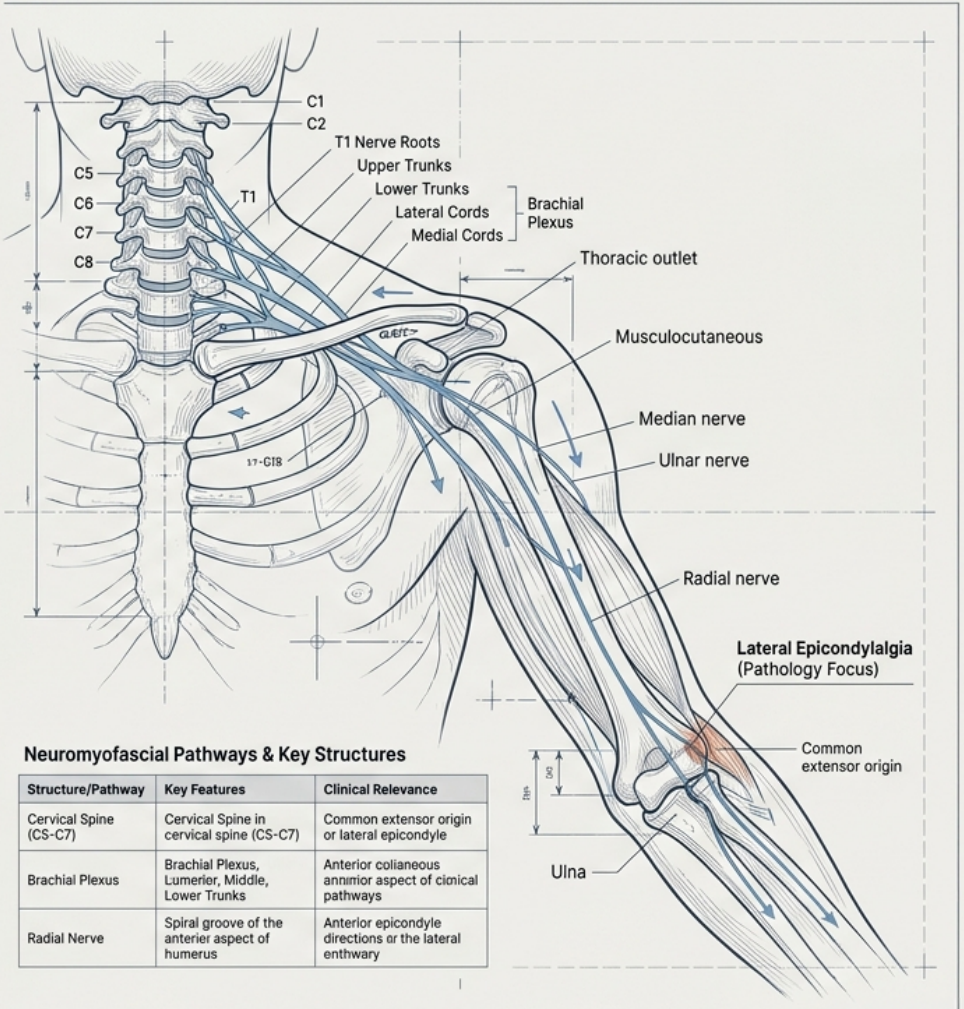
Neurogenic Contribution to Tendinopathy:

A Neuromyofascial Perspective on Lateral Epicondylalgia

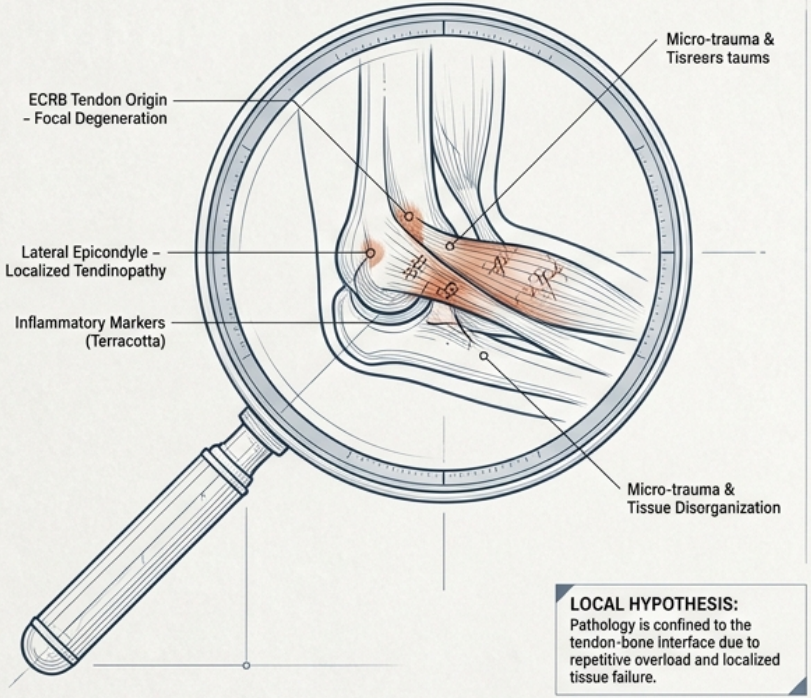


Neuromyofascial Pathways & Key Structures

Structure/Pathway	Key Features	Clinical Relevance
Cervical Spine (CS-C7)	Cervical Spine in cervical spine (CS-C7)	Common extensor origin or lateral epicondyle
Brachial Plexus	Brachial Plexus, Lumerier, Middle, Lower Trunks	Anterior colianeous anmior aspect of cinical pathways
Radial Nerve	Spiral groove of the anterier aspect of humerus	Anterior epicondyle directions or the lateral enthwary

