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Review

Pain and motor control: From the laboratory to rehabilitation

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ABSTRACT

Movement is changed in pain and is the target of clinical interventions. Yet the understanding of the physiological basis for movement adaptation in pain remains limited. Contemporary theories are relatively simplistic and fall short of providing an explanation for the variety of permutations of changes in movement control identified in clinical and experimental contexts. The link between current theories and rehabilitation is weak at best. New theories are required that both account for the breadth of changes in motor control in pain and provide direction for development and refinement of clinical interventions. This paper describes an expanded theory of the motor adaptation to pain to address these two issues. The new theory, based on clinical and experimental data argues that: activity is redistributed *within* and *between* muscles rather than stereotypical inhibition or excitation of muscles; modifies the mechanical behaviour in a variable manner with the objective to "protect" the tissues from further pain or injury, or threatened pain or injury; involves changes at multiple levels of the motor system that may be complementary, additive or competitive; and has short-term benefit, but with potential long-term consequences due to factors such as increased load, decreased movement, and decreased variability. This expanded theory provides guidance for rehabilitation directed at alleviating a mechanical contribution to the recurrence and persistence of pain that must be balanced with other aspects of a multifaceted intervention that includes management of psychosocial aspects of the pain experience.

Contents

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1. Introduction

Rehabilitation of control of movement and muscle activity is a mainstay of management of many pain conditions related to the musculoskeletal system. This is based on the premise that pain and movement are intimately linked. In the acute phase, the motor system provides an opportunity for the nervous system to respond and remove or reduce a threatening noxious stimulus (mechanical, chemical or thermal) to the tissues. If the nervous system concludes that a situation is threatening (this may be in response to discharge of nociceptive afferents or the threat of a noxious input) it can move or change the mechanical behaviour of the body to remove the threat, and reduce the potential for further pain/injury to the tissues. In the chronic phase the motor response may be less meaningful, less accurate or unnecessary as the threat to the tissues may be less relevant as a result of the range of physiological and psychological issues that change the gain of the pain system. Thus, the pain that a person experiences does not necessarily match the input from the nociceptive afferents and pain may not reflect harm or potential harm to the tissues. Maintenance of a motor adaptation in chronic pain may not provide benefit to the system.

Many clinical interventions target changes in motor control that accompany pain. These include motor learning strategies (e.g. exercise with error correction, augmented feedback, part-practice), some psychological interventions (e.g. treatments to reduce threat value of pain), and hands-on techniques (e.g. manual therapy, muscle stretching, needling techniques). However, the mechanisms that underlie the motor adaptation to pain are surprisingly poorly understood and two primary theories have been proposed: "vicious cycle" (Roland, 1986) and "pain adaptation" (Lund et al., 1991). These theories explain some observations in clinical and experimental pain. However, there are two major limitations. First, many clinical and experimental observations are inconsistent with predictions made by these theories; and second, the link between these theories and rehabilitation is weak. The aim of this paper is to review the limitations of current theoretical models of the motor adaptation to pain, to review a new theory (Hodges and Tucker, in press) that accounts for many of the observations that cannot be explained by existing theories, and to consider the implications for rehabilitation.

2. Current theoretical models for the motor adaptation to pain and their relationship to rehabilitation

The "vicious cycle" theory proposes a stereotypical increase in activity of muscles that are painful or move the painful region. This muscle activity induces ischaemia from vascular compromise and becomes a source of further pain due to accumulation of pain metabolites (Roland, 1986). Various mechanisms have been proposed to explain the increase in muscle activity, including increased sensitivity of muscle spindles (Johansson and Sojka, 1991). Treatments based on this theory include the use of electromyography (EMG) biofeedback to train muscle relaxation, such as reduced activity of the erector spinae muscles in back pain (Flor et al., 1983) and the temporalis muscle in tension headache (Holroyd et al., 1984). Although this approach received some initial support (Flor et al., 1983; Nouwen, 1983) two issues compromise the validity of the approach. First, although there is evidence of increased muscle activity, this is not uniform and many studies show decreased (Wolf and Basmajian, 1977; Zedka et al., 1999) or no change (Kravitz et al., 1981; Cram and Steger, 1983) in activity. Second, clinical improvement has been reported despite no changes in muscle activity (Holroyd et al., 1984). This suggests clinical efficacy of may be related to cognitive aspects rather than rehabilitation of the motor adaptation. Alternative theories are required to explain the non-uniform changes in muscle activity and movement with pain and to guide interventions (see Fig. 1).

