

Interaction Between Pain, Movement, and Physical Activity

Short-term Benefits, Long-term Consequences, and Targets for Treatment

Paul W. Hodges, PhD, MedDr, DSc, BPhy(Hons)*
and Rob J. Smeets, MD, PhD†‡

Abstract: Movement is changed in pain. This presents across a spectrum from subtle changes in the manner in which a task is completed to complete avoidance of a function and could be both a cause and effect of pain/nociceptive input and/or injury. Movement, in a variety of forms, is also recommended as a component of treatment to aid the recovery in many pain syndromes. Some argue it may not be sufficient to simply increase activity, whereas others defend a necessity to consider how a person moves. There is unlikely to be a simple relationship between pain and movement, as both too little and too much movement could be suboptimal for the health of the tissues. The interaction between pain, (re)injury, and movement is surprisingly unclear. Traditional theories to explain adaptation in the motor system in pain are unable to account for the variability observed in laboratory and clinical practice. New theories are required. Treatments that focus on physical activity and exercise are the cornerstone of management of many pain conditions, but the effect sizes are modest. There is limited consensus when, if, and how interventions may be individualized and combined. The aim of this narrative review was to present current understanding of the interaction between movement and pain; as a cause or effect of pain, and in terms of the role of movement (physical activity and exercise) in recovery of pain and restoration of function.

Key Words: pain, physical activity, movement, motor control, adaptation

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Movement changes in pain across a spectrum from subtle changes in muscle coordination to complete avoidance of a function (Fig. 1). Movement in a variety of forms is recommended as a treatment to aid recovery in all kinds of pain syndromes, and this can range from encouraging regular physical activity^{1,2} to specific exercise approaches that modify how people complete a task.³ Although these issues seem straight forward, the reality is far from clear.

It is undeniable that a person with pain uses his/her body differently, yet the underlying mechanisms and their

complexity are only beginning to be understood.^{4,5} Important questions continue to be debated. Is movement “inhibited” as a simple mechanism to avoid further pain and injury? Is avoidance of movement driven by cognitive learning processes that can be changed? Are movement changes beneficial in the short- and long-term or are they part of the problem? Current theories cannot answer these questions or explain the diversity of changes observed in clinical and experimental contexts.

As a component of management of the person in pain it is generally accepted that maintenance or resumption of activity is important, at least to reduce disability and increase quality of life.⁶ Clinical trials,⁷ systematic reviews,^{8,9} and clinical practice guidelines¹⁰ all recommend that advice to remain or become active is better than advice to rest for management of acute and chronic back pain. Exercise is considered to have an analgesic effect^{11,12} and prevent development of chronic pain.¹³ When pain persists and becomes chronic, cognitive-behavioral approaches are advocated that use graded exposure to physical activity while challenging cognitions that cause avoidance of movement and activities; or operant conditioning techniques to encourage and reward gradual increase of physical activity and discourage attention to pain. But is it that simple? Recent evidence indicates exercise-induced analgesia may be absent in musculoskeletal pain as a result of central sensitization, and exercise can increase pain.¹⁴ Some argue it may not be sufficient to simply increase activity, but is it necessary to also consider *how* a person moves?¹⁵ There is unlikely to be a simple relationship between pain and activity as both too little and too much movement could be suboptimal for tissue health.^{16,17} The effectiveness of active treatments is likely to depend on the individual and are unlikely to be optimal if implemented in a generic manner without consideration of the individual. The scope of issues that have an impact on the application of exercise approaches to pain management must be explicitly explored.

The aim of this narrative review was to consider the contemporary view of how movement changes in pain; the potential benefits and consequences of these changes in the short-term and long-term; and the role of movement and physical activity in recovery from pain and restoration of function. Spinal pain is drawn on as an example as it is associated with a wealth of research investigation, but has far from ideal clinical outcomes.

HOW IS MOVEMENT CHANGED IN PAIN?

When pain is acute the nervous system takes action to remove real or anticipated threat to the tissues and this can be achieved by motor output. A limb may be withdrawn by

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From the *The University of Queensland, NHMRC Centre of Clinical Research Excellence in Spinal Pain, Injury and Health, Brisbane, Qld, Australia; †Department of Rehabilitation Medicine, Maastricht University, School for Public Health and Primary Care, CAPHRI, Maastricht; and ‡Adelante Centre of Expertise in Rehabilitation, Hoensbroek, The Netherlands.

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Reprints: Paul W. Hodges, PhD, MedDr, DSc, BPhy(Hons), School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane, Qld 4072, Australia (e-mail: p.hodges@uq.edu.au).

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simple nociceptive reflex mechanisms from the source of threat to the tissues; muscle activation may be modified to splint the painful part¹⁸; muscle activation may be “inhibited” to reduce the amplitude or velocity of a painful movement¹⁹; or an activity involving the painful area (in)directly may be avoided²⁰ (Fig. 1). When pain is chronic the motor system changes and its relevance for “protection” of tissues is less clear. Although the presence of changes in motor control is undeniable, the explanation for how and why it occurs and its relevance for ongoing pain remain debated.⁴ Although older theories of motor adaptation in pain provide some understanding^{21,22} they do not explain the complexity of response identified in clinical and experimental contexts.

