Focus Article

Orofacial Pain and Jaw Muscle Activity: A New Model

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Two major theories proposed to explain the effect of pain on muscle activity are the Vicious Cycle Theory and the Pain Adaptation Model. Comprehensive reviews demonstrate conflicting or limited evidence in support of a critical aspect of the Vicious Cycle Theory, namely that pain leads to increased muscle activity. The Pain Adaptation Model proposes that changes in muscle activity limit movement and thereby protect the sensorimotor system from further injury. This model is generally considered the most appropriate explanation of the effect of pain on muscle function. Although there is much literature consistent with the model, there are a number of lines of evidence that appear inconsistent with it. Possible reasons for the lack of consistency between studies include the functional complexity of the sensorimotor system (eg, the possibility of different pain effects at different sites within functionally heterogeneous muscles), and the multidimensional nature of pain. The latter consists of sensory-discriminative, cognitive-evaluative, and motivational-affective components, where factors such as pain location, intensity, and characteristics and other supraspinal/suprabulbar influences may modify the effects of pain on motor activity. The variety of changes in electromyographic (EMG) activity features during pain suggests that pain and motor function are not hardwired. The authors propose that the existing Pain Adaptation Model is a subset of a broader model that could be called the Integrated Pain Adaptation Model. Given the recent view of pain as a homeostatic emotion requiring a behavioral response, this new model states that pain results in a new, optimized recruitment strategy of motor units that represents the individual's integrated motor response to the sensory-discriminative, motivational-affective, and cognitive-evaluative components of pain. This recruitment strategy aims to minimize pain and maintain homeostasis. J OROFAC PAIN 2007;21:263–278

Key words: experimental pain, jaw muscle activity, muscle movement, Vicious Cycle Theory, Pain Adaptation Model

The following article is based on the literature cited in a number of major recent reviews[1–4] and a Medline search from 2000 to June 2006 with the search terms *muscle contraction* or *muscles* or *muscle, skeletal* or *electromyography* or *Temporomandibular Joint Dysfunction Syndrome* or *masticatory muscles* and *low back pain* or *abdominal pain* or *facial pain* or *myofascial pain syndromes* or *back pain* or *neck pain* or *pain* or *shoulder pain*. The references were initially screened on the basis of titles and/or abstracts, and those containing a statistical comparison of electromyographic (EMG) activity and pain were included for detailed review.

Fig 1 *The basic proposals for* (a) *the Vicious Cycle Theory and* (b) *the Pain Adaptation Model. Summary diagrams and legends reprinted, with permission, from Lund and Sessle[21] and Lund.[22]*

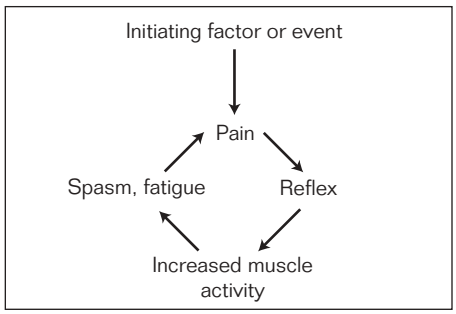

Fig 1a Suggested neural pathways forming the basis of the pain-spasm-pain cycle. Some initiating factor or event causes pain, which reflexively causes increased muscle activity or "muscle hyperactivity," which leads to further spasms, fatigue, and further pain.

