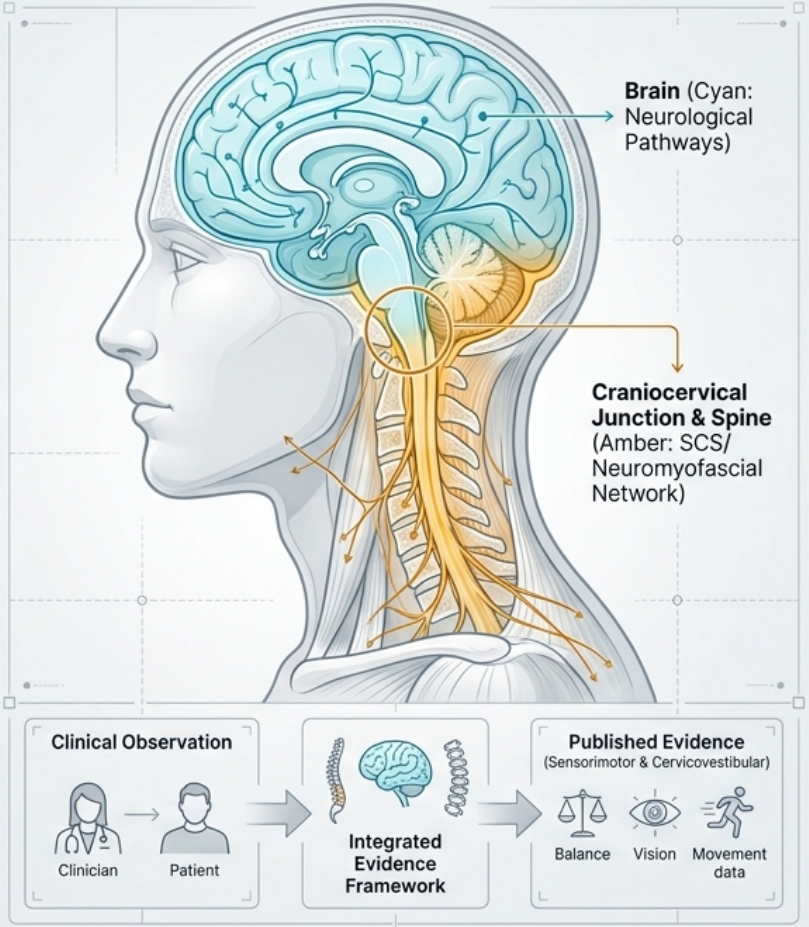
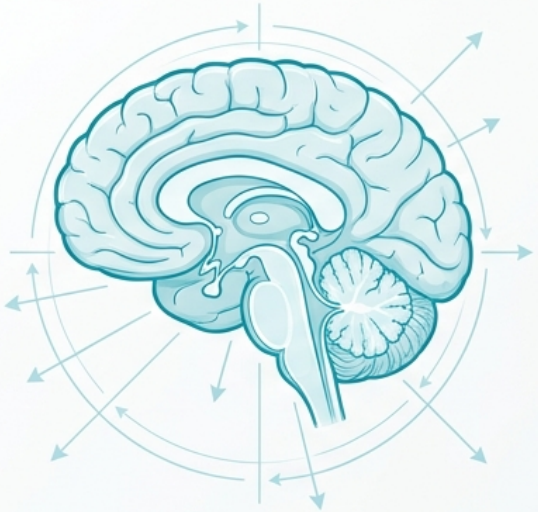
Beyond the Brain: **The Neuromyofascial Science Perspective on Persistent Concussion Symptoms**

Are persistent concussion-like symptoms always brain-based? Exploring the Whole-Injury framework and Spinal Concussion Syndrome (SCS).

