks (3 × 15–20 s) with breath continuity; no Valsalva.

Goal: improve map precision so the CNS allows movement without default bracing. [22,23,25,30,50,58,59,71,80–87,100]

4.4.10 Sitting ergonomics and micro-break architecture

Chair and desk.

-Seat height so hips are slightly above knees; pelvis able to find neutral without gripping extensors.

-Backrest supports thoracolumbar junction without forcing extension.

-Keyboard/mouse close enough to avoid protracted reach (upper limb tension can up-regulate trunk co-contraction). [55–57,77,80–83,100]

Crest tunnel protection.

-Avoid belts or hard waistband edges pressing on the posterior iliac crest; if unavoidable, use padding and vary belt position through the day. [20,21,88–92]

Micro-breaks (every 30–40 min).

-Stand 60–90 s; two micro-sliders (each ~20–30 s); one hinge rehearsal with exhale; walk 30–60 s if possible.

-“Snack frequency” is more important than snack size—aim for 4–6 brief breaks per workday block. [55–57,64–70,77,100]

Alternative positions.

-Intermittent sit-stand if available; avoid static standing without mini-sways or heel-to-toe rock.

End-of-day decompressor (3–5 minutes).

-Breathing (1 min) → SLR sliders (1–2 min) → gentle gait with arm-swing (1–2 min).

These strategies directly target the sedentary driver—low excursion and sustained compression—while reinforcing therapeutic gains. [24–26,31,39–41,55–57,64–70,80–83,93–99]

4.4.11 Week-by-week clinical plan

Weeks 0–2: Desensitize, establish glide, protect crest

Session arc.

-Priming 2 min (breathing + imagery).

-Sciatic sliders: supine SLR (2–3 × 8–12), seated slump (2 × 8–10).

-Cluneal superficial glides: 30–45 s × 1–2 bouts; no pressure.

-Manual therapy: gluteals/posterior pelvis low-load 2–4 min → re-test quality at fixed SLR angle.

-Movement: hinge 2 × 3–4; pelvic tilts 3 × 10; cat-cow 1–2 min.

-Proprioception: trunk repositioning 2–3 min; balance eyes open 3 × 30 s/side.

-Ergonomics: chair setup; micro-break schedule adopted.

-Home: 8–12 min 2×/day—same sequence; add a 60–90 s “walk minute” afterward to spend the win.

Expected changes.

-Quality at 50° SLR (left) shifts from neural toward neutral/stretch; slump modulation increases; crest soreness tolerates tiny glides; fewer breath-holds on testing. [1–3,20,21,22–24,27–29,30,31,33,36–41,38–40,50,58,59,88–92]

Weeks 3–4: Expand excursion; begin mid-range tensioners

Criteria to progress.

-Quality shift at fixed angle; after-effects ≤24 h; crest tolerates hygiene well.

Additions.

-Sciatic mid-range tensioners: 5–8 s holds, bracketed by sliders; 2–3 exposures; increase time-in-range before angle.

-Movement: hinge 2 × 5; introduce short plank 3 × 15–20 s with breath continuity.

-Proprioception: balance eyes closed (10–15 s holds).

-Ergonomics: micro-break compliance review; add a standing email block with mini-sways if tolerated.

Expected changes.

-SLR left 55–60° or better quality at 50°; time-to-relax after bend improves ~25%; sitting tolerance increases by 10–15 minutes without spike. [1–3,22–24,27–29,30,31,33,36–41,50,58,59,67–70,72–76,93–99]

Weeks 5–6: Consolidate excursion; integrate context; reduce guarding

Neurodynamic.

-Tensioners: 8–10 s holds; small angle increase if after-effects stable; maintain frequent sliders.

-Crest hygiene: continue; potentially reduce frequency if calm.

Movement.

-Hinge with light load (6–8 kg) 2 × 5; anti-rotation holds (10–15 s).

-Expand cat-cow arc slightly, avoiding neural quality.

Proprioception.

-Balance eyes closed toward 20–25 s; laterality/imagery before sliders on workdays.

Function.

-Integrate sit-to-stand sets (2 × 3–5) during micro-breaks; add short hallway walks.

-Cue “exhale-to-move” on rising from chairs.

Expected changes.

-SLR left 65–70°; slump much less reactive and more differentiation-sensitive; crest tenderness minimal to light pressure; sitting tolerance ~60–75 minutes with planned micro-breaks. [1–3,20,21,22–24,27–29,30,31,33,36–41,38–40,50,58,59,67–70,88–92,93–99]

Weeks 7–8: Generalize; stress-test sitting; pre-empt relapse

Neurodynamic.

-Optional brief forays toward near end-range with 3–5 s holds, always bracketed by sliders; no provocation.

Movement.

-Hinge load optional to 10–12 kg only if breathing remains quiet; maintain soft ribs.

Function & ergonomics.

-Trial 90-minute desk block with timed micro-breaks (every 30–40 minutes).

-Add daily 10-minute walk after lunch.

-Continue crest padding policy if belts/waistbands are used.

Outcomes (target).

-NRS reduced ~60%; SLR left ~75° with neutral/stretch quality at previous angles; sitting tolerance ≥90 minutes with micro-breaks; posterior pelvic discomfort rare and controllable. [39,40,55–57,58,59,93–99]

4.4.12 Measurement trajectory and interpretation

Expected sequence of true mechanism change.

1.Quality at fixed angle improves first (e.g., SLR left at 50° shifts neural → neutral/stretch).

2.Range increases (50° → 55–60° by weeks 3–4; → 70–75° by week 8).

3.Tone/coordination: time-to-relax halves; breath-holds extinguish in testing and sit-to-stand.

4.Proprioception: trunk repositioning smoother; eyes-closed balance approaches 20–30 s bilaterally.

5.Function: sitting tolerance increases; standing transition smoother with less bracing; hallway walk “resets” quicker.

6.Crest behavior: tenderness minimal and non-zingy; superficial glide remains comfortable.

If SLR improves but sitting tolerance does not, target context: stricter micro-break timing, add between-set walking, verify belt/waistband compression is not re-irritating the crest, and emphasize breathing continuity during work tasks. [1–3,20,21,22–24,27–29,30,31,33,36–41,38–40,50,58,59,80–87,88–92,93–99]

4.4.13 Home program (8–12 minutes twice daily)

1.Priming (2 min): slow diaphragmatic breathing + 30–45 s imagery (“smooth nerve glide; easy crest slide”) + cue “long spine, soft ribs.” [23–25,60–63,71,80–83,100]

2.Sciatic sliders (4–6 min): supine SLR and/or seated slump; 2–3 × 8–12 cycles; no breath-holds. [1–3,11–14,27–29,33,36,39,51–54,72–76]

3.(When indicated) Tensioners (1–2 min): 3–4 exposures of 5–10 s, bracketed by sliders. [27–29,33,36,39,51–54,72–76]

4.Movement/proprioception (2–3 min): hinge or pelvic tilts + 30–60 s trunk repositioning/balance. [22–24,30,31,58,59,80–87]

5.Functional minute (1–2 min): walk minute (hallway), or 2–3 sit-to-stands with exhale-to-move. [24–26,31,39–41,55–57,64–70,93–99]

Flare plan (posted on the fridge).

-Roll back one variable (time, range, or reps) by ~30–40% for 48 hours.

-Add hourly micro-sliders (45–60 s) for one day.

-Keep breathing and walk minute; avoid belt/waistband pressure over crest. [15,16,24–29,33,36,39,51–54,60–63,72–76,77,100]

4.4.14 Workplace coaching: scripts and checklists

Morning setup (60 seconds).

-Pelvis neutral, ribs soft, feet grounded; belt/waistband not pressing on the crest; timer set for 40-minute cue.

Micro-break (90 seconds).

-Stand → 2 gentle slider cycles → one hinge on exhale → walk to printer and back; sip water.

Meeting strategy.

-If seated >30 minutes, subtly alternate postures and perform ankle pumps; if allowed, stand briefly to speak; avoid static stand—use mini-sways.

End of block (2–3 minutes).

-Breathing + 1–2 slider sets; short hallway walk; optional superficial crest glide for 30–45 s if comfortable.

-Self-talk script.
“Polish, don’t provoke. Change one thing at a time. Spend the win—walk a little after gliding.” [24–26,31,39–41,55–57,64–70,93–99,100]

4.4.15 Decision points and troubleshooting

-Mechanosensitivity-dominant (large differentiation effects, rapid quality changes): remain longer in sliders; keep tensioners very short and well-bracketed; prioritize breathing and micro-frequency. [27–29,33,36,39,51–54,60–63,72–76]

-Excursion-dominant (stubborn early SLR limit with little modulation): introduce mid-range tensioners earlier (5–8 s) with strict time-in-range before angle; pair with interface-oriented manual therapy; confirm pelvis stability during SLR. [1–3,11–14,20,21,27–29,30,33,36–41,38–40,51–54,72–76,88–92]

-Crest remains irritable: reduce local glide frequency; address external compression; bias proximal/distal superficial motion (trunk side-glide, small rotations) without local pressure; re-check chair back edge location. [20,21,38,40,88–92]

-Sitting still provokes despite improved tests: increase micro-break frequency instead of duration; use walk minutes; insert 2–3 sit-to-stands with exhale every break; consider sit-stand desk with mini-sways. [55–57,64–70,77,100]

-Flare after tensioners: invoke rollback + viscosity reset; retain functional walking; revisit breathing continuity and cueing. [24–29,33,36,39,51–54,60–63,72–76,100]

4.4.16 Safety and boundaries

-Avoid end-range neural tension in high-irritability states; emphasize sliders and breathing.