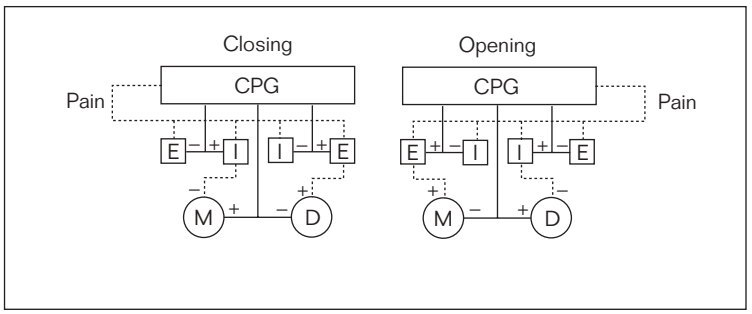

Fig 1b Theoretical model illustrating the effects of tonic pain (broken lines) on the masticatory central pattern generator (CPG) and on excitatory (E) and inhibitory (I) interneurons. Pain afferents converge with CPG inputs onto common interneurons, which project to motoneurons (M: masseter; D: digastric). During closing in pain, nociceptive inputs enhance the excitability of the inhibitory interneurons that project to jaw-closer motoneurons (eg, masseter motoneurons) and enhance the excitability of excitatory interneurons projecting to jaw-opener motoneurons (eg, digastric motoneurons). The pattern is reversed during opening.

Temporomandibular disorders (TMD) are a group of conditions that involve 1 or more of the following symptoms: pain in or about the jaw joint and/or jaw muscles, limitation of jaw movement, and joint sounds.[5] They are the most prevalent chronic pain condition in the orofacial area.[6–11] Their severity prompts 5% to 10% of the population to seek treatment.[6,12] TMD can considerably affect a patient's work, family life, and social activities and can also result in nutritional deficiencies because of the discomfort associated with eating.[4,13] While the etiology of TMD remains elusive, a number of recent reviews have shed light on the possible pathophysiology of TMD and other musculoskeletal disorders.[13,14]

Myofascial pain is a common subset of TMD, and it is well known that jaw muscle pain and motor function are interrelated. The exact nature of the interrelationship between muscle pain and motor function has been the topic of much research over the years.[1,3,4,12,15–18] Two major but conflicting theories have been proposed to explain the effect of pain on muscle activity: the Vicious Cycle Theory and the Pain Adaptation Model (Fig 1). The Vicious Cycle Theory essentially proposes that an initiating factor, such as an abnormality in structure, posture, movement, or stress, results in pain that reflexively leads to "muscle hyperactivity." This, in turn, leads to spasm or fatigue and thereby to further pain and dysfunction, thus perpetuating the cycle (Fig 1a).[4,12,15–17,19] It was recently proposed that activation of muscle nociceptive afferents through local inflammatory substances causes increased firing of spindle afferents via direct excitation of γ-motoneurons. This increased spindle firing is in turn associated with a deterioration in the fidelity of spindle afferent transmission, a loss of proprioceptive acuity, less efficient muscular coordination, and increased accumulation of inflammatory substances.[16,20] Management has been advocated to attempt to break the vicious cycle.[15,19]

The Pain Adaptation Model, on the other hand, proposes that pain arises from causes other than muscle hyperactivity (causes the model does not presume to explain) and that pain leads to alterations in muscle activity that limit movement and thereby protect the skeletomotor system from further injury and promote healing.[2,21,22] It is generally considered to be the most appropriate explanation of the effect of muscle pain on muscle function in sensorimotor systems in the body.[1,4,18] Activity in thin nociceptive afferents is said to influence motor function by acting on the central motor program directly (eg, via the masticatory central pattern generator [CPG] shown in Fig 1b) and on interneurons (E and I in Fig 1b). The result is a facilitation of pathways that are inhibitory to muscles when the muscles act as agonists and facilitation of excitatory pathways during antagonist

activity (Fig 1b), which generates slower and smaller movements to reduce the chance of aggravating the injury and therefore aids healing. The effect of the pain is not restricted to the muscle in which it arises, and the effect occurs irrespective of whether pain originates in the muscle or the joint around which the muscles act.