The "pain adaptation" theory was developed to explain changes in voluntary movement and argues that activity of a muscle that is painful or produces a painful movement is uniformly inhibited, whereas that of the muscles that oppose the movement (antagonist) is facilitated (Lund et al., 1991). The outcome would

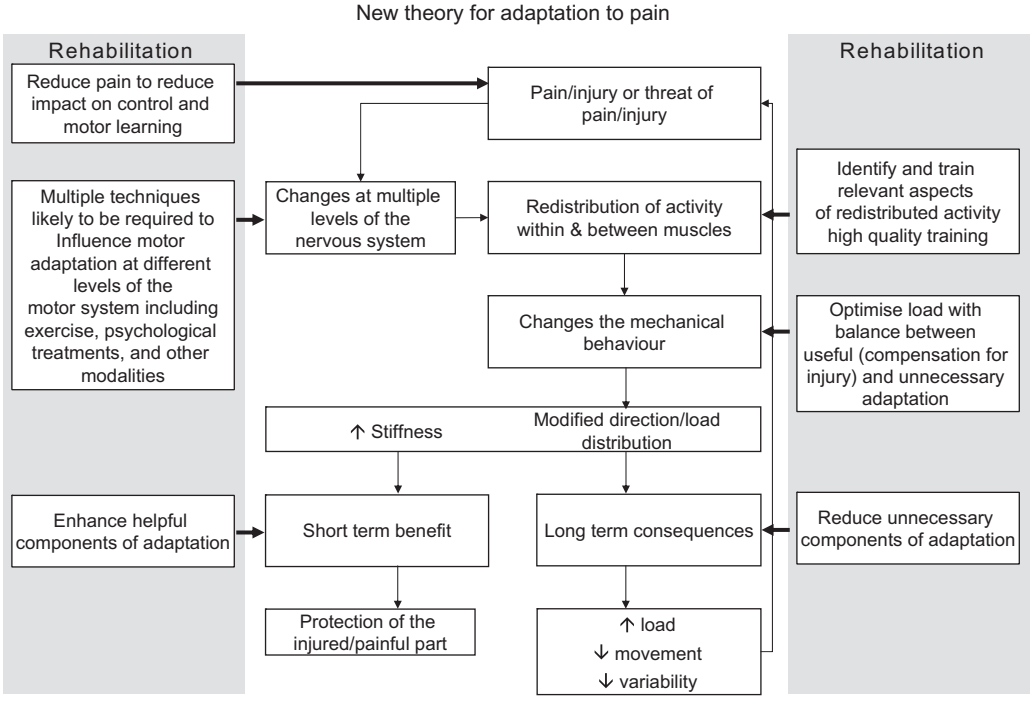

Fig. 1. New theory of motor adaptation to pain and implications for rehabilitation.

be decreased displacement, velocity or force to reduce pain provocation and further injury. These changes in excitability were proposed to be mediated by networks in the spinal cord or brainstem (Lund et al., 1991). Data in support of the theory can be identified largely from the effect of acute experimental pain. For instance, agonist muscle activity is reduced during voluntary movements of the jaw (Svensson et al., 1995), trunk (Zedka et al., 1999), neck (Falla et al., 2007) and limbs (Farina et al., 2005) and a combination of reduced and increased activity of opposing muscle groups has been shown in dynamic movements of the leg (Graven-Nielsen et al., 1997). However, the observations are not universally supported. First, recent work of motoneuron excitability by evaluation of the response to excitation of corticospinal axons at the level of the mastoid processes showed increased excitability of the motoneurons innervating both the painful muscle and its antagonist (Martin et al., 2008), rather than the predicted reduction of excitability of the agonist muscle. Second, activity of motoneurons in a painful muscle is not uniformly decreased (Tucker et al., 2009). Although discharge rate of some motoneurons is decreased (Farina et al., 2004a,b; Sohn et al., 2004), if the person is asked to generate a constant force between trials with and without pain, activity of other units increases to maintain the force output (Tucker and Hodges, 2009). Other data of motor behaviour (coordination of whole muscle activity) show that experimentally induced low back pain is accompanied by redistribution of activity between the multiple muscles of the trunk rather than a stereotypical and predictable change in muscle activation during slow voluntary trunk movements (Hodges et al., 2006). This is consistent with the high degree of variability observed in changes in trunk muscle control across many studies of clinical low back pain (van Dieën et al., 2003). There are similar observations from other body systems such as the jaw (Murray and Peck, 2007).

