Why Functional Assessment Is Superior to Structural Observation in Neuromusculoskeletal Conditions

Introduction

In clinical practice, musculoskeletal assessment has traditionally relied heavily on what we can **see** and **measure structurally**—radiographs, MRI scans, posture analysis, or visual inspection of joint alignment. These tools undoubtedly offer valuable information about anatomy, but they do not necessarily explain how a patient **functions**. Many individuals present with severe degenerative findings on imaging yet experience minimal or no pain, while others suffer from disabling symptoms despite "normal" scans. This discrepancy highlights a critical truth: **function, not structure, determines health and performance** in the neuromusculoskeletal system.

The Limitations of Structural Observation

1. Imaging does not reflect dynamic capacity.
Radiographs and MRIs are static snapshots of a dynamic system. They depict bones, joints, and tissues in a fixed position but reveal nothing about how those structures behave during motion or under load. A perfectly aligned joint on x-ray may move abnormally during gait, while a degenerative joint may still function efficiently because of compensatory neuromuscular control.

2. Structural variation is common and often asymptomatic.
Studies consistently show that imaging findings such as disc bulges, rotator cuff tears, or osteoarthritis appear in a large proportion of asymptomatic adults. Structural "abnormalities" may simply represent normal biological aging rather than pathology. Over reliance on imaging risks medicalising normal variation and directing attention away from the true determinants of pain and performance.

3. Observation alone misses underlying control mechanisms.
Visual assessment—posture, swelling, or muscle atrophy—can provide clues but rarely reveals the **neuromuscular coordination** responsible for maintaining or disrupting movement patterns. Two individuals with identical postures may experience entirely different motor control strategies and, consequently, different clinical outcomes.

The Central Role of Function

1. Function integrates structure, control, and adaptation.
The musculoskeletal system's purpose is not simply to exist in anatomical harmony but to move, stabilise, and adapt to load. Functional testing—measuring muscle activation, joint mobility, timing, endurance, and movement efficiency—evaluates how the nervous system organises muscular effort to achieve these goals. Function thus represents the integration of structure (anatomy), physiology (neural control), and behaviour (movement).

2. Pain and dysfunction are often neurophysiological, not structural.
Pain perception arises from the interaction between sensory input and central processing. Dysfunctional motor patterns, altered proprioception, or inhibited stabilising muscles can produce pain even in the absence of structural pathology. Conversely, restoring normal neuromuscular coordination can eliminate pain long before any structural "abnormality" resolves. Function therefore serves as both a **diagnostic window** and a **therapeutic target**.

3. Function predicts prognosis and guides treatment.
Functional measures—such as range of motion under active control, muscle recruitment timing, or load tolerance—provide actionable information. They allow clinicians to identify weak links, design individualised interventions, and track progress objectively. Unlike imaging findings, which may remain unchanged even as the patient improves, functional tests demonstrate **real recovery**.

Clinical Implications

Evaluating function requires dynamic, hands-on examination. Techniques such as manual muscle testing, movement screening, and joint-specific functional analysis reveal inhibited or overactive muscle groups, altered synergy patterns, and proprioceptive deficits. By addressing these functional disturbances through neuromuscular retraining, manual therapy, and targeted exercise, clinicians restore the system's capacity to adapt and self-stabilise.

This perspective reframes treatment goals. The objective is not to "fix" what appears abnormal on imaging but to **optimise the patient's functional capacity**—to improve movement efficiency, load distribution, and neuromuscular coordination. Structural changes often follow functional improvement, not the other way around.

Conclusion

When dealing with neuromusculoskeletal conditions, **function is the most meaningful parameter** to assess and restore. Structure provides context;

function provides truth. A clinician who evaluates how the system behaves in motion gains insight into the root cause of dysfunction, the effectiveness of intervention, and the real measure of health. In short, while x-rays show us **what is there**, functional examination reveals **what it can do**—and in clinical reality, that difference defines recovery.

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Deep Dive into Research:

Neuromuscular Inhibition After Joint Trauma: Evidence from Recent Studies

Arthrogenic Muscle Inhibition (AMI) and Reflex Inhibition Mechanisms

Definition and Mechanism: Joint trauma – especially injuries involving excessive stretch beyond the normal passive range of motion – can trigger a protective neural response called **arthrogenic muscle inhibition (AMI)**frontiersin.orgmdpi.com. In AMI, sensory signals from injured joint tissues (e.g. stretched ligaments, inflamed capsule) cause a **presynaptic reflexive inhibition** of the alpha motor neurons supplying muscles around the jointfrontiersin.org. In simpler terms, the body "shuts down" or reduces activation of certain

muscles to protect the joint from further damage. This reflex inhibition is **involuntary** and occurs even without direct muscle injurymdpi.com. Key contributors to AMI include joint pain, swelling (effusion), and damage to mechanoreceptors, which alter afferent signals and spinal reflex loops – ultimately preventing full muscle contraction. Recent research confirms that **AMI is common after acute joint injuries**, such as ligament tears or joint sprains, and can **persist long after the initial trauma**frontiersin.orgfrontiersin.org.