-Screen continually for red flags (new neuro deficits, constitutional signs). [4–7,47–49,55–57,60–63,77]

-Respect comorbidities (e.g., osteoporosis, anticoagulation): favor small arcs, slow tempo, superficial techniques. [4–7,55–57,77]

-Do not compress suspected cluneal tunnels; use padding and positional variation. [20,21,38,40,88–92]

4.4.17 Outcomes at 8 weeks and interpretation
Subjective/function.

-Pain NRS reduced by ~60% at worst (from 6/10 to ~2–3/10 after longer desk blocks).

-Sitting tolerance improved from 30–40 min to ≥90 min with planned micro-breaks (every 30–40 min), without next-day spike.

-The patient reports greater confidence standing from chairs without bracing and less "hamstring tightness."

Objective.

-SLR L improved from 50° to ~75° with neutral/stretch quality at prior angles; SLR R ~80°.

-Slump: left low back ache minimal and highly differentiation-sensitive; overall intensity down.

-Crest tenderness: minimal with light palpation; no "zing"; superficial glides entirely comfortable.

-Tone/coordination: time-to-relax after forward bend reduced by ~50%; breath-holds no longer evident in testing or sit-to-stand.

-Proprioception: trunk repositioning smooth; single-leg eyes-closed 20–25 s bilaterally.

-Gait: longer stride, improved trailing-hip extension, arm swing less damped. [1–3,20,21,22–24,27–29,30,31,33,36–41,38–40,50,58,59,80–87,88–92,93–99]

Mechanistic inference. The sequence of change aligns with the model: quality → range → tone/relaxation → proprioception → functional automaticity (sitting tolerance). Improvements in quality at fixed angles and crest comfort preceded functional gains, supporting the role of restored excursion and reduced segmental gain, not merely compensation. [23–25,30,31,39–41,50,58,59,67–70,80–83,93–99]

4.4.18 Limitations, caveats, and generalizability

-Multi tissue loading. SLR and slump load nerve, muscle, fascia, and joint; neural attribution relies on structural differentiation and intra-session change at fixed angles—never on a single test. [1–3,11–14,27–29,33,36–39]

-Behavioral contributors. Stress, sleep debt, and deadline cycles can amplify protective gain; the plan should incorporate graded activity, micro-recovery, and basic sleep hygiene. [26,55–57,60–63,78–80,100]

-Chair constraints. Not all offices permit sit-stand or frequent breaks; when constrained, emphasize micro-motion while seated (ankle pumps, small pelvic tilts, rib softening) and brief walk minutes between tasks. [55–57,64–70,77,100]

-Expect plateaus. At 4–6 weeks some patients plateau; revisit dose choreography (time-in-range vs angle), breathing, and transfer. If no shift occurs after good adherence, expand the differential (hip OA, SI drivers, mood/sleep disorders). [4–7,55–57,60–63,78–80]

4.4.19 Clinician checklist (per visit)

1.Priming (breathing + imagery) completed?

2.Sliders delivered without breath-holds?

3.If tensioners used: time-in-range progressed before angle?

4.Manual therapy non-provocative and followed by sliders?

5.Cluneal hygiene applied; any external compression sources mitigated?

6.Re-test recorded (quality at fixed SLR angle; crest tenderness; time-to-relax)?

7.Ergonomics/micro-break adherence reviewed and adjusted?

8.24-hour plan: progress, consolidate, or flare protocol? [1–3,20,21,22–24,27–29,30,31,33,36–41,38–40,50,55–57,58,59,72–76,77,80–83,88–92,93–99,100]

4.4.20 Patient one-minute brief (fridge card)

“Breathe slow (1 minute). Picture the nerve gliding and the belt-line area sliding easily. Do gentle sliders—polish, don’t provoke. If it feels easy tomorrow, hold a few seconds at the edge. Keep ribs soft. Spend the win—walk a minute or do a couple of sit-to-stands. If it’s cranky tomorrow, roll back one notch and do tiny glides each hour. Change one thing at a time.” [24–26,31,39–41,55–57,60–63,64–70,93–99,100]

4.4.21 Conclusion

This case exemplifies the sedentary CLBP phenotype in which long periods of sitting, low movement variability, and modest psychosocial load converge to sustain subclinical neural restrictions—notably sciatic mechanosensitivity/excursion loss and superior cluneal irritation. The patient's hallmark signs—early SLR limit with neural quality that modulates under structural differentiation, posterior crest tenderness consistent with cluneal tunnels, tonic co-contraction, poor hinge, and relief with walking—map tightly to the proposed mechanism. [1–3,20,21,23–25,27–29,30,31,33,36,38–41,50,58,59,88–92]
A mechanism-aligned intervention—sciatic and cluneal sliders, interface-friendly manual therapy, breathing-anchored motor control, proprioceptive tune-up, and ergonomic micro-breaks—produced clinically and functionally meaningful change over eight weeks: ~60% pain reduction, SLR improvement to ~75°, and sitting tolerance ≥90 minutes with planned micro-breaks. [1–3,20,21,27–29,33,36,38–41,39,40,50,58,59,67–70,72–76,88–92,93–99] Crucially, the sequence of improvements (quality → range → tone → proprioception → function) supports genuine mechanism modification rather than compensatory work-arounds. [23–25,30,31,39–41,50,58,59,67–70,80–83,93–99]
The case also highlights pragmatic levers for sedentary patients: micro-dose frequency, walk minutes, crest padding, and one-variable progressions. When progress stalls, clinicians should revisit dose choreography, verify breathing continuity, strengthen functional transfer, and reassess context (stress, sleep, workstation constraints). Above all, the pathway remains falsifiable—every session pairs a small, safe exposure with a re-test of quality at a fixed angle or crest comfort. If those markers don't budge, the model adjusts. That disciplined loop is why this approach is not only effective in the clinic but also testable in research and scalable to the everyday realities of patients who spend most of their waking hours in a chair. [29,33,36,39–41,55–57,77,93–99,100]

4.5 Integration with Existing Models

4.5.1 Why integration is necessary (and overdue)

Chronic low back pain (CLBP) is not a single disease; it is a syndrome whose presentation emerges from multiple, partially independent subsystems—biological tissues and interfaces, sensorimotor control, and psychosocial context—interacting in nonlinear ways over time. The last two decades have moved practice away from a "find-the-lesion" paradigm toward multidimensional models: the biopsychosocial framework, central sensitization theory, fear-avoidance and graded exposure approaches, predictive processing accounts of pain perception, and contemporary motor control and behavior-change models (5,6,25,26,43,57,60–63,77). Yet, in this shift, neural mechanics—how peripheral nerves and their interfaces slide, strain, and signal—have often been relegated to a niche corner of manual therapy or treated as synonymous with "sciatica" (1,2,7,9,29). The thesis advanced here is that subclinical neurodynamic restrictions—modest, often non-radicular reductions in neural excursion with accompanying mechanosensitivity—constitute a specific and modifiable dimension within CLBP that is complementary to, not competitive with, the broader models (1–3,11,29,33,39,44,51–54,74–76). The neurodynamic lens adds

an actionable driver that may sustain protective motor behavior and pain even when psychosocial education and biomechanical exercise have been optimized (5,6,25,26,39–43,51–54,74–76).

This section articulates how the neurodynamic model fits inside the dominant frameworks—not as a rival theory but as a plug-in mechanism that clarifies assessment, dosing, and outcome interpretation. We show concrete pathways for integration, clarify where the models overlap and where they diverge, and provide clinical decision rules that keep practice falsifiable and patient-centered (5,6,25,26,39–43,51–54,74–76).

4.5.2 The biopsychosocial model: where neurodynamics nests

The biopsychosocial (BPS) model posits that biological factors (tissue status, nociception), psychological factors (beliefs, expectations, mood), and social factors (work demands, family roles, culture) jointly shape pain and disability (5,6,57,60–63,77). This breadth prevents reductionism, but its very generality can limit day-to-day mechanistic precision: clinicians agree the system is multifactorial, yet struggle to decide which lever to pull first and how to measure whether a chosen lever actually moved the needle.

Neurodynamics supplies a concrete biological lever that links directly to psychological and behavioral domains:

1.Biological layer. Subclinical reductions in neural excursion and increases in mechanosensitivity can amplify afferent noise from the lumbosacral region. This noise need not reflect frank pathology; it is enough to bias spinal gain and fuel protective reflexes, producing co-contraction and movement variability loss (23,27,28,39–41,44,50). Neurodynamic testing (e.g., quality at a fixed SLR/slump angle) provides a repeatable biomarker for this layer (1–3,11,29,33,36,37,45,46).

2.Psychological layer. If neural tissue feels “zingy” at particular arcs, the brain learns “that range is unsafe.” Fear-avoidance can consolidate around these arcs even in the absence of structural disease (25,26,60,61,100). Brief, successful exposures using sliders can reconsolidate memories with corrective prediction errors, especially when paired with pain neuroscience education (PNE) and graded activity (25,26,64–66).

3.Social/behavioral layer. Jobs that require long sitting (sedentary accountant) or repetitive lifting (warehouse worker) present contextual loads that repeatedly bias certain neural postures (5,6,57,77). Integrating micro-glide breaks or task sequencing at work changes the environmental contingencies that keep the problem alive (39,40,74–76,88–92).

By making the “bio” in BPS measurable and trainable—with clearly defined dosing rules (glide → time-in-range → angle; one variable at a time)—the neurodynamic

approach clarifies what to treat first and how to know it worked, while leaving ample room for parallel psychological education and social/context modification (5,6,25,26,39–43,51–54,57,74–76,77).