Integrating Clinical Observation with Published Sensorimotor and Cervicovestibular Evidence



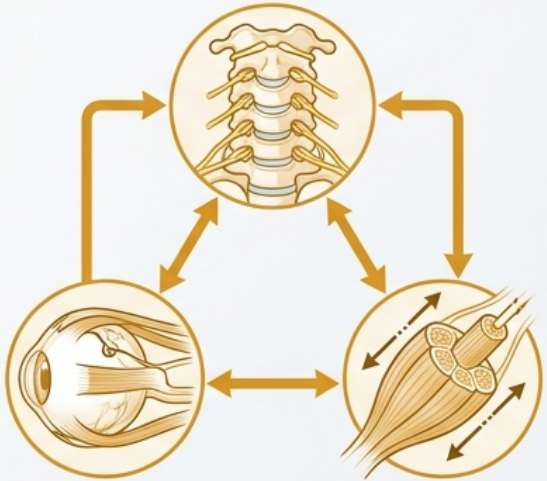
The Central Paradox in Concussion Rehabilitation



Diagnostic Label: mTBI

- Classified strictly as mild Traumatic Brain Injury
- Treated conceptually as a neurometabolic cascade

If PCS is purely a brain disorder, why does clinical recovery so often depend on treating the neck, spine, and vestibular systems?



Rehabilitation Reality

- Rehabilitation heavily targets:
  - Cervical spine & posture
  - Vestibulo-ocular integration
  - Musculoskeletal restriction

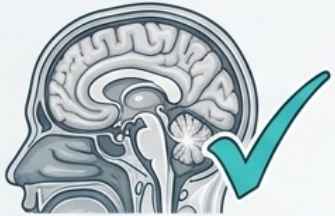
Expanding the Clinical Lens: PCS versus SCS

Post-Concussion Syndrome (PCS)	Feature	Spinal Concussion Syndrome (SCS)
Brain tissue (mTBI focus)	Primary Tissue Target	Cervical/Thoracic spine, fascial, neural pathways
Often standard normal MRI	Imaging Profile	Grossly normal structural MRI; functional soft-tissue pathology missed
Neurometabolic cascade	Primary Symptom Drivers	Myofascial contracture, nerve root/cord tension, proprioceptive mismatch
Migraine, Anxiety, PTSD	Diagnostic Overlap	Whiplash, Fibromyalgia, Cervicogenic conditions

The Core Premise: A patient may have brain injury alone, SCS alone, or a simultaneous mixed presentation

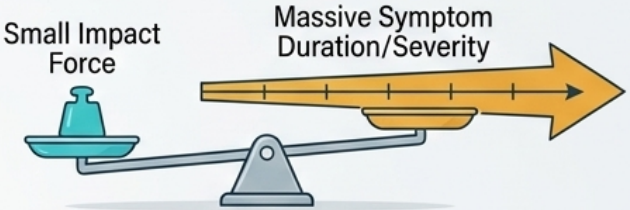
Clinical Gaps in the Brain-Only Diagnostic Model

1. Normal Brain Imaging



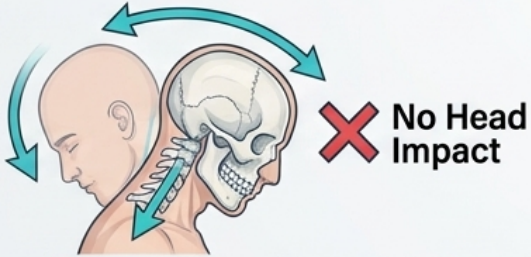
Standard MRIs frequently appear normal and fail to explain severe, persistent somatic symptoms.

2. Severity Mismatch



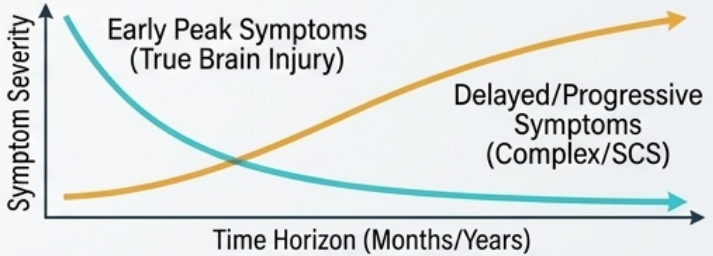
Head impact force does not reliably correlate with symptom chronicity or severity.