The link between the pain adaptation theory and rehabilitation is unclear. What would inhibition and excitation to reduce voluntary movement suggest for rehabilitation of pain? It may suggest that activity should be discouraged to relieve pain, but this is unlikely to be successful in restoring function. Consideration of the motor adaptation to pain in this paradigm has not led to clear implications for management and new models are required to drive improvements in rehabilitation.

3. New theory of the adaptation to pain

A new theory has been developed on the basis of data from micro (motoneuron discharge) to macro (whole muscle behaviour) levels of the motor system in order to reconcile the complex nature of the adaptation in motor control that accompanies pain (Hodges and Tucker, in press). This theory is a progression from the vicious cycle and pain adaptation theories and is inclusive of the observations associated with the basis for those theories. The key progression of the theory is that it aims to account for variation in the adaptation in motor control rather than the stereotypical adjustments in behaviour predicted by existing theories and includes consideration of multiple concurrent possible mechanisms throughout the nervous system that can mediate such changes. The theory also has an objective to provide guidance for the development and refinement of treatments. The following provides a summary of the main elements of the theory, the associated evidence, and implications for rehabilitation.

The new theory proposes that pain is associated with an adaptation in motor behaviour that: (i) involves redistribution of activity *within* and *between* muscles (rather than inhibition or excitation of muscles in a stereotypical manner); (ii) changes the mechanical behaviour such as modified movement and stiffness; (iii) leads to "protection" from further pain or injury, or threatened pain or injury (as a result of a variety of changes such as decreased activity to reduce voluntary movement, increased activity to splint a body region, or change distribution of activity to modify the distribution of load on a structure); (iv) is not explained by simple changes in excitability, but involves changes at multiple levels of the motor system and these changes may be complementary, additive or competitive (rather than isolated effects of nociceptor afferent input at the spinal cord); and (v) has short-term benefit, but with potential long-term consequences due to factors such as increased load, decreased movement, and decreased variability (Hodges and Tucker, in press) (Fig. 1).

3.1. Redistribution of activity within and between muscles

The new theory proposes that rather that a uniform increase or decrease of activity there is an adaptation that may vary between individuals and tasks to change the mechanical response to protect the tissues or remove threat. A variety of options are available to meet the overall objective of protection. In some cases this may involve reduced activity (such as the inhibition of masseter muscles during painful jaw movement (Svensson et al., 1997) consistent with the pain adaptation theory), increased activity (such as increased activity of sternocleidomastoid in neck pain (Johnston et al., 2008) consistent with the vicious cycle theory) or a combination of both (such as increased and decreased abdominal muscle activity with slow trunk movements during experimental pain (Hodges et al., 2006). Studies of changes in responsiveness of corticomotor pathways indicate that muscles can be affected differentially. For instance, noxious stimuli to that hand increase responsiveness of hand muscles to magnetic stimuli over the motor cortex, but simultaneously decrease responsiveness of proximal arm muscles (Kofler et al., 1998). Recent work shows similar differential affects on the distribution of activity between abdominal muscles with experimental back pain; responsiveness of pathways to transversus abdominis is reduced whereas those to obliquus abdominis externus are more responsive (Tsao et al., submitted for publication).

The redistribution of activity may also occur *within* a muscle. Although reduced discharge rate of active motor units during experimental pain has been interpreted to suggest inhibition (Farina et al., 2004a,b; Sohn et al., 2004), recent work shows the force output is maintained by recruitment of additional units that were not active during contractions without pain (Tucker and Hodges, 2009). This cannot be explained by uniform inhibition of the motoneuron pool as new motor units are recruited despite reduced discharge of others.

