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Review

Moving differently in pain: A new theory to explain the adaptation to pain

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1. Introduction

People move differently in pain. Although this statement is unquestioned, the underlying mechanisms are surprisingly poorly understood. Existing theories are relatively simplistic, and although their predictions are consistent with a range of experimental and clinical observations, there are many observations that cannot be adequately explained. New theories are required. Here, we seek to consider the motor adaptation to pain from the micro (single motoneuron) to macro (coordination of whole-muscle behaviour) levels and to provide a basis for a new theory to explain the motor changes in pain.

2. Contemporary theories of the motor adaptation to pain

Two major theories have emerged to explain the changes in movement that accompany pain. These are the vicious cycle [70] and pain adaptation theories [48]. The vicious cycle theory hypothesizes that muscle activity increases in a stereotypical manner in pain, regardless of task, yet sustained activity leads to ischaemia and accumulation of algesic agents, producing pain [70]. A range of mechanisms underlying the increased activity has been proposed, including increased sensitivity of muscle spindles via inputs from group III and IV afferents (nociceptive muscle afferents) onto gamma motoneurons [39]. This is supported by increased response to stretch reflexes in human jaw [87,100,101] and calf muscles [57], and cat hind limb muscles [91].

The pain adaptation theory, first proposed by Lund et al. [48], explained more variable changes in muscle activity with pain. The theory, which is based on experimental observations, proposed that activity of muscles that are painful or that produce a painful movement reduces during voluntary efforts, whereas that of opposing/antagonist muscles increases [48]. This adaptation reduces the amplitude and velocity of the painful movement, and it decreases the force produced by the muscle. A range of data underpins this theory. For instance, experimentally induced muscle pain in humans decreases maximal force [23,55], and when pain is induced in a jaw muscle, the velocity and amplitude of jaw movement decreases [86]. Evidence of differential effects, depending on function of the muscle, comes from studies of induced pain in muscles such as the erector spinae during gait [2] and forward bending [105]. In these cases, activity increased when the muscle is normally inactive and decreased when the muscle is normally active. Furthermore, during dynamic leg movements, muscle pain decreased agonist muscle electromyographic activity (EMG) and increased antagonist muscle EMG [24]. At a micro level, observations of reduced motoneuron discharge rate (a determinant of muscle force) during constant force contractions were interpreted to be consistent with this theory [15,80]. The pain adaptation theory proposed that the inhibitory and excitatory inputs were mediated at the spinal cord (via interneurons or direct inputs from nociceptive afferents onto motoneurons) or brain stem, although the mechanisms were not clearly defined.

3. Problems with existing theories

3.1. Pain does not have a uniform effect on excitability of the motor pathway

There is no doubt that some observations are congruent with predictions of existing theories [2,11], but numerous observations are not. In terms of the vicious cycle theory, although increased muscle activity and spindle discharge are reported during pain [9], many observations are inconsistent with this theory [48]. A critical inconsistency is evidence of variable changes in muscle activity. Although injection of glutamate into the temporomandibular joint in rats induces a prolonged increase in EMG activity of muscles that close (masseter) and open (digastric) the jaw [7], induced pain in humans can increase [11,79,86], decrease [11,14] or not change [16,56,77] muscle activity. Furthermore, if EMG increases, it does not last the duration of the painful stimulus [85]. Finally, changes in muscle activation cannot be accounted for by effects at the muscle spindle because activity of jaw-opening muscles is modified by pain, despite the absence of muscle spindles [69]. Taken together, these data contradict the generalisability of the predictions of the vicious cycle theory. A more comprehensive theory is required that accounts for variable patterns of increased and decreased muscle activity.

An underlying premise of the pain adaptation theory is that of uniform inhibition of motor drive to muscles that are painful or produce a painful movement. Although this premise is supported

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by some recordings of muscle activity using surface electrodes (e.g., decreased masseter muscle activity during chewing [83]), there is also contrary evidence [79]. Furthermore, measures of excitability along the motor pathway from the motor cortex to the motoneuron are variable. Direct recordings of motoneuron membrane properties in animals show both excitatory and inhibitory postsynaptic potentials in response to inputs from group III and IV nociceptive muscle afferents [41]. In humans, the effect of nociceptive afferent discharge on motoneuron excitability has been investigated with several methods. The Hoffman reflex (H reflex) is the electrical analogue of the stretch reflex whereby a muscle response is evoked by reflex excitation of the motoneuron in response to an afferent volley (mainly 1a afferents from muscle spindles) excited by electrical peripheral nerve stimulation. H reflex amplitude is reduced in wrist flexor muscles during homonymous muscle pain [46] but is not changed in human leg [56], hand [17], or jaw muscle pain [84]. However, the H reflex does not provide a pure measure of motoneuron excitability because its size is affected by mechanisms in addition to changes in motoneuron excitability. This includes presynaptic inhibition of the 1a afferent terminal [73], amongst other problems. The stretch reflex, which depends on various factors including motoneuron excitability, spindle sensitivity, gamma motoneuron drive to the intrafusal muscle fibres, and presynaptic effects on the Ia afferent synapse, increases [87,100,101] or decreases [4,88] in pain.