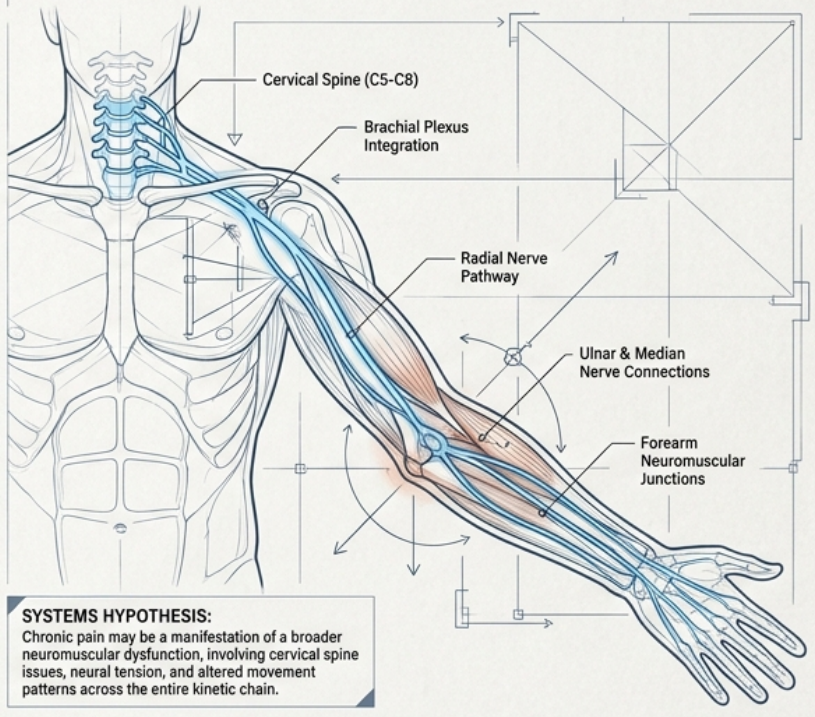
Is chronic tennis elbow always a local tendon disorder, or can it reflect a broader neuromuscular systems problem?

Local View



LOCAL HYPOTHESIS:
Pathology is confined to the tendon-bone interface due to repetitive overload and localized tissue failure.

Systems View



SYSTEMS HYPOTHESIS:
Chronic pain may be a manifestation of a broader neuromuscular dysfunction, involving cervical spine issues, neural tension, and altered movement patterns across the entire kinetic chain.

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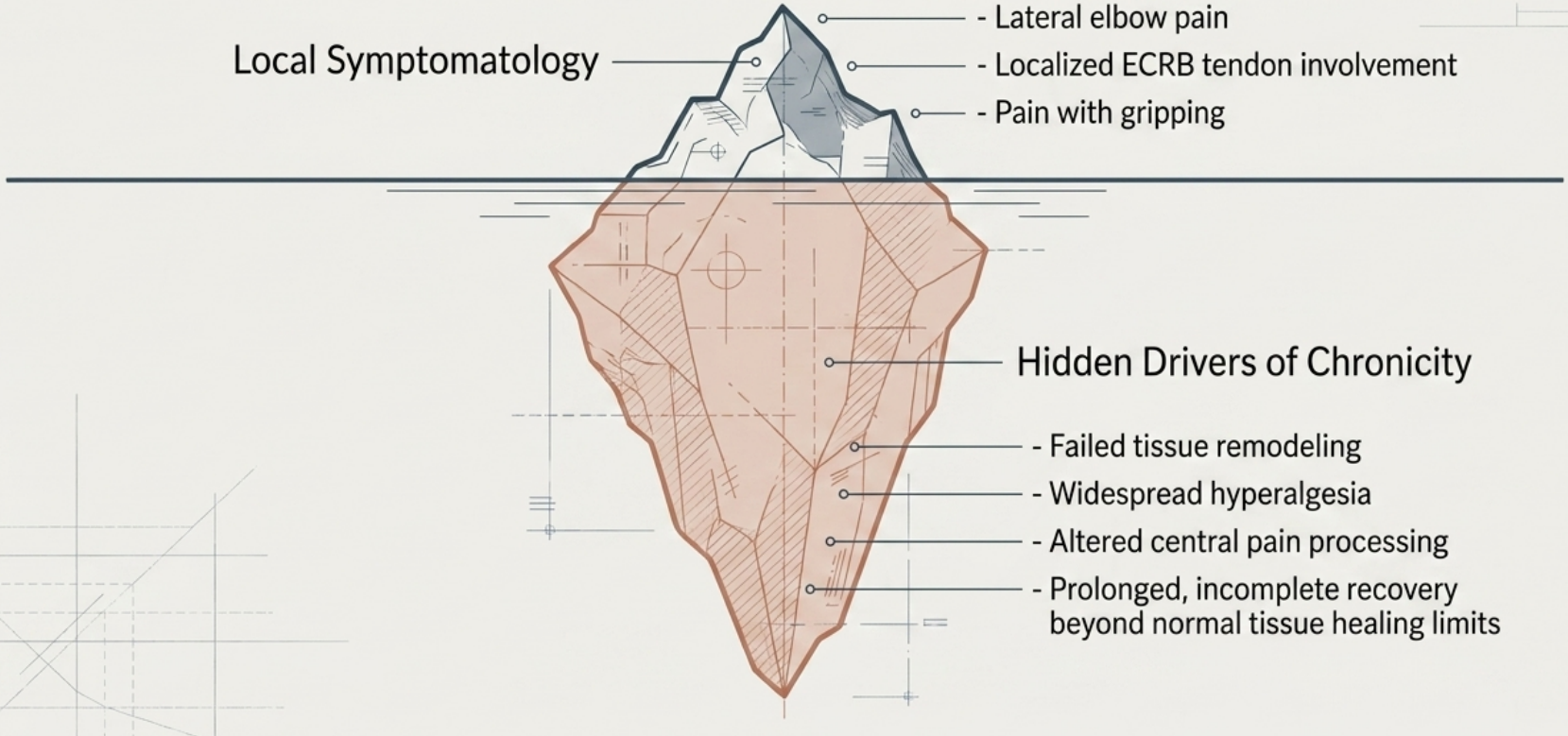
The Clinical Reality of Persistent Lateral Epicondylalgia