3. Absence of Head Strike



Severe PCS presentations regularly emerge from pure acceleration-deceleration events.

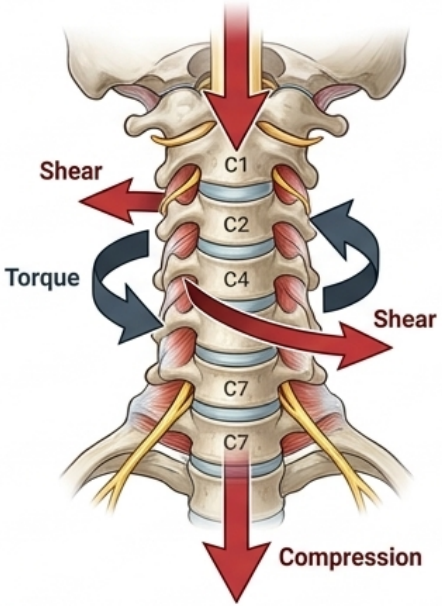
4. Symptom Chronology



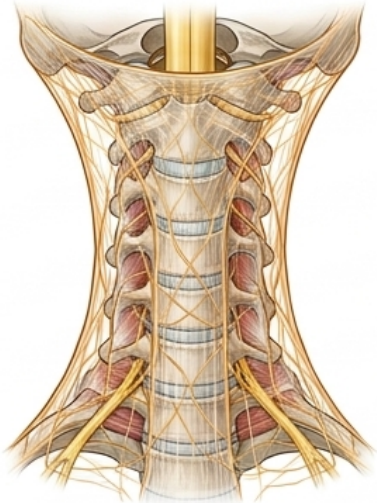
True brain injury symptoms typically peak early; complex symptoms often worsen months later.

The Evolutionary Injury Response

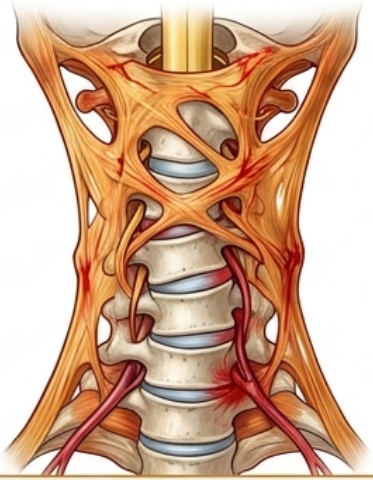
Stage 1: Acute Trauma
Tissues subjected to extreme shear, torque, and compression forces.



Stage 2: Biological Stabilization
The body initiates an internal cast mechanism, forming dense neuromyofascial tissue.