The response of a muscle to electrical stimulation of the descending corticospinal axons at the cervicomedullary junction has been studied [21,55]. Because this input to motoneurons is not affected by presynaptic inputs [64], it provides a more accurate measure of motoneuron excitability. By means of this method, biceps brachii muscle pain has been shown to facilitate the motoneurons to flexor and extensor muscles, contradicting the pain adaptation theory's prediction of opposite effects on antagonist muscles. Further, sustained discharge of group III and IV afferents does not maintain changes in motoneuron responsiveness to cervicomedullary stimulation after fatiguing muscle contractions [21]. These observations question the uniform inhibition of motoneurons innervating a painful muscle.

Transcranial magnetic stimulation (TMS) over the motor cortex has been used to study the responsiveness of the corticomotor system, including the cortex. Motor-evoked potentials (MEPs) to TMS decrease [17,42,46,55,98], increase [1,11,12], or do not change [71] during local muscle pain. MEP amplitude also changes variably during remote pain: biceps MEPs reduce during hand pain [97] but increase during pain at the tip of the index finger [42]; abductor digiti minimi MEPs are reduced during pain in the first dorsal interosseus, but not the opposite hand [42,46]. Recent work highlights that the effect of pain on corticomotor responsiveness can vary between muscles. Experimental back pain in the interspinous ligament decreases MEP amplitude in a deep abdominal muscle, transversus abdominis, but increases MEP amplitude in overlying abdominal muscles (obliquus externus abdominis) and lumbar erector spinae (Tsao, Tucker, and Hodges, unpublished data). In contrast, the threshold for erector spinae MEPs increases in chronic low back pain [81]. Changes in excitability are associated with reorganisation of cortical representation (posterolateral shift) of inputs to transversus abdominis in chronic low back pain [92] (Fig. 1).

Although changes in the response to TMS of the motor cortex have been interpreted to reflect excitability of cortical networks, these responses are affected by both cortical and motoneuron excitability. Several studies sought to distinguish effects between sites. Le Pera et al. [46] aimed to control motoneuronal effects by investigation of the H reflex and showed reduction of both MEP and H reflex amplitudes during pain, but this conclusion is compromised by problems with interpretation of H reflexes (see above). Martin et al. [55] showed depressed excitability of biceps and triceps MEPs, but increased excitability of motoneurons studied using volleys excited by electrical stimulation at the cervicomedullary stimulation. These data suggest opposite effects at cortical and spinal sites. This is not predicted by the pain adaptation theory. Similar differential effects have been shown for the deep paraspinal muscles after injury to an intervertebral disc in pigs by means of similar techniques, but with the opposite pattern of increased cortical and decreased spinal excitability [33]. Other data show no change in MEP amplitude evoked by electrical stimulation of the cortex (because this technique activates cortical cells directly, it is not affected by cortical excitability, and the finding suggests that spinal motoneuron excitability is not changed), but decreased responsiveness to TMS (which activates cortical cells transynaptically and is affected by excitability of cortical cells) [98]. This comparison allows interpretation of changes at the cortex. Recent work investigating intracortical inhibitory and facilitatory circuits shows increased inhibition and decreased facilitation after pain [76], again focussing attention on the cortical components.

In summary, data of excitability along the corticomotor pathway fail to support the predictions of existing models that there will be uniform inhibition (pain adaptation) or facilitation (vicious cycle) of muscles that are either the source of pain or that produce a painful movement. Responses vary between muscles and tasks, and this must be accounted for in theories that explain the adaptation to pain.

3.2. Changes in motor control during pain are not always stereotypical or predictable

Existing theories predict relatively stereotypical change in whole-muscle behaviour, but this has not been observed, and variable patterns of adaptation are identified in clinical populations and in response to experimental pain (e.g., [35,99]). Although some aspects of the motor adaptation to pain are consistent between individuals (e.g., [36,37,50]), changes in behaviour of other muscles are unique to the individual and possibly to the task [36,99]. This is most common in complex systems such as the trunk, where the muscle system has considerable redundancy (multiple muscles achieve a similar goal) [35,99]), and jaw [63,74], where there is complex muscle anatomy [26]. New theories must account for the variability.

