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Clinical Commentary

The interaction between pain and movement

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ABSTRACT

Study Design: Clinical commentary.
Introduction/Purpose: Pain and movement are universally relevant phenomena that influence human experiences in readily observable ways. Improved understanding of pain-movement relationships can guide medical and rehabilitative approaches to recovery and decrease risk of dysfunctional long-term consequences of otherwise normal neuromuscular responses. Therefore, the overall intent of this article is to elucidate the relationships between pain and movement as they relate to clinical decision making.
Conclusions: Motor output is highly adaptable, can be influenced by multiple mechanisms at various levels along the nervous system, and may vary between individuals despite similar diagnoses. Therefore, interventions need to be individualized and consider both the types of motor response observed (ie, whether the response is protective or maladaptive), and the patient's acute physical activity tolerance when prescribing exercise/movement.
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Introduction

Pain and movement are universally relevant phenomena that impact human experiences in readily observable ways. Clinically, we observe that pain produces a large range of motor adaptations from subtle motor compensations during task completion to complete avoidance of painful movements and/or activities.[1] Although pain is a normal protective response to injury and potentially harmful stimuli, prolonged or dysfunctional neuromuscular adaptations in response to pain may contribute to disability and chronicity in a variety of pain conditions. Alternatively, movement (including global physical activity,[2-6] individualized exercise programs,[2,6-10] and specific motor learning/functional tasks[10-12]) is often prescribed to help decrease pain and restore/improve function. Understanding pain-movement relationships can guide medical and rehabilitative approaches to recovery and decrease risk of dysfunctional long-term consequences of otherwise normal neuromuscular responses. Thus, the overall intent of this article is to elucidate the relationships between pain and movement by discussing the primary pain-related motor control theories that guide interventions, observed relationships between movement and pain, and current evidence of neuromuscular adaptations in response to pain.

Impact of pain in the United States

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[13] According to the 2011 Institute of Medicine Report on Pain, more than 100 million Americans are affected by chronic pain resulting in $635 billion in annual health care costs.[14] Neuromuscular adaptations in response to muscle pain have been implicated in the transition from acute to chronic musculoskeletal

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pain conditions[15,16] and can lead to functional impairment and disability. Thus, an understanding of the interplay between pain and motor responses may facilitate improved medical and rehabilitative outcomes.

Pain-related neuromuscular adaptation hypotheses and theories

Clinically, pain is often the primary motivator for people to seek medical care, while simultaneously serving as a common barrier for adherence to prescribed movement[17,18] (eg, global exercise, individualized exercise programs, and/or specific motor learning/functional tasks). In addition, there is compelling evidence demonstrating various muscle changes related to pain/injury including atrophy, fatty infiltration, and fibrosis.[19] Currently, 3 primary hypotheses attempt to describe the relationships between movement/motor control and pain/injury. These hypotheses are described by Hodges[20] in more detail elsewhere but outlined briefly here: (1) suboptimal movement/tissue loading can lead to injury/pain,[21-23] (2) actual or perceived threat of injury/pain can impair or interfere with motor output at various levels of the neuromuscular system, and (3) altered movement patterns in response to injury/pain or the perceived threat of injury/pain serve to protect the affected body region (includes altered movement as a learned or conditioned response to pain with movement even in the absence of an ongoing painful stimulus). These concepts can overlap. For example, a neuromuscular adaptation to pain may serve to protect an injured/painful extremity, yet lead to suboptimal tissue loading and further pain/injury. Each of these pain-motor interactions has different implications for clinical management, which is discussed in more detail in Clinical Implications section later. Many studies have focused on suboptimal tissue loading as a result of motor patterns, ranging from overuse injuries (eg, carpal tunnel syndrome from work-related repetitive tasks) to traumatic injuries (eg, ulnar collateral ligament rupture at the elbow while pitching). Thus, we will instead focus on the motor responses to pain and the protective vs non-protective nature of these potential neuromuscular adaptations.