Features of the Motor Adaptation to Pain and Limitations of the Existing Theories

Early physiological theories argued for simple stereotypical changes in motor output in response to nociceptive input.^{21,22} The “vicious-cycle” theory advocated increased muscle activity (spasm) and subsequent stimulation of nociceptive afferents by accumulation of metabolites secondary to ischemia induced by muscle contraction.^{22,23} The failure to observe these changes universally led to the proposal of more versatile, but still predictable, “pain adaptation” theory that predicted reduced activation (inhibition) of agonist muscles and increased activation (facilitation) of antagonist muscles.²¹ Such changes were observed during simple voluntary movements and force-controlled tasks of one or few joints,²⁴ and were related to an objective to reduced amplitude, velocity, or force of a painful movement. These theories explained some subtle deviations in muscle coordination (Fig. 1) using simple brain stem and spinal cord mechanisms.²¹ Animal studies revealed changes in excitability of spinal motoneurons that could underpin some of these effects.²⁵

In parallel, other theories aimed to explain changes at the other end of the spectrum (Fig. 1) and proposed cognitive-emotional mechanisms for complete or relative avoidance of motor functions. It was argued that movement and function are avoided (kinesiophobia) as a result of fear of pain or (re)injury, mediated by catastrophizing thoughts about the pain experience and hypervigilance

toward painful sensations, with the net outcome of disuse and disability.²⁰ Although this is supported by evidence²⁶ and is relevant for subgroups in the pain population,²⁷ it cannot and does not attempt to explain more subtle deviations in motor behavior and changes in individuals who do not express avoidant behaviors.²⁸

There is a void between existing theories and the spectrum of presentations. Several issues require consideration. First, clinical conditions are rarely typified by stereotypical and systematic changes in muscle activation.^{4,29} Analysis of more complex systems such as the trunk, which involves many muscles and multiple possible solutions,¹⁸ and complex tasks that involve multijoint movements and postural challenges^{30,31} has revealed non-stereotypical changes in control that vary between individuals. New theories must explain this variability and the interaction with function. Second, although early theories proposed spinal and brain stem mechanisms, motor control also changes when pain/nociception is anticipated in the absence of peripheral nociceptive input.^{26,32–35} This implies a “top down” rather than “bottom up” mechanism. Third, although cognitive-emotional mechanisms account for “top-down” effects in some individuals, they do not attempt to explain subtle changes in motor control or changes in individuals without negative cognitions. A comprehensive understanding of motor adaptation to pain must account for changes across the spectrum. Fourth, theories should account for the possibility that changes in movement may precede and contribute, at least in part, to the development and maintenance of pain and/or injury. Fifth, models must recognize the different types of pain (eg, nociceptive, neuropathic, and centrally maintained pain³⁶) and pain presentations from highly disabled individuals to those who maintain high level function with occasional recurring episodes of pain.^{37,38} The relationship between movement and pain is likely to differ among the heterogeneous population with pain. Finally, theories must dismiss the notion of a simple relationship to nociceptive stimulation and consider the subjective experience of pain. Although early theories may not be wrong, they fail to explain the diversity of adaptation identified in the presence of pain. New models can be inclusive of the existing theories, but must account for the issues presented above.

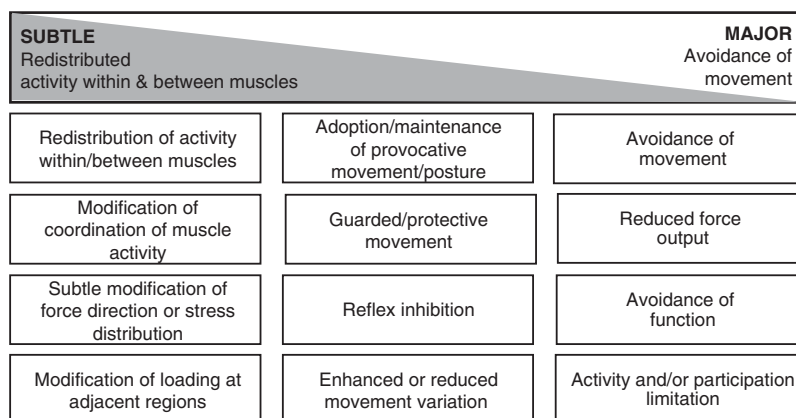


FIGURE 1. Spectrum of changes in motor behavior expressed by people in pain. Motor adaptation can present in many phenotypes, from subtle changes in the manner that a task is completed, to complete avoidance of movement/function, with varying impacts on activity and participation and potentially important implications for selection of treatments that involve movement (physical activity/exercise).

