EDITORIAL

Changes in Brain Structure and Activation May Augment Abnormal Movement Patterns: An Emerging Challenge in Musculoskeletal Rehabilitation

The Concept of Central Changes Driving Maladaptive Movement Patterns

New research reveals key evidence that possibly explains why some, but not all, patients who sustain musculoskeletal (MSK) injuries go on to develop pain that persists long after the tissues have healed. Several recent publications used brain imaging technologies such as transcranial magnetic stimulation (TMS) and functional magnetic resonance imaging (fMRI) to demonstrate meaningful differences in brain structure and function between individuals with persistently painful MSK disorders compared with those in the asymptomatic population. These observations have led to the hypothesis that alterations in brain structure and activation are linked to the perseveration of abnormal movement and muscle activation patterns, that is, altered motor control [1–3]. Further exploration of this hypothesis may help us develop more effective interventions for MSK disorders.

This hypothesis is addressed by Te and co-authors in this month’s Pain Medicine article entitled “Primary Motor Cortex Is Altered in Persistent Patellofemoral Pain.” Using TMS, Te and colleagues identified that organizational changes (overlapping muscle activation regions and loss of number of discrete peaks of activation) in the primary motor cortex (M1) are associated with disordered quadriceps muscle firing in patients with persistent patellofemoral pain (PFP). The authors suggest that these brain changes in individuals with PFP are associated with a loss of the normal intermuscular coordination of activation, which contributes to adoption of more “simplified movement patterns,” resulting in the altered motor control that is often observed in this population.

Similar organizational changes in M1 have also been reported in other populations of patients with persistent pain related to MSK injuries. For example, in patients with chronic, recurrent low back pain, M1 reorganization occurred for trunk extensor muscles [4] and was associated with pain severity [2] and altered muscle activation timing [1]. In addition to activation changes, variations in brain structure, including reduced gray matter, have been observed in people with a wide array of MSK problems. These conditions include back and neck pain, patellofemoral pain, knee osteoarthritis, and anterior cruciate ligament (ACL) injury [5–8].

These intriguing studies suggest that the identification of changes within M1, and the linkage of their association with reduced function and persistent pain, may be a good first step toward developing new approaches to target those conditions associated with abnormal motor control. However, it is important to note that these findings in M1 organization currently only represent the change in the overall output from the motor cortex that controls movement. Evidence also suggests that M1 function is highly influenced by communication with other cortical regions including primary (S1) and secondary (S2) somatosensory cortices, premotor cortex, and subcortical regions (cerebellum) via the thalamus [9,10].

A good illustration of this interplay is the observation that injuries to peripheral neural tissues significantly change the representation of somatosensory information in the S1 and S2 cortical regions [9]. During recovery from these injuries, activation of S1 is necessary to restore normal movement [11]. Interestingly, practicing a motor task has been associated with “rewiring” of S1 regions activated during the task [12]. Together, these findings would suggest that somatosensory input to M1 is critically important to regaining normal motor control.

The variety of brain changes and the resulting movement dysfunction require us to explore models of how and why these changes evolve in some individuals but not in others. Hodges and Tucker (2011) presented one such hypothesis related to MSK pain in their paper “Moving Differently in Pain: A New Theory to Explain the Adaptation to Pain” [13]. According to Hodges and Tucker, the dominant model of persistent MSK pain focuses on the way in which peripheral drivers of persistent pain—such as specific biomechanical changes and altered somatosensory information—are associated with M1 changes. For example, MSK injury can result in immediate responses to pain within the motor system that are reflected as muscle activation and movement pattern alterations that may act to minimize the load on injured MSK tissues. However, the repetition of the adapted movements and muscle activation patterns over time results in neuroplastic changes represented as reorganization within the motor cortex. Following the histologic healing of originally injured MSK tissues, these altered movement and muscle activation patterns may linger. The unconscious (implicit) selection of these new maladaptive patterns may result in a cycle of persistent abnormal stress to MSK structures that leads to further injury and pain [9,10,13]. If left untreated, the repeated stress and injury from the maladaptive movements...
contributes to the transition to an ongoing chronic pain condition.

There is, however, growing consensus that the peripheral driver model (pain generated from injured peripheral tissues) does not account for all of the mechanisms that contribute to persistent MSK pain. A complementary model proposes additional involvement of central mechanisms, including central pain processing,* as well as an individual’s pain experience and beliefs about pain [10]. This complementary model focuses on the central mechanisms as the driver of persistent pain and has gained support as it has provided clinicians with additional treatment approaches [14].

An individual’s pain, or the sensations related to pain, can drive pain persistence and be associated with M1 changes. Evidence suggests that different cortical mechanisms can be responsible for this phenomenon [9,10]. For example, the pain experience can be modulated by spinal cord mechanisms, such as temporal summation, or be directly influenced via cognitive factors such as anxiety and hypervigilance regarding potentially painful movements. These potential “threats to the body” increase parietal cortex activation, which in turn facilitates the sensory experience of pain by exciting the primary sensory cortex. Over time, this facilitation can result in pain amplification or central sensitization that can drive abnormal movement and muscle activation patterns.