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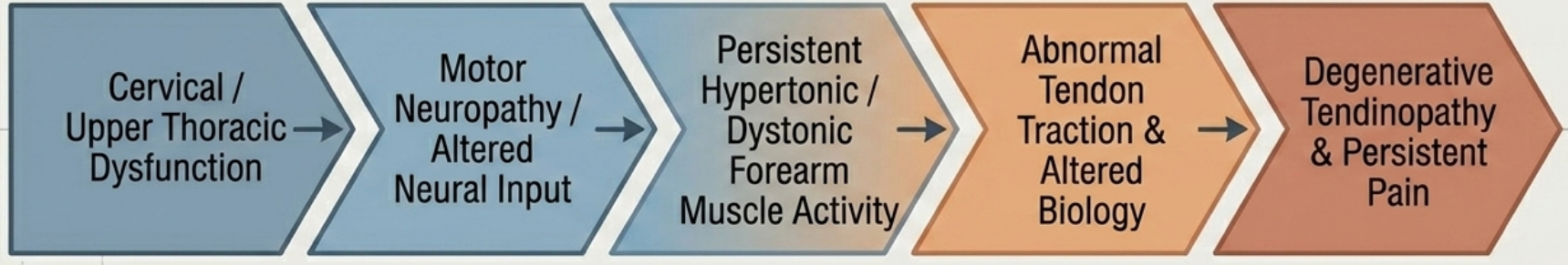
Why Chronicity Challenges the Purely Local Model

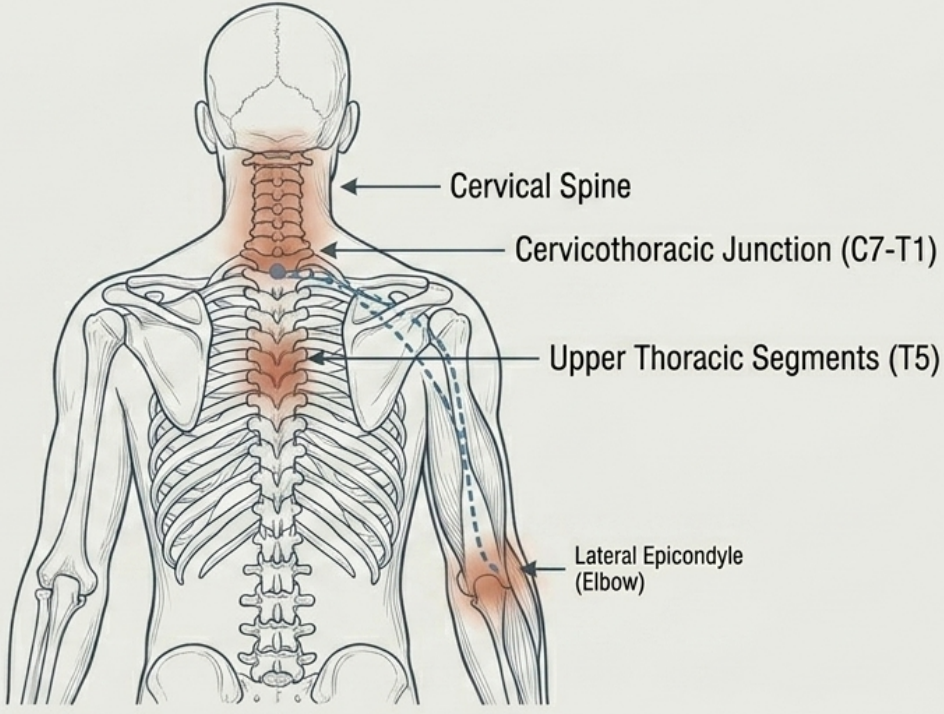
	Purely Local Tendon Model	Neuromyofascial Systems Model
Primary Driver	Mechanical wear-and-tear	Upstream neuromuscular dysfunction
Scope of Pathology	Isolated to common extensor tendon	Involves nerve, muscle, and tendon biology
Healing Expectation	Predictable tissue healing timelines	Persistent as long as upstream neural drivers remain active
Unexplained Variables	Struggles to explain autonomic changes or bilateral hyperalgesia	Accounts for sympathetic responses and spreading hyperalgesia