Four primary classes of theories currently exist to help explain the relationships between pain/nociception and common motor responses: (1) vicious cycle theory,[24,25] (2) strength inhibition theory (SIT), (3) pain adaptation theory,[26] and (4) protective response theory.[1,16,27-29] Because these theories can influence our clinical practice, advocacy, and educational efforts implicitly and explicitly, they warrant a brief review.

Vicious cycle theory

The vicious cycle theory (Fig. 1) proposes that pain will result in sustained increases in muscle activity (ie, muscle spasms) that result in ischemia and accumulation of metabolites that cyclically produced more pain and dysfunction.[24,25,30,31] This theory reasons that accumulation of metabolites stimulates group III (Aδ, thinly myelinated) and group IV (C, unmyelinated) afferent nociceptors (transmit pain information from peripheral nerve endings) which, via facilitated gamma motor neurons, lead to increased muscle spindle sensitivity and reflex-mediated muscle stiffness.[32] Trigger points,[33] temporomandibular disorder etiology in some people,[34] and some experimental animal-model evidence of transient increases in jaw muscle electromyography activity[35,36] are consistent with aspects of the vicious cycle theory. Furthermore, treatment of musculoskeletal pain with muscle relaxants fits this model, where breaking the muscle tension cycle may serve to treat the pain. However, major criticisms of this theory include evidence of variable muscle response in the human jaw with induced pain,[27,29] decreases as opposed to increases in muscle activity in some chronic pain conditions,[26] and evidence demonstrating decreased tension headache activity regardless of electromyography activity.[37]

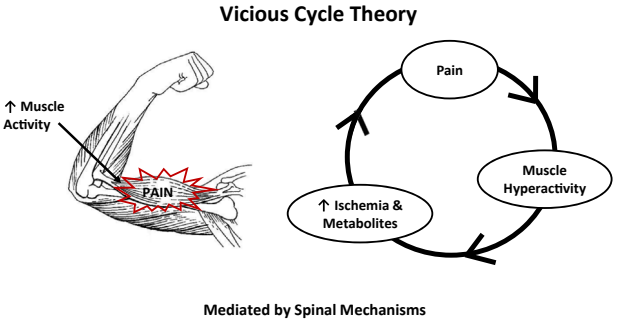

Fig. 1. The vicious cycle theory proposes that pain results in predictable increases in muscle activity regardless of task. Muscle hyperactivity in turn leads to ischemia and accumulation of metabolites, which then cyclically produces more pain. Anatomic arm graphic adapted from Wikimedia Foundation with permission from Pearson Scott Foresman.

Strength inhibition theory

Although not traditionally given a name, the theory that peak muscle force is inhibited by pain has been generally accepted, thus we refer to it simply as the SIT. Strength assessments may be considered invalid when in the presence of pain due to generalized inhibition. Evidence supporting this theory has been demonstrated with decreased peak torque generation for both knee flexion and extension after induction of experimental knee pain (infrapatellar fat pad injection of hypertonic saline) that largely recovers once the pain has resolved.[38] Thus, the SIT is in direct contrast to the vicious cycle theory.

Pain adaptation theory

The pain adaptation theory (Fig. 2) proposes both facilitated and inhibited motor responses depending on their relationship to the painful region, thereby combining to some degree both the vicious cycle theory and the SIT. That is, agonists (painful muscles and muscles that produce painful movements) will demonstrate decreased activity, whereas antagonist muscles (those opposing the painful muscle) will demonstrate increased muscle activity.[26] Lund et al[26] proposed that via a feedforward mechanism, nociceptive afferent input will converge on group II interneurons in the spinal cord and brain stem resulting in reduced muscle force; and decreased amplitude, velocity, and displacement of the painful part to prevent further pain/injury.