Contemporary Theory of Movement and Pain

Contemporary theories of motor adaptation to injury/pain or the threat of these argue that the motor system adaptation: (1) may precede or follow the onset of pain/injury; (2) involves a diversity of changes from subtle redistribution of activity within and between muscles to complete or relative avoidance of movement; (3) is specific to the individual and perhaps the task, and this may be influenced by a range of issues including psychosocial features; (4) has a general aim, at least in the short term, to protect the painful or threatened body part from real or anticipated further pain or injury and has “real” or perceived short-term benefit; (5) has potential long-term consequences if it is maintained, excessive, or inappropriate but the relationship between the motor adaptation and further pain/injury depends on the underpinning mechanism for persistence of symptoms; and (6) is underpinned by multiple mechanisms at various levels of the nervous system that are influenced by biological, psychological, and social aspects of pain. The components of the contemporary theory of motor system adaptation in pain are presented in Figure 2 and require consideration in greater detail.

Movement Adaptation May Follow or Precede Pain/Injury

Adaptation in motor behavior in pain may be: (1) a reaction to nociceptor input/pain or injury; (2) a reaction to the threat of these; or (3) secondary to some tertiary factor and contribute to the development of pain. In an acute pain episode motor function may adapt in response to an acute noxious input from a chemical, thermal, or mechanical stimulus. Abnormal mechanical tissue loading or injury that ultimately leads to nociceptor stimulation can result from a sudden large amplitude load such as in whiplash injury,³⁹ a repetitive or sustained low-level load,⁴⁰ or a combination. There is a complex relationship between loading, dosage of exposure, and injury, and why some people become injured, whereas others do not.⁴¹ The motor system adapts quickly and may do so as a process of trial and error as an optimal solution is searched for.⁴²

Alternatively, a motor behavior may be suboptimal, leading to abnormal tissue loading and injury that ultimately stimulates nociceptors. Factors that could lead to suboptimal motor behavior as a precursor to pain include habitual postures or movement patterns such as early

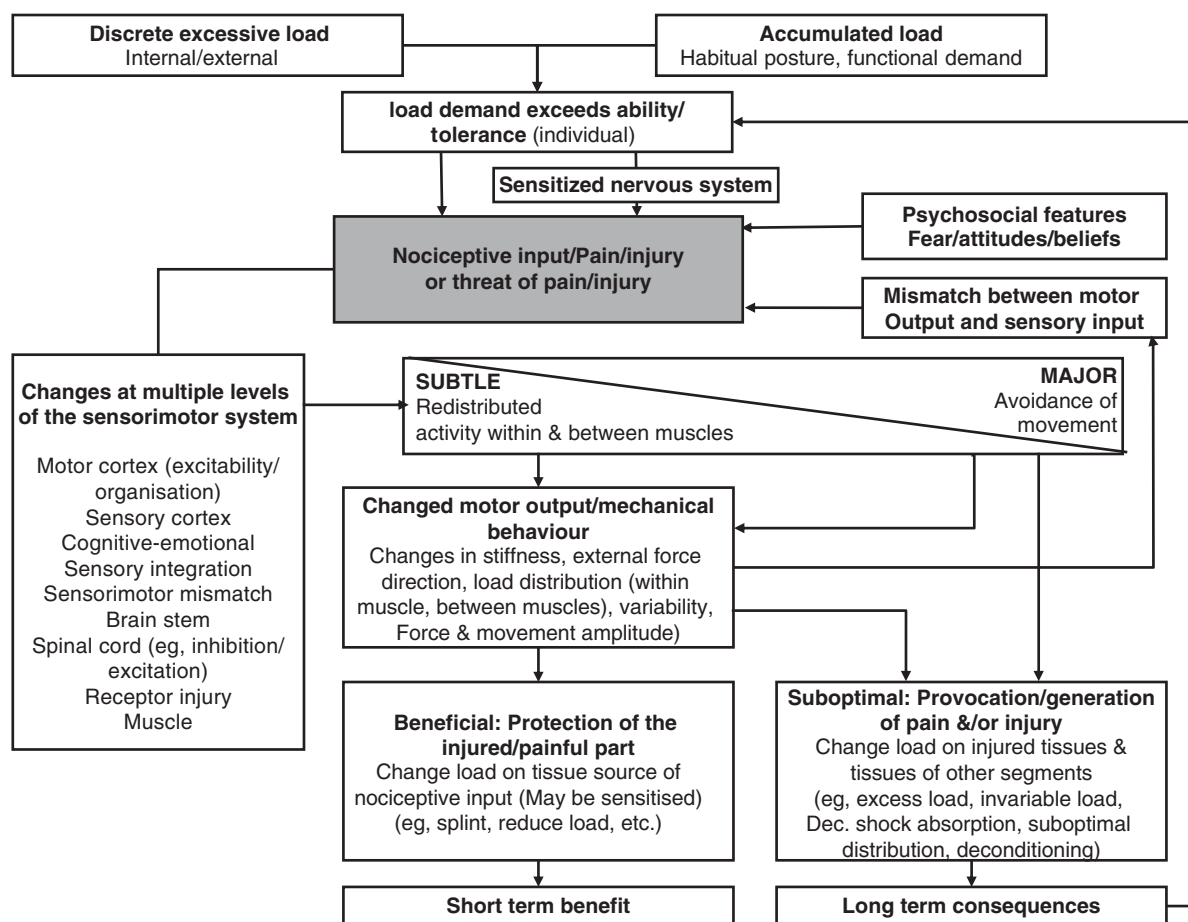


FIGURE 2. Contemporary theory of motor adaptation in pain. Changes in the motor system across the spectrum from subtle modification to avoidance of function can be both a cause and effect of injury, nociceptive input, and/or pain. Using a range of mechanisms at multiple regions in the distributed sensory and motor systems, motor control is modified and may present both positive and negative outcomes for the individual. Changes are variable and individual specific. When, if, and how movement should be addressed to influence this cycle is a topic of ongoing debate, but will require consideration of the multiple underlying mechanisms.