Although the adaptation of muscle activity may be predictable in some situations (e.g. voluntary jaw (Svensson et al., 1997) and limb movements (Graven-Nielsen et al., 1997)) this is not the case for complex systems such as the trunk. There is considerable redundancy amongst the trunk muscles (i.e. multiple muscles perform similar functions) and the objective of protection could be achieved via multiple strategies such as co-contraction of antagonist flexor and extensor muscles, or increased activity of many combinations of these muscles (van Dieën et al., 2003; Hodges et al., 2006). This is supported by modelling work (Van Dieën et al., 2003). Although the large variation in adaptation in trunk muscle activity could be interpreted to suggest that no underlying theory can explain the adaptation, the alternative argument is that each individual develops a protective strategy that is unique based on experience, anthropometrics, posture, task, etc. The multiple possible solutions are likely to be related to the clinical subgroups of low back pain that have been proposed (Sahrman, 2002; O'Sullivan, 2005). If a goal of rehabilitation (e.g. using motor learning strategies) is to modify the adaptation (remove, modify or enhance,

see below) then this needs to be considered on an individual basis with respect to the unique solution adopted by the patient. This would require development or refinement of methods to assess the adaptation in motor control (e.g. with ultrasound imaging or electromyography).

3.2. Altered mechanical behaviour

Removal or reduction of threat to the tissues can be achieved via multiple changes to the mechanics of a system. This could be removal of the body part from the threat (e.g. nociceptive withdrawal reflex (Clarke and Harris, 2004), fright/flight response), reduced displacement/velocity/force (e.g. reduced displacement and velocity of voluntary jaw movement (Svensson et al., 1995)), stiffening a segment to prevent movement that is association with pain or the threat of pain or injury (e.g. trunk stiffening (Hodges et al., 2009b)), or modification of the distribution of load on a painful structure (e.g. changed angle of knee extension force (Tucker and Hodges, 2010)). Although some of these changes could be coordinated at a basic level of the nervous system (e.g. spinal cord control of the nociceptive withdrawal reflex), other changes are more complex involving changes in higher processing and planning or even voluntary adjustment of force (e.g. search for a less painful movement option). It is likely that such adaptation would generally occur subconsciously and in the case of threat of pain it would not be necessary to have explicit conscious recognition of fear of pain/(re)injury. Although, the adaptation may be beneficial in the short term, it may pose problems in the long-term and training an individual to reduce or modify the adaptation may form the basis of effective treatments (see below).

3.3. "Protection" from further pain or injury, or threatened pain or injury

A basic assumption, like that proposed in the vicious cycle and pain adaptation theories, is that the adaptation aims to protect the body segment from pain or injury, or the threat of pain or injury. Although the vast majority of experimental and clinical observations can be reconciled on this basis (e.g. nociceptive withdrawal reflex, reduced agonist activity during voluntary movement), recent work provides additional support. When stepping down from a stair, activity of gluteal muscles precedes foot contact to control hip loading (Zazulak et al., 2005) and this activity is earlier and greater to enhance the protection for the hip when the stair height increases (e.g. 5 cm vs. 15 cm step height) (Hodges et al., 2009a). Consistent with the hypothesised protective nature of the adaptation of motor control with pain, when the contact of the foot with the floor is associated with pain or anticipated pain (e.g. painful electrical shock to the back triggered by foot contact) the strategy normally reserved for the 15 cm stair height is used for the 5 cm stair (Hodges et al., 2009a) (i.e. a solution reserved for a high load task is used for a lower load task). Many permutations of adapted motor behaviour may lead to enhanced protection such as decreased muscle activity and force (Svensson et al., 1995); muscle splinting (Kaigle et al., 1998); withdrawal (Clarke and Harris, 2004) as described in the previous section.

3.4. Changes at multiple levels of the motor system

Initial theories assumed relatively simple mechanisms to explain the adaptation to pain, such as direct input from nociceptive afferents on motoneurons (Kniffki et al., 1979), inhibitory and facilitatory interneurons and circuits in the spinal cord and brain stem (Lund et al., 1991), and increased spindle sensitivity (Matre et al., 1998; Wang et al., 2000; Svensson et al., 2001; Thunberg et al., 2002). Work over the past few decades has highlighted multiple mechanisms. In addition to well-established spinal mechanisms, changes have been identified in excitability (Le Pera et al., 2001; Strutton et al., 2003; Martin et al., 2008; Tsao et al., 2008) and organisation (Maihöfner et al., 2007; Tsao et al., 2008) of the motor cortex, and more complex changes in the planning of motor responses that may be mediated "upstream" of the motor cortex (Hodges and Moseley, 2003). This latter class of changes include changes in the pattern of activity of muscles of the trunk initiated prior to predictable perturbations to the body, such as anticipatory postural adjustments (Hodges and Richardson, 1996).