The pain adaptation theory is supported by evidence of reduced agonist muscle activity during voluntary jaw movement,[39,40] reduced motoneuron discharge rates during painful constant-force jaw contractions,[41] differential agonist/antagonist muscle activity at the trunk,[42,43] and with dynamic leg contractions.[44] However, the theory does not adequately explain many other findings, such as both excitatory and inhibitory postsynaptic potentials occurring in response to group III and IV muscle afferent activation in animal models.[45] Evidence demonstrating antagonist inhibition and agonist facilitation during jaw pain,[39] low back pain,[46,47] and arm pain[48] is in opposition to this model. Furthermore, the pain adaptation theory fails to explain the phenomenon of new motoneuron recruitment within a motoneuron pool during pain despite force maintenance,[49,50] and it may be challenging to

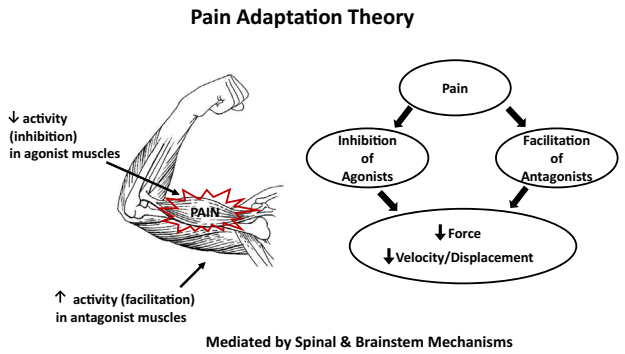

Fig. 2. The pain adaptation theory predicts inhibited muscle activity in painful agonist muscles with simultaneous excitation of nonpainful antagonist muscles. Anatomic arm graphic adapted from Wikimedia Foundation with permission from Pearson Scott Foresman.

define which muscles serve as agonist vs antagonist for nonmuscle pain origins, such as with generalized joint pain.

Protective response theory

This theory (Fig. 3) is an alternative, but inclusive, model, which attempts to describe and explain the observed variability in the neuromuscular system response to pain that cannot be fully explained by other theories.[1,27] Two authors have described similar tenets of this model, one referred to as the contemporary theory of motor adaptation in pain[1] and the other as integrated pain adaptation model.[28,29] Because they are very similar, we refer to this approach as the protective response theory, focusing largely on the contemporary theory of motor adaptation in pain model for brevity as it also encompasses the integrated pain adaptation model tenets. One of the theory's central premises is that short-term neuromuscular adaptations in response to pain serve to protect the painful or threatened body part, thus we will refer to it as the protective response theory for simplicity. This theory is different from the previous 3 theories in that it is not a direct pain-motor response theory per se but rather suggests that the unifying intention of any motor response to pain is that of protection. The theory asserts that injury, pain, or the threat of such can cause a wide range of motor behavior changes from subtle redistribution of activity within and between muscles to avoidance of movement. For example, it has been shown that during pain, a muscle can maintain force output through decruitment of some motor units and new recruitment of others,[50] which does not support a generalized facilitation or inhibition of muscle consistent with the other 3 models. The protective response theory argues that the changes in motor output (ie, changes in force and movement amplitude, changes in load distribution, etc.) can have real and/or perceived short-term benefits of protecting the affected body part from real or anticipated pain/injury.[16,27] However, this model acknowledges that these same adaptations, which may have short-term protective benefits, can also have dysfunctional long-term consequences that may decrease function and/or increase risk of