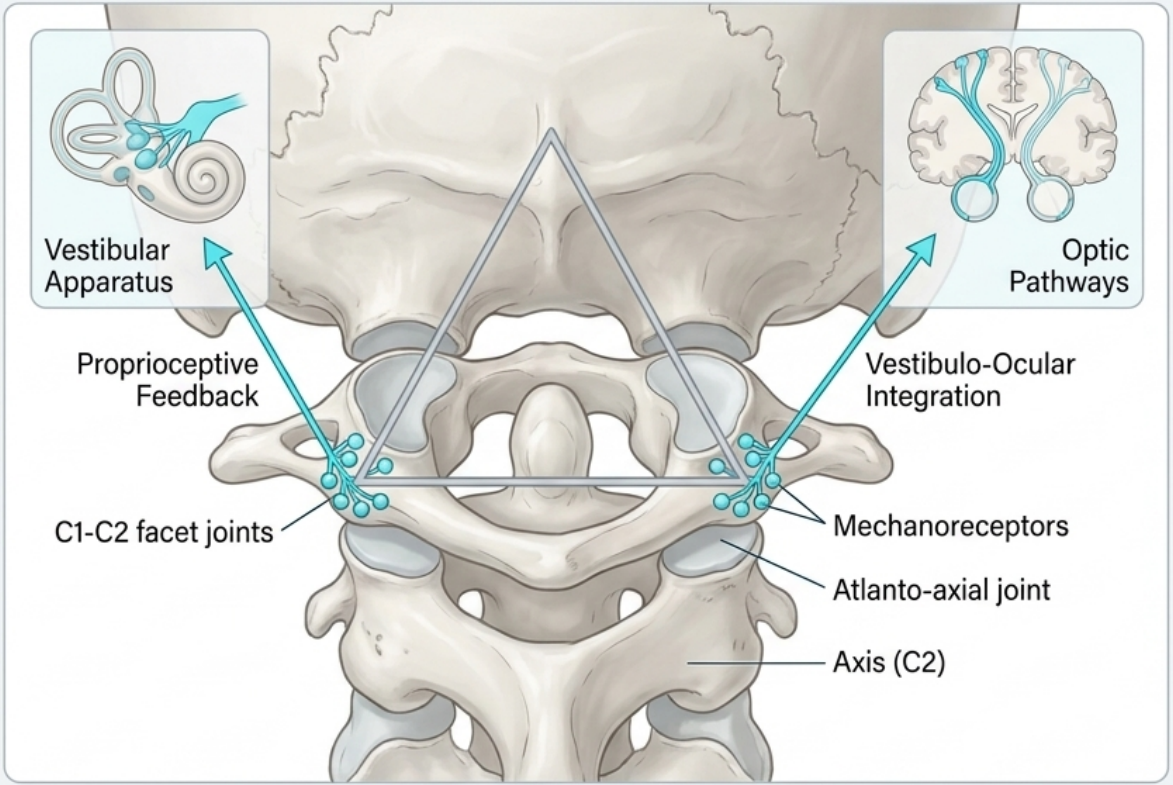
Stage 3: Pathological Progression (SCS)
Accumulation of dense, scar-like tissue results in:
- Loss of normal tissue glide
- Segmental spinal restriction
- Dynamic nerve entrapment



Outcome: Progressive pain and delayed neurological symptoms as tissue compliance decreases.

The Cranial-Cervical Junction: C1-C2 Sensorimotor Disruption

Anatomical Focus: Atlas (C1) and Axis (C2)

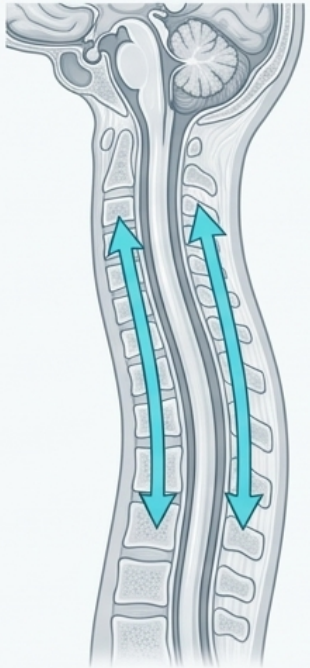


Key SCS Overlaps:

- Head position and precise neck rotation mechanics
- Cranial-facial headache referral pathways
- Proprioceptive feedback loops and spatial orientation
- Vestibulo-ocular mismatch (driving brain fog and light sensitivity)

Clinical Observation: Rapid visual clarity and symptom shifting are frequently reported during targeted upper-cervical physical treatment.

The Spinal Cord Tethering Hypothesis

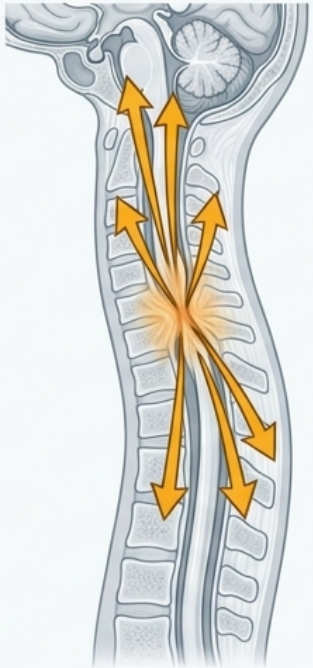


Normal Function

The spinal cord glides freely within the canal during flexion, extension, and rotation.