The Proposed Neuromyofascial (NMF) Sequence



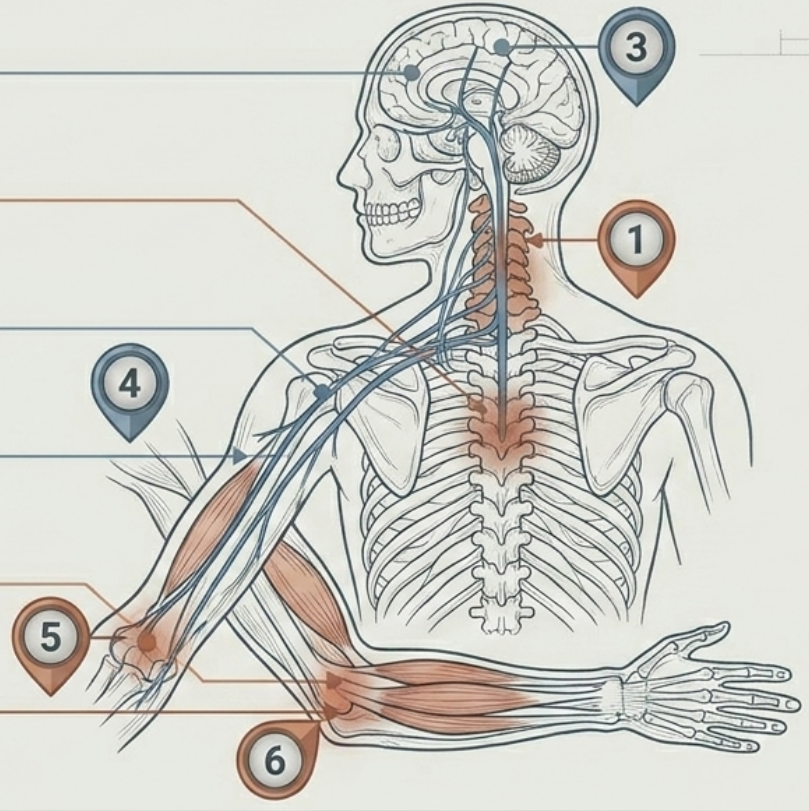
Clinical Baseline: Upstream Spinal Associations

Based on clinical observations from the Lamb Pain Clinic, chronic LE cases frequently present with concurrent, clinically relevant dysfunction in these proximal spinal regions, rather than isolated elbow pathology.

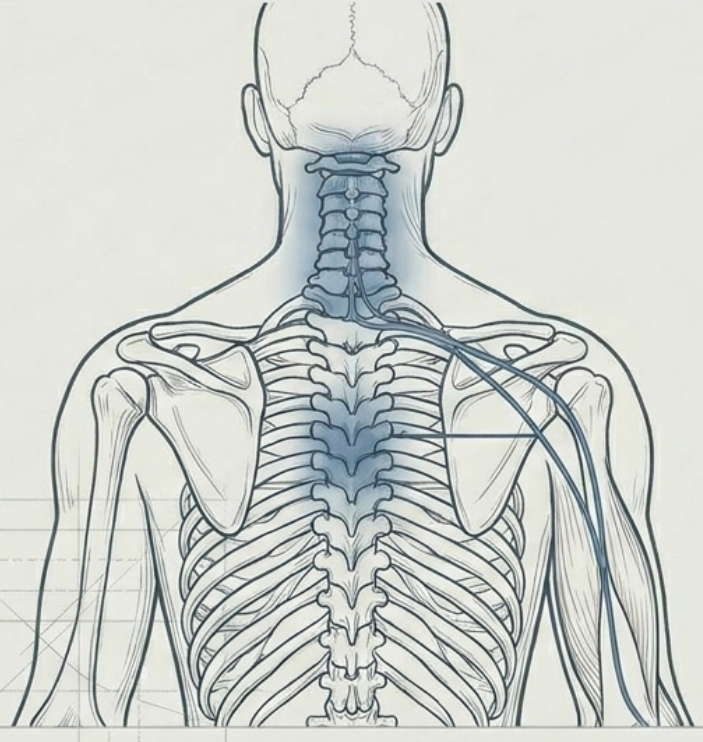


Evidence Map: Validating the NMF Plausibility

1. Cervical Modulation (Spine)
2. Thoracic Modulation (T-Spine)
3. Sensitization & Sensibility (Brain/Systemic)
4. Radial Nerve Involvement (Arm pathway)
5. Altered Motor Control (Forearm muscles)
6. Neurogenic Tendon Biology (Tendon insertion)