Theories also do not explain reduced or delayed activity of some deeper muscles of the trunk in pain (e.g., transversus abdominis [18,36,37] and multifidus [50]). This occurs regardless of movement direction and despite the trivial moment arms of these muscles to generate torque, which means they have trivial potential to act as agonists or antagonists to movement. Furthermore, because these muscles contribute to control of spine motion, the vicious cycle theory may predict their activity would increase to splint a painful spine. However, this is opposite to the reduced activity observed in clinical and experimental pain [18,36,37,50].

3.3. Existing theories do not account for changes in all classes of movement

The pain adaptation theory only makes predictions regarding voluntary movements and ignores changes in other automatic functions such as postural control. The proponents of that theory argued that pain causes little change in postural functions [48]. However, the literature increasingly refutes this claim. There is evidence of changes in balance [6,59] and whole-muscle behaviour in anticipatory [36,37,47] and reactive postural [51,53] mechanisms in experimental and clinical pain. Predictions of the pain adaptation theory cannot be extrapolated to coordination of these auto-

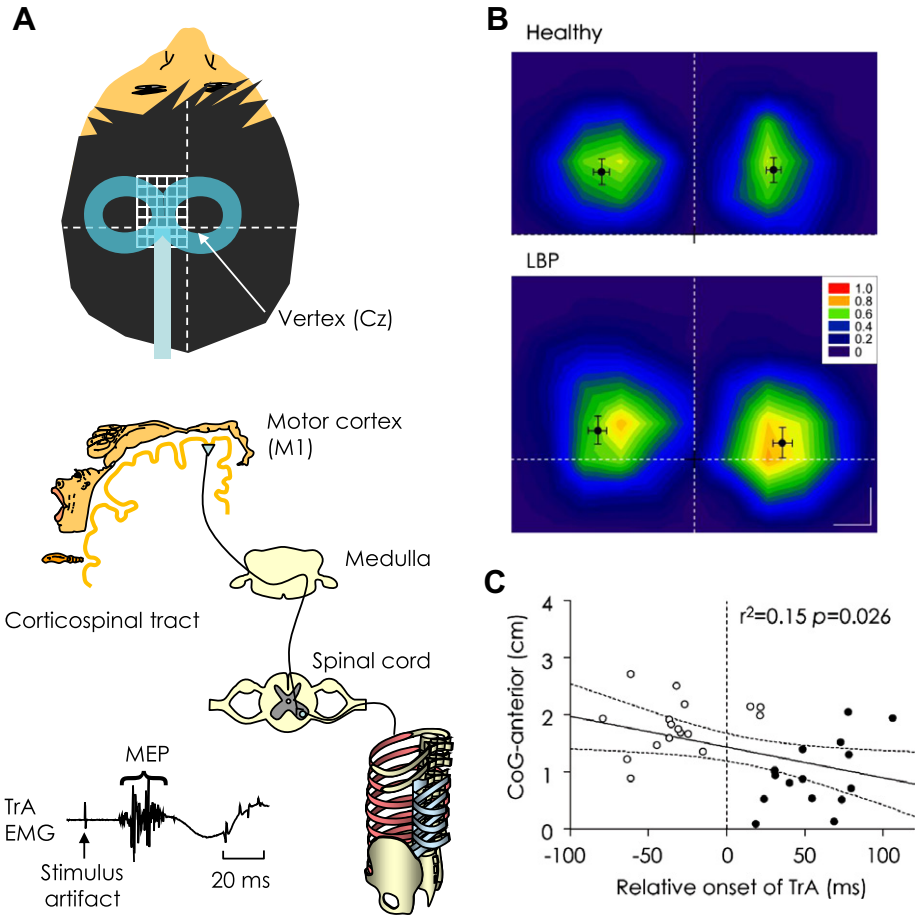

Fig. 1. Changes in motor cortical map with pain. (A) Mapping of the motor cortex using transcranial magnetic stimulation (TMS). Stimuli over the motor cortex (M1) excite intracortical neurons that excite corticospinal cells. Descending volleys excite the spinal motoneurons to produce a motor-evoked potential (MEP) in the contralateral transversus abdominis (TrA) muscle. (B) Average normalised motor cortical maps generated from the MEPs evoked at points on a grid over the cortex on the left and right hemisphere are shown for a healthy and low back pain (LBP) group. Mean (standard deviation) of the centre of gravity (CoG) show a more posterior and lateral location of the CoG relative to the vertex in the LBP group (calibration, 1 cm). (C) Relationship between location of the CoG (distance anterior from the vertex) and timing of TrA electromyographic (EMG) activity during arm flexion relative to that of the arm mover deltoid at time = 0. Individuals with later TrA activation (mostly individuals from LBP group [open circles]) had TrA CoG located more posterior to the vertex. Adapted from Tsao et al. [92].

matic postural adjustments. Postural adjustments can precede arm movement to overcome the perturbation to the body (e.g., early erector spinae activity to overcome the trunk flexion perturbation from arm flexion [38]). If the muscle producing this adjustment was painful and therefore inhibited (e.g., such as may be predicted with erector spinae pain during arm flexion), this would tend to increase the perturbation due to reduced opposition to the reactive moments, rather than reduce it.