4.5.3 Central sensitization and predictive processing: a place for peripheral truth

Central sensitization (CS) describes a state of increased central nervous system responsiveness to sensory input, leading to amplified pain and spread beyond the original tissue source (5,6,48,49,60–63,78,79). Predictive processing models extend this view: the brain is a Bayesian predictor, integrating sensory evidence with prior beliefs to generate the best guess about threat and needed action; pain, in this view, is not a direct readout of nociception but a percept shaped by priors and precision weighting (23,25,62).

Where does neurodynamics fit?

-As a precision tool. In CS, the system may assign high precision to noisy afferents and low precision to contextual safety cues (48,49,60–63,78,79). Small improvements in peripheral signal quality (e.g., less neural "sting" at a fixed angle) reduce the variance of incoming data. This allows the CNS to down-weight threat predictions. The repeated experience of "this range is now comfortable" constitutes a prediction error that updates priors (23,25).

-As a safe exposure vehicle. Sliding techniques deliver controlled, low-threat sensory evidence precisely at the range that had been coded as dangerous (1,2,29,33,39,51–54,72–76). When paired with breath control and non-harm framing (PNE), they produce convergent learning signals: sensory input says "safe," interoception says "calm," and cognition says "this makes sense" (23,25,26,62,71).

-As a gatekeeper for progressions. If a patient's CNS is highly sensitized, tensioners and end-range holds may be too "loud." The neurodynamic dosing rules (sliders first, time-in-range before angle) function as a graded exposure dial that respects central sensitivity while still engaging the peripheral contributor (25,39,51–54,74–76).

Thus, neurodynamics is neither peripheralist nor centralist; it acts as a bridge that shapes the information stream upon which central models operate, creating conditions for belief updating and behavior change (5,6,23,25,48,49,60–63,78,79).

4.5.4 Fear-avoidance, graded exposure, and motor adaptation: aligning languages

The fear-avoidance model explains how catastrophizing and fear of movement lead to avoidance, deconditioning, and sustained pain (25,26,60,61,100). Graded exposure treats this by systematically confronting feared activities, beginning well below panic thresholds (25,26,64–66). In CLBP, there is a tight kinship between feared movements and angles that increase neural strain (e.g., long-stride hip flexion, deep sitting slump) (1–3,11,29,33,36,37,39,50).

The neurodynamic model provides:

-Precise stimuli for exposure: rather than generically "bend forward," we can ask patients to perform two sets of 8–10 SLR sliders with breathing, calibrated to keep quality neutral. The target is not "more range at all costs," but "consistent quality at a fixed angle," a metric that patients can feel and clinicians can measure (1–3,11,29,33,36,37,39,51–54,72–76).

-Immediate reinforcement loops: After a slider set, the patient spends the win in a micro-task that previously felt risky—e.g., a 60–90 second gait bout emphasizing trailing-limb hip extension. This exploits short-term metaplasticity: the nervous system is more willing to update movement policies immediately after a low-threat exposure (25,26,41,50,71).

-Breath-anchored control: Fear manifests as breath-holding and bracing. Mandating breathing continuity converts every exposure dose into a dual intervention: mechanical (glide) and autonomic (down-shifting gamma gain). This is graded exposure + motor control in one move (23,25,41,50,71,72).

The end result is a coherent narrative the patient can adopt: "We're teaching your nerves to glide where they used to feel sketchy, while your breathing tells your brain you are safe; then you immediately use that safer range in a real task so your system remembers it" (25,26,41,50,71).

4.5.5 Biomechanics and manual therapy: from "aligning bones" to "improving interfaces"

Traditional biomechanical approaches in CLBP have focused on joint alignment, stability, and muscle length/strength imbalances. Manual therapy aimed to "correct" these issues via mobilization and manipulation. Modern evidence encourages a shift in language from correction to capacity and options (39,40,43,57,77). Neurodynamics can enrich this reframing:

-Joint techniques as interface facilitators. Rather than claiming a thrust "realigns" a joint, we can say it reduces guarding and temporarily increases options for movement. Immediately following manipulation with sliders leverages that window to improve neural glide—a plausible biological mechanism for the clinical effect (39,40,43,72,73,74–76).

-Soft tissue work as friction management. Low-load myofascial techniques around the gluteal or posterior crest regions may reduce superficial friction around sciatic and cluneal corridors (27,28,38,39,40,88–92). The test of relevance is not the "release feel" but a change in quality at a fixed neurodynamic angle. If quality changes, the manual dose is mechanism-relevant; if not, it was pleasant but non-essential (1–3,11,29,33,36,39,45,46,72–76,88–92).

-Strength and mobility training as consolidation. Once glide improves, compound movements (hinge, squat variants, carries) consolidate the new options. Cueing emphasizes elastic posture over rigid bracing, keeping with the neurodynamic view that low-threat movement sustains the gains (41,50,68–70,71,72,77).

In short, biomechanical tools remain valuable, but their aim is clearer: make the interfaces more permissive, then teach the system to use those options under real-world constraints (39,40,43,51–54,68–70,71–76,77).

4.5.6 A practical integration map (clinician-ready)

To make the integration tangible, consider a four-lane map—Neurodynamic, Education, Behavior, and Strength/Capacity—advanced in parallel, with traffic rules to avoid crashes (5,6,25,26,39–43,51–54,60–63,72–76).

Lane 1: Neurodynamic (mechanical information)

-Start with sliders in the most relevant position(s) (SLR, slump, PKB) (1–3,11,29,33,36,39,40).

-Dose by irritability; change one variable (time-in-range → angle) (33,39–41,51–54).

-Re-test quality at fixed angle each session to validate the mechanism (33,36,37,39,45,46).

Lane 2: Pain education & beliefs (cognitive information)

-Deliver brief PNE micro-lessons that contextualize sliders ("nerves like to glide; safe movement rewrites maps") (5,6,25,26,60–63,64–66).

-Link neurodynamic change to the meaning of safety: "When the same angle feels normal, your nervous system updates its prediction" (23,25,26,62).

Lane 3: Behavior & exposure (experiential information)

-Embed functional “spend the win” moments between slider sets (gait, hinge, sit-to-stand) (39–41,50,71–73).

-Use graded exposure rules (below flare threshold, frequent, successful) (25,26,60–63,64–66).

Lane 4: Strength/Capacity (consolidation information)

-Introduce low-threshold loading with breathing continuity (short planks, anti-rotation holds) (41,50,68–70,72,77).

-Progress only when neurodynamic quality and after-effects are stable (33,39–41,51–54).

Checklist rules:

-If neurodynamic quality worsens post-session, roll back one variable (range/time/reps) and keep education and behavior lanes flowing (33,39–41).

-If beliefs remain rigid despite quality change, increase PNE dose and highlight objective wins (angles, balance time) (5,6,25,26,60–63).

-If behavior is sticky (e.g., breath-holding persists), allocate time to breath-anchored drills before re-attempting tensioners (23,25,41,50,71,72).

This map converts abstract integration into daily scheduling and dose choreography (39–43,51–54,72–76).

4.5.7 Mediation logic: how to know which lever mattered

Integrative care must remain falsifiable. We therefore pair each lane with candidate mediators—variables that should change first if the lane is working (3,5,6,23,25,44–46,57):

-Neurodynamic mediator: quality at fixed angle (SLR/slump/PKB) improves before range expands (1–3,11,29,33,36,37,39,45,46).

-Education mediator: a shift in threat appraisal (reduced fear on movement-specific items, improved pain self-efficacy) (5,6,25,26,60–63,64–66).

-Behavior mediator: fewer breath-holds, quicker time-to-relax, increased movement variability in a target task (23,25,41,50,71,72).

-Strength/Capacity mediator: improved dose tolerance (e.g., more time-in-range without after-effects), not just heavier loads (39–43,68–70,72,77).

If symptoms improve without the expected mediator changing, re-examine attribution: perhaps the relief came from sleep improvement or social support that week (BPS), or from non-specific alliance effects (5,6,57,77). If the mediator improves without symptom change, suspect measurement mismatch (e.g., testing an irrelevant angle) or transfer failure (benefits not being used in daily life) (3,25,44–46,57).

4.5.8 Integrating with central sensitization-oriented care

For patients with high CS features (widespread pain, allodynia, fatigue, sleep disturbance),[48,49] the entry point for neurodynamics is smaller and gentler (5,6,23,25,60–63,78,79):

-Micro-sliders (30–60 seconds, hourly on flare days) with strict breathing continuity (23,25,39–41,51–54).

-No tensioners until after-effects are predictably ≤24 hours (39–41,51–54,74–76).

-Higher emphasis on education (explaining safety), sleep hygiene, and stress-dose management (5,6,25,26,57,60–63,78,79).

-Early wins may be non-pain (smoother movement, less guarding, better balance); celebrate and link them to safety learning (23,25,41,50,71,72).

This keeps the central focus while still addressing a peripheral contributor that can be softened without provoking the system (5,6,23,25,48,49,60–63,78,79).

4.5.9 Integrating with graded activity and pacing

Graded activity schedules function like behavioral contracts that ensure frequency and gradualism (25,26,60–63,64–66). Neurodynamic work slots naturally into these contracts:

-Treat each slider set as a “step” with a box to check (25,26,39–41,51–54).

-Use time-based rather than symptom-based progression early (e.g., 2 minutes/day of sliders, then 3 minutes) (25,26,39–41).

-Pair each step with a micro-walk or hinge rehearsal to generalize learning (39–41,50,71–73).

Pacing protects against over-dosing tensioners in enthusiastic patients. The single-variable rule (change one thing at a time) is pacing by design (33,39–41,51–54).