Changes in sensory function will have profound effects on control of movement and have been identified in many painful conditions of the musculoskeletal system (e.g. ankle sprain (Garn and Newton, 1988), shoulder pain (Warner et al., 1996), back pain (Newcomer et al., 2000), neck pain (Treleaven et al., 2006), knee osteoarthritis (Sharma and Pai, 1997)). Changes include reduced sensory acuity (Sharma and Pai, 1997), increased errors in repositioning (Brumagne et al., 1999), reduced responsiveness to sensory input (Brumagne et al., 2004), and reorganisation of the somatosensory regions of the brain cortex (Flor et al., 1997).

The multiple mechanisms that may influence movement could be complementary, additive, or competitive. For instance, competing effects have been identified following injury to a porcine intervertebral disc with reduced responsiveness of spinal pathways, but increased responsiveness to stimulation over the motor cortex (Hodges et al., 2009c). In contrast, activation of groups III and IV muscle afferents (nociceptive afferents) by hypertonic saline injection into human biceps brachii facilitates motoneurons innervating elbow flexor and extensor muscles but depresses excitability of motor cortical cells projecting to these muscles (Martin et al., 2008). The net effect may be to limit voluntary activation but enhance responsiveness to other inputs. Other work cites complementary inhibition at both the cortex and motoneurons, but with a different time-course (Le Pera et al., 2001). The critical message for clinical practice is that the motor output observed in patients may have multiple underlying mechanisms and each may have unique implications for rehabilitation.

3.5. Short-term benefit, but with potential long-term consequences

Although adaptation in motor behaviour may be beneficial in the short-term, failure of the adaptation to resolve after the initial period following injury (at which time a protective response may be appropriate for "survival") may pose risks for the tissues and nervous system. The vicious cycle theory also proposes a consequence of the adaptation to pain, yet this was simply explained by chemical irritation of nociceptive afferents due to ischaemia from sustained contraction (Roland, 1986). In addition to this possibility, the new theory proposes consequences from modified loading of the tissues as a result of altered mechanical behaviour. This has important implications for rehabilitation as techniques to resolve the adaptation may help to reduce persistence or recurrence of pain.

There are a number of reasons why the changes in mechanical behaviour may not be ideal if maintained. First, the protective solution may compromise the quality of movement. For instance, increased trunk stiffness in low back pain is associated with decreased damping (i.e. control of velocity) (Hodges et al., 2009b), which is likely to be important to minimise the effect of perturbations. Although people normally move the spine in advance of predictable challenges to the spine, this is less frequently used in people with pain (Mok et al., 2007), and is accompanied by greater perturbation to the spine (Mok et al., 2007) and decreased quality of the postural recovery after the perturbation (Mok et al., 2009). In the lower limb, decreased knee flexion (i.e. increased knee

stiffness) is present in non-copers following ACL tear (Rudolph et al., 1998) and may underlie ongoing problems.

Second, the adaptation may increase load on the tissues (e.g. if muscle activity is increased) or change the distribution of load (e.g. change distribution of activity within or between synergist muscles), which may lead to tissue irritation, particularly if there is sensitisation. For instance people with back pain have greater load on the spine during lifting than pain-free individuals as a direct result of changes in muscle activation (Marras et al., 2004) and this may lead to structural change over time (Kumar, 1990). Although changes in the distribution of load may theoretically benefit the system by unloading a painful structure with short-term benefit to decrease pain and injury, the adaptation would inevitably increase load on other structures. Many other examples have been presented in the literature. For instance, movement adaptations follow ankle sprain such as reduced ankle dorsiflexion (Friel et al., 2006) to reduce loading of the injured ankle structures leads to modified kinematics at proximal joints (Davis and Seol, 2005). If maintained in the long-term these changes may lead to problems elsewhere in the body (Davis and Seol, 2005).

Third, adaptations in motor behaviour such as increased muscle activity to splint the painful pain may reduce movement variability. Although too much variation is not ideal (Tzagarakis et al., 2010), so too is too little variation (Hamill et al., 1999). If movement is performed in the same manner with each repetition this will load the same structures, same joint surface, same muscles each time. With some variation the load is shared resulting in potential benefits for tissue health. Reduced variation has been identified in a range of conditions, such as lower limb pathologies and variability in gait (Hamill et al., 1999).

