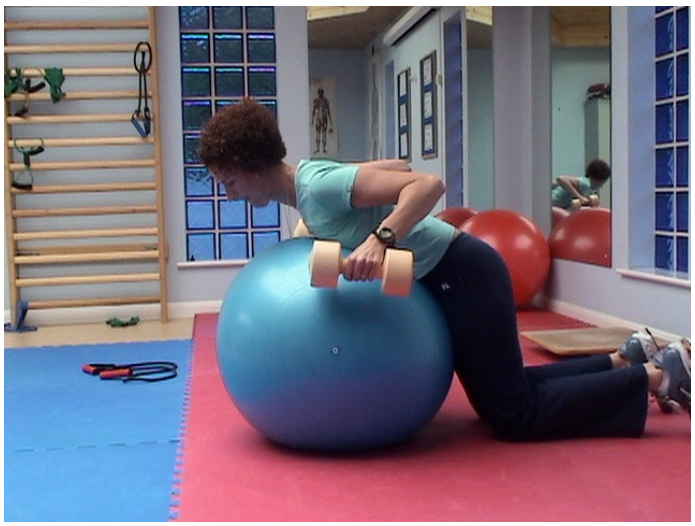
When Exercise Meets a Switched-Off Muscle

The problem with "rehab as usual" when old inhibition is still running in the background



In everyday MSK practice, we routinely prescribe strengthening and motor control exercises after pain or injury: squats after ACL reconstruction, core work after low back pain, balance drills after ankle sprain. We assume that if we load the right muscles in the right way, the system will adapt.

But there is a fundamental problem with this logic:

You cannot rehabilitate a muscle the nervous system is still reflexively inhibiting.

If a past injury has left behind unrecognised neuromuscular inhibition, the muscle is not simply "weak" – it is **not being allowed to fully switch on**. The patient can grimace, focus, and push as hard as possible, but the descending drive is being blocked by altered reflex loops at spinal and supraspinal levels. In this context, even the best exercise program becomes a "software problem" treated with a "hardware solution."

Arthrogenic muscle inhibition: the classic example

Arthrogenic muscle inhibition (AMI) is a well-described phenomenon after joint trauma, especially around the knee. After ACL rupture or reconstruction, joint injury and inflammation alter afferent input from mechanoreceptors and nociceptors in the joint capsule, ligaments, and surrounding tissues. This abnormal input triggers a **presynaptic reflex inhibition** of the quadriceps motor neuron pool in the spinal cord – AMI Frontiers.

Clinically, that means:

- The quadriceps, particularly the Vastus Medialis, **cannot fully activate**, even when the patient is pain-free and trying hard.
- This is not just atrophy; EMG and motor unit studies show impaired neural drive and persistent activation deficits months after surgery FrontiersThe Knee Journal.
- Scoping reviews identify AMI as a major barrier to restoring strength, normal gait, and sport performance, and as a contributor to early osteoarthritis when it remains unresolved British Journal of Sports Medicine.

Crucially, several recent papers emphasise that AMI **can persist for months or years** despite conventional rehabilitation, leading to strength asymmetry, altered biomechanics, and increased risk of re-injury MDPIThe Knee Journal. In other words, patients can "complete" rehab, yet still have a chronically inhibited quadriceps.

From a neuromuscular perspective, this is exactly the scenario where exercise alone is insufficient: you are trying to strengthen a muscle that the nervous system is actively holding back.

Low back pain: Multifidus inhibition and failed "core training"

The same mechanism appears in the spine. Multifidus is a key stabiliser of the lumbar segments and a major contributor to fine motor control of the "neutral zone." Multiple reviews now highlight **Multifidus dysfunction as a distinct phenotype in chronic low back pain** OUP Academicijssurgery.com.

Key points:

- Multifidus atrophy and poor activation often arise from **pain-related reflex inhibition** following an initial insult (facet joint pain, disc injury, strain, etc.) ScienceDirectEinstein.
- This inhibition can continue even after pain improves, reducing segmental stability and predisposing to recurrent episodes ScienceDirectEinstein.
- Restorative neurostimulation studies show that when you directly **normalise the neuromuscular drive to Multifidus**, patients with this phenotype improve significantly – and that standard "optimal medical management" and exercise often provide very limited benefit in this subgroup SpringerLinkijssurgery.com.

If Multifidus is reflexively down-regulated, the usual prescription of "core stability" exercises may not achieve what we think. Patients often compensate with global muscles (Erector Spinae, Obliques, Gluteals,QLs), creating bracing patterns that look strong but do not restore precise segmental control. Clinically, this appears as:

- Good effort but limited true segmental control
- "Core" workouts that fatigue global muscles while the key stabilisers remain under-recruited
- Recurrent episodes once the patient returns to normal activity

Again, the **neuromuscular control problem precedes and undermines** the exercise program.

Ankle sprains and chronic instability: when balance training isn't enough

Lateral ankle sprains provide another familiar pattern. After the initial injury, many athletes develop **chronic ankle instability (CAI)** with residual pain, episodes of giving way, and recurrent sprains. Research shows that:

- A large proportion of those with prior ankle sprain have persistent **proprioceptive deficits and neuromuscular control impairments**, not just mechanical laxity MDPI.
- These sensorimotor deficits are closely tied to altered postural control and increased re-injury risk NatureSAGE Journals.
- Proprioceptive and neuromuscular training can substantially improve function in CAI, highlighting that restoring sensorimotor control is central to recovery PLOSphysiotherapyjournal.org.

If the peroneal muscles and other stabilisers are still inhibited or poorly integrated sensorimotorly, prescribing generic strengthening or balance tasks can generate compensation and fatigue, but not genuine restoration of dynamic stability at the joint.

