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Distinguishing sources of pain: Central vs peripheral mediation

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ABSTRACT

Research in chronic pain often fails to distinguish the pain source: central vs peripheral. In this review, I lay out a case for a greater consideration of a peripheral pain source, namely myofascial trigger points. Findings from our group are presented indicating that trigger points are alpha sympathetically innervated and are not directly related to the cholinergic neuro-muscular system. Treatment implications are discussed.

Keywords: Myofascial pain trigger points; sympathetic muscle innervation

INTRODUCTION

Chronic pain, although difficult to define, is now clearly recognized as a major health problem creating a world-wide burden.[1] It is useful to classify chronic pain as having a primarily central vs a peripheral etiology. Clear cut examples of centrally mediated pain are phantom limb pain, Fibromyalgia (allodynia and hyperalgesia), and perhaps Complex Regional Pain Syndromes. A great deal of work has been published to try and elucidate the neural, behavioral and cognitive aspects of centrally mediated pain. An excellent recent review tries to model brain plasticity in brain organization for these pain syndromes.[2]

"A large body of converging evidence suggests that chronic pain is not simply a temporal extension of acute pain but involves distinct mechanisms. The transition of acute pain into a chronic disorder involves activity dependent changes (that is, functional plasticity) at many different interconnected levels, ranging from the molecular to the network level, at several anatomical avenues in the nociceptive pathway.[3,4] This interconnectivity can explain why even small molecular changes, such as a single point mutation, can result in large changes at the behavioural or clinical levels that are caused by amplification along multiple scales of plasticity. Mechanisms involving functional plasticity have been studied extensively and have revealed a range of modulatory factors that change the sensory, emotional and cognitive components of pain (reviewed in.[5-8] However, recent data show that functional plasticity changes are accompanied by structural re-modelling and reorganization of synapses, cells and circuits that can also occur at various anatomical and temporal scales[8,9] thereby further adding complexity and a large dynamic range, and potentially accounting for the development of pain that extends over longer periods of time. Structural re-modelling of connections has not been studied as widely as functional plasticity, and it remains unclear whether it represents a cause or a consequence of chronic pain"[2](p.20).

While this perspective is clearly informative and relevant to pain syndromes involving allodynia or hyperalgesia, it may be missing a large number of chronic pain disorders that may have a peripheral source. In this paper, I would like to focus on myofascial or muscular pain, which appears to constitute the largest number of chronic pain patients[10,11] and may be relatively independent of the central sensitization discussed above.

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Our group has struggled with the lack of a clear mediational model connecting psychological stress and muscle pain (Diagram 1). There seem little doubt that psychological/emotional features play a role in pain, but other than postulating a central nervous system pain amplification system, little was known about this "mind to muscle connection". Naïve theories postulated that stress caused striate muscle groups to contract chronically, thus producing overuse and pain. The problem here is that there is no evidence that painful muscles have byproducts associated with pain, nor are they chronically contracted [based on surface electromyogram (EMG) studies]. There is some indication that a fascial contracture may occur, but this new line of research has not been widely recognized.[12] In fact, an exhaustive search for a peripheral biomarker associated with chronic pain, has turned up no meaningful answers.

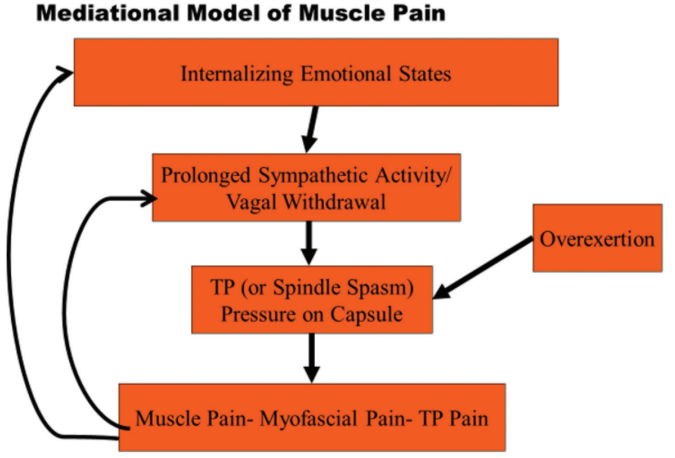

Diagram 1. Mediational model of Muscle pain
TP: Trigger point

Increasingly, pain from local nodules called trigger points has emerged as a promising source of muscle pain syndromes such as tension headache, neck pain, low back pain, pelvic floor pain, etc.

