Looking through the NeuroLens: The Missing Link in the everyday practice of MSK differential diagnosis and treatment - A White Paper

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Introduction

It's truly mind boggling. To look at the manner in which clinical examinations are taught to professions responsible for assessing MSK injuries, you would think the neural system is a remote contributor to the patient presentation at best, and completely irrelevant to the matter at hand at worst. The initial MSK assessment includes a quick reflex test and key muscle screen (or not), perhaps a Lasegue or ULTT test to see if the exact symptoms are reproduced (or not), then on we go to the joints and muscles and how people feel about their jobs and personal relationships, with barely a sideways glance at any finding that isn't grossly obvious. (Tawa et al, 2017, Bender et al, 2023)

With that in mind, it's no wonder that debate rages as to the best approach to patient care using the evidence we have available. We are effective in our interventions only some of the time, or we

get just part of the way to restoring the desired level of function and painfree existence that many people expect when they seek physiotherapy treatment.

Worse, especially for the newer physiotherapist without the benefit of context and experience to fall back on, they are left with making decisions based on research where evidence not only doesn't clearly support any intervention over another, but everything seems to work at least a little bit. Here, 'everything' describes a wide range of commonly taught and used interventions: pain education, manual therapy, general aerobic exercise, targeted strength exercise, global non specific strength programs, deadlifts, no deadlifts, sometimes with a rounded back, sometimes not. Most physiotherapists who have pushed through this mishmash of evidence and try to apply the haze of imperfect Clinical Practice Guidelines to the patient in front of them have a long list of those who should have responded to their intervention strategy based on the best available clinical recommendations but didn't.

This leaves the patient frustrated with ineffective or slow treatment and the physiotherapist aiming to be a good clinician similarly frustrated, likely even moreso than their client.

Here is who comes to see us in the clinic: the client with calcaneal pain diagnosed as 'plantar fasciitis' who doesn't have plantar fascia that is painful to the touch, but has all the rest of the signs and symptoms – morning pain, relief with a change in foot mechanics, and pain on stretch into DF with toe extension; someone with 'tennis elbow' describing pain that moves from the lateral to the medial epicondyle then back to the lateral side on a daily or weekly basis; the person with an imaged TFCC tear which was surgically repaired but they still suffer from the exact same pain as they did before surgery. Each of those is an example of a difficult case you may have seen in the clinic yourself and were frustrated with the lack of clarity on how to approach their treatment because their presentation doesn't quite make sense. These clients don't respond to treatment for plantar fasciitis/tennis elbow/TFCC post-surgical protocols the way you were taught they would.

Here is where we must pull up to a full stop. Refocus your analysis and look at these problems through the perspective of the function of the neural system. The neural system is lying behind and supporting all of the tissues you suspect as being the problem in these difficult clinical cases. It's behind the muscles and the fascia that envelops them. And the bones. And the joints with all their supporting ligaments. And it doesn't stop at anatomic structures – the neural

system is also behind the feelings and attitudes the biopsychosocial model emphasizes as being a key source of pain signal interpretation.

The neural system activates muscles with action potentials, is responsible for the coordination and grading of contractions, receives information from the joints of the body and all the tissues that cross that joint, including joint position and the amount of force being transmitted through the muscles and tendons, then sends this information through a spinal reflex arc that can protect you from harm even before a message gets to the brain, and has another system altogether to filter the collected input from a vast array of nerve fiber types where the brain ultimately decides how the body will move in response to that input.

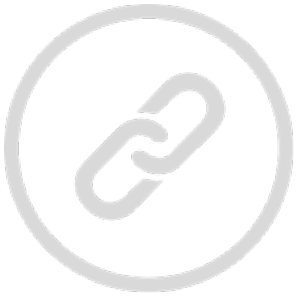
All of this is done with a complex network of structures that require blood supply and oxygen metabolism to produce the massive amount of energy required to support the activities of the neural system: production and transport of neurotransmitters, ion channels and receptors; the maintenance of the infrastructure of the nerve itself; the triggering of action potentials and subsequent repolarization to prepare for the next action potential; and healing areas of the nerve that have been damaged, just to name a few.