3.4. Theories cannot explain the maintenance of force when motoneuron discharge reduces in pain

Although reduced discharge rate of motoneurons innervating muscle fibres in a painful muscle has been interpreted to be consistent with inhibition of the motoneuron pool predicted by the pain adaptation theory [80], several features of this adaptation are not consistent. First, because the experimental tasks required force matching between contractions with and without pain, the adaptation in motor unit discharge did not decrease the force output. Second, because motoneuron discharge rate is a determinant of force, reduced discharge rate during pain must be accompanied by other changes in motor output in order to maintain force. Recent studies show that new motoneurons are recruited during pain, and this implies nonuniform inhibition of the motoneuron pool [93,95] (see below; Fig. 2). This observation may imply that reduced muscle activity is a reflection of processes to change the manner in which the muscle generates force rather than uniform inhibition of a painful muscle. A more complex model of adaptation in pain is required that can account for the changes in motor control.

4. New theory for the motor adaptation to pain

A theory to explain the adaptation to pain must account for each issue highlighted above, particularly the variability between individuals and tasks. We propose a new theory based on existing data at the micro (motoneuron discharge) and macro (whole-muscle behaviour) levels. The theory has 5 key elements that expand on the basic premise that the adaptation to pain aims to reduce pain and protect the painful part, but with a more flexible solution than currently proposed (Fig. 3). We propose that the adaptation to pain (1) involves redistribution of activity within and between muscles; (2) changes the mechanical behaviour such as modified movement and stiffness; (3) leads to protection from further pain or injury, or from threatened pain or injury; (4) 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; and (5) has short-term benefit but has potential long-term consequences due to factors such as increased load, decreased movement, and decreased variability. Each aspect and the supporting data are described below.

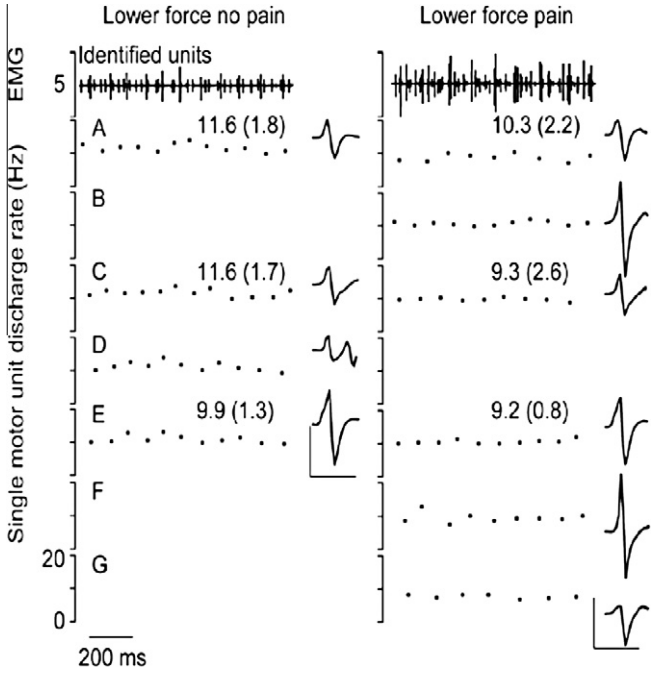

Fig. 2. Redistribution of activity within a muscle. Recordings of flexor pollicis longus (FPL) single motor unit electromyography (EMG) are shown with their discharge rates and spike-trigger averaged electrical profile. Three motor units recruited during no-pain and pain trials (A, C, E) decreased their discharge rate during pain. Unit D was derecruited during pain. Three new units (B, F, G) that were not active in the no-pain conditions were recruited during pain. These features indicate a change in the population of active units during pain in order to maintain force output. Adapted from Tucker et al. [93].

4.1. Pain leads to redistribution of activity within and between muscles

A key aspect of the new theory is that rather than uniform inhibition or excitation of muscles or motoneuron pools, we propose that inputs to motoneurons may be unequally distributed with redistribution of activity between regions within a muscle or between muscles in an individual- and task-specific manner, but with a common goal to protect the painful part from further pain or injury (Fig. 3). There is evidence of such redistribution from studies of muscle activation at micro and macro levels. Redistribution of activity within a muscle provides an alternative explanation for decreased motoneuron discharge rate during pain [16,32,80]. Although discharge rate is consistently reduced or ceased in motoneurons active before and during pain, recent work shows force is maintained by recruitment of a new population of units that were not active before pain [93,95] (Fig. 2). This could not occur with uniform inhibition of the whole motoneuron pool and may be explained by either a change in motoneuron recruitment order to recruit larger units at lower forces (perhaps to enhance the rate of force development as part of a fright/flight response), or by a change in the distribution of activity within a muscle (perhaps to preferentially activate muscle fibres with a specific force direction to change load distribution on the painful structure).