Neurophysiological Changes: Modern studies have begun to unravel the complex neural mechanisms behind AMI. Following knee ligament injury (e.g. ACL rupture), patients exhibit both **spinal and supraspinal alterations** in muscle control. For example, researchers have documented **increased excitability of spinal reflex pathways** (heightened inhibitory reflexes) alongside **reduced excitability of corticospinal pathways** (diminished voluntary drive from the brain) in the months after injuryfrontiersin.org. Electrophysiological analyses up to one year post-ACL reconstruction show persistent changes in motor unit recruitment and firing rates in the thigh musclesfrontiersin.orgfrontiersin.org. These findings suggest that joint trauma induces not only local reflex changes but also central nervous system adaptations (e.g. altered cortical motor output) that **inhibit muscle activation**. In summary, AMI represents a multi-level neuromuscular response: a combination of spinal reflex inhibition (driven by joint afferents) and central activation deficits, both of which serve to guard the injured joint at the cost of normal muscle functionfrontiersin.org.

Functional Consequences of Joint Injury on Surrounding Musculature

Muscle Weakness and Atrophy: One immediate consequence of AMI is measurable **muscle weakness** in the affected limb. Since the nervous system is not fully activating the muscle, strength output drops significantly. For instance, after knee injuries like ACL tears, patients experience marked quadriceps weakness very early post-injury frontiersin.org. This weakness is not simply due to pain or disuse – it is a direct result of reflex inhibition preventing full recruitment of muscle fibers. Over time, chronically reduced activation can lead to **muscle atrophy** (loss of muscle mass), as commonly seen in the quadriceps after knee trauma mdpi.com. A 2022 narrative review by Pietrosimone et al. noted that AMI-induced quadriceps activation failure **impairs muscle strength and leads to aberrant movement biomechanics** in ACL-injured individuals pubmed.ncbi.nlm.nih.gov. Even with rehabilitation, such reflexive muscle shutdown can be "resistant to traditional rehabilitation techniques," resulting in **persistent neuromuscular deficits** long after the joint itself has healed pubmed.ncbi.nlm.nih.gov.

Altered Movement and Joint Function: Because key stabilising muscles cannot fire normally, joint injuries often cause **functional movement impairments**. Inhibited muscles fail to provide proper joint support, leading to compensatory movement patterns or instability. Clinically, this may manifest as gait abnormalities, poor balance, or decreased functional performance. Pietrosimone et al. (2022) explain that AMI-driven quadriceps weakness contributes to **altered knee biomechanics** and can hinder safe return to activity

pubmed.ncbi.nlm.nih.govpubmed.ncbi.nlm.nih.gov. Surrounding muscle groups may not coordinate effectively, so patients develop limp or avoidance of certain ranges of motion. In the upper extremity, a similar phenomenon can occur – for example, after shoulder capsule overstretch or dislocation, reflex inhibition of the rotator cuff can compromise shoulder stability. In ankles, chronic instability after severe sprains is now partly attributed to lingering AMI in muscles like the peroneals and soleus mdpi.com.

Persistent Deficits and Re-Injury Risk: Perhaps most concerning, neuromuscular dysfunction from AMI can **persist long after the acute injury phase**, creating a latent risk for future injuries. Recent longitudinal research confirms that muscle activation deficits are not just an acute response but can become a chronic issue. McPherson et al. (2023) followed ACL-injured athletes and found that even **12 months after ACL reconstruction**, motor unit firing patterns in the thigh muscles remained abnormal compared to healthy controls frontiersin.org. Notably, both the injured and even the "uninjured" contralateral limb showed smaller motor unit potentials and altered firing rates, indicating a bilateral neuromuscular impact of the unilateral injury frontiersin.org. Such deficits in muscle activation and coordination may **predispose individuals to re-injury** or insufficient functional recovery frontiersin.org. In fact, McPherson et al. observed that these lingering neural deficits can exist **even when standard strength tests show ~90% limb symmetry** at return-to-sport, potentially contributing to the high re-injury rates post-ACL frontiersin.org. Beyond re-injury, chronic AMI has longer-term consequences: it can accelerate **degenerative changes**. Reduced shock absorption and altered joint loading due to muscle inhibition are thought to contribute to early **osteoarthritic changes** in the joint cartilage pubmed.ncbi.nlm.nih.gov. Thus, the reflex inhibition mechanism not only hampers short-term function but can also influence long-term joint health.