movement of the spine during hip rotation,⁴³ or competing functions of the muscle system such as competition between roles of the trunk muscles for spine control and breathing in respiratory disorders.⁴⁴

Regardless of whether the modified movement behavior was the *cause* or *effect* of an initial tissue injury/pain or threat of these, suboptimal tissue loading could be relevant for the ongoing health of the tissues. This may drive a peripheral contribution to the pain cycle, particularly in the presence of peripheral and central sensitization (see below).

Movement Adaptation Involves a Spectrum of Presentations

Although motor system adaptations in pain is diverse and ranges from complete avoidance of a task to redistribution of activity within and/or between muscles, the overarching purpose may be a universal objective to protect the painful part from further pain or injury, at least in the acute phase. There are numerous examples of complete or relative avoidance of movement, activity, and participation, such as avoidance of flexion to end range,⁴⁵ avoidance of sitting, and avoidance of work or sport, respectively.

More subtle motor adaptations include redistribution of activity between and within muscles,¹⁸ which may occur in a manner that either sustains the motor output such that the goal of the task is maintained, but with modified distribution of load in the tissues, or fails to maintain motor output. There are numerous examples of each. In the case of the former, force may be maintained, but with a different mechanics. For instance the distribution of force may be modified by redistribution of activation of motor units within a muscle,⁴⁶ altered activation of synergist muscles,¹⁸ or redistribution of load between limbs. Alternatively, the output might not be sustained. Reduced gait speed⁴⁷ and decreased capacity to maintain maximal force⁴⁸ are both examples. These subtle adaptations may not prevent activity and participation, but instead change mechanics in a manner that has short-term benefit. There may be long-term consequences.

There is a potential conflict between protecting the painful part from further pain or injury at the one hand, and realizing current activities in daily life despite pain in order to protect important life goals. These conflicts are conceptualized within the avoidance-endurance⁴⁹ and goal pursuit⁵⁰ model of pain.

Movement Adaptation is Individual Specific

Older theories of adaptation to pain and injury predicted stereotypical changes in motor output. Although some stereotypical changes have been observed, such as reduced masseter muscle activity during acute experimental pain,¹⁹ this is not universal. For instance, when pain is induced experimentally in the low back, although most participants achieve a net outcome of increased spine protection, they all engaged different patterns of modified muscle activity.¹⁸ Individual variation may be caused by differences in fear-avoidance beliefs, habitual postures and movements, experience, and differences in anatomy and structure. The diversity of changes to motor behavior means that mechanisms other than simple changes in excitability at the spinal cord must be involved.

Some clinical approaches aim to reduce the complexity of the individual variation by allocation of patients into subgroups, based on clustering of features, including motor

behaviors such as movement characteristics that provoke or relieve symptoms.^{51,52} Lending support to this approach, features of these subgroups seem to be grossly consistent with variants of motor adaptation induced by an acute noxious stimulus.¹⁸

Adaptation in the Motor System Aims to Protect the Painful, Injured, or Threatened Body Region

Whether the adaptation involves subtle change or complete avoidance of participation, the overarching purpose seems to be reduced risk for further injury, nociceptive input and pain; that is, to *protect the body part*. Subtle changes in the manner in which a task is performed may unload a painful, injured, or threatened tissue. Avoidance of participation aims to overtly prevent exposure to a situation that may load or threaten the injured part.⁵³ In the context of acute tissue damage, and in the context of sensitization, this adaptation seems logical and relates to the fundamental principal that pain is a motivator to remove threat.

One issue highlighted recently is that adaptation to pain is not always the most obvious solution. When presented with an obvious alternative movement strategy to relieve pain, such as a particular direction of movement that is pain free, this is not always selected by the nervous system (Bergin M, Tucker K, Vicenzino B, Hodges P, unpublished data, 2013). This implies that multiple factors have to be considered in addition to the benefit of reduced nociceptive discharge and tissue protection. In some cases movement behaviors are provocative. For instance, people who experience pain when sitting in lumbar flexion often continue to adopt this position.⁵⁴ This apparently maladaptive behavior might be most prevalent in cases where it preceded the onset of pain.

Movement Adaptation has Long-term Consequences That Will Depend on the Type of Pain

Although potentially beneficial in the short-term to “protect the part,” adapted motor behaviors may have long-term consequences if they persist *beyond* when they are necessary (eg, beyond the time of tissue healing), *exceed* what is necessary (eg, modify load in a manner that is greater than that which could be beneficial), or are *inappropriate* for what is necessary (eg, change in motor control when there is no real threat to the tissues, such as when pain is threatened but without any tissue damage or nociceptive input³³; or complete avoidance of movement with resultant physical deconditioning⁵⁵).