Shah et al.[13] provide a detailed historical perspective on myofascial or trigger point pain. While the concept of myofascial trigger points (MTrPs) can be traced back to Guillaume de Baillous (1538-1616) of France, the pioneering work of Janet Travell marks the coming of age of this pain conceptualization.[14,15] Travell et al.[15] catalogued the location of MTrPs throughout the body in great detail in two volumes of their handbook. Their exhaustive analyses are widely accepted as accurate. While the underlying physiology of MTrPs is quite controversial, a number of features are widely accepted: Associated stiffness

Localized point tenderness in muscle

Stimulation produces local and referred pain Often with a palpable taut band

Twitch

Trigger because like a gun trigger is initiated with pressure Produces pain in another place-(target).

While the myofascial perspective is now widely known as applying to tension headache, neck pain, upper body pain, lower back pain, etc., recent work has focused on pelvic floor pain. Jantos et al.[17] have described in detail a peripheral pain model that incorporates the myofascial perspective.[16-18]

Thus, MTrPs represent a peripheral source of pain, at least partially, and I would argue, substantially independent of central sensitization. For this reason, more emphasis on these pain syndromes is warranted.

MATERIALS AND METHODS

Mediational model

Our group began investigating MTrPs about 20 years ago. We began by trying to find a biological signature using surface electromyographic methods. This approach proved unfruitful. Needle electrodes were then proposed, but where should the needle be inserted in the muscle? A medical student (Dawn Bravata), working with us on various projects suggested a trigger point in the trapezius muscle. At that time Travell's work[19] was not well known nor accepted in medical circles, so we were quite skeptical at this suggestion. But in a rare fit of open-mindedness we tried inserting the needle into a tender nodule in the trapezius and—Eureka,—the needle EMG (nEMG) monitor lit up at a very specific locale. A second needle nearby in non-tender tissue remained electrically silent. Figure 1 shows a typical finding.

This method was then perfected and the first systematic study run and published.[19]Almost all of the subsequent studies used this methodology. We estimate that we have tested over 300 patients in this manner. With rare exception, we get the same pattern.

A series of pharmacological studies followed[20] that showed that the nEMG activity in the trigger point (TP) was unaffected by curare (a powerful cholinergic blocker that blocks all motor neuron activity), but dampened by phentolamine (an alpha-sympathetic blocker, see Figure 2). Unaffected by acetylcholine, the usual motor neuron neuro-transmitter, but blocked by a sympathetic blocker, this work seemed to offer a hypothesis about the pathways for sympathetically mediated peripheral pain in muscle.

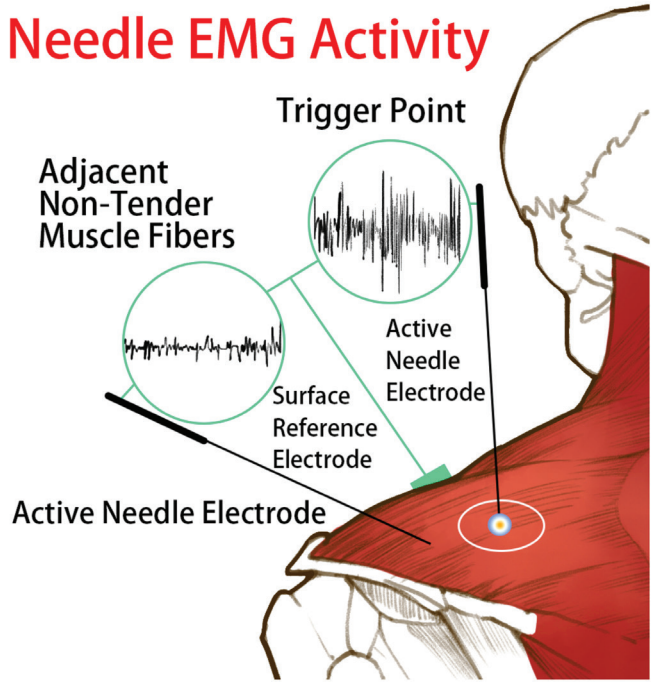

Figure 1. Needle EMG (nEMG) paradigm. Left circle shows activity in the non-tender site, right circle for the trigger point
EMG: Electromyogram

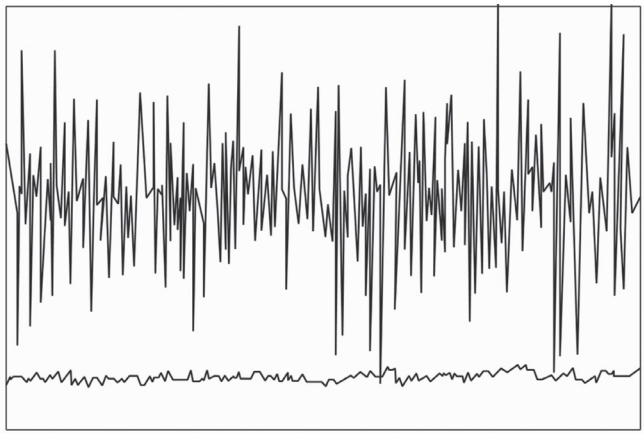

Figure 2. It shows the nEMG action potentials for Curare, lower is the non-tender adjacent site
nEMG: Neddle electromyogram