Proximal Intervention, Distal Response: Cervical & Thoracic Modulation



Cervical Effects

(Vicenzino 1996; Herd & Meserve 2008)

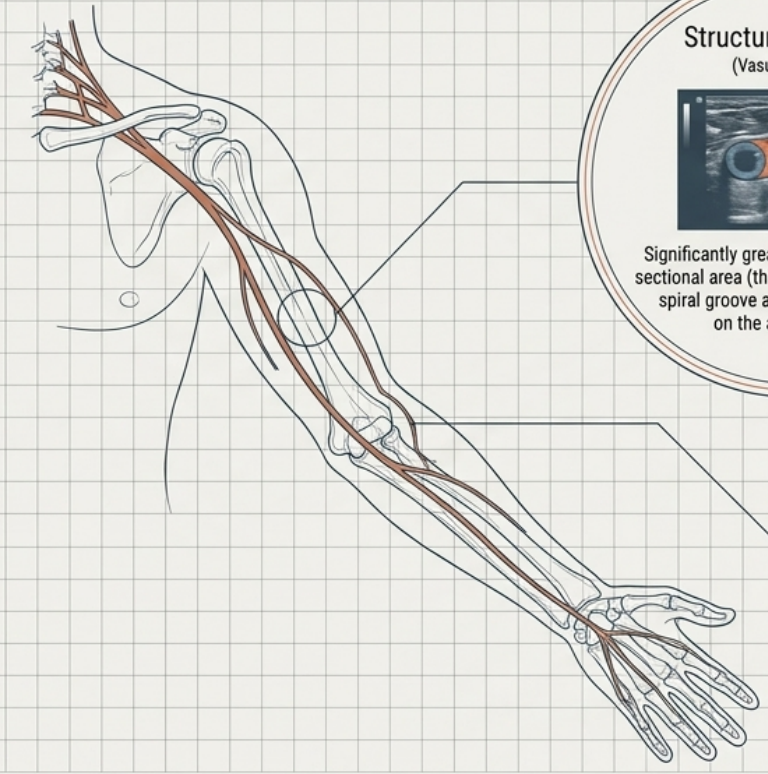
Cervical manipulative therapy produced immediate changes in pain, grip strength, pressure pain threshold, and upper limb neurodynamics in lateral epicondylalgia.

Thoracic & Autonomic Effects

(Zunke 2020; Paungmali 2003)

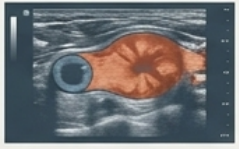
A 2-minute T5 costovertebral mobilization increased pain-free grip by 4.6 kg, increased skin conductance, and produced immediate hypoalgesic and sympathoexcitatory responses.

Radial Nerve Pathology and Spreading Sensitization



Structural Evidence

(Vasudeva 2021)



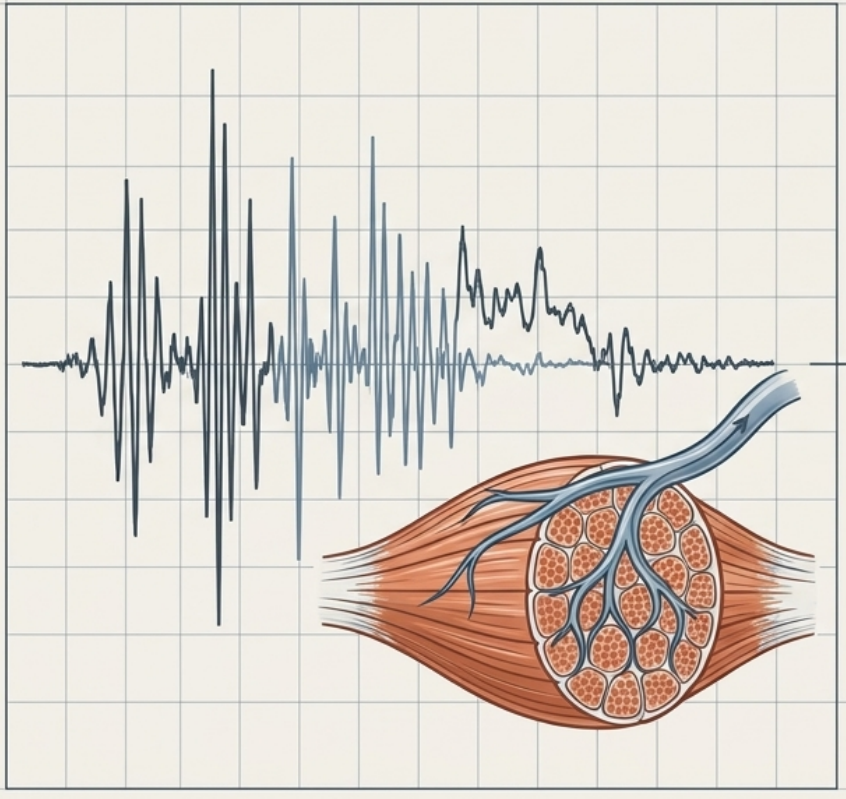
Significantly greater radial nerve cross-sectional area (thickness/swelling) at the spiral groove and antecubital fossa on the affected side.