In general, it is considered that motoneurons are recruited in an orderly manner from small to large [27,28], on the basis the assumption that drive to a motoneuron pool is evenly distributed and that electrical properties mean that small motoneurons reach their threshold for discharge earlier. Few examples of contravention of this order have been identified, but it has been reported with nonphysiological electrical afferent stimulation [22,78] and can be achieved volitionally [68,89,90]. Earlier recruitment of larger units may have the benefit of enabling faster development of force to facilitate escape of the individual from threat such as pain or injury. An unequal balance of excitatory and inhibitory inputs [40] may mediate the departure from orderly recruitment from nociceptive afferents onto motoneurons. Inhibitory inputs have been suggested to evoke larger inhibitory postsynaptic potentials on smaller motoneurons [49], leading to slowing or derecruitment of smaller units. Higher drive, and activation of higher-threshold units, would then be required at a lower force. Furthermore,

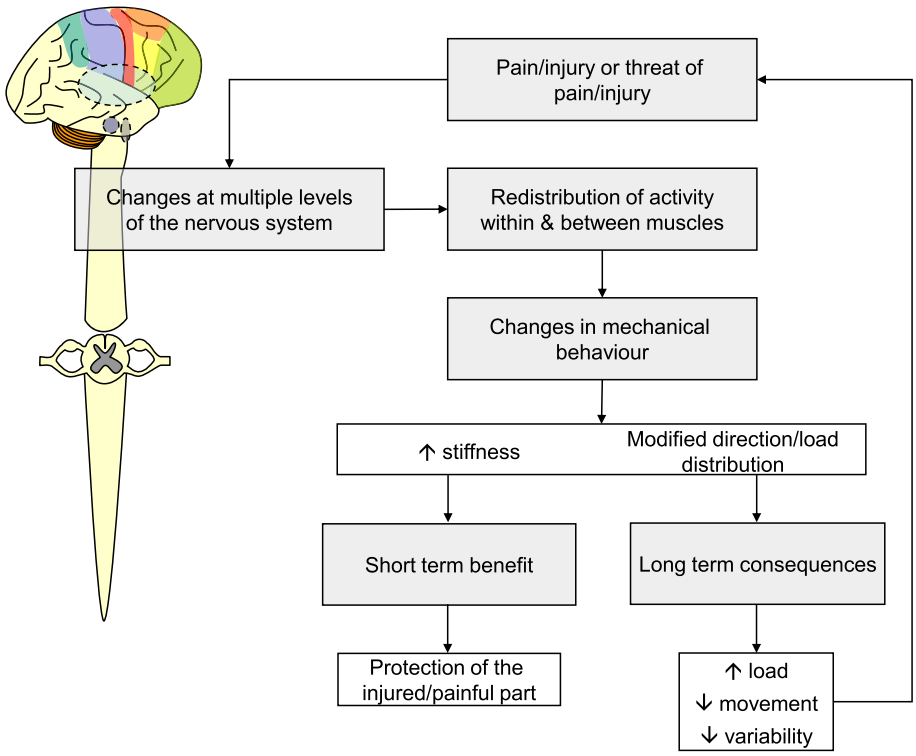

Fig. 3. New theory of motor adaptation to pain.

introduction of Renshaw-cell-mediated recurrent inhibition could slow or inhibit discharge of smaller low-threshold motor units upon recruitment of larger units [10,19,72]. Nonuniform effects on the motoneuron pool may explain the variability observed in studies of motoneuron excitability. For instance, facilitation of larger units would lead to greater MEP amplitude to cervicomedullary stimulation [55], despite inhibition of smaller motoneurons.

The alternative argument is that the population of active units is changed to alter the distribution of force in the muscle with or without a change in the direction of net force generated by the muscle [96]. A new distribution of force or net force direction would change load distribution and may be less painful or less injurious for the painful tissue. Individual motor units within a muscle have slightly different directions of force production as a result of variation in muscle fibre angle and attachments [5,82] and may be associated with contractions of different type or orientation [68,89,90,104]. This spatial redistribution of activity may occur in conjunction with a change in recruitment order, or it could be misinterpreted as a change in recruitment order because units may be activated at a lower force (appearing as a change in recruitment order) in the new direction if it is the muscle fibre's preferred direction of force. Consistent with the proposed change in population of active units, several studies show spatial redistribution of activity between regions of muscle. For instance, of 53 vasti muscle EMG recording sites (up to 7 per subject) during pain induced by injection of hypertonic saline into the infrapatellar fat pad, 38% had a >20% increase in EMG amplitude and 25% had a >20% decrease [95]. Spatial redistribution of activity has also been recorded with array electrodes, revealing a shift of activity away from the site of pain injection in the upper trapezius [52]. Spatial redistribution would not be detected with a single pair of surface electrodes placed over the whole muscle. This could contribute to variability between studies [2,3,13,15,29,77].