An individual’s beliefs about pain, or how an individual believes pain is caused and treated, can also drive pain persistence with evidence, again suggesting multiple underlying mechanisms [9,10]. One example of this cortical mechanism relates to the “attention switching” activity associated with fear avoidance of pain. Attention switching, which is under the control of the prefrontal cortex, could result in cognitive fatigue from constant inhibition of the awareness of the threat of potentially painful stimuli. This cognitive fatigue can result in loss or reduction of the prefrontal cortex control that influences sensori-motor integration by downplaying the role of sensory information in movement control.

Another potential mechanism, within the category of beliefs about pain, involves being in a state of depression, which can heighten the degree to which incoming sensory signals are interpreted as unpleasant and painful. Individuals with persistent pain can often become depressed as they cope with their pain. Depression and the stress-related components can react synergistically to alter pain processing.

A lesser-known model involves “sensory-motor conflict,” defined as incongruence between motor intention and movement associated with altered sensory cortex maps or poor sensory information integration. A failure to integrate sensory feedback into motor patterns may result in pain [15,16], damage to MSK structures, and/or abnormal biomechanics associated with development of abnormal movement and muscle activation patterns [10].

This mechanism includes damage to peripheral sensory/proprioceptive systems, resulting in inaccurate feedback as well as disrupted neural processing of sensory information in the cortical regions [17]. Although this model is well described within the literature relating to limb amputation, it has only recently been applied to altered motor control associated with MSK injury [10]. Finally, peripheral MSK tissue damage can result in pain signals interrupting normal sensory feedback. Over time, the interruption of normal sensory feedback by an “interference pattern” associated with pain may contribute to maladaptive motor control. This is particularly likely when combined with central mechanisms that impact sensory feedback by either 1) locally facilitating sensory information inappropriately (amplification or reweighting) or 2) globally through the unlinking of the communication between the sensory and motor systems.

The development of persistent pain is therefore most likely dependent on an interaction between varying degrees of peripheral and central mechanisms. In order to remediate persistent pain, a treatment approach integrating peripheral and central mechanisms is important, along with treatments that target maladaptive sensory motor communication. For example, unconscious (implicit) selection of maladaptive movement patterns encoded in memory are triggered in varying degrees by MSK injury (e.g., ACL tear, cartilage degradation, degenerative disc disease), residual altered somatosensory information (e.g., proprioceptive impairment or reweighting), changes in central pain processing, or emotional regulation results in clinical subgroups of persistent pain. Therefore, an assessment of these aspects and matched integration approaches is likely to be necessary to target treatment to the individual patient.

**Considerations for Musculoskeletal Rehabilitation**

By examining factors common to individuals who recover from MSKs without persistent pain, we might identify components that should be included in therapeutic strategies. In order to recover from MSK injury without the development of persistent pain, maladaptive movement and muscle activation patterns must be temporary and not predominate within the movement repertoire. This scenario may occur when 1) the tissue injury and pain are not severe or long-lasting; 2) the musculoskeletal system is generally healthy with an abundant and flexible movement repertoire; and 3) there exists adequate psychosocial support and reward for recovery.

Historically, interventions that address only the predominant model of peripheral drivers such as specific muscle strengthening exercise and locally applied pain reduction modalities have had limited success in the rehabilitation of people with persistent MSK pain. Traditionally, the targeting of the peripheral system has focused on the motor system (muscles), with less consideration to the somatosensory aspects (e.g., proprioceptive exercises, postural control challenges, recovery from perturbations). In addition, there is often failure to
move beyond “exercises” to focus on retraining both simple and complex functional movement patterns to restore appropriate and flexible movement strategies. Perhaps this approach has been less successful given potential sensorimotor conflict that can contribute to a patient’s difficulty in learning exercises and new movement patterns without direct intervention and intensive sensorimotor retraining [18]. As previously mentioned, practice of a motor task has been associated with expansion of S1 regions activated during the task and, importantly, may assist in improving the appropriate weighting of proprioceptive information in the region of the MSK injury. Thus, rehabilitation for painful MSK disorders may require specific intervention to address the sensory-motor conflict that hinders retraining of functional movement patterns. Suggested interventions include explicit training of sensory discrimination, practicing movement and muscle activation patterns while providing adequate feedback (e.g., visual or auditory cuing, sensory cuing) and reward (must occur simultaneously) with the execution of correct patterns.

Exciting new clinical research has investigated these models and suggests that cognitive-behavioral approaches to treatment (e.g., graded exposure, graded exercise, motor imagery, neurosciences of pain education) that target central mechanisms associated with fear avoidance, catastrophizing, and pain amplification may be valuable when interfaced with patients who present with motor control impairments associated with MSK disorders. The effect sizes reported in these studies are, however, modest, suggesting that this approach alone may not be the answer [19]. Our challenge is to improve the clinical utility of these interventions by enhancing the understanding of the linkages between central drivers of altered motor control and clinical manifestations of persistent MSK disorders. The exciting advances in brain imaging will greatly help us address this challenge.

*Central pain processing is used here to refer to brain processing of painful stimuli, rather than a brain injury resulting in a “central pain syndrome.”

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References