Upward Traction Effects

Tension toward the brainstem, cranial nerves, and optic pathways.



Tethering Dysfunction

Scar-like neuromyofascial contractures create focal restriction points, preventing normal glide.

Upward Traction Effects

Tension toward the brainstem, cranial nerves, and optic pathways.

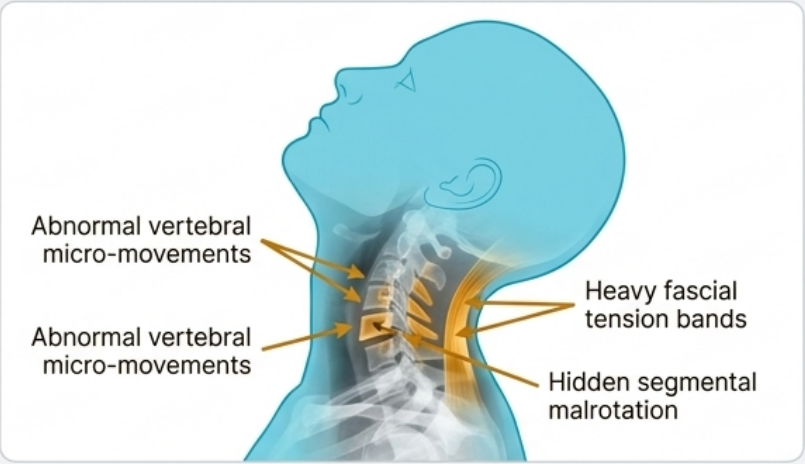
Downward Traction Effects

Manifests as leg heaviness, balance issues, or autonomic disruption.

Note: While supported by clinical treatment responses, systemic tethering mechanisms remain a theoretical component of the NMFS framework.

Hypermobility and the Diagnostic Audit

The Range of Motion Fallacy



Normal or high Range of Motion (ROM) does not equate to spinal health in hypermobile patients.

The Vulnerability: Hypermobile spines experience exaggerated excursion during trauma, leading to extensive, pathological internal casting.

The Historical Audit Protocol:

- [ ] 1. Trace precise **trauma kinematics** (acceleration forces).
- [ ] 2. Map the exact timeline of **delayed symptom onset**.
- [ ] 3. Identify **psychiatric symptoms** emerging secondary to chronic pain.

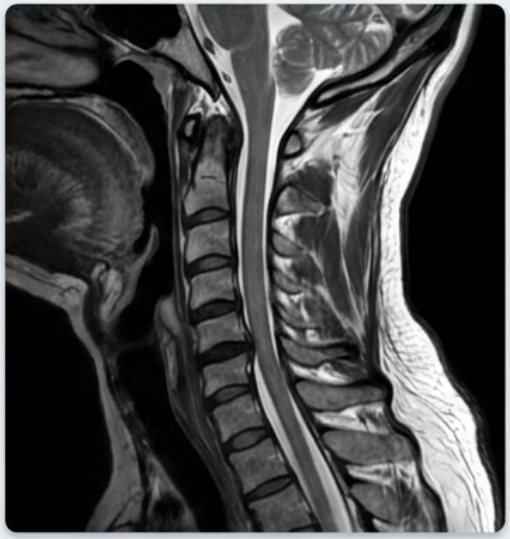
Targeted Physical Exam:

- Focus on **fascial restriction**, tissue density changes, and **dynamic segmental dysfunction** rather than just global ROM.