4.5.10 Integrating with acceptance and commitment therapy (ACT) and mindfulness

ACT emphasizes values-based action in the presence of persistent symptoms and cognitive defusion from unhelpful thoughts. Mindfulness trains non-reactivity and interoceptive awareness (25,26,60–63,67). Neurodynamics complements both:

-During sliders, patients practice noticing sensations without fusion ("that's a stretch, not danger") and maintain breathing continuity—an embodied mindfulness (23,25,41,50,67,71,72).

-Values link: assign "spend the win" tasks that connect to values (walking the dog, gardening), reframing exposures as meaningful, not merely clinical (25,26,60–63,67).

This pairing is especially potent in patients whose primary barrier is vigilance and catastrophizing rather than stubborn mechanical restriction (25,26,60–63,67).

4.5.11 Integrating with sleep, lifestyle, and metabolic considerations

Sleep deprivation and metabolic stress increase pain sensitivity and reduce learning rates (5,6,57,60–63,78,79). Integrating brief evening decompressor routines—two minutes of breathing + sliders + a short walk—serves both neurodynamic and sleep-facilitating ends (23,25,39–41,51–54). Low-intensity aerobic conditioning with nasal/diaphragmatic breathing acts as a systemic desensitizer, reinforcing the autonomic balance targeted during neurodynamic exposures (57,60–63,68–70,78,79). Lifestyle shifts therefore become contextual amplifiers of mechanical work, not "extras" (5,6,25,26,57,77).

4.5.12 Interfacing with imaging and structural findings

In many CLBP cases, imaging is non-specific or normal. In others, incidental disc bulges or Modic changes are found (44–46,57,77). The neurodynamic model avoids simplistic structural determinism:

-If imaging is unremarkable but SLR/slump quality is clearly neural at fixed angles that improve with dosing, neurodynamic restriction is a plausible maintainer of pain despite “nothing on MRI” (1–3,11,29,33,36,39–41,44–46).

-If imaging shows degenerative features but neurodynamic markers are quiet, structural findings may be clinically silent; chase function, not pictures (44–46,57,77).

-If both imaging and neurodynamic tests are positive, set shared decision rules: we will pursue a six-week mechanism-aligned plan and track objective mediators; if they do not budge, reconsider the structural pathway (3,5,6,44–46,57).

This protects patients from nocebo while keeping open avenues for escalation when warranted (5,6,25,26,43,57).

4.5.13 Safety, ethics, and the “nothing magical” stance

Integrating neurodynamics ethically means:

-No cure claims. We are testing a mechanism, not promising a miracle (5,6,25,43).

-Dose transparency. Explain why sliders come before tensioners and why breath matters (23,25,33,39–41).

-Stop rules. New neurological deficits, night pain, fever, weight loss—follow red-flag pathways (5,6,57,77).

-Shared decisions. Patients choose progression pace; clinician provides structure and guardrails (5,6,25,26,43,57).

This stance aligns with modern ethical practice across BPS-informed care (5,6,25,26,43,57).

4.5.14 A stepped-care template using integrated logic

Step 1 (2–3 weeks): Education + sliders + breathing + micro-functional "spend the win." Outcome: quality at fixed angle improves; after-effects ≤24 h (1–3,11,25,26,33,39–41,51–54).

Step 2 (Weeks 3–6): Add mid-range tensioners; expand functional contexts (gait, sit-to-stand); introduce low-threshold loading; continue PNE and pacing. Outcome: range expands; tone (time-to-relax, breath-holds) improves (23,25,33,39–41,50,68–70,71–73).

Step 3 (Weeks 6–10): Consolidate with strength and conditioning; normalize gait/hinge; progress exposure to previously feared tasks. Outcome: transfer to life roles; symptom/PSFS improvement; stable self-management (5,6,25,26,39–43,68–70,72,77).

Step 4: If mediators do not shift despite adherence, re-phenotype (consider hip/SI, sleep/mood contributions, alternative drivers) and adjust plan or escalate (3,5,6,23,25,44–46,57,77).

This structure integrates the models (BPS, CS, graded exposure, motor control, conditioning) with neurodynamics as the biological keystone where relevant (5,6,23,25,26,39–43,48,49,51–54,60–63,68–70,72–76).

4.5.15 Research implications: testing the integrated hypothesis

Integration should invite better trials, not blur mechanisms. We propose:

-Design: randomized pragmatic trial comparing (A) education + generalized exercise vs (B) education + generalized exercise + neurodynamic dosing; both arms receive equal contact time (3,39,43,51–54,57).

-Primary mediator: quality at fixed neurodynamic angle at 2 weeks (33,36,37,39,45,46).

-Secondary mediators: breath-hold frequency, time-to-relax, fear of movement, balance time (23,25,41,50,60–63,64–66,68–70,71–73).

-Outcomes: pain, ODI/RMDQ, PSFS, gait metrics, return-to-function (5,6,25,26,39–43,57,77).

-Hypothesis: the integrated arm shows earlier mediator shifts and greater "spend the win" transfer, predicting superior functional outcomes at 6–12 weeks (3,25,39–43,51–54,57).

Such a trial honors the BPS model while specifying how the “bio” component contributes (5,6,25,26,43,57).

4.5.16 Case-mapping exemplars (condensed)

-Athlete with tempo-provoked CLBP (Section 4.3): BPS lens flags training load and beliefs about bending; CS lens warns against over-exposure; neurodynamics identifies sciatic excursion as a tractable driver; integrated program restores glide, then consolidates in running mechanics (39,41,48–50,68–70).

-Sedentary worker with crest pain (Section 4.4): BPS lens highlights desk ecology and stress; CS lens recommends gentler doses; neurodynamics targets sciatic + cluneal restrictions with superficial glides; ergonomic micro-breaks change the social/context layer (39,40,48,49,57,77,88–92).

-Fear-avoidant patient with “fragile back” beliefs: Education addresses beliefs; neurodynamics delivers safe sensory proof; graded exposure rebuilds activity; motor control replaces bracing with elastic posture (25,26,39–43,60–63,64–66,71–73).

These vignettes show that integration is not a slogan but a work flow (5,6,25,26,39–43,48–50,57,77).

4.5.17 What integration is not

-It is not doing everything to everyone all the time. It is sequencing the fewest effective levers that fit the phenotype (5,6,25,26,39–43).
-It is not abandoning strength training; rather, it uses strength to consolidate neurodynamic wins (39–43,68–70,72,77).

-It is not ignoring central factors; it uses peripheral clarity to make central learning easier (5,6,23,25,48,49,60–63,78,79).

-It is not a rebrand of stretching; sliders and tensioners obey distinct rules (reciprocal motion first, time-in-range before angle, structural differentiation to validate) (1–3,11,29,33,36,39–41,45,46,51–54,72–76).

4.5.18 Putting it all together: the one-page integration algorithm

1.Screen for red flags; classify irritability (5,6,33,39–41,57).

2.Test Slump/SLR/PKB and record quality at fixed angles; check crest for cluneal signs (1–3,11,29,33,36,38–40,45,46).

3.Decide: Is a neural pathway implicated (mechanosensitivity and/or excursion loss)? (1–3,11,29,33,36,39–41,44–46,50).

4.If yes, launch sliders + breathing; deliver PNE micro-lesson; insert one functional spend; set micro-goals for the week (5,6,23,25,26,33,39–41,51–54).

5.Re-test a marker in the same session; if improved, consolidate 48–72 h (33,39–41).

6.Progress to mid-range tensioners only after stable quality and after-effects; keep education and behavior lanes active (39–41,51–54,60–63,64–66).

7.Integrate low-threshold strength; keep cues external and breath-anchored (39–43,68–70,71–73,77).

8.If markers stall, adjust dose or re-phenotype (consider CS emphasis, sleep/stress, alternative tissue drivers) (3,5,6,23,25,44–46,48,49,57,78,79).

9.Discharge with a minimalist maintenance plan (breathing + brief sliders + values-based functional bouts) (25,26,39–41,51–54).

The algorithm is agnostic to practitioner background; it simply choreographs the lanes (5,6,25,26,39–43,57).

4.5.19 Limitations and cautions

-Specificity constraints. Neurodynamic tests are multitissue; structural differentiation and intra-session change bolster attribution but cannot make it perfect (1–3,11,29,33,36,39–41,45,46).

-Heterogeneity of CLBP. Some patients improve chiefly through context change (sleep, workload) with minimal need for neurodynamic work. Integration means being selective, not dogmatic (5,6,44,45,57,77).

-Risk of over-dosing. Enthusiastic patients may push tensioners prematurely; the single-variable rule and after-effect checks protect safety (33,39–41,51–54,74–76).

-Language risks. Avoid implying “trapped nerves.” Prefer “glide and information quality,” consistent with PNE (25,26,39,43,60–63).

4.5.20 Conclusion: a shared grammar for modern CLBP care

The biopsychosocial model tells us what domains matter; central sensitization and predictive processing explain how perception and behavior lock in; fear-avoidance/graded exposure clarifies how to restore function despite threat; motor control and strength training give us how to stabilize gains (5,6,23,25,26,43,57,60–63,68–70,77). The neurodynamic model plugs into this architecture by specifying a common, testable peripheral contributor—restricted neural mobility with mechanosensitivity—and by offering precise, low-threat interventions (sliders → tensioners) that generate the kind of sensory evidence the CNS can trust, consistent with emerging clinical evidence for neural mobilization in LBP.[51–54]

Clinically, integration means sequencing the fewest effective levers: begin with education that reframes pain, apply sliders that improve quality at fixed angles, insist on breathing continuity to down-shift gain, and spend the win immediately in function. Progress with time-in-range before angle, add tensioners judiciously, and consolidate with strength and graded exposure tailored to values (25,26,33,39–43,51–54,60–63,68–70,71–73,77). Measure change along mediator pathways so your model stays falsifiable. When progress stalls, re-phenotype—sometimes the driver is central tone or life load more than neural glide (5,6,23,25,44–46,48,49,57,78,79).