In some body systems, particularly those with substantial redundancy such as the trunk muscles, spatial redistribution of activity has been observed between muscles. For instance, delayed/reduced activity of transversus abdominis is accompanied by an individual-specific increase in activation of other abdominal and back muscles as a part of the postural adjustment before arm movement [36]. Furthermore, although the net activity of the trunk muscles increases during simple trunk movements with experimental pain, this increase is achieved with different patterns of increased and decreased activity in each individual participant [30]. Pain is also associated with a change in relative timing of activation of medial and lateral heads of the quadriceps [8,34], which is coupled with reduced synchronisation of discharge of motoneurons in these two muscle heads [58].

4.2. Adaptation to pain changes mechanical behaviour

A central premise of the new theory is that the redistribution of activity within and between muscles changes the mechanical outcome of contraction. Recent work shows that changes in the population of active units within the quadriceps during experimental

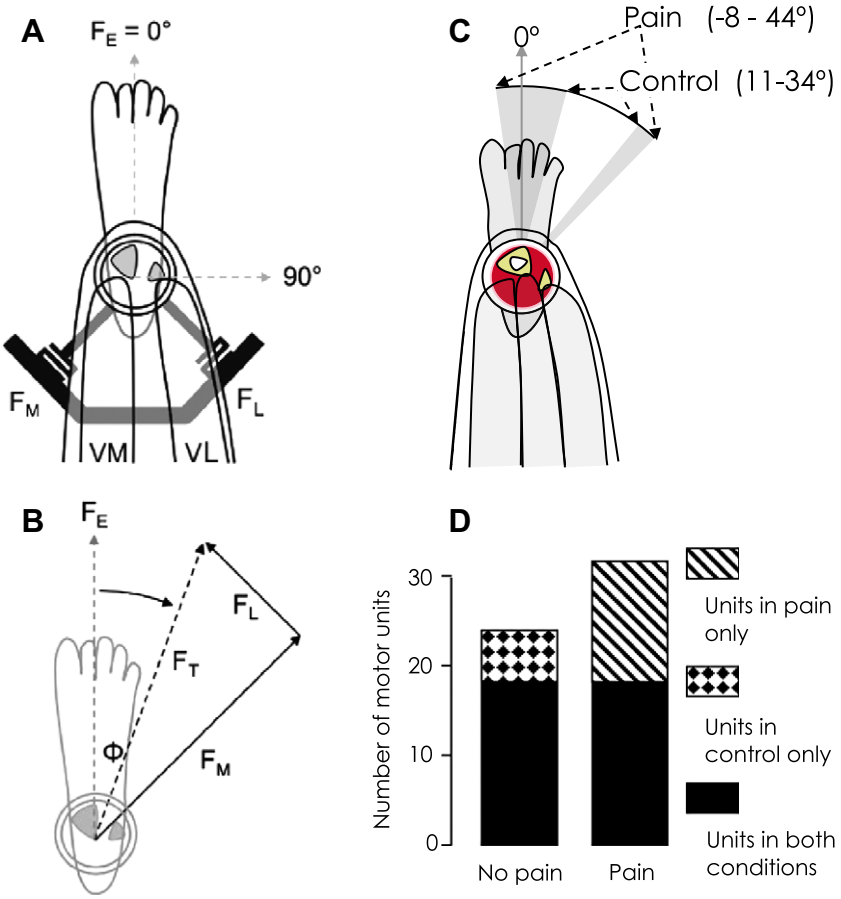

Fig. 4. Changes in knee extension force direction with pain. (A) Isometric knee extension force was measured from two force transducers (force medial [FM] and force lateral [FL]) positioned at 90° to each other and attached above the subject's ankle. (B) Knee extension force (FE), total force (FT), and angle of FT (Φ) were calculated from FM and FL. (C) Before pain, knee extension was performed at an angle of 11–34° to the sagittal plane. During pain, the angle changed either medial or lateral to this control angle. (D) Changes in force direction were associated with redistribution of activity within the quadriceps muscles. This included reduced discharge rate of units active before and during pain, derecruitment of some units, and recruitment of a new population of units. Adapted from Tucker and Hodges [96].

knee pain (redistribution within muscle) change the direction of knee extension force a few degrees medial or lateral to that in the pain-free trials [96] (Fig. 4). Similar to this variation in mechanical change between individuals (medial vs. lateral change in direction of knee extension force), studies of jaw movement highlight that adaptation is common, but the specific changes vary between individuals [63,75]. Redistribution of activity between trunk muscles also changes kinematics and mechanical properties of the spine. During walking, the normal counterrotation of the thorax and abdomen is changed to more en bloc movement in clinical [45] and experimental pain [44]; stiffness (i.e., control of displacement) of the trunk is increased in clinical back pain, but this is at the expense of damping (i.e., control of velocity) [31] (Fig. 5); and movement of the trunk in anticipation of arm movement is reduced [60]. In each of these cases, the gross features of the task are maintained, but the quality is affected, and this may have consequences for the individual.