The Diagnostic Imaging Gap

Static Structural View

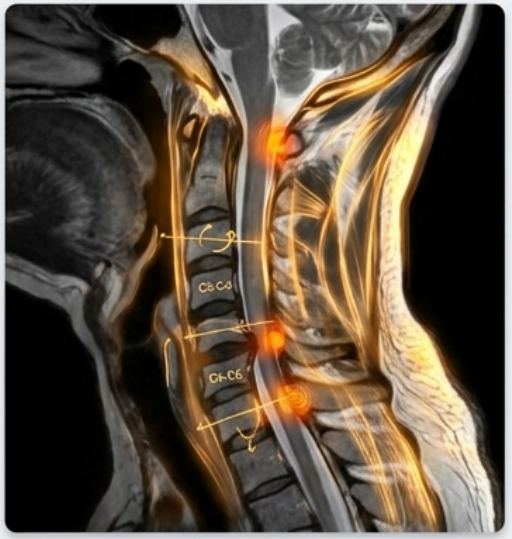
What Standard Imaging Detects:



- Excellent for ruling out fractures, severe disc herniations, and gross structural abnormalities.

Dynamic Soft-Tissue View

What Standard Imaging Misses (SCS Pathology):



- Fascial contracture and abnormal tissue density
- Subtle vertebral malrotation
- Dynamic instability under physiological load
- Soft-tissue nerve root entrapment

The Future Diagnostic Requirement:

Advancements in fat-water indexing MRI, upright/gravity-loaded MRI, Dynamic Motion X-ray (DMX), and molecular markers for scar pathology.

Published Evidence: Cervicovestibular Integration

Evaluating the Core Six Medical Studies

Cervical Dysfunction Prevalence

Published studies suggest a high incidence of cervical impairments documented in prolonged concussion-like presentations.

Upper-Cervical Focus

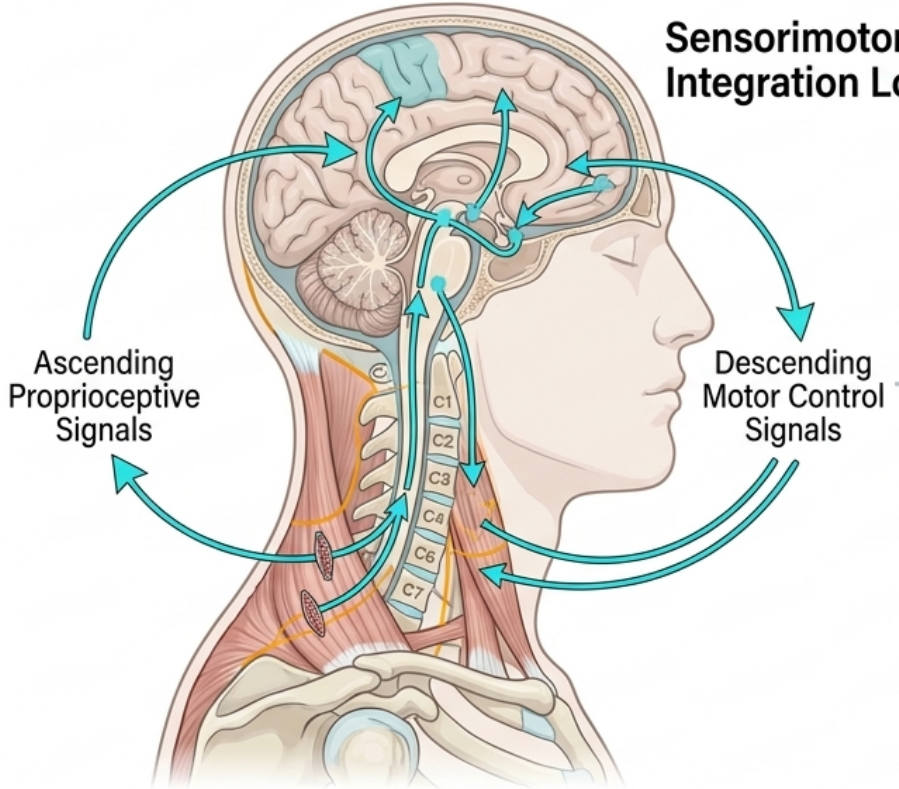
Literature strongly supports the upper cervical spine's role in perpetuating persistent dizziness, cervicogenic headache, and visual motion sensitivity.