Although ongoing peripheral nociception is not necessary for development of chronic pain, motor adaptation will continue to be relevant in the subgroup of patients with ongoing excitation of nociceptive sensory neurons.⁵⁶ This may be particularly important when sensitization is present, or if a new source of peripheral nociception is initiated as a result of changed loading on structures secondary to modified movement patterns. Although the initial motor adaptation may have been beneficial to protect the painful/injured body region, if maintained there are a number of mechanisms for the adapted strategy to contribute to ongoing pain. Complete or relative avoidance of movement as explained by the fear-avoidance model will lead to deconditioning and disuse²⁰ and a compromised capacity to meet physical demands. More subtle adaptations to movement to protect the tissues may suboptimally load the same or other tissues as a result of augmented muscle contraction/co-contraction,¹⁶ decreased movement, decreased “shock absorption,”⁵⁷ decreased variation of movement,⁵⁸ and so on.

Why does motor adaptation persist beyond when it is necessary? There are several possibilities. (1) The nervous system may fail to return to its preinjury/pain motor strategy. Although pain motivates a change to movement, removal of pain may not provide impetus for movement recovery. If feedback is immediate the nervous system is motivated to change behavior, but as the consequences of tissue loading takes time to develop, the outcome is unlikely to be anticipated and no such motivation to modify behavior exists. (2) Ongoing anticipation, threat, or fear of potential pain and/or injury may motivate the maintenance of the adapted strategy. For example, fear of pain maintains absence of trunk extensor muscle relaxation during flexion.⁴⁵ (3) Central or peripheral sensitization render the link between motor adaptation and the pain experience less clear, with neither maintenance of the adaptation nor its recovery directly related to pain. (4) The modified motor behavior could lead to secondary biomechanical or neurophysiological changes that prevent return to the prepain state. Although not observed universally,⁵⁹ atrophy and fatty infiltration of the multifidus muscle in back pain,^{60–62} and the supraspinatus muscle in tendinopathy,⁶³ adaptive muscle length/stiffness changes,⁶⁴ and increased or decreased muscle strength⁶⁵ would limit the potential for the motor system to recover motor function.

If a patient's symptoms are *neuropathic* with pain attributable to a lesion or dysfunction in the nervous system⁶⁶ the motor adaptation may still be relevant regardless of whether there is a mechanical aspect to the neuropathic symptoms. This could present as augmented muscle activation to reduce load on neural tissues such as hamstrings muscle activation in the presence of sciatica⁶⁷ or simple avoidance of movement of the painful segment. Although the adaptation may have little direct relevance to the development or maintenance of pain, the adapted behavior is likely to lead to secondary changes in other tissues. In this case modification of motor system would not modify the neuropathic source of pain, but prevent or reduce nociceptive input from secondary issues.

Central sensitization involves amplification of neural signaling at multiple regions of the CNS.^{68,69} After the initial insult, central reorganization occurs, and in some this underpins the maintenance of pain without ongoing peripheral nociception as a result of hyperalgesia, allodynia, compromised central pain modulatory mechanisms (descending and/or ascending), cognitive-emotional issues, and potential conflict between sensory and motor events. Movement adaptation requires consideration for a number of reasons. First, persistent nociceptive input can contribute to the maintenance of central sensitization.⁷⁰ Suboptimal control of movement may contribute to the ongoing input and, in the presence of a sensitized system, lesser stimuli will be required to sustain nociceptive input. Second, pain may involve mechanisms that relate to central processing of motor and sensory activity⁷¹ such as a mismatch between motor output and sensory input, and disturbed body image. Movement may be relevant; not from the perspective of abnormal tissue loading, but the disturbed body image may make it difficult to learn movement patterns. Third, stress-induced analgesia from physical activity that is normally mediated by descending opioid and nonopioid brain circuits is absent and the link to motor adaptation will be less clear.^{72–77}

In summary, although the initial adaptation may benefit the system, if maintained in the long term, it can

become part of the problem for the primary involved tissues or could be responsible for establishment of secondary changes and sources of nociception, regardless of the type of pain. Whether rehabilitation of the adapted motor behavior is relevant for recovery of symptoms, depends on the underlying mechanism. It is important to consider that the mechanism for persistence of pain may differ from the mechanism that initiated pain. Although biomechanics may underpin the onset of nociceptive input and pain, and ongoing biomechanical issues could mediate maintenance of nociceptive pain, other cognitive and biological processes may also be responsible; and each requires a different treatment approach.

Movement Adaptation Involves Multiple Mechanisms That are not Mutually Exclusive

There is ongoing debate regarding the neural mechanisms that underlie motor adaptation in pain. Pain, injury, or their threat can change motor control at multiple levels of the nervous system, from cognitive-emotional aspects of the pain experience to the spinal cord. These mechanisms are not mutually exclusive and can interact.