Sensory Evidence

(Cancela-Cilleruelo 2023; Jespersen 2013)

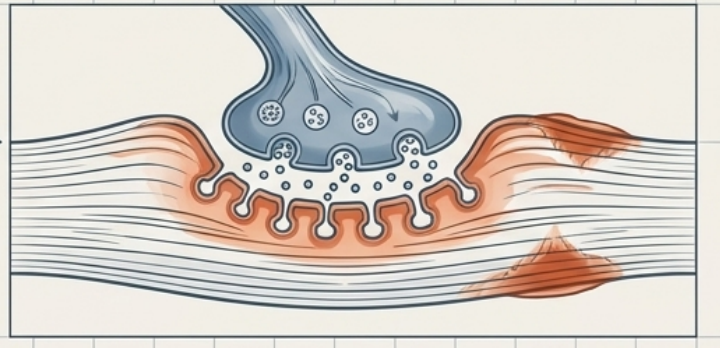
Generalized pressure-pain hyperalgesia over the radial nerve, altered pressure-pain thresholds, and evidence consistent with central sensitization and spreading hyperalgesia.

Altered Neuromuscular Control & Focal Hypertonicity



EMG Findings (Heales 2016)

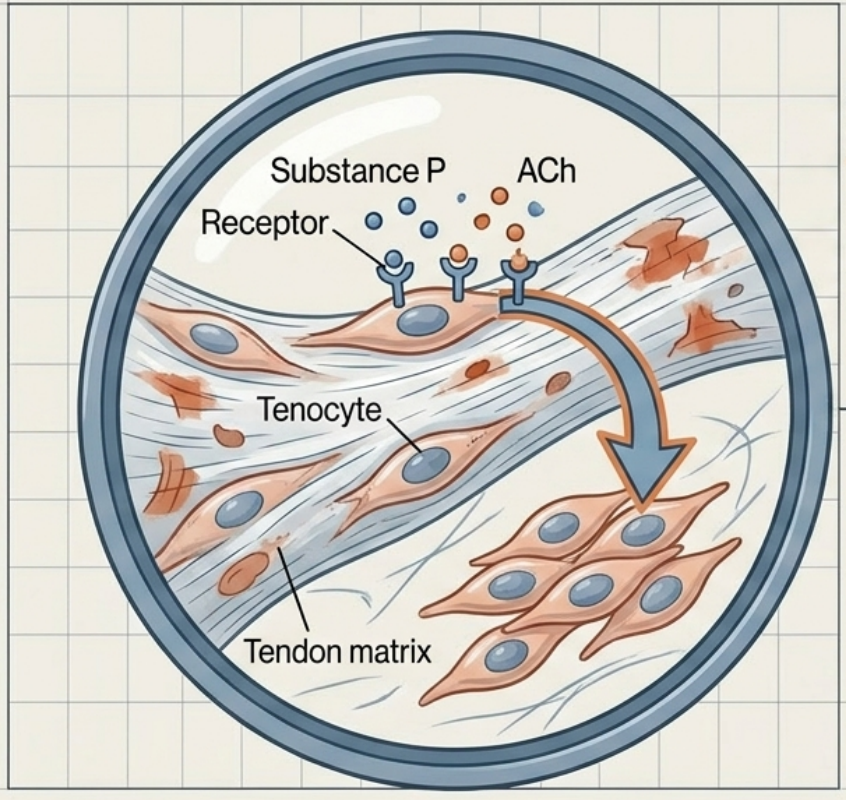
Differences in motor unit potentials, increased time between wrist extensor recruitment and grip force onset, and altered motor cortex organization.



Motor Endplate Activity (Ge 2011)

Spontaneous electrical activity at myofascial trigger points represents extrafusal motor endplate activity, leading to focal contraction, cramp potentials, and ischemia.