Cervicovestibular Rehabilitation

Targeted spinal and vestibular therapies demonstrate distinct, measurable efficacy over rest or brain-only cognitive therapies.

Published Evidence: Sensorimotor and Myofascial Deficits

Evaluating the Core Six Medical Studies



Proprioceptive Disturbance

Objective measurements confirm altered cervical joint-position error and sensorimotor integration issues in persistent PCS populations.

Myofascial Impairment

Documented presence of myofascial trigger points, tissue density changes, and restricted mobility driving head/neck pain.

Symptom Generation

The literature validates that the cervical spine and soft tissues are capable of generating the exact symptom cluster (brain fog, dizziness, fatigue) traditionally attributed to mTBI.

Delineating Scientific Evidence from Framework Theory

Published Evidence (Component Mechanisms)

What the Literature Strongly Supports:

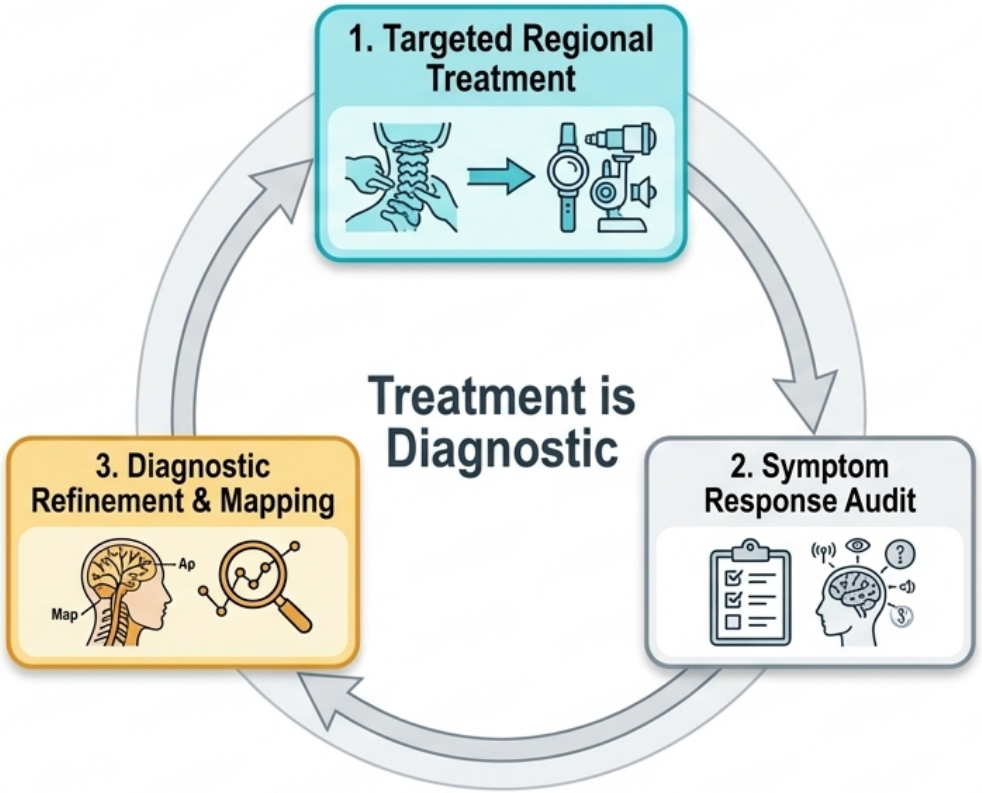
- Cervical spine dysfunction is highly prevalent in persistent PCS.
- Targeted cervicovestibular therapy improves patient outcomes.
- Sensorimotor and proprioceptive mismatches are active symptom drivers.

NMFS Framework (Theoretical Integration)

What Remains Theoretical (Framework Interpretation):

- The complete Evolutionary Injury Response cascading model.
- Spinal cord tethering as the primary mechanism for distal retinal/visual changes.
- A singular, unified Neuromyofascial Science (NMFS) etiology for all chronic pain and PCS overlap.