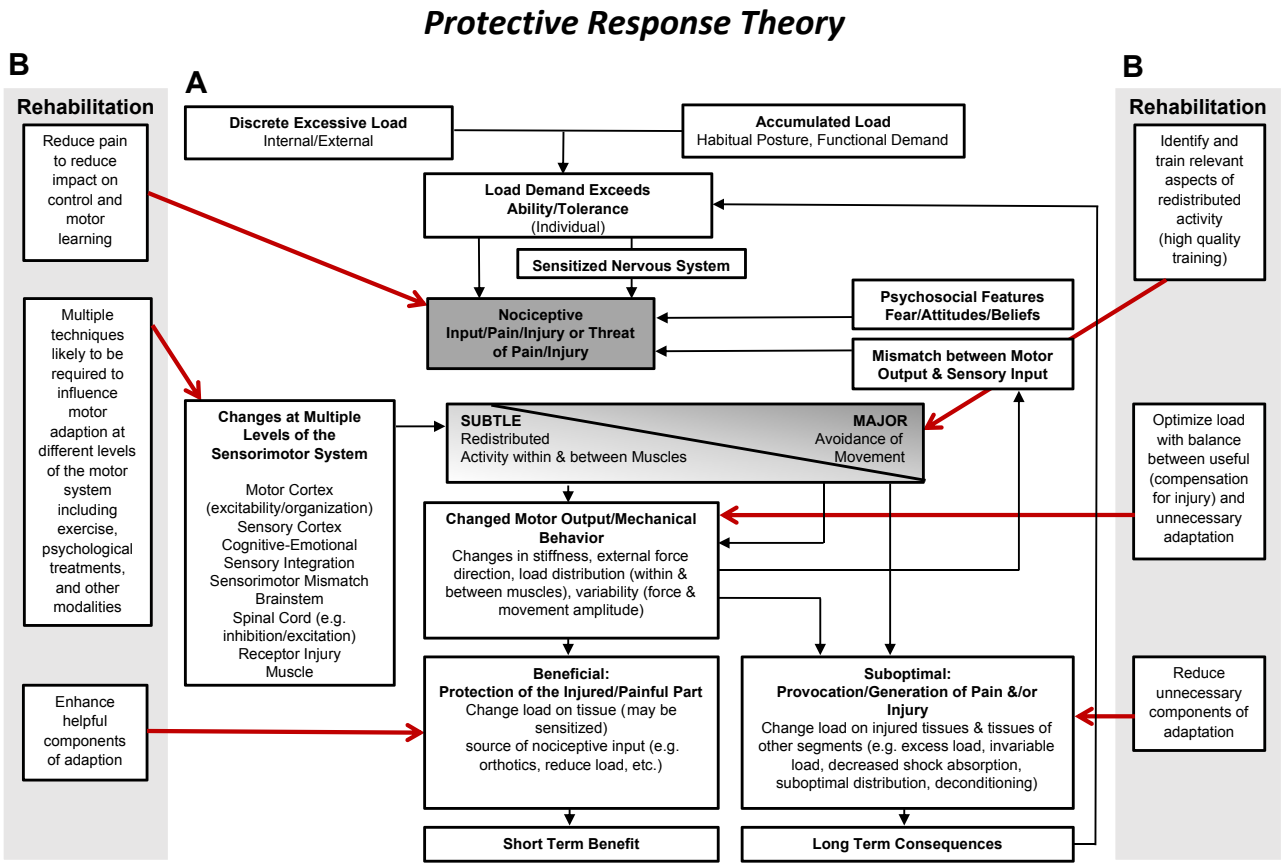

Fig. 3. Protective response theory with (**A**) motor adaptations to pain and (**B**) rehabilitative implications. Variable changes in motor responses can have both positive and dysfunctional outcomes that have implications for intervention. (**A**) With permission from Wolters Kluwer Health, Inc, from Hodges and Smeets.[1] (**B**) With permission from Elsevier from Hodges.[16]

further pain/injury. Furthermore, this theory concedes that biopsychosocial influences may help explain individual variability in motor behaviors, potentially affecting neuromuscular responses at multiple levels of the nervous system. The variability of neuromuscular responses to pain differentially impacts activity and function[1] and has important rehabilitative implications. These concepts provide an additional platform on which to consider and/or investigate nonuniform neuromuscular adaptations to pain.

Reflexive neuromuscular adaptations to pain

Despite the realization that transition from acute to chronic pain states is often accompanied by changes in motor activation patterns,[51] the high variability in neuromuscular adaptations in response to pain makes identifying dysfunctional adaptations and their corresponding interventions elusive. Reflexes are one type of motor response that can change in the presence of pain. Reflexes are present throughout the life span and are foundational components of, and integrated into, voluntary movements. The nociceptive withdrawal reflex (NWR) is of particular interest because it evokes an efficient coordinated limb withdrawal in response to painful stimuli, thereby inherently linking the pain and motor symptoms. Facilitated NWR responses in the lower extremity have been suggested to reflect central nervous system hyperexcitability in many chronic musculoskeletal pain populations.[52] For example, those with knee osteoarthritis have shown facilitated NWR responses when compared with those without knee osteoarthritis.[53,54] Consistent with our growing understanding of the complexity of the neuromuscular system, NWR activity is highly adaptable and influenced by a variety of factors not limited to but including task,[55] pain,[56] movement,[57] and muscle,[57] sex (males have higher thresholds), age (adults have higher thresholds), inactivity and obesity (lower thresholds), and pharmacology.[58]