An underlying premise of the new theory of motor adaptation to pain is that the adaptation is less relevant or even detrimental in the long-term. This is aligned to current pain theory that proposes that when pain persists beyond the period of tissue healing the mechanics at the tissue level may be less important and the central mechanisms for maintenance of pain may be more important. Both physiological and psychosocial factors underlie a mismatch between the nociceptive discharge in the periphery and the pain experienced by the individual (Waddell, 1998; Latremoliere and Woolf, 2009). These changes include: plastic changes in the responsiveness and function of neurons and circuits in the spinal cord that change the gain of the pain system (Cook et al., 1987); modification of function of the descending facilitatory and inhibitory pain systems (Arendt-Nielsen et al., 2010); cognitive aspects of pain such as catastrophising and fear of pain that modify threat value (Sullivan et al., 2001). In each of these cases there may be a mismatch between the relevance of nociceptive afferent discharge and the experience of pain, and at the tissue level the adaptation in movement control may no longer be relevant or appropriate. The alternative argument is that some degree of tissue-level change may have a maintained contribution to the perpetuation of pain. Panjabi (1992) proposed that adaptation in muscle and neural control is required to compensate for any loss of passive support from joint structures as a result of tissue injury. In this case some degree of adaptation may be beneficial (van Dieën et al., 2003). Although this may be true in some cases, the possibility to replicate changes in motor control (e.g. increased protection) simply by the threat of pain, in the absence of injury (Moseley et al., 2004), implies the relationship between injury and adaptation is not linear; i.e. "input" is not directly related to "output". In some cases the adaptation may be appropriate leading to reduced pain in the long-term, and this could explain the mismatch between structural joint changes and pain where some people experience little pain despite significant joint damage. The lack of pain in this context may be due to a combination of an appropriate adaptation of the motor system to compensate for the structural damage (i.e. appropriate protective strategy) and the fact that pain is an output of the central nervous system based on interpretation of inputs, and not directly explained by the input from nociceptor discharge. If the nervous system has taken action to protect the injured/painful part (i.e. adapted the motor behaviour), then the pain "output" may be reduced. Taken together it would seem reasonable to conclude that it is necessary find the right balance between restoration of control to some baseline and the maintenance or retention of elements of the adaptation in order to meet the demands of function.

Although the proposal that failure of resolution of the adaptation to pain may contribute to recurrence or persistence of pain is based on sound reasoning, there is lack of data from high quality longitudinal studies to support this hypothesis. Some data support the association between non-resolution of acute atrophy of the multifidus muscle and future recurrence of back pain (Hides et al., 2001). However, that study was small and the group with muscle recovery also received a different treatment package than the control group and this may have independently affected the outcome. Investigation of the relationship between adaptation and long-term outcome will not be simple as there is potential for enormous variation in adaptation and the interaction between biological, psychological and social aspects when pain persists is complex. A final consideration is that adaptation leading to long-term changes may be caused by factors other than an initial injury or pain related to a musculoskeletal structure. For instance, motor control may adapt in response to more widespread or centrally mediated pain states or other issues that challenge the motor system (e.g. respiratory (Hodges et al., 2001) and continence (Smith et al., 2007) challenges affect trunk muscle control in a similar manner to spinal pain). Like the adaptation to an acute pain/injury these changes may lead to further problems (Smith et al., 2009).

4. Implications of the new theory of motor adaptation in pain for rehabilitation of musculoskeletal pain

How can the new theory inform rehabilitation for people with musculoskeletal pain? Although existing models of the adaptation to pain provide limited guidance for rehabilitation, the expanded theory of motor adaptation to pain provides a rich array of implications that can be used to shape interventions. Such interventions may include exercise (e.g. training of motor control or movement strategy), psychological interventions (e.g. treatments that change the threat value of pain), and physical modalities (e.g. modalities that can influence muscle activation such as manual therapy techniques). It is important to acknowledge that it is not possible to predict the size of a clinical effect on the basis of physiological data, and clinical trials are needed to determine whether changes to clinical practice lead to better outcomes. However, hypotheses can be proposed for testing in appropriate clinical trials.