That's a massive amount of complexity. And described above is basically an executive summary of the neural system's activity in a simple reflex test. Now, imagine the demand on that system when it must physically contort to follow the positions of the body under load during high energy demand activities such as playing football or rugby, dancing or gymnastics.

Think about it again for a second. This system maintains the function of all the parts of the body; muscles, tendons, joints, organs, the chemical and hormonal balance in the blood, and even monitors and repairs itself. Yet, it is inadequately emphasized during the teaching of the clinical examination in most physiotherapy university curricula around the world and in the post-graduate training for physiotherapists working to develop and refine our craft in MSK assessment and treatment techniques.

The Missing Link

The neural system is everywhere and influences everything – how have we managed to ignore it? How have we, leading experts in diagnosing MSK dysfunction, (*Moore et al. 2005)* underrecognized such an important factor in our client presentation?



What follows is a list of contributing factors that have brought us here. It's not a complete list, but these points are either documented in the literature or self-evident to those who have practiced physiotherapy in a Western medicine model.

Physiotherapy curricula worldwide

The traditional pathoanatomical model of assessment and treatment taught to physiotherapy students has largely been based on the theories of James Cyriax, Freddy Kaltenborn and Geoffrey Maitland. These three have been the mainstays of orthopedic curriculum development for physiotherapy students all around the world. This is not to minimize the contributions of other giants of orthopedic manual therapy such as Robin McKenzie, Gregory Grieve, Brian Mulligan, Shirley Sahrmann and David Butler, but most physiotherapists aren't exposed to the concepts of these thinkers until they have graduated and are taking advanced post-graduate education courses.

From a basic training perspective, the physiotherapy curriculum is anchored in the thoughts of the first 3. Cyriax, an orthopedic surgeon who invented an elegant assessment system organizing the physical test into contractile and inert tissue categories in order to logically rule in or out the offensive or injured tissue to arrive at a diagnosis. Kaltenborn, a physiotherapist and osteopath

who further delved into the influence of inert tissue, using the emerging science of biomechanics to apply it to manual therapy, proposing a scientific model of osteokinematics and arthrokinematics based on the shape of the joint surfaces. Maitland, another physiotherapist who took this biomechanical application of force to joints and refined it further according to the need – grading his manual techniques at the low end for pain modulation, gradually increasing in force or duration to effect tissue mobilization/improving range of motion at a joint. With this mobilization grading system that ranges between 1 and 5, he also refined the assessment of joint endfeels and emphasized integrating client feedback on their level of pain into the treatment technique.

Here lies the groundwork for physiotherapy diagnosis and treatment. The history of the presenting problem is extracted from the client, a hypothesis is created, then tested by trying to isolate the tissue at fault. The Western approach to medical education, and therefore physiotherapy education, has been to deconstruct the body into systems, then break down the systems into their parts, the parts into their functions, then test those functions to very specifically identify a fault.

This system of assessment, which is deep, complex and detailed from an anatomical and logical perspective, considers the present structural health of the muscles, joints and ligaments. Done well, it captures and accurately identifies many MSK pathologies.

However, in this approach the nervous system generally gets only a rudimentary look – we test reflexes, sensation and key muscles. Once those are cleared for red flags, the consideration for the neural system as a contributor to the diagnosis is finished, critically overlooking the supply and maintenance function of the tissues we have just tested.

Death of the clinical exam and overreliance on imaging in medicine

The death of the clinical examination in medicine has been described for decades – alternately being dismissed as unimportant or the loss being lamented. The hallmark of expert practice

used to be the combination of a low-tech but careful physical examination combined with the accumulated personal and shared experience from peers. The development of laboratory tests and imaging technologies promised a new gold standard of diagnostic accuracy and a movement away from the quaint, low-tech visual and manual examination. At the same time, with health care expenses rising at an unsustainable pace and there being an unending demand for a physician's time, the thinking became 'what is the point in wasting time trying to figure out why someone's shoulder hurts if we can just get an ultrasound or MRI that will show us what the problem is?'