Neurogenic Inflammation: Neural Mediators Alter Tendon Biology



Substance P & ACh upregulate TGF-beta1, driving abnormal tenocyte proliferation

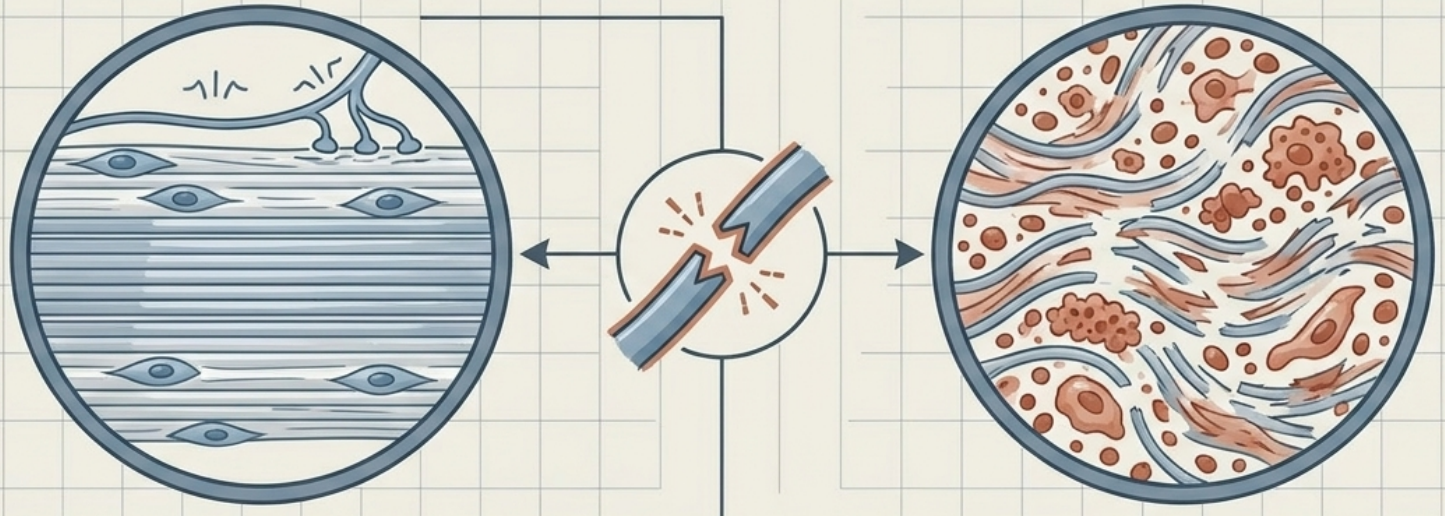
(Wasker 2023)

Systematic review confirming neurogenic inflammation, nerve ingrowth markers, and glutamatergic/sympathetic involvement in tendinopathic tissues.

(Fong 2017 & Backman 2011)

Substance P acts as a mechanoresponsive, autocrine regulator bridging mechanical loading and cellular remodeling.

Denervation Forces Tendinosis-Like Structural Change



Study Summary (El-Habta 2018)

Unilateral sciatic nerve transection in rat Achilles resulted in hypercellularity, disfigured cells, disorganized collagen, increased type III collagen, and increased NK-1R expression after just two weeks.