Specific exercise/movement regimens and pain

It is difficult to identify specific evidence-based individualized exercise programs or motor learning/functional tasks that definitively decrease pain. However, a number of studies provide evidence for the effectiveness of exercise in general for the treatment of pain.[59-62] For example, moderate quality evidence supports the use of scapulothoracic and upper extremity strengthening and endurance regimens for immediate post-treatment pain relief for those with mechanical neck pain.[63] Similarly, static-dynamic cervicoscapulothoracic strengthening and endurance exercises can improve pain and function at long-term follow-up for those with chronic cervicogenic headache.[63] Furthermore, a review of 12 studies concluded that including eccentric exercise as part of a multimodal treatment program is effective for decreasing lateral epicondylitis symptoms and improving function.[64] Despite improving methodology and quality of evidence, many questions remain regarding individualizing exercise/movement interventions for those with various pain conditions. Therefore, for those with chronic pain conditions, current evidence suggests that ongoing client education and a client-centric approach to interpreting dose-response relationships to movement interventions may be necessary.

Integrated motor control treatment model

One approach to personalizing nonpharmacologic rehabilitation for patients with pain follows the integrated motor control model, described recently by Chimenti et al.[65] This model integrates the 4 motor control-pain response theories described previously as well as considerations of the protective nature of any motor adaptations, posed as 4 questions for clinicians to consider when developing a treatment plan (Fig. 4). Depending on each patient's individualized assessment, the clinician may be better able to address motor adaptations that may be maladaptive and nonprotective (eg, reducing spasms or activating inhibited muscles), volitional vs reflexive (may require different treatment strategies to address), and whether suboptimal tissue loading results from these motor adaptations.

Acute vs chronic effects of physical activity on pain

Being routinely physically active has many health benefits, including decreased risk of chronic disease and disability for a wide range of conditions.[66-69] Similarly, European epidemiologic studies support that those engaging in regular physical activity is associated with less frequent back pain[70] and reduced incidence of musculoskeletal pain,[71,72] suggesting the benefits of physical activity on preventing pain conditions. Furthermore, pain sensitivity assessments in healthy adults have been shown to be similar or

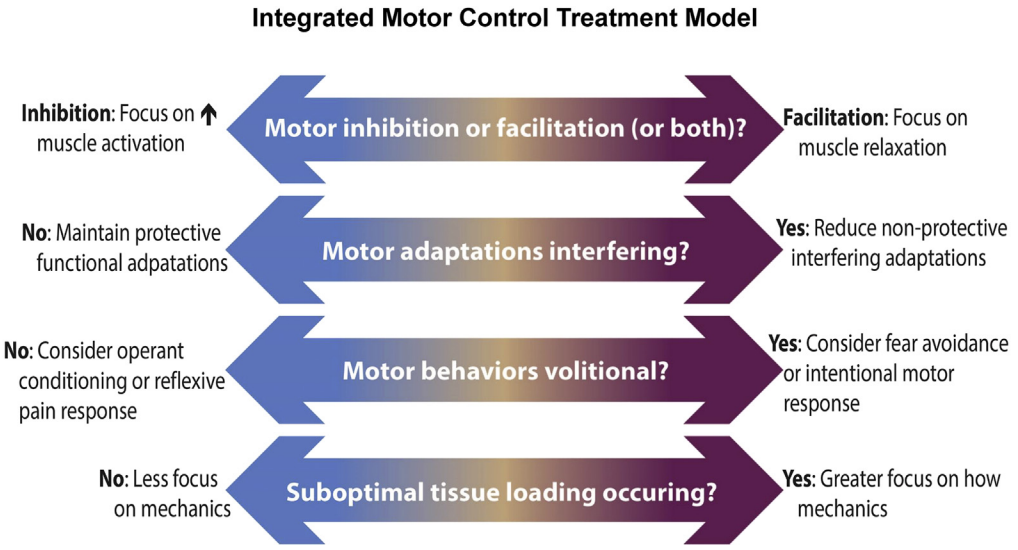

Fig. 4. The integrated motor control treatment model suggests considering these 4 questions when determining optimal motor control interventions for patients with pain. With permission from Chimenti et al.[65]

reduced in those participating in vigorous physical activity on a regular basis.[73] Thus, physical activity does not always result in improved pain sensitivity in healthy adults, but greater pain sensitivity has never been observed.

