Why a neuromuscular lens changes what you see (and what you can fix) with hands and movement

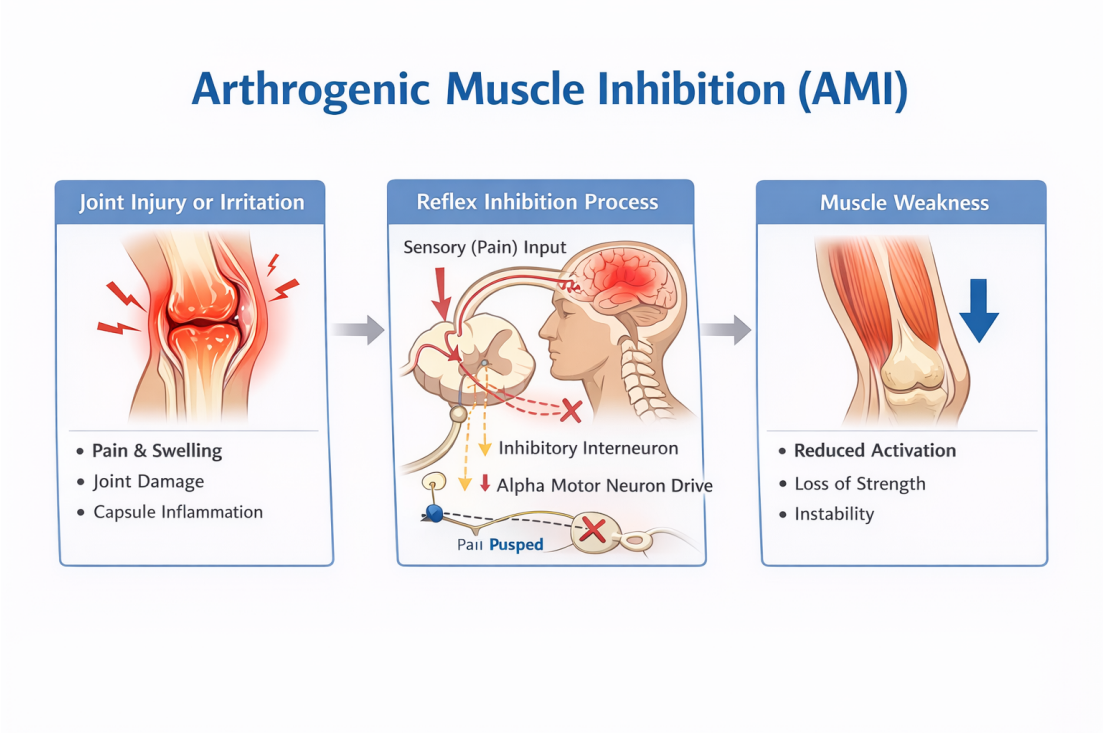
Most of us were trained to "localise the lesion" by tissue: tendon, muscle belly, joint, nerve. That model still matters—but it often fails in a familiar scenario: the patient who "should be strong" is inexplicably weak, unstable, or painful despite good imaging, reasonable ROM, and honest effort.

A neuromuscular approach starts from a different first principle:

Motor output is conditional. The CNS continuously gates force production based on the salience of incoming afferent information—especially nociception, joint mechanoreception, and perceived threat. When threat rises or afferent clarity drops, the system doesn't simply "hurt"; it **downregulates** muscles, alters timing, and changes coordination as a protective strategy.

That protective strategy has names in the literature (e.g., arthrogenic muscle inhibition), has measurable neurophysiology, and—crucially—often responds immediately to skilled manual input and/or well-chosen movement.

Arthrogenic muscle inhibition as the clinical "smoking gun"



Arthrogenic muscle inhibition (AMI) is a well-described example of how joint pathology produces persistent weakness **without primary muscle damage**. After joint injury, effusion, inflammation, or mechanoreceptor disruption, reflex pathways and corticospinal

changes reduce voluntary activation of surrounding musculature—classically the quadriceps after knee insult, but the principle generalises.

Experimental work also shows that even *artificial* joint effusion can reduce corticomotor excitability and alter intracortical inhibition—i.e., not just peripheral reflexes, but central gating.

Clinical translation: when a muscle "won't test," you may be looking at a **signal problem** (joint/nerve/threat), not a strength problem.