Value of Neuromuscular Functional Testing (NMFT) vs. Imaging and Passive Exams

Limitations of Structural Imaging: Traditional diagnostic tools like X-rays or MRI are excellent for visualising structural damage (e.g. torn ligaments, bone lesions) but **cannot detect neuromuscular inhibition**. After a trauma, imaging might show that a ligament has healed or a surgical graft is intact, yet this reveals nothing about how well the patient's muscles are firing. Many patients are cleared by imaging or passive range-of-motion criteria, yet still harbour significant muscle activation deficits. For example, persistent quadriceps inhibition after knee injury is **"invisible" on an MRI** – the scan may look structurally normal while the patient still cannot fully activate the quad during dynamic tasks. This disconnect was highlighted by Sonnery-Cottet et al. (2022), who reported that **arthrogenic muscle inhibition often goes underrecognized** in standard exams, prompting them to propose a clinical grading scale for AMI based on the ability to volitionally contract the Vastus Medialis oblique (VMO) muscle sciencedirect.com. Such a scale (Grade 0 = normal VMO contraction; higher grades = VMO inhibition and atrophy) can reveal deficits that imaging or a cursory exam would miss. In short, structural imaging gives an incomplete picture of joint injury recovery because it **misses the functional status of the neuromuscular system**.

Active Neuromuscular Testing – A Superior Diagnostic Tool: To truly assess and treat joint injury consequences, clinicians are increasingly turning to **neuromuscular functional testing (NMFT)** and dynamic muscle exams. These include techniques like manual muscle testing (MMT) with careful observation of contraction quality, isokinetic strength assessments, reflex response tests (e.g. H-reflex measurements), electromyography (EMG), and functional movement analyses. Such tests directly evaluate whether the muscle can be activated appropriately and how it performs under load or in coordinated movement. Research supports that these functional tests have greater sensitivity to the residual effects of injury. For instance, Kim *et al*. (2024) used an **H-reflex ratio test** in patients with acute ankle sprains to quantify AMI in the soleus muscle pubmed.ncbi.nlm.nih.govpubmed.ncbi.nlm.nih.gov. They found the S**oleus H_max/M_max ratio was significantly depressed** (indicating reflex inhibition) in acutely sprained ankles compared to healthy controls, **even when the joint appeared otherwise intact** pubmed.ncbi.nlm.nih.gov. Importantly, the degree of Soleus inhibition correlated strongly with the patients' pain and self-reported ankle disability, but not with a standard static balance test pubmed.ncbi.nlm.nih.gov. This suggests that **dynamic neuromuscular testing uncovered deficits tied to patient function that a basic balance assessment did not**. In a rehabilitation context, such NMFT results guide clinicians to target specific muscles or neural pathways to restore proper function.

Guiding Treatment and Rehabilitation: Beyond diagnosis, neuromuscular tests provide prognostic and therapeutic guidance that imaging simply cannot. If a patient has persistent AMI, a therapist knows that traditional strengthening alone may not suffice; interventions must aim to *reactivate* the inhibited muscle (through techniques like neuromuscular electrical stimulation, cryotherapy to reduce joint swelling, reflex retraining, or motor imagery). For example, the presence of AMI may prompt use of modalities to down-regulate joint afferent signals (cryotherapy, TENS) or up-regulate muscle activation (biofeedback, electrical stimulation) pubmed.ncbi.nlm.nih.gov. Sonnery-Cottet *et al*. (2019) in a scoping review noted that treatments specifically addressing AMI – rather than just healing tissues – were critical for **improving quadriceps strength and functional outcomes after ACL reconstruction** pubmed.ncbi.nlm.nih.gov. In practice, **manual muscle testing (MMT) can be more informative than an MRI** at a follow-up visit: an MRI might show a well-healed graft, but an MMT revealing poor VMO firing or side-to-side strength asymmetry immediately signals that the patient is not ready for pivoting sports. Likewise, performance-based tests (single-leg squat quality, jump-landing mechanics, etc.) or specialized neuromuscular evaluations (like dynamic balance or perturbation response tests) can uncover deficits in coordination and muscle control that imaging and passive range tests miss. By examining *how* the body is functioning (not just its structure), clinicians obtain a superior roadmap for rehabilitation. They can tailor neuromuscular training to reverse the inhibition, thereby restoring joint stability and normal movement patterns.

In summary, **recent evidence strongly supports the primacy of neuromuscular functional assessment over structural imaging or passive observation in post-trauma care**. Joint injuries frequently induce reflexive muscle inhibition (AMI) that impairs strength and neuromuscular control around the joint pubmed.ncbi.nlm.nih.gov. These deficits have clear functional consequences – from muscle atrophy and weakness to altered

biomechanics and elevated re-injury risk pubmed.ncbi.nlm.nih.govfrontiersin.org – which imaging cannot capture. Clinical studies over the past five years consistently emphasise that evaluating muscle activation and functional performance is **crucial for accurate diagnosis and effective rehabilitation** pubmed.ncbi.nlm.nih.govfrontiersin.org. By focusing on neuromuscular function through tools like NMFT and MMT, healthcare providers can detect hidden problems, tailor interventions to restore proper muscle activity, and ultimately improve patient outcomes beyond what structural healing alone would predict.

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