We then began a series of psychophysiological studies. My student, McNulty et al.[21], gathered subjects with palpable TPs and found that the TP, but not the adjacent site responded vigorously to psychological stress. As our theory predicted, the non-tender site remained silent during the stress. It seems that the muscle contraction we see in the clinic during stress can be inhibited with instruction (and perhaps the fear of moving a needle in the muscle). Another student, Banks[22] replicated the McNulty paradigm but added a relaxation component. TPs were clearly activated during stress but activation decreased during Autogenic Relaxation. Lewis et al.[23] found the same relationships existed in pain patients.

Since we were unsure of the nature of the psychological stimuli that would invoke a TP response, we then undertook a series of studies to elucidate the emotional stimuli. Gadler et al.[24] used interview techniques to try and elicit TP responses and found that during recall, very high nEMG activity was produced (up to 120 micro-volts). Professional actors were used by Cafaro[25] to see if dramatically expressed or inhibited emotions would produce higher TP activity. They didn't; it seems that recall of an emotional event drives the TPs at about the same rate as an angry or volatile outburst.

RESULTS

Based on these results, we postulate a mediational model of chronic muscle pain that yields potential treatment models. Figure 3 illustrates the broad model. It is hypothesized that chronic muscle pain syndromes result from activation of MTrPs by overstretch, emotional stress, or other sources that create a powerful afferent signal to pain perception centers in the brain. Thus, chronic sources of stress such as anxiety, worry, or other internalizing emotional states can fuel activity in the MTrP.

We have postulated that the MTrPs are actually muscle spindles (the intrafusal fibers within capsules responsible for stretch perception and regulation). This idea has been challenged, but is worthy of further exploration.[13]

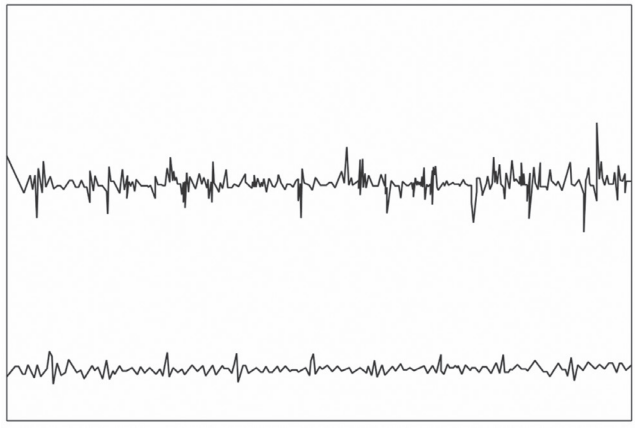

Figure 3. It shows the nEMG action potentials for Phentolamine, lower is the non-tender adjacent site
nEMG: Neddle electromyogram

DISCUSSION

Treatment models

It has long been recognized that techniques such as acupressure, massage, or dry needling are effective in reducing pain in MTrPs, at least temporarily (c.f.)[26, 27] Longer term relief has been a more elusive goal. Based on the above model, we have postulated that combining techniques that reduce sympathetic outflow with manual release techniques might offer longer term relief. One such technique, that has gained increasing support is Heart Rate Variability Biofeedback (HRVB).

In this protocol, patients learn to breathe at a specific rate diaphragmatically so as to greatly increase beat to beat heart rate accelerations during inhalation and decelerations during exhalations. Regular practice at this resonance frequency increases autonomic flexibility such that the vagus can limit sympathetic outflow to various target organs.[28-31] By combining this type biofeedback-based home practice technique, we have found that pain relief is prolonged dramatically, presumably because re-stimulation of the MTrPs is limited by better autonomic balance and a more robust parasympathetic (vagal) tone.[32-34]

The rapid response to the above treatment protocols seems to imply that central sensitization plays a relatively minor role in these syndromes, as compared to Fibromyalgia for example.

CONCLUSIONS

The growing recognition of the burden of chronic pain requires complex analyses that recognize the sources of pain. It is argued here, that recognizing the important role of peripheral sources of pain, specifically MTrPs, can greatly enhance treatment models so as to reduce pain, restore functionality, and reduce treatment costs.

DISCLOSURES

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