Early animal studies showed spinal mechanisms for motoneuron excitation and inhibition from noxious input.²⁵ Other work showed inhibition of extensor muscles by afferent input from injured tissue.⁷⁸ These mechanisms imply stereotypical adaptation to nociceptive input, and although they may contribute to motor adaptation, they cannot explain the diversity of presentations,¹⁸ the non-linear relationship between nociceptive input and motor adaptation,³⁰ or motor adaptation induced by the threat of pain without nociceptive input.^{32,33}

Reflex adjustments constitute the simplest mechanisms for adaptation of movement to remove threat. Flexor withdrawal reflexes are the most widely studied.⁷⁹ Although reflexes may maintain involvement in chronic pain, such as protective muscle spasm when sensitized nerve is stretched,⁸⁰ they cannot explain the diversity of adaptation.

Higher nervous system mechanisms have been identified. Reorganization of the motor and sensory cortical representations in pain imply modified sensorimotor interaction⁷¹ as evidenced by shift in motor area for specific muscles,⁸¹ “smudging” between adjacent motor⁸² and sensory regions,⁸³ and changes in cortical map threshold or volume.⁸⁴

Cognitive-emotional mechanisms are a major factor in motor adaptation.^{49,85} Although fear of pain and injury underpins avoidant behaviors, considerable research has highlighted the specific psychosocial features that mediate and moderate the effect on movement. According to the *Fear-Avoidance Model*, when confronted with a new episode of pain, individuals^{86,87} with a tendency to negative affectivity, will start to develop catastrophizing thoughts (erroneous beliefs) leading to fear of pain, injury, and movement.^{86,87} This process can be further fueled by hypervigilance to signals that might indicate harm or pain. This leads to avoidance or escape from movement and development of safety behaviors. Although this behavior seems adaptive in the short term, in the long term this leads to functional disability, disuse, and depression, which enhance the vulnerability to pain. The *Avoidance-Endurance Model*⁴⁹ implies a patient's cognitions and beliefs can underpin a strategy to complete a task despite pain, with the outcome of overuse of the motor system leading to additional pain and injury and subsequent rest until the pain subsides.⁸⁵

Sensory dysfunction also plays a role in motor adaptation. Sensation is critical for movement to interpret position, motion, and interaction with the environment. In pain, there is increased threshold to detect sensory inputs,^{88,89} and distorted body scheme,^{90–93} cortical representation,⁸¹ and interpretation of body space.⁸⁹ These changes may “drive” modified motor behavior⁹⁴ or compromise optimal motor performance as a result of distorted perception.

It is important to consider that multiple mechanisms can interact, and these may be complementary, summative, or competitive. For instance, immediate changes in excitability of motor output of spinal cord and cortical regions are opposite after experimental intervertebral disk lesions.⁹⁵ Thus, diversity in motor adaptation to pain is not surprising. It is also clear that the optimal treatments to change motor control and the potential benefit of such change will depend on many factors, not least the type of pain and the relevance of the motor adaptation for ongoing symptoms.

WHAT IS THE ROLE OF MOVEMENT IN RECOVERY FROM PAIN?

Movement and physical activity (including general physical activity, exercise, specific motor learning) are advocated in the management of many musculoskeletal conditions to reduce pain and disability in acute and chronic conditions, and to prevent the transition from an acute to chronic state. It is also advocated to reduce disability despite persistence of pain. Important and largely unresolved issues require consideration. First, the mechanisms by which movement and physical activity affect pain are debated. Treatment is likely to be optimized if the mechanism is known. This may differ between conditions, types of pain, individuals, and different movement interventions. Second, there is a paradox; physical activity is encouraged in pain, but physical activity may provoke pain.

Regardless of the mechanism for maintenance of modified movement behaviors in persistent pain states, it is essential to consider whether restoration or rectification of these issues is a relevant target for treatment, that is, if and how the adaptation contributes to the ongoing pain. Various biological, psychological, and social factors are potential mediators of the transition to persistent pain. Key factors that include peripheral and central sensitization,⁶⁸ cognitive-emotional features of the pain response,²⁷ social issues such as those related to high job demands, low work support, or satisfaction⁹⁶ contribute to, this transition. Although restoration of motor adaptations may be neither sufficient nor necessary for recovery in a number of these cases, movement issues cannot be dismissed. The motor system is a biological factor within the bio-psycho-social model and interacts with other domains. For instance, attitudes to pain influence movement behavior.^{42,45} The relative importance of biological, psychological, and social factors will vary between individuals; movement may be critical for the primary pain condition for some, and for others movement changes may be relevant for secondary conditions, such as development of knee pain secondary to modified movement patterns in back pain.⁹⁷

Mechanisms for Movement and Physical Activity to Change Pain and Disability and the Implications for Treatment

Movement and physical activity may influence pain and disability by several mechanisms. These range from

analgesic effects to a contribution to cognitive-behavioral processes and require further consideration.