In this integrated view, no single model owns CLBP. Instead, each provides complementary coordinates on the same map. Neurodynamics adds one crucial axis—peripheral sliding capacity and signal quality—that clinicians can train safely and patients can feel rapidly (1–3,11,29,33,36,39–41,44,45,51–54). Addressing it alongside psychosocial and biomechanical levers is more than additive; it is synergistic, because better peripheral signals make central learning easier, and calmer central states make peripheral exposures cheaper. That synergy is the practical promise of integration—and a plausible route to more reliable, patient-centered outcomes in chronic low back pain (5,6,23,25,26,39–43,51–54,57,60–63,68–70,71–73,77).

4.6 Practical Considerations for Clinicians

Implementing neurodynamic interventions in chronic low back pain (CLBP) is both an art and a science. The science gives us constructs (excursion vs mechanosensitivity), tests (SLR, Slump, PKB), and dose rules (sliders → time-in-range → angle). The art is deciding when, how much, and what to combine for this person, in this context, today. This section translates the model into day-to-day practice. It expands the bullet points you provided into a complete playbook covering: clinical judgment, dosing and progressions, monitoring and flare management, integration with other therapies, patient education and scripts, documentation standards, and service delivery logistics (time constraints, home adherence, and ethical communication). (1–3,7,9,11–13,18,19,22,23,25,26,29,30,33,36,39,41–43,45,47–49,50,51,55–57,60–63,69–71,74–77,80–83,84–87)

4.6.1 Core principles: how to think before you treat

1.Glide before load. Start with low-intensity sliders to reduce mechanosensitivity and improve excursion. Delay tensioners and end-range holds until quality improves at a fixed angle and after-effects are mild. This respects the tissue's viscoelastic behavior and the nervous system's preference for low-threat inputs (39,51,53,73–76).

2.Quality precedes range. Your first success metric is not "more degrees," it's a change in quality at a fixed test angle (e.g., SLR at 55° feels neutral/stretch rather than "zingy"). When quality improves reliably, then chase degrees (33,39,44,45,51,53).

3.Dose one variable at a time. To protect learning (and attribution), change only one parameter per step: time-in-range, angle, repetitions, tempo, context, or complexity. Consolidate wins over 48–72 hours before progressing (1–3,11,19,29,33,39,51,53,73–76,97,98).

4.Treat the mechanism—but also the person. Mechanism-aligned dosing is foundational, but the plan must match irritability, beliefs, stress/sleep, work demands, and goals (biopsychosocial alignment). Combine neurodynamic techniques with strengthening, flexibility, and psychosocial care for best outcomes (4–6,22,24,26,43,55–57,60–63,64–66,69–71,77,80–83).

5.Make improvements matter immediately. Always "spend the win": embed a short functional bout (e.g., a 60–90 s gait drill with trailing-hip focus) right after sliders so the nervous system uses the safer range while it's available. This accelerates consolidation (22,24,39–41,50,58–60,69–71,80–86).

6.Narrate safety. Pacing, breathing, and analogies (e.g., "nerves are cables that need to slide freely") reduce fear-avoidance and increase adherence (25,26,60–62,64–66,95–97,100).

4.6.2 Starting low—how low is "low" (and how to progress)

Preferred entry technique: a slider that biases the implicated pathway (e.g., supine SLR slider for sciatic; prone knee bend variant for femoral; superficial crest glides plus short-arc trunk side-glide for cluneal) (1–3,7,11,19,29,33,36,39,41,42,45,73–76,88–92).

Initial dose (typical):

-Sliders: 2–3 sets × 8–12 cycles; slow tempo (2–3 s per half-cycle); 1–2 times/day (1–3,11,19,29,33,39,51,53,73–76).

-Time-in-range: start with zero holds (pure sliding). Add micro-holds (3–5 s) only after quality improves at fixed angle.

-Patient-reported discomfort ceiling: ≤3/10 during and immediately after a set; symptoms should settle to pre-set baseline within minutes (33,39,44,51,53,72,73–76,97,98).

Progression sequence:

1.Increase time-in-range at the same angle (e.g., micro-holds from 3–5 s → 5–8 s).

2.Then increase angle by a few degrees, keeping quality neutral.

3.Then increase repetitions modestly (e.g., 8–10 → 10–12).

4.Then consider a second context (e.g., add seated Slump sliders to the supine SLR set).

5.Finally, add complexity (e.g., multi-segment sequencing, light functional loading).(1–3,11,19,29,33,36,39,44,45,51–53,69–71,73–76)

When to introduce tensioners:

-Clear quality shift at a fixed angle, with after-effects ≤24 hours, and no breath-holds or guarding on observation.

-Start with 5–8 s holds at mid-range (never at hard end-range), bracketed by sliders.

-Keep total exposures 2–3 per set, 1–2 sets. Grow time before angle (2,11,29,33,39,42,51,53,72–76).

Do not progress two variables simultaneously (e.g., time-in-range and angle in the same session) (29,33,39,51,53,73–76).

4.6.3 Monitoring response in real time

Three domains to monitor during every session:

1.Symptom intensity (NRS) and quality (neural pull, sting, burn vs neutral/stretch). Target ≤3/10 during/after, and quality improvement at fixed angle within the session or across 48–72 hours (33,39,44,51,53,72–76,97,98).

2.Motor behavior: breath-holds, bracing, grimacing, or movement “stiffness.” These are proxies for gamma gain and perceived threat. If they appear, pause, reset with breathing, and reduce dose (22,23,24,50,58–62,69–71,80–83,84–87,95–97,100).

3.After-effects: next-day report. Acceptable: mild stiffness that resolves within 24 hours. Unacceptable: flare >24–48 hours, sleep disruption, or new neuro symptoms.

Use a single-variable rollback if unacceptable (see flare protocol) (33,39,44,51,53,72–76,97,98).

Practical in-session checklist (60–90 seconds):

-“How does this feel (0–10)?”

-“What kind of feeling is it (stretch vs nerve-like)?”

-“Is your breathing easy?” (watch, don’t only ask)

-“Let’s check the same test angle again—does the quality feel different?”

If quality is unchanged and symptoms climb, stop progression and switch to adjuncts (manual interface work, breathing, short-arc sliders) (1–3,11,18,19,22–24,27,29,30,33,38,39,41–43,45,51–53,60–63,69–71,72–76).

4.6.4 The flare management algorithm (viscosity reset)

Flares happen—especially when tensioners are added too quickly or when patients “chase angle.” A crisp protocol prevents derailment:

Step 1: Single-variable rollback (48 hours).

-Reduce one of the following by ~30–40%: angle, time-in-range, or repetitions. Keep the rest unchanged.

-Maintain breathing and education.

Step 2: Viscosity reset (24 hours).

-Add hourly micro-sliders (45–60 s) while awake to keep interfaces gliding with minimal load.

-Use paced breathing (6–8 breaths/min) before each micro-bout.

Step 3: Language and expectations.

-“This is feedback, not failure. We overshot dose. We’ve dialed it back and kept the good parts (breathing, gentle gliding).”

If a flare recurs despite rollback, consider (a) tensioner deferral, (b) psychosocial drivers (stress/sleep), (c) alternate phenotype (hip/SI contributions), or (d) lower-frequency but higher-quality dosing (1–3,11,19,23,25,29,33,39,41–43,51–53,60–63,69–71,72–76,97,98).

4.6.5 Safety and contraindications

-High irritability (severe mechanosensitivity, allodynia): sliders only; tiny arcs; short bouts; frequent rests; no tensioners.

-Acute radicular pain or evolving neuro deficit: avoid tensioners; coordinate with the patient's medical team.

-Red flags (new weakness, saddle anesthesia, constitutional symptoms): stop and refer.

-Osteoporosis/anticoagulation: avoid high-force techniques and end-range spinal loading; use superficial or low-load methods.

-Cluneal tunnels: never compress; superficial glides only; pad belts/waistbands.

Always document the safety screening at evaluation and at key progression points (4–6,21,22,23,47–49,55–57,60–63,77,78,88–92,97).

4.6.6 Integrating neurodynamics with strengthening and flexibility

Why integrate? Neural glide provides options; strength and mobility stabilize those options in daily life. Integration reduces relapse risk and improves performance (22,24,39–43,50,58–60,69–71,77,80–87).

Strength (low-threshold → moderate):

-Start with breath-continuous, low-threshold drills (short planks, anti-rotation holds, dead-bug/bird-dog).

-Emphasize external focus cues: "push the floor," "touch the wall with your hips," which reduce self-monitoring and bracing.

-Load slowly, only after neurodynamic quality and after-effects are stable across sessions (22,24,50,58–60,69–71,80–83,84–87).

Flexibility:

-Early phase: avoid long passive holds at provocative end-ranges (may increase interface friction). Prefer active mobility in mid-ranges and short, frequent exposures (1–3,11,19,29,33,39,51,53,73–76).

-Mid-late phase: add longer holds after glide improves and only within non-provocative quality. For hamstrings, blend hip-hinge patterns to bias joint over nerve (1–3,11,19,22,24,29,33,39,41–43,50,69–71,80–87).

Session choreography:

-Priming → sliders → (if indicated) tensioners → immediate functional "spend" (e.g., gait, sit-to-stand) → low-threshold strength/mobility → re-test quality at fixed angle.

-Keep the end-of-session re-test sacred. If quality worsens, re-think the mix (1–3,11,19,22,24,29,33,39,41–43,50,51–53,69–71,73–76,97,98).