4.3. Adaptation to pain leads to protection from pain or injury, or threatened pain or injury

The change in distribution of activity within and between muscles and the resultant change in mechanical behaviour is proposed to protect against further pain, injury, or both. This is consistent with the theoretical proposal of Murray and Peck [63] that the nervous system may search for a movement pattern that is less painful during painful mastication. The examples provided in the preceding section can be interpreted in this context. Change in direction of knee extension force associated with redistribution of activity within the vasti muscles would change load on the infrapatellar fat pad that lies under the patellar tendon (the structure injected with hypertonic saline in those experiments), and this could modify mechanical irritation of this structure [96]. Increased trunk stiffness [31] and decreased counterrotation of thorax and pelvis in gait [45] would splint the spine and prevent ongoing irritation of sensitive or sensitised structures. Such adaptation would also be expected with the threat of pain, injury, or both, in the absence of current pain, injury, or both. Changes in distribution of activity within [94] and between muscles [62] have been reported when pain is threatened (anticipation of painful electrical shocks).

Many different adaptations in muscle activity may achieve protection. In addition to the examples presented above, this would include inhibition of agonist muscles to reduce voluntary movement force and displacement (predicted by the pain adaptation theory [48]); increased muscle activity to splint the painful part (predicted by the vicious cycle theory [70]); and other observations, such as a lowered threshold for flexor withdrawal reflexes as a result of central sensitisation [102,103].

The unique feature proposed in the new theory is that rather than a stereotypical change that is the same in all conditions, we propose the nervous system has a range of options to achieve the goal of protection, and this may involve increased, decreased, or redistributed activity. This will involve more complex neural processes than those proposed by the existing theories that advocate stereotypical change.

4.4. Adaptation to pain involves changes at multiple levels of the motor system

Although changes in excitability of motoneurons may underlie or contribute to changes in muscle activity during pain [55], this is not sufficient to explain the complexity of adaptation. Changes at the multiple sites along the motor pathway may be complementary, additive, or competitive. As mentioned earlier, motoneuron excitability can be increased but accompanied by decreased cortical excitability [55] and increased intracortical inhibition [76]. The mechanisms at each site may be different. For instance, spinal effects may be mediated by direct input of nociceptive afferents on