Immediate Analgesic Effects of Exercise and Impact on Central Pain Processing

In pain-free individuals, aerobic exercise and resistance training trigger centrally mediated pain inhibition presumably by the release of endogenous opioids [β -endorphins from the pituitary (peripherally) and hypothalamus (centrally) activating the μ -opioid receptors] and growth factors, through the periaqueductal gray, activating the descending nociceptive inhibitory systems.^{98,99} Catecholamines suppress the release of excitatory transmitters of primary afferent nociceptors and postsynaptic responses of pain relay neurons in the spinal cord. Exercise can also: enhance experience of other bodily sensations such as heart beat, sweating that diverts attention from pain; activate gate control through afferent input from skin and muscles; and activate conditioned pain modulation by nociceptive afferent input from other regions secondary to ischemia and lactate accumulation.¹⁴ As evidence, movement reduces pressure pain thresholds.¹⁰⁰ Although effective in pain-free individuals, the effectiveness in chronic pain is unclear.¹⁰¹ Exercise-induced endogenous analgesia may be dysfunctional in patients, and exercise might therefore increase pain, particularly in individuals with central sensitization, which is characterized by nonsegmental spreading of pain, difficulties with concentration and fatigue, stress-intolerance, and hypersensitivity to stimuli such as bright light and touch (for review see Nijs and colleagues^{14,29}). However, this may only apply to a subgroup as central sensitization is the dominant pain mechanism in only 23% of chronic low back pain patients. This concurs with the finding that exercise-induced analgesia is experienced in chronic back pain, but not in chronic fatigue syndrome and whiplash-associated disorder.^{102,103} Whether this is activated by exercise such as static muscle contractions has not been studied.

Exercise That Aims to Correct Motor Control

Several approaches to optimization of motor control have been proposed^{52,104} with the objective to correct features of muscle activation, postural alignment, and movement patterns that abnormally load tissues. Although most advocate individualized treatment based on assessment, others prescribe a generic approach.¹⁰⁵ The science underpinning this approach is sound, and mechanics may explain the onset of injury or nociceptor discharge, but tissue loading may or may not be involved in transition to chronicity. Thus, restoration of motor control might not be relevant for recovery for some individuals and some argue that attention to motor control is unnecessary.^{106,107} Although clinical trials and subsequent systematic reviews point to the efficacy of interventions that address these issues,^{108–115} evidence is not yet conclusive as other trials have not shown higher effectiveness than more general exercise.^{116,117} Issues such as optimal selection of patient subgroups and individualization of treatment have been questioned and need to be addressed. Theoretically, individuals with a persistent nociceptive component should benefit from improved motor control. Several issues require consideration. First, optimal outcomes are expected if exercise is targeted to the individual presentation based on careful assessment. Second, optimization of motor control may be particularly relevant for a sensitized system. Third,

interaction between motor adaptation and psycho-social features requires consideration. Fourth, motor control training requires consideration of multiple mechanisms from muscle, to the spinal cord and the supraspinal centers. Fifth, as pain is a potent stimulus for adaptation, pain relief in conjunction with exercise would be relevant whether pain is nociceptive, neuropathic, or centrally maintained.

Treatments to Modify of Self-efficacy, Catastrophizing Thoughts, and Fear of Pain/Injury

Approaches to modify self-efficacy, catastrophizing thoughts, and fear of pain/injury can take many forms. In fact, positive effects of aerobic exercise and back extensor muscle strengthening have been shown to be mediated by reduced catastrophizing thoughts, rather than increased aerobic capacity or strength.¹¹⁸ Likewise, cognitive-behavioral treatments such as graded physical activity increase self-efficacy and decrease catastrophization leading to secondary gain of functional ability.^{106,119,120}

Graded exposure designed to challenge catastrophic thoughts regarding pain, injury, or movement is relatively new. It is characterized by a systematic and repeated exposure to sensations, movements, or activities the patient fears. This is achieved by: identification of fears; explanation why the feared stimuli, movement, or activities can be performed without further harm; challenge of catastrophic thoughts by experience with the movement/activity associated with fear. This process aims to lower threat-value of stimuli, and reduce perception of harmfulness of the movements and activities. Although, this proposed sequential relationship between the psychological risk factors described in the Fear-Avoidance Model has not been proven and disability is only partly dependent on these risk factors,⁸⁶ efficacy of graded exposure has been demonstrated in low back pain, whiplash-associated disorders, and complex regional pain syndrome (CRPS).^{106,107,121–123} In CRPS pain reduction is accompanied by improved dystrophic changes, which demonstrates a complex interaction between psychological and physiological factors.

Strategies to Reverse/Prevent Disuse and Deconditioning

Moderate to strong evidence indicates that a more active lifestyle is better for the general health and prevention of morbidities.¹²⁴ Although, it remains debated whether deconditioning is common in patients with chronic pain,^{125,126} the association between a less active lifestyle and greater pain and disability is strong.¹²⁷ The possibility to resume activities has broad benefits for those with chronic pain including enhancement of opportunities to re-engage in social roles.^{1,128,129}

Multimodal Integration of Movement and Activity Interventions That Target Different Mechanisms

Integration of approaches that target the different mechanisms outlined above has potential benefits, but has not been well investigated. Notably, whether attention to how a person moves adds additional benefit to increasing how much they move should be a major priority in pain research. Recent work showed greater clinical effect of treatment based on comprehensive classification of patients on the basis of movement characteristics and psychosocial presentation to select treatment, than manual therapy and exercise in chronic low back pain.¹⁰⁸ Despite methodological issues, this is the first trial to tease out the factors,

across multiple dimensions, which may contribute to maintenance of pain and disability. Other studies show that graded exposure without consideration of movement quality does not have adverse effects in back pain and CRPS patients,¹³⁰ follow-up has been limited to 6 or 12 months, so no definite conclusions can be drawn.