4.6.7 Psychosocial integration: education, pacing, and graded exposure

Education (micro-dosed, everyday language):

-Analogy: "Nerves are like cables in flexible sleeves—they work best when they can slide freely."

-Re-frame pain: "Pain is an alarm tuned by recent experiences; gentle motion gives the alarm good data."

-Expectation: "We'll change one thing at a time to let your system learn calmly." (25,26,60–62,78,79,95–97,100)

Pacing:

-Replace "do as much as you can" with "do exactly this dose, twice a day."

-Protect consolidation windows (48–72 h) after each progression (26,55–57,60–63,64–66,69–71,77,97,98,100).

Graded exposure:

-Use neurodynamic tests to identify feared angles and convert them into safe exposures (sliders → time-in-range → angle).

-Celebrate quality shifts as prediction-error wins; immediately spend them in a functional task tied to values (walking the dog, playing with kids) (25,26,60–62,64–66,69–71,78,79,95–97,100).

4.6.8 Communication scripts you can use tomorrow

Introducing sliders (first visit):
“Your tests suggest the nerve’s sliding is a bit limited in this range. Rather than stretching hard, we’ll use small, smooth movements that help it glide. You should feel at most 2–3/10 and no zing. If it feels sharper, we stop and make it easier. After we do a set, we’ll try a quick walk or a sit-to-stand so your body uses that easier range right away.” (33,39,42,51–53,72–76)

Explaining progressions:
“When this angle starts to feel normal at the same setting, we’ll add a few seconds in that zone—then later, a few degrees. We always change one thing at a time, so your body knows exactly what’s happening.” (1–3,11,19,29,33,39,51,53,73–76,97,98)

Handling a flare:
“We turned the dial a bit too far. We’ll roll back one variable and do tiny glides more often just for a day—think of it like unsticking a zipper gently. Most people settle within 24–48 hours.” (29,33,39,51,53,72–76,97,98)

Linking to strengthening:
“Gliding opens the door; strength keeps it propped open so daily life doesn’t close it again.” (22,24,39–43,50,58–60,69–71,80–87)

Addressing fear-avoidance:
“Your nervous system is trying to protect you by tightening. These gentle movements are a way to teach safety so the protection eases. We’ll only move forward when your breathing stays easy.” (25,26,60–62,64–66,78,79,95–97,100)

4.6.9 Practical templates (notes you can copy)

A) Initial evaluation—neurodynamic section

-SLR (R/L): angle at first symptom; quality; modulation with ankle/neck; after-effect at 5 min.

-Slump: symptom location; modulation with cervical/ankle; post-test feel.

-PKB: (if indicated) angle/quality; change with pelvic stabilization.

-Cluneal screen: crest tenderness Y/N; superficial glides tolerated Y/N.

-Irritability: high/mod/low; breath-holds Y/N.

-Provisional phenotype: sciatic mechanosensitivity vs excursion; cluneal superficial irritation.

-Safety: red flags screened; contraindications noted.(1–3,11,19,21,22,29,33,36,39–41,45,51–53,72–76,88–92,93–99)

B) Daily treatment note—flow

-Priming completed (breathing/imagery) Y/N.

-Sliders: position(s), sets × reps, quality, NRS, breath-holds Y/N.

-Tensioners: holds (s), bracketed Y/N, after-effect.

-Manual therapy: region, non-provocative, re-test effect.

-Strength/mobility: drills, cues, adherence.

-Functional spend: task, duration, cue.

-Re-test at fixed angle: quality, angle, NRS.

-Plan: progress / consolidate / rollback; home program specifics.(1–3,11,19,22,24,29,30,33,39–43,50,51–53,69–71,72–76,93–99)

C) Home program one-pager

-Breathe slow (1–2 min).

-Do gentle sliders (2–3 × 8–12 reps).

-If easy for 48–72 h, add 3–5 s holds.

-“Spend the win” (walk 60–90 s or sit-to-stand 3–5 reps).

-Stop if zing or breath-hold; roll back one notch.

-Goal: quality first, range later. (1–3,11,19,25,26,29,33,39,41–43,51–53,69–71,72–76,95–99)

4.6.10 Time management in busy clinics

20–30 minute sessions can still be mechanism-aligned:

-Minute 0–3: Priming (breathing + imagery) while you review the last note.

-Minute 3–10: Sliders (1–2 positions) with live coaching; quick manual facilitation (2–3 min) if needed; re-run a brief slider set.

-Minute 10–15: Movement re-education (hinge or pelvic tilts) + a brief balance or trunk repositioning drill.

-Minute 15–18: Functional “spend” (gait or sit-to-stand).

-Minute 18–20: Re-test quality at fixed angle; set precise home dose; document.

For longer sessions, add tensioners and strength work—but never skip the re-test (4–6,22,24,39–43,50,55–57,58–60,64–66,69–71,77,80–87,93–99).

4.6.11 Adherence levers: how to help patients actually do the work

-Habit stacking: attach sliders to existing routines (after brushing teeth; during coffee brew).

-Micro-scheduling: 8–12 minutes twice daily beats 25 minutes once.

-Visible tracking: checkboxes on a fridge card; pair with a simple reward schedule.

-Meaningful goals: tie “spend the win” to values (walk with partner; play with kids).

-Language: “polish, don’t provoke”; “change one thing”; “spend the win.”

If adherence dips, simplify: one slider position + one functional minute, twice daily. Success grows from doable (25,26,55–57,60–63,64–66,69–71,77,93–99,100).

4.6.12 Special populations and adaptations

A) High-irritability / widespread pain (central sensitization features).[48,49]

-Very short, frequent micro-sliders (30–60 s hourly) with strict breathing.

-No tensioners until after-effects ≤24 h and quality consistently improves.

-Emphasize education, sleep hygiene, graded activity; early wins are non-pain (smoother motion, fewer breath-holds) (25,60–63,78,79,93–99).

B) Older adults / osteoporosis risk.

-Avoid end-range spinal loading and ballistic moves; favor supported positions (reclined slump).

-Prioritize ankle and cervical differentiation to keep spinal loads low.

-Shorter sets; longer consolidation between progressions (4–6,21,22,29,33,36,39,41–43,47–49,55–57,60–63,69–71,77,88–92).

C) Athletes in-season.

-Keep frequency; reduce density (no back-to-back high loads).

-Insert gait drills and “spend the win” between slider sets during warm-ups.

-Use 24-hour markers to decide if tensioners can be added without compromising performance (22,24,39–43,50,58–60,69–71,80–87,93–99).

D) Occupation-driven constraints (e.g., drivers, desk-bound).

-Engineer micro-breaks (every 30–40 min): stand, two slider cycles, hinge once, walk 30–60 s.

-Pad waist/crest; vary belt positions for cluneal irritation.

-If breaks are limited, practice seated micro-sliders (tiny ankle/cervical moves) (4–6,21,22,39–41,55–57,60–63,69–71,77,88–92).

4.6.13 Decision points and when to pivot

-No change in quality at fixed angle after 2–3 visits (with good adherence):

-Verify dose choreography (was only one variable changed?).

-Check breathing and cueing (are they breath-holding?).

-Try manual interface facilitation → immediate re-test.

-Re-phenotype: consider joint/hip/SI drivers; increase psychosocial emphasis; adjust sleep/stress plan. (4–6,21–24,29,30,33,39–43,50,55–57,58–63,64–66,69–71,77,80–83,88–92,93–99)

Quality improves, range improves, but function doesn’t:

-Increase functional spends; add task-specific graded exposure; ensure carryover to daily roles (22,24,26,39–43,50,55–57,58–63,64–66,69–71,77,80–87,93–99,100).

Frequent flares at small doses:

-Reduce arc size; increase frequency; expand education; consider co-management for stress/sleep (23,25,29,33,39,41–43,51–53,60–63,69–71,72–76,78,79,93–99).

Persistent cluneal tenderness:

-Audit compressive inputs (belts, chair edges); emphasize superficial glides only; refine sitting ergonomics; add trunk side-glide micro-motions (21,22,39–41,88–92).

4.6.14 Ethical communication and expectations

-Avoid structural determinism ("trapped nerve," "out of place"). Prefer function-forward language: "We're improving slide and signal quality."

-Be transparent about uncertainty: "We're testing a plausible mechanism and watching how your markers respond."

-Set bounded optimism: celebrate small wins (quality shifts, fewer breath-holds) as evidence the system is learning.

-Document shared decisions, flare plan, and stop rules.

-This protects the therapeutic alliance and reduces nocebo (25,26,55–57,60–63,64–66,77,93–99,100).

4.6.15 Minimal clinician toolkit (equipment and skills)

-Inclinometer or phone goniometer app for SLR/PKB angles.

-Timer for time-in-range and breathing pacing.

-Sticky notes / handout with one-page home plan and flare protocol.

-Language toolkit (scripts above) practiced until fluent.

-Video capture (optional) for gait/hinge feedback—short clips, not analysis paralysis.

What matters most is consistency and clarity, not fancy tech (1–3,11,22,24,29,33,36,39–43,50,55–57,69–71,77,93–99).

4.6.16 Outcome tracking and quality assurance

Track a minimum viable dataset every 1–2 weeks:

-Neurodynamic markers: angle at first symptom and quality at fixed angle (SLR/Slump/PKB) (33,36,39,44,45,51–53,72–76,93–99).

-Motor behavior: breath-hold count across standardized tasks; time-to-relax after forward bend.

-Function: PSFS items; sitting tolerance; gait bout comfort; trailing-limb hip extension (estimate acceptable).

-Symptoms: NRS at rest and after provocation; sleep disruption yes/no.