This 'advancement' in technology and clinical reasoning has resulted in important knock-on effects: a filtering down of the attitude that the clinical examination is unnecessary in because we can just take a picture of MSK pathologies; and the associated loss of nuance in the clinical examination over time. (*Jauhar S. 2006; Tanael M. 2021)*

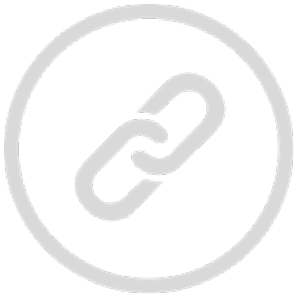
The critical flaw in applying technology and minimizing the importance of a clinical examination in favour of data is the difference between MSK conditions and medical conditions involving the organ systems. While imaging tests for organs (lung x-ray for pneumonia or cancerous tumours; kidney ultrasound for the presence of stones) or lab tests measuring blood sugar, creatinine, potassium or white cell counts are exceedingly accurate and can often be tightly tied to a clinical diagnosis, the imaging tests we have for diagnosing MSK conditions are painfully unreliable when it comes to diagnosing the presenting problem. *(Brinjikji et al 2015; Jensen et al 1994; Boden et al 1990; Englund et al 2008; Tempelhof et al, 1999; Stabler et al, 2001).* Further complicating the picture around the neural system, tests for the health of this organ are restricted to the base function of measuring action potential conduction. The EMG detects this well, but does not reflect the potential subclinical or suboptimal states of this system that occur with chronic constriction or ischemia.

Having someone present with positive imaging findings does not absolve the physiotherapist of doing their own thorough clinical exam, but despite the instruction to 'not treat the image', clients have a need to have their diagnosis acknowledged, and some physiotherapists are happy to treat the image because it's easy. Unfortunately, in many cases, it will also be ineffective.

On the other hand, we may have a person present with pain who has had imaging that did not reveal any structural damage or reasonable explanation for the origin of their pain. They are either

accused of malingering or told there isn't anything wrong with them, and dismissed from the system.

We as physiotherapists have a professional responsibility to cut through the messaging that is dominant in the health system and look more deeply at why these types of cases exist. Looking through the NeuroLens should make things clearer, whether or not the root of their symptoms is neurally mediated or influenced. If you never consider the impact of the neural system on someone's pain presentation, especially in cases that don't respond the way you expect, you are inviting a cycle of frustrating client interactions.



'Evidence Based Practice'

There has been a strong push for using the scientific method to rigorously test treatment interventions to ensure the best treatments are identified, the optimal dose of those treatments defined, the having the appropriate treatment doses applied to the appropriate patient at the appropriate time. This is an admirable goal, and certainly the gold standard to which all health care interventions should be held. Billions of dollars and years of time have gone into developing research to this end. To the misfortune of everyone involved, the clinical applicability of most MSK research is, shall we say, poor. *(Ratnani et al, 2023)* The scientific method doesn't lend itself well to the messy world of MSK pain, with its poor localization, its varied lengths of onset and severity, and potentially most critically, poorly grouped subjects, with the status of the neurological system almost never part of the inclusion/exclusion criteria. Trying to apply a standard treatment intervention to a wildly varied group of individuals and hoping to be able to come up with a solution for the best single intervention just doesn't work.

Let's look at this another way: Imagine trying to find the best shoe for everyone in the world with feet, using the classic scientific method. We will use a validated measurement scale for both

comfort and function. Our independent variable will be the type of shoe – a dress shoe, a flip flop, and a cushioned running shoe. Since the scientific method dictates there can only be one variable, everyone in the world, randomly allocated and matched, puts on a standard size of one of the above types of shoe. All subjects wear a size 8 for all of their activities, then rate how comfortable and functional they are. Imagine – all the mountain climbers, soccer players, dog walkers, and people living in extreme temperatures all putting on the same size of shoe, participating in their usual daily activities as widely varied as they are, then rating how they felt.