Physical Activity as a Cause or Consequence of Pain

Advice to stay active is the cornerstone of management of the patient with acute back pain and advocated in clinical practice guidelines internationally. In chronic pain, graded return to activity is advocated as the optimal approach. Although there is no convincing evidence that a specific type of exercise or cognitive-behavioral treatment is most effective,¹³¹ some argue that the latter is more cost effective,⁷ yet, the overall clinical effect size is low to moderate.¹³¹ Regardless, it is generally accepted that back pain outcome is better in those who remain active, with some provisos such as early identification of more serious pathologies. Again there are issues to consider. First, the relationship between pain and movement is complicated by the reality that movement may both reduce and increase pain. Is management as simple as the suggestion to remain active? Second, it remains unclear how important the way a patient moves is to their symptoms. Is it enough to encourage a person to move more, regardless of how they move, or should effort be made to change the way a person moves?

Only 1 population-based cross-sectional study has studied the relationship between chronic low back pain and physical activity.¹³² Engagement in sport was associated with less back pain, and, in women, there was a moderate risk for greater back pain for participants both with a sedentary lifestyle and involvement in strenuous physical activities. This implies a nonlinear U-shaped relationship. Further work is needed to resolve this quandary.

When considering the role of movement and activity as a treatment, dysfunctional exercise-induced analgesia has been reported in chronic pain syndromes that exhibit central sensitization (eg, fibromyalgia and whiplash). However, these studies involved excessive bouts of exercise, and randomized controlled trials provide evidence for efficacy of exercise in these conditions,^{2,133} despite the presence of central sensitization. From another perspective, symptom flare-ups after exercise often lead to early dropout in treatment. The likely solution is to individually tailor the increase of exercise load to avoid symptom flare-ups and use aerobic exercise rather than eccentric/isometric contractions, which are thought to enhance central sensitization,¹³⁴ and focus on training nonpainful body parts. Cognitive-emotional sensitization should be addressed concurrently with cognitive-behavioral techniques such as education, graded activity, etc., as well as other techniques to modulate pain such as pain relieving modalities. These could include transcutaneous electrical nerve stimulation, virtual reality, and pharmacological agents. However, the evidence for transcutaneous electrical nerve stimulation¹³⁵ and virtual reality^{136,137} is scarce, and effects of medication are small with risk of side effects.¹³⁸

What could go wrong if pain is not taken into account? Can increased walking, bending, and lifting cause additional damage in a patient with a movement pattern that potentially loads the tissues suboptimally? Motor adaptation to nociception and pain often persist despite the relief

of nociception.^{139–141} For instance, multifidus muscle atrophy is not reversed when back pain resolves.¹³⁹ Why the adaptation does not resolve is not clear. One explanation is that although the nervous system has a refined capacity to respond and adapt to immediate feedback such as pain provocation, there is limited potential to predict and adjust to long-term outcomes such as the potential consequences of maintenance of a suboptimal strategy. This may justify implementation of motor control (re)training strategies for patients with chronic pain. Given the disrupted body image this also seems reasonable. Further, if chronic nociception inhibits motor output, as predicted by some models of motor-nociception interaction, this could be interpreted to be contradictory and even fruitless.⁷¹ Evaluation of whether the failure of resolution of motor control dysfunction is relevant for recovery requires more valid and reliable assessments for motor control,¹⁴² and more methodologically sound and adequately powered studies. Although no evidence shows motor control exercise is harmful, how this approach interacts with other interventions requires consideration. For example, graded exposure approaches emphasize not paying attention to pain and this may be hampered if a motor control intervention is added that advocates attention to aspects of movement that provoke pain.

If a structural lesion is present but not well diagnosed and treated, starting to move might increase the risk of compounding the problem. For example, an ankle sprain with concomitant limitation of dorsiflexion that is not well treated will likely induce compensatory external rotation of hip, decreased knee flexion at heelstrike, and increased hip circumduction during swing phase. Consistent with contemporary models of pain adaptation, the short-term benefit of these adaptations to enable walking could underpin long-term consequences such as development of secondary problems at the knee, hip, and back through excessive loading. This example highlights the potential relevance of addressing suboptimal features of movement in association with increased activity.

CONCLUSIONS

Contemporary models that aim to unify understanding of divergent changes in the sensorimotor system are enhancing understanding of why people move differently in pain, yet there is much still to learn, particularly with respect to the interaction between pain and movement over time and the ideal methods to apply and combine (or not) treatments. From a clinical point of view, 2 options could be justified. One option would be to first trial training that increases activity with dosage controlled to limit flare-ups, followed by attention to optimization of motor control once the patient is more active. Another option would be to first train motor control to optimize tissue load before augmenting activity. There is insufficient evidence to justify one approach over the other, and limited evidence that exercise of either dimension contributes to a normalization of central nervous system processing of pain. This issues should be a priority for ongoing research.

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