A key aspect of the new expanded theory for motor adaptation to pain with implications for rehabilitation is the proposal that the adaptation may have *short-term benefit but with long-term consequences*. It is proposed that if the motor adaptation is excessive or fails to resolve after it is no longer helpful it may contribute to the perpetuation or recurrence of injury and pain. The clinical conclusion is that clinicians need to identify aspects that may be excessive/inappropriate and develop a strategy to train the patient to restore more optimal control. The alternative and not mutually exclusive view is that the adaptation may be necessary to compensate for deficit in the passive support for the segment (e.g. injury to intervertebral disc) and that enhanced control is required to meet the demands for control of the segment (Panjabi, 1992; van Dieën et al., 2003). In rehabilitation the challenge will be to identify the

relevance of the adaptation and find the balance between that which is required and that which is not.

According to the theory, resolution of the motor adaptation would involve *redistribution of activity between and within muscles* to modify the *mechanical behaviour* of the body segment. Redistribution of activity between muscles is already considered as part of existing interventions. For instance rehabilitation of the coordination between the medial and lateral vasti muscles is effective for the management of anterior knee pain (patellofemoral pain syndrome) (Crossley et al., 2002) and treatment of back pain that includes modification of the coordination between trunk muscles reduces pain and disability and prevents recurrence of episodes (O'Sullivan et al., 1997; Hides et al., 2001; Stuge et al., 2004; Ferreira et al., 2006; Macedo et al., 2009). Clinical tools are used to aid redistribution of activity between muscles such as techniques to provide feedback (e.g. electromyography (McConnell, 1986; Crossley et al., 2002) and ultrasound imaging (Hides et al., 1996)) and techniques that enhance the function of specific muscles (e.g. application of therapeutic tape (Cowan et al., 2002b)). Redistribution of activity *within* the upper trapezius has been applied in shoulder pain (Samani et al., 2010). A similar approach may have potential in other conditions.

As changes may occur at *multiple levels of the nervous system*, a range of clinical strategies are likely to be required to restore/retrain ideal control. If the motor adaptation was simply due to input from nociceptive afferents on motoneurons (Kniffki et al., 1979) or due to reflex inhibition (Spencer et al., 1984) it could be assumed that the most appropriate treatment should be application of techniques to reduce pain (e.g. analgesic agents) or techniques that may increase motoneuron excitability (e.g. peripheral electrical stimulation). Treatment of pain is unlikely to be sufficient to restore motor control as it has been shown that many aspects of the motor adaptation persist between episodes, despite resolution of pain (Hodges and Richardson, 1996; MacDonald et al., 2009), that is, motor control adaptation does not require ongoing nociceptor stimulation for maintenance. Interventions that target higher levels of the motor system are likely to be required. These include motor learning strategies to change planning and coordination of movement and muscle activity (such as biofeedback techniques to change distribution of activity described above), and management of unhelpful cognitions such as catastrophising and fear-avoidance, which may influence the muscle activation strategies. The greatest challenge will be to validate clinical techniques to determine which aspects of the motor adaptation are necessary to change.

5. Can motor adaptation be changed with intervention and does it make a difference?

A variety of clinical interventions have been proposed to retrain motor control in musculoskeletal pain. These interventions vary in their approach and are based on a multitude of clinical theories such as aiming to *modify* loads on painful structures (Crossley et al., 2000) and *enhance protection* of a painful part (McGill, 2002; van Dieën et al., 2003). The common feature is the use of relearning strategies to change motor features considered to contribute to the perpetuation or recurrence of pain.