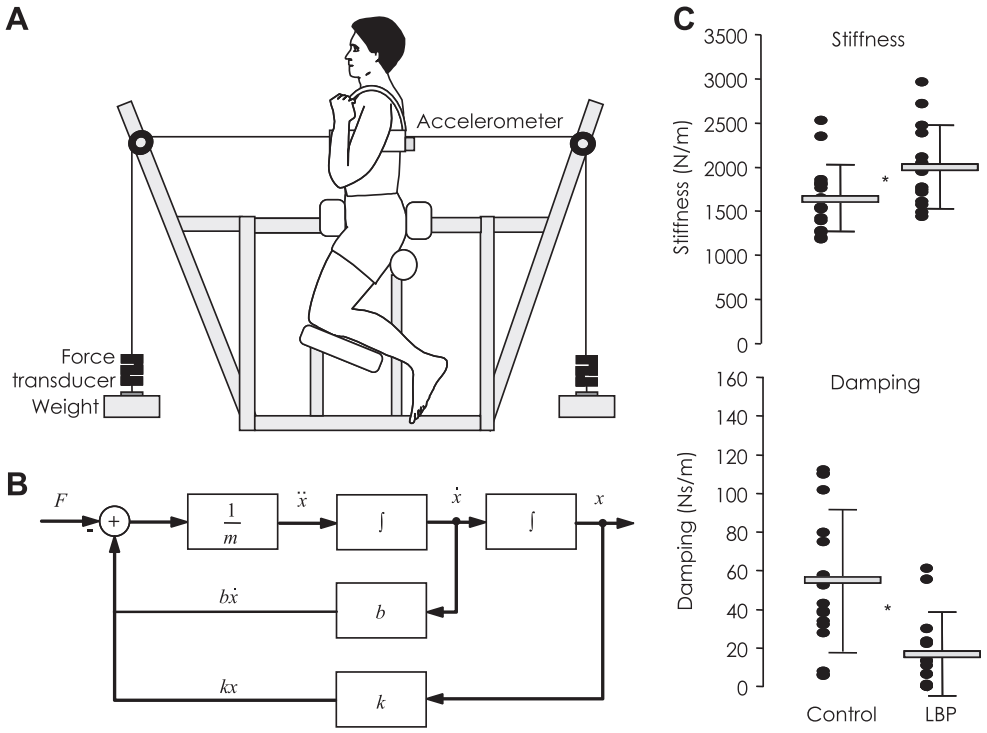

Fig. 5. Changes in spinal stiffness and damping with pain. (A) In sitting, a load was released from one side of the trunk by deactivating an electromagnet to perturb the trunk. (B) Linear second-order feedback-control model. *F* is an input force acting on mass *M*. The resulting velocity ($\dot{x}$) and displacement (x) are then fed back with gains *B* (damping) and *K* (stiffness), respectively, to achieve the desired output displacement x ($\ddot{x}$ – acceleration). (C) Effective trunk stiffness was increased and damping was decreased for people with low back pain (LBP) compared to control participants. Mean (± standard deviation) and individual data are shown. * $P < .05$. Adapted from Hodges et al. [31].

motoneurons [40] or functional plasticity in the spinal cord elicited by nociceptive primary afferent inputs (i.e., central sensitisation [102]), whereas cortical changes may be due to changes in motor planning, such as the recruitment of a more protective strategy in advance of movement (e.g., manipulation of the sequence of trunk muscle activation before arm movement in anticipation of threatened pain in the absence of nociceptor discharge [62]) or reorganisation of cortical regions [92] (Fig. 1). The net output of the motor system would be dependent on the relative impact of the events throughout the motor system, and this may vary between individuals and tasks, which may account for some of the variability in experimental findings.

4.5. Adaptation to pain has short-term benefit, but with potential long-term consequences

A final aspect of the theory is that although the adaptation achieves a short-term goal of protection from further pain, injury, or both, the adaptation may have consequences that could lead to further problems in the long term [35]. We argue that if it is assumed that movements are performed in an optimal or efficient manner in a nonpain state, departure from this state may not be ideal. This could be due to increased or modified load, decreased movement, decreased variability, or other changes. Redistributed or increased muscle activity to splint or protect a painful part during the acute episode will change [96] (Fig. 4) or increase load [54] on the painful part, and this may have detrimental effects in the long term. For instance, high cumulative load on intervertebral discs, which would be a likely outcome from trunk muscle splinting [54], may lead to mechanical and physiological changes in the disc [43,65]. Furthermore, modified mechanics of the proximal lower limb joints during gait to avoid painful ankle dorsiflexion after ankle sprain [20] could lead to further problems as a result of decreased shock absorption from modified joint position at heel strike. Spinal movement is necessary to dampen forces. However, movement is reduced in pain [45,60], and damping is reduced [31] (Fig. 5), which may enhance force impact on the spine. Finally, some variability in performance of movement has the advantage of varying the areas of joint load, muscle activity, and ligament stress. This would be compromised if the adapted protective strategies lead to a reduction in variation [25].

The proposed negative outcomes of adaptations are not likely to be immediate and would require a period of maintenance/repetition to influence tissue health. This would limit the capacity of the nervous system to identify any potential negative impact, thus limiting any motivation to overcome the adaptation. Although pain provides a potent stimulus to change the movement strategy to protect the painful or injured part, resolution of pain or injury does not necessarily provide a stimulus to return to the initial pattern. In terms of motoneuron recruitment, discharge rate of active units recovers with resolution of pain, but the redistribution of activity within a muscle does not [94]. At the level of whole-muscle behaviour, some individuals, particularly those with unhealthy attitudes about pain, are less likely to restore muscle recruitment patterns to a prepain state [61].

Recurrence/persistence of pain is common after an initial episode (e.g., 73% of those with an acute episode of back pain experience a recurrence within 1 year [67]). Although it is possible that the failure of the adaptation to pain to resolve after the initial episode may contribute to the ongoing problems, an alternative view is that the adaptation compensates for a failure of support by injured passive joint structures and is therefore necessary for normal function [66,99]. There is likely to be a delicate balance between positive and negative aspects of the adaptation. Longitudinal studies are required to confirm whether nonresolution of adaptation is associated with long-term consequences.

5. Conclusion

We present a new theory for the motor adaptation to pain that is consistent with clinical and experimental observations and provides a range of testable hypotheses. A key aspect that requires further clarification is that although the adaptation has immediate potential benefit for the system, there may be long-term consequences for the health of the individual. Our theory presents candidate targets for new and refined treatments for rehabilitation of people in pain.

6. Conflict of interest

There are no conflicts of interest.

Acknowledgments

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