Here's the problem: some of these shoes were completely inappropriate for the activity they were being used for and would get a poor rating for comfort and function, even if they fit perfectly. Others would be perfect for the activity the subjects were participating in, but fit badly, so would also get low ratings. There is another group - a much smaller group, who have size 8 feet and wore the right shoe for the right environment and would give high comfort and function ratings for the shoe they had that was matched with the activity. But this data reflecting the best shoe for a particular activity would be statistically washed out in the data analysis.

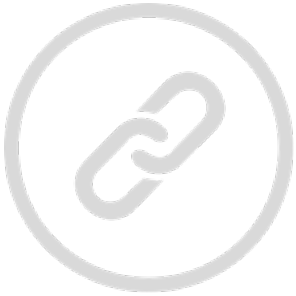
In the end of this study, the conclusion would show no statistically significant difference between the groups, therefore no shoe is better than any other – a blanket statement for all shoes at all times. We have the wrong shoe in the wrong size given to most of our subjects, and the conclusion is that no shoe is any better than any other, regardless of what the subject actually needed. Sounds ridiculous, doesn't it?

Unfortunately, that has been the quality of a lot of 'evidence' published around MSK intervention: there isn't enough strict inclusion and exclusion criteria; not enough subgrouping; not enough study of long-term vs short-term effects of treatment; and not enough consideration of the contextual factors that influence MSK pathology.

This includes a lack of consideration of the influence of the neural system contribution because in the interest of reaching a conclusion around the single best intervention for immediate change, system influencers like the neural system generally aren't considered. It is this manner of research that led the American College of Physicians and the American Pain Society to state in their CPG for acute low back pain that the only non-pharmacological intervention supported by research is applying superficial heat. (Chou R, *2007)*

Models of care evolution

There has been a massive advancement in the understanding of pain and pain processing. Thanks to researchers who have justified the physiological basis for centralization of pain, the biopsychosocial model originally proposed in 1977 has had a resurgence. This has resulted in serious implications for clinicians responsible for identifying a diagnosis. If the factors that underlie a client's pain are mostly psychological or social in nature, the therapist has very little control over those with their physiotherapy intervention, and therefore very little ability to directly create change in the condition. The clear risk with applying this model too widely to MSK disorders is that it absolves the clinician of careful biomechanical analysis of a presenting condition. (Cayrol et al, 2021) The absence of an accurate and detailed clinical examination (and dismissal by some of the importance of needing one at all) leaves clients who have a true biomechanical or structural basis to their condition frustrated at their lack of improvement. These people disappear, saying physiotherapy doesn't work for them, or they find a different clinician to address their problem.



Basic anatomic principles

If all of the above hasn't convinced you that you need to know more about the role of the neural system in common MSK disorders, let's go back to first principles thinking around the structure and function of this amazing organ system that is responsible for every sensation you feel and every movement you make.

- The neural system isn't a set of passive wires attached to your brain. It is an interconnected signaling network of billions of neurons that uses chemical, electrical and mechanical signaling to transmit information. (Low et al, 2006; Egri et al, 2012)
- This organ system is active and hungry. Your brain accounts for approximately 2% of your body mass, but this system uses 20% of the circulating glucose and about 20% of the oxygen . (Mergenthaler et al, 2014) That seems outrageous, so let's look deeper at some of what is happening in this system to consume so much energy:
- It is constantly transporting within itself – there are two anterograde systems with different functions and speeds, and a retrograde system of intraneural transport. (Butler, 1991)
- It creates its own infrastructure components such as microtubules and neurofilaments, sends them to areas that need maintenance and repair, then uses energy to perform that maintenance. (Butler, 1991)
- It builds its own chemicals (neurotransmitters), ion channels and transport vesicles, then sends them to synapses where they are essential parts of creating action potentials. (Butler, 1991)
- It brings used transmitter vesicles, neurite growth promoting factors and collects 'trophic messages' updating status of the environment via retrograde transport, which are energy intensive processes. (Butler, 1991)
- 75–80% of the energy consumed by individual neurons is used at the synapses to restore the neuronal membrane potentials lost during depolarization – these happen constantly, so disruptions to the system can have massive impact on a functioning body. (Butler, 1991)