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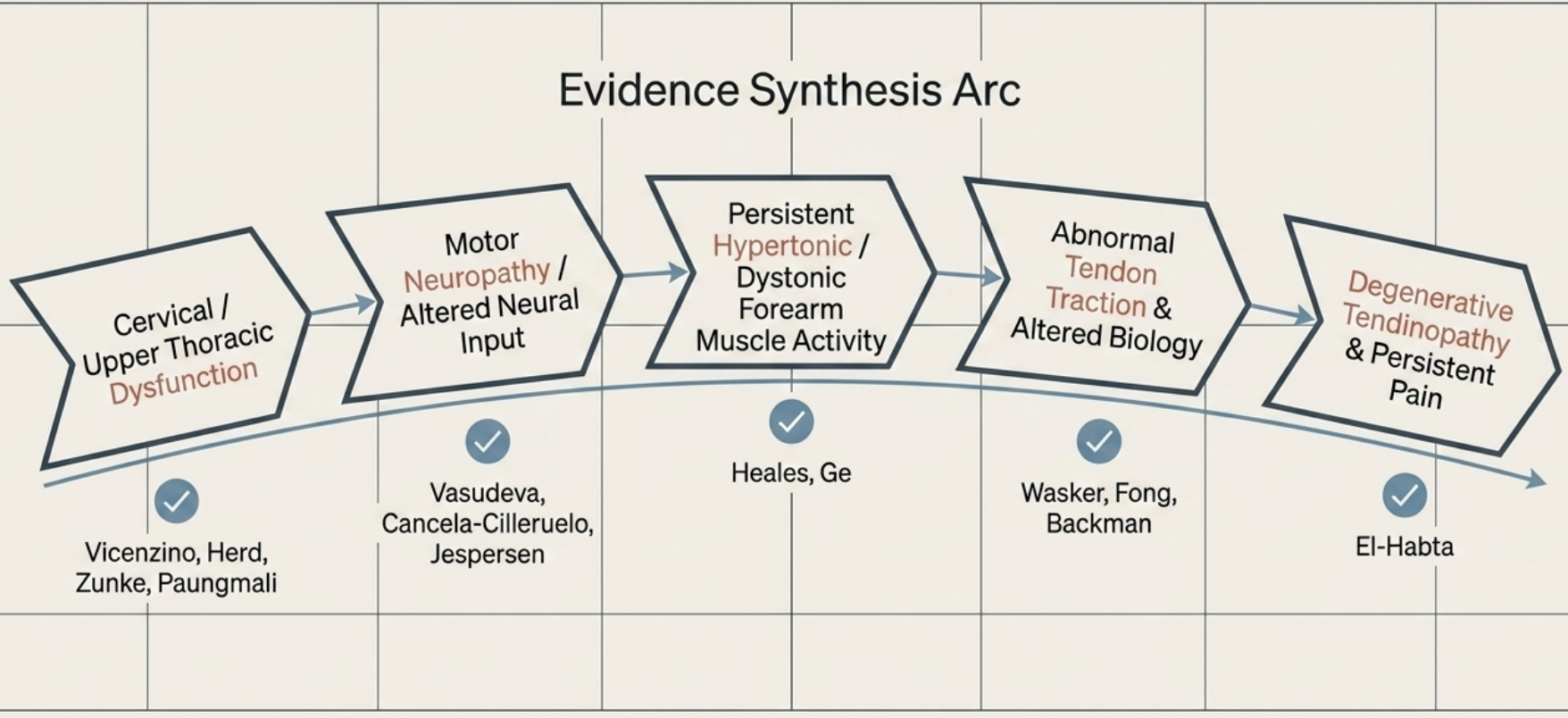
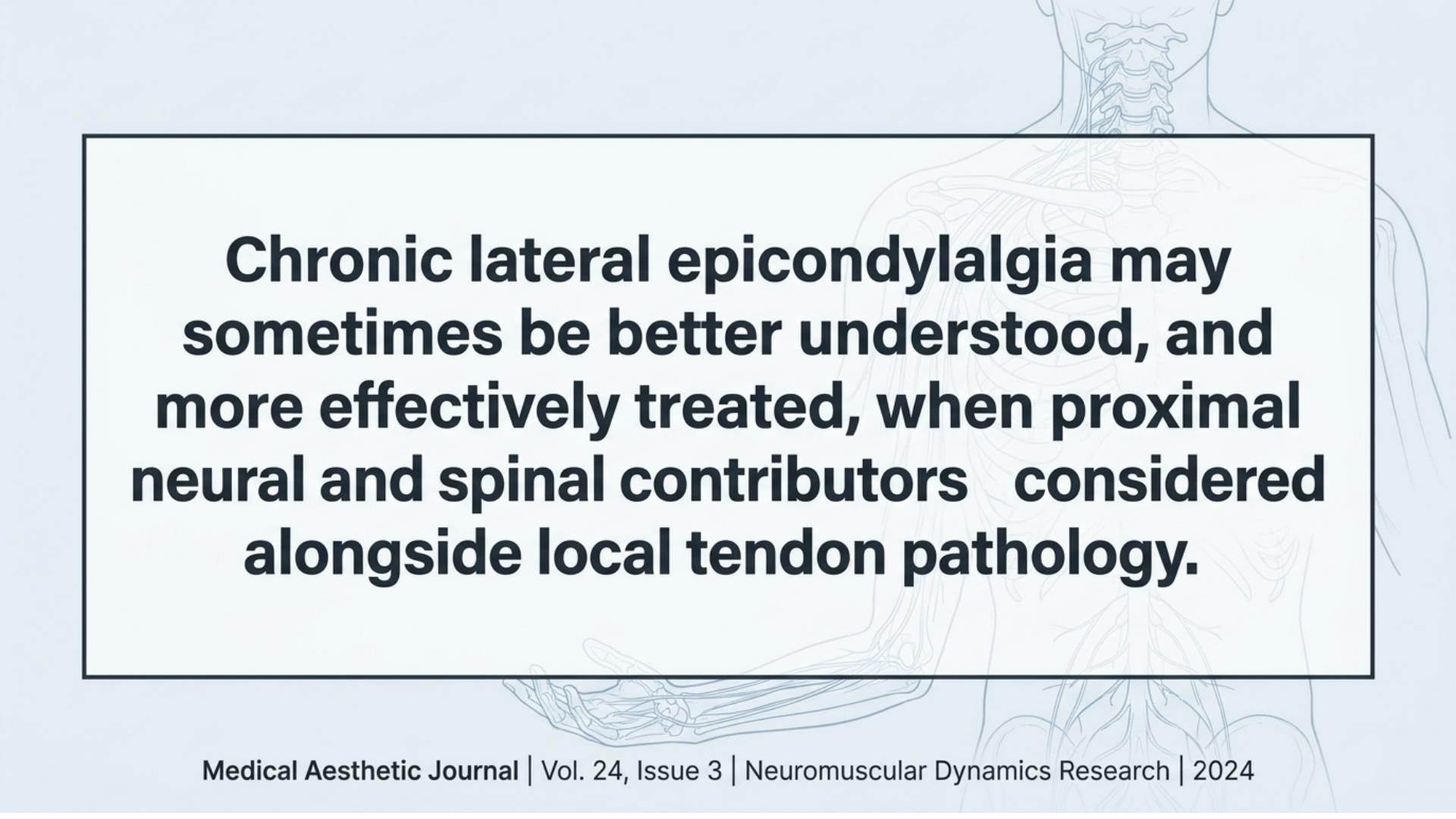
Synthesis: Validating the Neuromyofascial Component Pathways



Clinical Context, Limitations, and Cautions

- **Component Support vs. Full Proof:** These studies validate component concepts; they do not claim experimental proof of the entire causal sequence in every patient.
- **Temporal Limits:** Many manual therapy studies report immediate or short-term neurophysiologic effects, not long-term disease modification.
- **Mechanistic vs. Clinical:** Animal denervation and in vitro tenocyte studies represent biological plausibility, not direct clinical proof for tennis elbow.
- **Patient Subsets:** Radial nerve sensitization supports nerve involvement in a subset of LE patients, but does not prove primary radial pathology in all cases.



Chronic lateral epicondylalgia may sometimes be better understood, and more effectively treated, when proximal neural and spinal contributors considered alongside local tendon pathology.

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