What Manuel Muscle Testing (MMT)/Neuro Muscular Function Testing (NMFT) are really testing (when used intentionally)

Manual Muscle Testing (MMT) is commonly framed as a strength test. In practice, it is often a **threat tolerance + motor control test under load**.

In an inhibited system, the failure pattern is rarely a true maximum:

- delayed recruitment
- "give-way" collapse
- inconsistent performance across positions
- rapid improvement after de-threat input (manual therapy, unloading, breathing/ANS shift, or motor re-patterning)

If you accept the CNS as a *predictive* controller, then MMT/NMFT becomes a fast way to probe:

1. **Does the CNS trust this joint/segment?**
2. **Is afferent input coherent?**
3. **Does load increase threat?**
4. **Can we change output immediately by changing input?**

That last question is the practical power of NMFT-style reasoning: if output changes quickly after a targeted intervention, the "weakness" was largely **neurophysiological gating**, not tissue capacity.

Why manual therapy can "switch a muscle back on" (and why it sometimes doesn't)

Manual therapy's most clinically useful effects are often **neurophysiological**, not structural. Contemporary reviews describe CNS and ANS changes following spinal/manual interventions—e.g., modulation of pain perception and measurable changes in excitability in some contexts—while also noting heterogeneity and methodological limitations.

From a neuromuscular viewpoint, the relevant mechanism is straightforward:

- **Joint and soft-tissue inputs are high-bandwidth sensory channels.**
- Skilled manual input can shift the balance away from high-salience nociception (Type III/IV dominance) and toward clearer mechanoreceptive input (Type I/II), reducing the CNS "need" to inhibit.
- If threat drops and afferent clarity improves, motor output often increases immediately.

Why it sometimes doesn't work:

- The primary driver may be nerve mechanosensitivity, sleep/stress load, or centrally maintained threat rather than a local joint signal.
- The manual input may not be specific enough to alter the dominant afferent stream.
- The patient's prediction of danger may overwhelm the sensory change.

This is not a reason to abandon manual therapy—it's a reason to link it to **a test–retest logic** and pair it with movement that consolidates new input.

Movement is often the stronger intervention (because it's the more meaningful input)

If manual therapy is a "reset," movement is the "re-write." The CNS weights sensory input by relevance: active control, load acceptance, and prediction error reduction are extremely salient.

Proprioceptive systems (muscle spindles, Golgi tendon organs, joint receptors) provide continuous feedback for motor control, and modern physiology work continues to detail how these sensors support robust movement.

Clinically, the fastest way to reduce inhibition long-term is to:

- keep the stimulus below the patient's threat threshold, and
- gradually increase meaningful load with high-quality afferent input (slow eccentrics, closed-chain stability, perturbation, graded exposure)

This is why “strengthening” often fails when it is applied too early: you’re asking for output while the CNS is still receiving “danger/noise” as the dominant input.

Why this approach attracts referrals (and better outcomes)

A neuromuscular model makes your clinical decisions look less like “try this technique” and more like **a repeatable, falsifiable process**:

- hypothesis → **input change → retest → consolidate**
  That’s not just persuasive to colleagues; it’s persuasive to the patient’s nervous system.

And it reframes “weakness” as a solvable problem of **signal quality and threat calibration**, not a moral failing (“not trying”) or an endless strength deficit.

Selected research references

1. Rice DA, McNair PJ. *Quadriceps Arthrogenic Muscle Inhibition: Neural Mechanisms and Treatment Perspectives*.(Review).
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3. Study on experimental knee effusion altering corticomotor/intracortical excitability (AMI mechanisms).
4. *The mechanisms of manual therapy: A living review of systematic, narrative and scoping reviews* (PLOS ONE).
5. *Short-Term Effects of Spinal Manual Therapy on the Nervous System…* (systematic review; CNS/ANS outcomes).
6. *Spinal manipulation therapy: Is it all about the brain?* (review discussing neurophysiological changes).
7. Review on neurological effects of spinal manipulative therapy (motor neuron excitability/ proprioceptive afferents discussed).
8. Physiology review on muscle spindles and their role in locomotion (proprioceptive control relevance).
9. Foundations/chapters summarizing spinal motor control and proprioceptive afferents (neuroanatomy context).
10. Manual muscle testing + handheld dynamometry clinimetrics in clinical populations (reliability/validity considerations).

Morten Wolff DC, BSc, NMFT

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