There are varying levels of experimental support for these approaches, but evidence as accruing that motor adaptation to pain can be resolved with rehabilitation and this is associated with positive clinical effects. One example is work in low back pain that has investigated temporal and spatial aspects of activation of the deep abdominal muscle, transversus abdominis, in trials of motor rehabilitation. The implication is not that this change constitutes the entirety of the adaptation, but that it is a common component that can be used as a "marker" of adaptation. Delayed and reduced activation of transversus abdominis is common in low back pain, it persists between pain episodes (Hodges and Richardson, 1996; Ferreira et al., 2004), and can be induced by experimental pain (Hodges et al., 2003). Delayed and reduced activation of transversus abdominis can be restored with motor relearning strategies (Tsao and Hodges, 2007). These changes persist after cessation of training (Tsao and Hodges, 2008), are related to the magnitude of improvement in pain/disability (Ferreira et al., 2010), and baseline values can indicate individuals who will respond to the intervention (Ferreira et al., 2010). These interventions also change the organisation of the motor cortical networks (assessed with transcranial magnetic stimulation of the motor cortex) that have input to this muscle (Tsao et al., 2010b) and the amplitude of change in timing is related to the magnitude of cortical reorganisation (Tsao et al., 2010b). Current data do not clarify whether symptom improvement is due to improved activation of transversus abdominis or the resolution of other aspects of the adaptation, such as more optimal control of other trunk muscles or movement and/posture. Regardless, the activation of transversus abdominis serves as a useful marker of the motor recovery.

Other work indicates that activation of another deep trunk muscle, multifidus, can be improved with motor control training and net activity of other more superficial trunk muscles reduced (Tsao et al., 2010a). Application of a similar program to the restoration of coordination of vasti muscles of the knee successfully changes relative timing of muscles (Cowan et al., 2002a; Cowan et al., 2003) and this is linked to clinical improvement.

An important observation in this work is that improvements in motor control appear dependent on conscious and precise correction of movement and muscle activity. Simple activation of the muscles without feedback or error correction, such as the activation of transversus abdominis during a sit-up, does not lead to changes in temporal or spatial features of muscle activation (Tsao and Hodges, 2007). This is consistent with the observation that cortical plasticity is dependent on the extent of conscious attention and skill during motor training (Karni et al., 1995; Plautz et al., 2000; Remple et al., 2001), and that movement repetition in the absence of skill or precision does not induce reorganisation (Plautz et al., 2000; Remple et al., 2001). Although other clinical techniques may also change coordination (e.g. application of tape over the skin modifies symptoms and muscle activation (Crossley et al., 2000; Cowan et al., 2002b; Macgregor et al., 2005); joint manipulation/mobilisation changes transversus abdominis activation is some (Marshall and Murphy, 2006), but not all trials (Ferreira et al., 2007)), current data indicate motor control training with conscious attention to correction of motor control is effective.

Does pain interfere with motor learning? It has been suggested that pain may interfere with plastic change in the motor cortex (Boudreau et al., 2007). That study showed when a tongue protrusion task was performed in the presence of pain, the expected training-induced increase in responsiveness of the corticomotor pathway was reduced. This was argued to be due to changes in cortical excitability in presence of pain (Le Pera et al., 2001). Although it is possible that pain may affect learning processes and animal studies indicate compromised capacity for learning due to pain-induced changes at the spinal cord (Hook et al., 2008), inspection of data from the study of Boudreau et al. (2007) suggest that failure of adaptation could be secondary to modified performance of the training task during pain. If quality of movement is maintained, the training-induced changes are unaffected by pain (Tucker et al., 2010). Thus pain may compromise learning by its affect on the performance of the task that is practiced rather than a direct effect of pain on plastic processes that are required for learning in the motor system. This highlights that quality of training is likely to be a key determinant of treatment success, and the poten-

tial benefit of combining training with other modalities to relieve pain. Other work also highlights the ability of an intervention to change motor function depends on the quality of training; higher quality training induces larger changes in temporal aspects of muscle activation (Tsao and Hodges, 2007). Taken together these findings indicate that motor plasticity may be enhanced by training without pain provocation and training with high quality feedback. Although some methods are available to assess motor adaptation and provide feedback (e.g. electromyography (Cowan et al., 2002a), ultrasound imaging (Richardson et al., 1999; Teyhen et al., 2007; Stokes et al., 2007) to change the redistribution of activity within and between muscles further technological development is likely to be required to optimise this approach across a range of conditions.

6. Conclusion

The new theory of motor adaptation to pain provides a more comprehensive explanation of clinical and experimental observations. The theory also provides a range of principles that can be applied and trialed for the rehabilitation of musculoskeletal pain. Some of these implications are supported by data from clinical trials whereas others provide predictions that require testing to confirm the magnitude of potential clinical effects.

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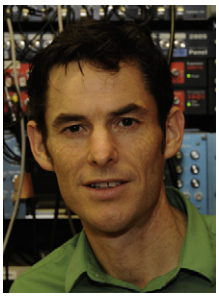

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