-Adverse events: flares >48 h; new neuro signs.

Use a simple run chart to visualize trends. If quality improves first, range next, function last, you're on track with the expected sequence (1,22,24,33,39–41,44,45,51–53,58–60,69–71,80–87,93–99).

4.6.17 Putting it all together: two brief vignettes

Vignette 1—Mechanosensitivity-dominant sciatic pattern (moderate irritability).

-Start SLR sliders (2–3 × 8–10), seated Slump sliders (2 × 8–10).

-No holds for 3 sessions; breathing policing; brief gluteal interface work.

-Re-test shows quality shift at 55°.

-Add 5–8 s holds at mid-range, bracketed by sliders; continue for 1–2 weeks.

-Functional spend: gait with trailing-hip cue, 60–90 s.

-At week 3–4: range improves; add low-threshold strength.

-Flares addressed with rollback + viscosity reset.

-Result: symptoms ↓, function ↑, adherence high.(1–3,11,18,19,22,24,29,30,33,38,39,41–43,45,51–53,58–60,69–71,72–76,80–87,93–99)

Vignette 2—Excursion-limited femoral bias (low irritability).

-Start PKB sliders with pelvic stabilization; sidelying femoral slider; pelvic tilt control.

-After stable quality shift, add very short mid-range holds (5–8 s).

-Integrate hinge and split-stance drills; "exhale-to-move."

-Re-test PKB: angle increases from ~90° to 110° with neutral quality.

-Functional transfer: sit-to-stand sets inserted between slider sets.

-Outcome: upright tolerance improved; daily function easier.(1–3,11,19,22,24,29,33,36,39–43,50,51–53,58–60,69–71,72–76,80–87,93–99)

These vignettes illustrate dose logic, sequencing, and transfer without overcomplication (1–3,11,19,22,24,29,30,33,36,38–43,50,51–53,58–60,69–71,72–76,80–87,93–99).

4.6.18 Common pitfalls (and how to avoid them)

-Pitfall: Chasing degrees too early.
Fix: Protect quality at fixed angle as the primary KPI for the first 1–2 weeks (1–3,11,19,29,33,39,44,45,51–53,72–76,97,98).

-Pitfall: Adding multiple progressions at once.
Fix: One variable per step; consolidate 48–72 hours (29,33,39,51,53,72–76,97,98).

-Pitfall: Provoking cluneal tunnels with pressure.
Fix: Superficial glides only; pad belts; avoid chair-edge compression (21,22,39–41,88–92).

-Pitfall: Ignoring breath.
Fix: Hard rule: if breath-holds appear, stop and reset. Breathing continuity is non-negotiable (22,23,24,50,58–62,69–71,80–83,84–87,95–97,100).

-Pitfall: No functional linkage.
Fix: Insert a spend-the-win minute after every slider set (22,24,39–43,50,58–60,69–71,80–87).

-Pitfall: Vague home plans.
Fix: Specify exact sets/reps/time and when to progress; give the flare card (25,26,55–57,60–63,64–66,69–71,77,93–99,100).

4.6.19 The 10-sentence mini-handbook (for your pocket)

1.Start with sliders, not stretches (39,42,51,53,73–76).

2.Keep symptoms ≤3/10; stop if quality feels “zingy” or breath tightens (33,39,44,51–53,72–76,97,98).

3.Look for quality change at a fixed angle before chasing range (33,39,44,45,51–53,72–76).

4.Change one thing per step: time → angle → reps → context → complexity (1–3,11,19,29,33,39,51,53,72–76,97,98).

5.Bracket tensioners with sliders, and only after qualities settle (2,11,29,33,39,42,51,53,72–76).

6.Use breathing to downshift gamma and protect dosing (22,23,24,50,58–62,69–71,80–83,84–87,95–97,100).

7.Spend the win immediately in a tiny functional task (41) (22,24,39–43,50,58–60,69–71,80–87).

8.Combine with strength/mobility and education for durable gains (43,25) (22,24,39–43,50,55–57,58–60,64–66,69–71,77,80–87,93–99,100).

9.If a flare hits, rollback one variable and do micro-sliders hourly for a day (29,33,39,51–53,72–76,97,98).

10.Document angles, qualities, breath, and a clear home plan every visit (33,39,44,51–53,72–76,93–99).

4.6.20 Conclusion: practical, safe, and falsifiable

Neurodynamic care becomes practical when it is structured and testable. Starting with low-intensity sliders honors mechanosensitivity (39); monitoring patient response with a ≤3/10 tolerance and breathing continuity protects safety (33); integrating strengthening, flexibility, and psychosocial components respects the multidimensional nature of CLBP (43,25,41,4–6,22,24,55–57,60–63,69–71,77,80–87). The clinician's craft is to titrate time-in-range before angle, to change one variable at a time, and to ensure every gain is spent immediately in meaningful function. When progress stalls, the plan is falsifiable: adjust dose, reinforce breathing and education, or re-phenotype. When progress flows, document it: improved quality at fixed angles, calmer motor behavior, and better function—the durable hallmarks of a nervous system that now trusts movement again (1–3,11,19,22,24,29,30,33,36,38–43,50,51–53,58–60,69–71,72–76,80–87,93–99).
These practical considerations ensure that neurodynamic interventions are safe, effective, and truly tailored to the individual across settings—from busy outpatient clinics to high-performance sport and desk-bound workplaces—while remaining squarely aligned with the broader evidence-based frameworks that guide modern CLBP care (4–6,22,24,25,26,39–43,50,55–57,58–63,64–66,69–71,77,78,79,80–87,93–99,100).

5.Conclusion

Chronic low back pain (CLBP) remains a heterogeneous, multifactorial condition whose persistence is best understood as the emergent product of interacting biological, psychological, and social processes over time (4–6,55–57,60–63,77,78,93–100).

Within this broad landscape, the present paper advances a specific, testable proposition: subclinical neurodynamic restrictions—mild, often non-radicular impairments in peripheral nerve excursion coupled with mechanosensitivity—constitute an underrecognized yet clinically relevant contributor to symptoms and disability in a subset of patients (1–3,7,9,11–13,18,19,27–29,33,36,38–41,44,45,51–53,72–76). These restrictions need not present with classic neuropathic signs; rather, they can subtly distort afferent signaling, bias segmental reflexes toward co-contraction, and disrupt sensorimotor control, thereby maintaining pain and functional limitation even when imaging is unremarkable and routine strengthening or flexibility programs stall [4–6,22,24,39–41,44,45,50,58–63,69–71,80–87].

5.1 **Core contributions and synthesis**

Across the paper we have:

1.Specified the construct—defining subclinical neurodynamic restriction by its behavioral and test characteristics (e.g., early symptom onset during SLR or slump with neural quality, modulation by structural differentiation, and improvement in quality at a fixed angle after dosing), rather than by structural imaging alone (33,36,37,39,42,45,51–53,73–76).

2.Explained plausible mechanisms—how reduced neural excursion and heightened mechanosensitivity can increase dorsal horn gain, elevate gamma drive, and promote protective bracing, degrading proprioceptive fidelity and motor planning (15–18,22–24,27,28,39,41,50,58–63,69–71,80–87).

3.Proposed a falsifiable assessment strategy—pairing standard neurodynamic tests (SLR, slump, prone knee bend) with mechanism-aligned anchors (e.g., quality at fixed angle, time-to-soften after a standardized forward bend, structural differentiation effects), thereby enabling disciplined progression and real-time attribution (1–3,7,9,11–13,19,29,33,36,37,39,41–43,45,51–53,72–76).

4.Outlined an integrated intervention model—neurodynamic mobilizations (sliders → carefully dosed tensioners) supported by interface-friendly manual therapy, movement re-education (breathing continuity, hip hinge, elastic control), proprioceptive retraining, and immediate functional spend of gains in contextually relevant tasks (gait, sit-to-stand, lifting). This approach aligns with and augments contemporary rehabilitation frameworks [4–6,22,24,39–43,50,55–57,58–63,64–71,72–76,77,80–87].

5.Demonstrated clinical feasibility—through detailed case studies (athletic and sedentary phenotypes) showing predictable sequences of change (quality → range → tone/relaxation → proprioception → functional automaticity) and pragmatic algorithms for dosing, flare management, and progression (33,36,39–41,44,45,51–53,58–60,69–71,72–76,80–87).

6.Situated the model inside the biopsychosocial and central sensitization perspectives, showing how small improvements in peripheral signal quality can ease central gain and facilitate belief updating, graded exposure, and behavior change (5,6,23,25,26,43,47–49,55–57,60–63,78,79,92,97–100).

Taken together, these elements present a coherent, mechanistic, and clinically actionable account of how modest peripheral constraints can sustain CLBP—and, crucially, how they can be addressed with low-threat, scalable interventions (1–3,4–6,22–24,33,36,39–43,44,45,50–53,55–57,58–63,69–71,72–76,77,80–87,93–100).

5.2 **Why this matters now**

5.3 Clinicians are frequently caught between two unsatisfying poles: (a) a search for discrete structural lesions that often fails in non-specific CLBP; and (b) a purely psychosocial emphasis that, while essential, can feel abstract to patients who experience very concrete movement-linked symptoms (4–6,55–57,60–63,77,93–100). The neurodynamic perspective bridges this divide. It neither reduces pain to a single peripheral cause nor dismisses peripheral contributions in favor of central ones. Instead, it clarifies one specific, modifiable peripheral dimension—the capacity of nerves to slide and signal cleanly—that interacts reciprocally with central processes, beliefs, and behaviors (1–3,7,9,11–13,18,19,23,25–28,33,36,39–43,44,45,47–49,50–53,58–63,69–71,72–76,78,80–87). In practice, this yields clear entry points for care (sliders, breathing, one-variable progressions) and clear exit criteria (stable improvement in quality at fixed angles, durable functional transfer, calm after-effects) (29,33,39–41,51–53,69–71,72–76,93–99).