Strategic, Staged, and Diagnostic Care



The Piecemeal Approach:

- **Example:** If headache clears after C1-C2 treatment, it had a cervical driver.
- **Example:** If anxiety improves with cranial-facial release, nociceptive signaling was a contributor.

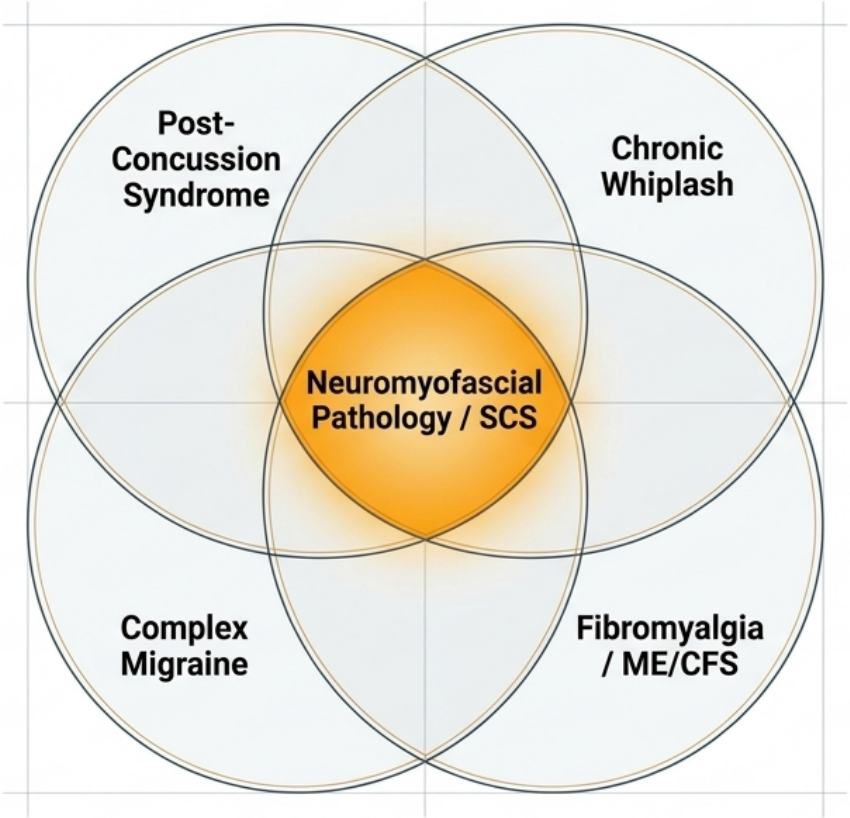
Multidisciplinary Modalities:

- Incorporates targeted neuromyofascial procedures, neuro-optometry, proprioceptive retraining, and osteopathy.

Clinical Objective:

Map the injury patient-by-patient; avoid single-variable panaceas.

Broader Implications: Overlapping Syndromes



The Psychiatric Overlap

Anxiety and PTSD-like symptoms may act as neurological side effects of unresolved **neuropathic pain** and autonomic disruption, rather than pure psychological trauma.

Public Health Mandate

Broadening the diagnostic lens to include soft-tissue structural injuries can reduce years of misdirected care and unnecessary disability.

Summary: Embracing the Whole-Injury Model

The Core Paradigm Shift: Moving from a Brain-Only Model to an integrated Whole-Injury Model.

Key Takeaways

1. **Persistent PCS-like symptoms** do not always equal persistent brain injury.
2. **Cervical, upper-thoracic, and neuromyofascial** injuries are proven active symptom drivers.
3. **Structural diagnostics** must advance beyond standard static MRI to capture dynamic tissue pathology.
4. **Individualized, staged treatment** is necessary to map and resolve specific injury sites.

Are persistent concussion-like symptoms always brain-based?
No. The spine requires equal diagnostic rigor.