What actually goes wrong when we overlook inhibition?

When unrecognised neuromuscular inhibition from an older injury is present, several predictable problems arise once we start exercise rehab:

1. **Compensation instead of correction**
   The inhibited muscle cannot produce its normal share of force, so other muscles step in. Patients recruit synergists, global stabilisers, or the contralateral limb to "get the job done." The clinician sees movement, but the underlying control strategy is altered. This can shift load to secondary regions – for example, hip or lumbar overload in a patient with unresolved quadriceps or Multifidus inhibition.

2. **Plateaus in strength and function**
   Because neural drive is capped, strength gains quickly plateau. Despite progressive loads, the inhibited muscle fails to hypertrophy or regain full output. This is well documented in the quadriceps after ACL injury, where lingering AMI prevents full restoration of strength even after standard rehabilitation timelines British Journal of Sports MedicineMDPI.

3. **Persistent asymmetries and abnormal biomechanics**
   AMI and similar phenomena produce side-to-side strength deficits, altered joint kinematics, and faulty movement patterns (e.g., reduced knee extension, gait asymmetry) that remain long after tissue healing British Journal of Sports MedicineThe Knee Journal. These biomechanical changes increase joint loading and are linked to early degenerative changes such as osteoarthritis British Journal of Sports Medicine.

4. **Mislabeling patients as "non-compliant" or "complex"**
   When exercises don't produce expected results, clinicians may assume poor adherence, psychological issues, or unusually complex pathology. In reality, the nervous system is still protecting the joint or region through reflex inhibition. The problem is not the patient's motivation – it is our failure to test and treat the **neurological gatekeepers** of muscle activation.

5. **Increased risk of recurrence and chronicity**
   Across knee, spine, and ankle literature, persistent neuromuscular activation deficits are repeatedly associated with recurrent injury, ongoing instability, and chronic pain MDPIEinsteinMainstay Medical. If we ignore the inhibition and focus only on "strengthening," we may be reinforcing maladaptive patterns rather than resolving them.

Why a Neuromuscular Approach Neuromuscular Function Testing (NMFT) Must Come *Before* Rehab

The emerging science – including the recent RCT showing that spinal manipulation can acutely up-weight lumbar proprioceptive input and improve sensorimotor integration in the spine Mainstay Medical – points to a clear clinical message:

We should evaluate and normalise neuromuscular control before, or at least alongside,

exercise rehabilitation.

Neuromuscular Functional Testing (NMFT) provides exactly this window into the control system. Before building a rehabilitation plan, NMFT can help the practitioner answer three critical questions:

1. **Is the target muscle actually available?**
   Through specific manual muscle tests and functional challenges, NMFT can identify muscles that *should* be strong but exhibit clear, reproducible weakness or delayed recruitment – out of proportion to pain or deconditioning. This pattern strongly suggests reflex inhibition rather than simple weakness.

2. **What is driving the inhibition?**
   NMFT does not stop at "weak or strong." By systematically challenging joints, ligaments, myofascial structures, and sensory inputs (vision, vestibular, cutaneous, proprioceptive), clinicians can identify aggravating or relieving stimuli. For example, a previously weak muscle that immediately strengthens following a specific joint mobilisation or cutaneous input suggests a neurogenic, afferent-driven inhibition – the exact type of problem seen in AMI, Multifidus dysfunction, and CAI phenotypes FrontiersEinstein.

3. **Can the reflex be reset?**
   Once the underlying trigger is identified (e.g., an old ligamentous lesion, segmental joint dysfunction, or a scar), targeted neuromuscular interventions – such as specific manual therapy, spinal manipulation, or reflex-oriented sensory stimulation – can be applied to normalise afferent input. When this is successful, the previously inhibited muscle often shows **an immediate improvement in strength and control**, confirming that the "software" has been updated.

Only after this neuromuscular "unlocking" does traditional exercise rehab reach its full potential. Now the muscle is truly available for strengthening, coordination training, and load progression. Rather than fighting the nervous system, rehab works *with* it.

Practical flow in a neuromuscular-first model

For MSK practitioners, a neuromuscular-first approach (integrating NMFT) might look like this:

1. **Screen for inhibition and sensorimotor deficits**
   - Test key stabilisers (e.g., Quadriceps, Gluts, Multifidus, Piriformis, TFL) with high-quality manual muscle tests and simple functional tasks.
   - Look for unexpected weakness, poor endurance, delayed activation, or side-to-side differences that do not match the structural picture.

2. **Identify and treat the neuromuscular drivers**

- Use NMFT to probe which joint, ligamentous, fascial, or sensory inputs modulate the inhibited muscle.
- Apply specific manual or neuromodulatory techniques (e.g., spinal manipulation, targeted joint mobilisation, soft tissue techniques, sensory cues). The recent work by Nyirö *et al*. shows that such interventions can meaningfully change proprioceptive weighting in the lumbar spine – a strong proof-of-concept for neuromuscular modulation Mainstay Medical.

3. **Retest immediately**
   - Confirm that muscle activation has improved after the intervention. Immediate changes give both clinician and patient confidence that they have addressed a control problem, not just symptoms.
4. **Layer exercise rehab on top of restored control**
   - Now prescribe strengthening, motor control, and return-to-sport exercises, knowing the key stabilisers are online.
   - Progress load and complexity while continuing to monitor neuromuscular function; if inhibition reappears, revisit NMFT and adjust.

By placing neuromuscular assessment and intervention **before** or alongside exercise, we dramatically reduce the risk of "pushing on a locked door" – investing time in rehab that the nervous system is not ready to support.

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