However, although even a single acute exercise session (ie, aerobic, isometric, and dynamic resistance) can systemically reduce experimental pain in healthy adults,[74] those with chronic pain often have mixed responses to acute exercise.[75-77] Whereas, routine exercise or physical activity is associated with improved function and decreased pain in several chronic pain conditions.[60-62,78-81] Animal studies similarly show acute increases in pain sensitivity after acute exercise,[82] yet the prevention of chronic pain development after regular physical activity.[83] This acute exacerbation of pain when first beginning an exercise program can be a barrier to treatment and should be differentiated from the long-term chronic adaptations that provide the greatest benefit to patient populations. An analogy that may be helpful to explain this normal yet disparate pain response to exercise to patients is that of exercise and heart disease. Acutely, exercise can be stressful and induce ischemia that may cause angina, yet if progressed slowly and continued regularly, the heart will strengthen, resulting in improved cardiorespiratory health and ultimately less angina with activity. Using this analogy, patients may gain an appreciation for the need to gradually increase their physical activity, with the goal of eventually reaching recommended guidelines of 30 minutes of moderate physical activity per day, 5 days per week, plus 2 days of strengthening. Therefore, distinguishing between acute responses to exercise and the benefits associated with routine exercise/physical activity when prescribing interventions for various pain conditions is necessary.

Clinical implications

Motor output is highly adaptable, can be influenced by multiple mechanisms at various levels along the nervous system, and may vary between individuals despite similar diagnoses. For example, the pain mechanisms influencing the perception of pain in a person with an acute musculoskeletal injury varies significantly compared with a person experiencing chronic regional pain syndrome with the former influenced more heavily by peripheral mechanisms and the latter influenced more heavily by central mechanisms. Furthermore, what may begin as a protective motor response after acute injury (eg, disuse after trauma) may transition into a maladaptive motor response (eg, continued disuse despite peripheral tissue healing). Therefore, interventions need to be individualized and consider the type of motor response observed (ie, whether the response is protective or maladaptive) and the patient's acute physical activity tolerance when prescribing exercise. For example, if suboptimal loading occurs as a result of shortened tissues from prolonged muscle spasm, stiffness, and guarding to protect an injured joint, then an intervention may need to initially target muscle relaxation and pain-free movement experiences. This may be achieved in a number of ways, from graded activities/movements in parallel with analgesic techniques, virtual movement/mirror therapy, to providing external forms of protection if necessary (eg, orthotics) allowing a more normalized motor pattern to emerge. Over time, treatment may progress to include strategies that retrain muscle activation patterns to achieve more optimal tissue loading, strengthening exercises targeting functional weakness, and/or generalized physical activity targeting reduced pain sensitivity through central mechanisms.

Conclusions

Although it is accepted that neuromuscular adaptations can occur in response to pain, researchers are only beginning to learn of the significance and extent of those adaptations. Furthermore, a preponderance of studies involves the lower extremity and trunk musculature, with fewer specifically investigating upper extremity motor adaptations. Pain can produce a large range of movement changes from subtle motor compensations during task completion to muscle spasm to complete avoidance of painful movements and/or activities.[1] Clinicians and researchers do not yet know the most effective intervention strategies to prevent dysfunctional long-term consequences of movement changes that result from pain. Improving methodology and quality of evidence (including controlling for potentially confounding demographic and psychosocial variables) should facilitate greater intervention efficacy. In addition, future studies addressing the influence of pain on reflexive and volitional motor control of the upper extremity will help elucidate these concepts. Improved understanding in these areas will lead to improved strategies for treatment of acute and chronic pain conditions.

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