- The PNS is not anatomically static, rather it undergoes a dynamic and active process of neuronal sprouting, rerouting and pruning according to the needs of the time. (Low et al, 2006)
- When considering bony sensitivity following fracture or other trauma, neuronal sprouting and retraction can play a part. For example, following standardized angular fractures to a femur, (Li et al, 2007) found that there was a greater density of CGRP sensitive nerves growing into the concave side of the fracture than the convex side. Once healing had progressed, they retracted back to the periosteum. This is a powerful example of how the neural system is

involved with a mechanism we had previously understood to be mediated by osteoblastic activity, but is in fact orchestrated by the neural system.

- In fact, bone cells express receptors for many of the neuronal messengers present in these skeletal nerve fibers, and activation of such receptors leads to profound effects on the activity of both osteoblasts and osteoclasts, strongly suggesting the existence of neuro-osteogenic or neuro-immuno-osteogenic interactions. (Abeynayake et al, 2021)
- There is a tight regulation of bone and marrow homeostasis modulated by the neural system. (Abeynayake et al, 2021)
- Scar tissue is real. Despite some physiotherapy related social media platforms that pretend otherwise, the basics of inflammation are irrefutable. Edema, fibroblasts and myofibroblasts create a network of essential cells for healing, but these can impede axoplasmic flow within the nerve structure and adhere to nerve tissue that is supposed to be able to move freely. (Butler, 1991; Shacklock, 2005)

The neural system in post-graduate courses

There are treatment approaches in the physiotherapy world that have placed the neural system at the forefront – David Butler, Michael Shacklock, Michel Coppieters in the world of neurodynamics; Diane Jacobs presenting the concept of dermoneuromodulation, and Chan Gunn's IMS technique based on the theory of denervation supersensitivity.

Each of these approaches look at the assessment and treatment of the neural system in a slightly different manner, but at their base, they are all working to optimize the function of this system in the management of pain disorders and MSK presentations.

Newer on the scene is the concept of neuroimmune reactions in pain states, the brain/gut axis, and more specifically to MSK disorders, the study of neuroinflammation and its effects on pain perception and MSK function. (Ren et al, 2010; Shraim et al, 2024)

With all of that, it is difficult to deny there should be a more robust look at whether the neural system is operating optimally, or if a suboptimal state exists that may be affecting your client's MSK presentation. This is what looking through the NeuroLens is all about. There needs to be a

shift in how we assess MSK disorders and in how we approach treatment. Not only of the MSK structures towards which we were initially directing our treatment, but the mechanical and/or physiological dysfunction of the neural organ system, and how including those considerations affect the rest of our treatment approach, our expectations for progress and pace of change in their disorder.

It sounds like a step too far, perhaps? Too radical a change in thinking?

Recall over the past 30 years, we have seen revolutions in MSK treatment – the concept that tendonitis is far less common than previously thought, and what had been believed to be an active inflammatory tendonitis is actually a tendinosis, featuring collagen disorganization, tissue death and no active inflammatory process at all. With this revolutionary change in understanding, the treatment for many tendon disorders changed from rest, antiinflammatory medication and corticosteroids to tissue mobilization, tissue loading and education on the robustness of the MSK system.

The biopsychosocial model of pain, which is now widely accepted (although perhaps too generously applied so as to relieve the physiotherapist of an expectation for detailed clinical reasoning) was not a consideration a few decades ago. The focus on MSK pain was strictly on the structures that must be damaged and painful, and if there was no tissue pathology evident through imaging or clinical examination, suffering people were dismissed as malingering or attention seekers. Now they receive robustly researched pain education, CBT, and are encouraged to be active in their recovery rather than dismissed as fakers and shoved out the door.

There are many more examples of massive shifts in thinking, but let's finish with this: in 1980, if you presented to your physician with an acute episode of back pain, you likely would have been prescribed 4 weeks of bed rest. Can you imagine that happening now? There are regular revolutions in thinking about how to manage MSK pain better than we did yesterday – recognizing and integrating the impact of the neural organ in MSK disorders should be the next one!

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