5.3 Clinical implications: a disciplined, patient-centered algorithm

A practical implication of this work is a succinct care pathway clinicians can apply across phenotypes:

1.Screen and phenotype. Exclude red flags. Use SLR, slump, and prone knee bend to probe for neural features. Record angle at first symptom and quality at a fixed angle. Note modulation with structural differentiation and observe breath-holds, guarding, and “time-to-soften” after standardized movements (33,36,37,39,41–43,45,51–53,72–76,93–99).

2.Decide if neurodynamics is implicated. If tests indicate mechanosensitivity and/or excursion loss—with immediate or short-term change after gentle dosing—plan to include neurodynamic care as a primary or strong adjunct (1–3,7,9,11–13,18,19,29,33,36,39–43,44,45,51–53,72–76).

3.Dose low and slow. Begin with sliders in the most relevant position(s), emphasizing smooth reciprocal motion, symptom ceiling ≤3/10, and breathing continuity. Use the rule: glide before load; time-in-range before angle; change one variable at a time (29,33,39,42,51–53,72–76,97,98).

4.Validate intra-session. Re-check quality at the same test angle. If quality softens with stable after-effects, consolidate for 48–72 hours. Introduce mid-range tensioners only when criteria are met, always bracketed by sliders (2,11,29,33,39–41,42,51–53,72–76).

5.Integrate synergists. Add interface-friendly manual therapy (non-provocative), movement re-education (hinge, pelvic control, breath), proprioceptive retraining, and immediate functional spend (brief gait or task bouts). Layer low-threshold strength and active mobility to consolidate new options (22,24,39–43,50,58–60,69–71,80–87).

6.Track the right markers. Expect the sequence: quality → range → tone/relaxation → proprioception → function. If the sequence stalls, adjust dose or re-phenotype; if it reverses, roll back one variable and run the viscosity reset (hourly micro-sliders for a day) (22,24,33,36,39–41,44,45,51–53,58–60,69–71,72–76,80–87,93–99).

7.Communicate ethically. Use simple, non-threatening language—"nerves like to glide," "we're improving slide and signal quality"—and frame progress as learning, not fixing a broken part (25,26,55–57,60–63,64–66,77,93–100).

This algorithm protects against over-treatment, improves attribution, and respects inter-individual variability—all while aligning with biopsychosocial best practices (4–6,25,26,39–43,55–57,60–63,64–66,69–71,77,93–100).

5.4 **Integration, not competition, with existing models**

The neurodynamic approach complements the biopsychosocial model by giving clinicians a specific biological lever to pull within a broader plan (4–6,55–57,60–63,77,93–100). It pairs naturally with pain neuroscience education, graded exposure, and values-based rehabilitation: sliders provide safe sensory evidence right at previously "dangerous" ranges, breathing down-regulates autonomic arousal, and functional spends help reconsolidate learning into daily habits (23,25,26,39–43,55–57,60–63,64–66,69–71,77,80–87,93–100). In central sensitization contexts, micro-dosed sliders operate as low-threat exposures that reduce input variability and facilitate belief updating (23,47–49,60–63,78,79,92,97–100). In biomechanical programs, joint and soft-tissue techniques become interface facilitators—ways to make sliders cheaper and more effective—and strength work functions as long-term consolidation (22,24,39–43,50,58–60,69–71,80–87). Thus, neurodynamics acts less like a new silo and more like a plug-in module that clarifies sequencing and dosing across care paradigms (1–3,4–6,23–26,33,36,39–43,44,45,51–53,55–57,60–63,69–71,72–76,77,78,80–87,93–100).

5.5 **Methodological and research agenda**

The paper identifies concrete directions to refine science and practice:

-Prevalence and impact. Establish how common subclinical restrictions are in different CLBP phenotypes and what proportion of variance in pain and disability they explain (3,39,41,44,45,51–53,60–63,69–71,72–76). Emerging tools—ultrasound elastography, dynamic MRI, and motion-capture proxies—could quantify excursion and friction at key interfaces (e.g., sciatic corridor during SLR), supporting mechanism-based stratification (39,41,44–46,51–53,72–76).

-Measurement validity and reliability. Standardize neurodynamic test protocols and train clinicians to capture quality at fixed angles, structural differentiation responses, and after-effects with acceptable reliability. Develop normative excursion data by age and activity level to sharpen diagnostic accuracy (1–3,7,11–13,19,29,33,36,37,39,41–43,45,46,51–53,69–71,72–76).

-Intervention efficacy and mechanisms. Randomized trials should compare education + generalized exercise versus education + generalized exercise + neurodynamic dosing with equal contact time, specifying mediators a priori (e.g., early change in quality at fixed angle; breath-hold frequency; time-to-soften). This strengthens causal inference beyond symptom change alone (3,11,18,19,33,38,39,41–43,51–53,60–63,69–71,72–76,93–99).

-Central correlates. Neuroimaging (e.g., fMRI) can test whether improved neural slide corresponds to altered somatosensory and salience network activity, clarifying peripheral–central coupling and the conditions under which peripheral improvements drive central re-weighting (23,25,47–49,60–63,78,79,80–83,92,97–100).

-Natural history. Longitudinal cohorts can reveal whether early neurodynamic markers predict chronicity, flare risk, or response to particular treatment bundles, enabling prognostic enrichment and smarter triage (4–6,39–41,44,45,51–53,55–57,60–63,69–71,72–76,77,93–100).

Collectively, this agenda moves the field from conceptual plausibility to quantitative precision, helping clinicians select the right patients and doses at the right time (3,4–6,22–24,33,36,39–43,44–46,50–53,55–57,60–63,69–71,72–76,77,80–87,93–100).

5.6 **Strengths, boundaries, and limitations**

A strength of this model is that it is clinically light-weight: tests require minimal equipment; interventions are scalable to home programs; and progress is captured with simple, repeatable markers (angles and qualities at fixed positions, breath behavior, short functional metrics) (1–3,11,19,22,24,29,33,36,39–43,50,51–53,58–60,69–71,72–76,80–87,93–99). It is also falsifiable—if sliders do not shift quality at fixed angles, if after-effects remain volatile despite careful dosing, or if function fails to improve once markers move, the algorithm demands recalibration (dose change, alternate phenotype, stronger psychosocial or sleep emphasis) (4–6,22–24,25,26,33,36,39–43,44,45,51–53,55–57,60–63,69–71,72–76,77,78,80–87,93–100). Limitations deserve equal emphasis. Neurodynamic tests are multitissue; while structural differentiation and intra-session change improve inference, they do not

confer perfect specificity (1–3,11,19,29,33,36,39,41–43,45,51–53,72–76). Some patients' dominant driver will be psychosocial load, sleep debt, or non-neural tissue pathology; in such cases neurodynamic care should remain adjunctive or be deemphasized if markers fail to budge (4–6,23–26,55–57,60–63,69–71,77,78,80–87,93–100). Finally, current evidence suggests benefit but still calls for higher-quality trials with mechanistic mediators—precisely the studies outlined above (3,39,41–43,45,51–53,60–63,69–71,72–76,74–76).

5.7 **A pragmatic message for clinicians and patients**

For clinicians: Start where the system will learn the most with the least threat. That often means small, smooth sliders delivered with calm breathing, tested against quality at a fixed angle, and followed by a brief functional spend. Change one variable at a time, and let your markers—not your preferences—tell you when to progress, consolidate, or pivot (29,33,39,41–43,51–53,58–60,69–71,72–76,80–87,93–99).
For patients: Your nerves are living tissues that like to glide. When they glide better, your movement feels easier and your body stops bracing so hard. We'll build that ability gently, show your system that these movements are safe, and then immediately use the improvement in things you care about—walking, lifting, getting through the workday. Each small success teaches your nervous system to trust movement again (23,25,26,39–43,55–57,60–63,64–66,69–71,77,80–87,93–100).

5.8 **Closing perspective**

CLBP will continue to challenge medicine precisely because it is not one thing (4–6,55–57,60–63,77,93–100). The promise of the neurodynamic perspective is modest and powerful at once: it offers a clear, mechanistically grounded way to address one frequently overlooked piece of the puzzle—the mechanical and informational behavior of peripheral nerves—and to do so in a manner that cooperates with, rather than competes against, biopsychosocial care (1–3,4–6,23–26,33,36,39–43,44,45,47–49,50–53,55–57,60–63,69–71,72–76,77,78,80–87,93–100). When neural slide improves, afferent noise falls; when afferent noise falls, protective tone loosens; when tone loosens, proprioceptive maps sharpen; when maps sharpen, movement regains its elastic quality—and clinical change becomes more durable (15–18,22–24,27,28,39,41,50,58–63,69–71,80–87).
This is not a rejection of central models or a return to structural reductionism; it is a synthesis that respects complexity while preserving clinical decisiveness. The nervous system is both signal and structure. Treating it as such opens practical avenues for assessment, for patient-centered dosing, and for research that links peripheral markers to central adaptation and functional life change (23,25,47–49,60–63,69–71,72–76,78,80–87,92,93–100). If pursued with methodological rigor and therapeutic humility, this line of work can meaningfully improve outcomes for patients whose symptoms have outlasted simpler explanations—and, in doing so, help move the field of CLBP management toward care that is at once mechanism-aligned, multidimensional, and genuinely hopeful (4–6,22–26,39–43,50–53,55–57,58–63,64–66,69–71,72–76,77,78,80–87,93–100).

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