Evidence Supporting and Refuting the Vicious Cycle Theory

There is conflicting or limited evidence in support of a critical aspect of the Vicious Cycle Theory, namely that pain leads to increased jaw muscle activity (Fig 1a).[1–4,22] For example, painful injections of hypertonic saline into the masseter[23] or the tibialis anterior muscles[24] did not change α-motoneuron excitability as determined by H-reflex amplitude assessments. Where increases in EMG activity have been associated with pain, it has been possible to explain these increases by possible contamination of surface jaw muscle EMG activity recordings from activity in muscles of facial expression,[2,25] which frequently become active with pain.[1] With clinical muscle pain, both increases and decreases, as well as a lack of change, in EMG activity have been observed. This variety of EMG effects may reflect a possible contamination of the EMG signal and/or improper matching of patient and control groups.[26] Although a number of well-controlled studies have demonstrated increased jaw muscle activity,[1,25,27] the increases are usually very small and not to a level that would be considered "hyperactive" or expected to cause pain.[1,2] An increase in jaw muscle EMG activity of only a few microvolts, as reported in these studies, is likely to be of no clinical significance. In addition, there is a poor association between the parameters of experimentally evoked pain and changes in EMG activity, although the Vicious Cycle Theory proposes otherwise. For example, hypertonic saline injections into the masseter muscle have resulted in pain lasting up to 600 seconds but in increases in masseter EMG activity of only 30 to 60 seconds in humans[28] and experimental rats.[29] However, reflexly induced increases in EMG activity of jaw-opening and jaw-closing muscles have been observed with injections of algesic chemicals into the temporomandibular joint (TMJ) or other orofacial tissues of rats.[13,30–32] Nonetheless, equivocal results have been obtained in reflex studies where chronic pain patients demonstrated significant decreases in masseteric reflex amplitude,[33] while a number of experimental pain studies have observed short-term increases in masseteric reflex amplitude.[34–39]

Although the Vicious Cycle Theory proposes pain would not change EMG activity associated with maximum voluntary contractions, significant reductions in EMG activity with experimental or clinical pain have been demonstrated in a variety of sensorimotor systems.[1–4] Furthermore, there was no evidence of differences between a TMD group and controls in jaw-muscle EMG activity measures (mean power frequency, bandwidth, or root-mean-square).[40]

With respect to the other arm of the Vicious Cycle, namely, that increased muscle activity leads to pain (Fig 1a), there is also conflicting evidence. Thus, while short-duration repetitive muscle activation led to muscle pain in humans,[41–43] these pain-related changes, while significant, were usually mild and short-lived. Interestingly, experimental clenching performed repeatedly over 5 days resulted in moderate pain levels that either reduced in intensity over the duration of the experiment[44] or, in a few subjects in another study, contributed to a diagnosis of TMD.[45] However, prophylactic intake of tolperisone hydrochloride, a muscle relaxant, provided no relief to postexercise muscle soreness pain, despite resulting in a reduction in isometric force.[46] It is possible, however, that in certain susceptible individuals, persistence of post-exercise muscle soreness pain may occur in association with central sensitization, which appears to be a feature of chronic pain patients.[13] It is also possible that changes in muscle activity in association with disability or dysfunction lead to further pain, injury, and disability for reasons that have yet to be elucidated.[47]

Evidence Supporting and Refuting the Pain Adaptation Model

There is indeed much data in the spinal and trigeminal literature consistent with or supportive of the Pain Adaptation Model (see reviews[1–4,18,21,22]), especially at moderate to maximal muscle activation levels.[48] In general terms, clinical or experimental pain in humans results in smaller and slower movements than in controls[49–52] (see reviews[1,3,4]). In chewing during pain, for example, there were significant reductions in displacements in the vertical and lateral axes (10% and 23%, respectively), in the opening and closing velocities (11% and 15%, respectively), and in the cumulative distance (11%).[50]

The abundant evidence that the level of resting EMG activity is either no higher or only slightly higher than normal in a range of musculoskeletal conditions is generally consistent with the Pain Adaptation Model, which proposes no change in muscle activity at rest (see reviews[1–4]). In addition, clear evidence for inhibition of agonist activity is seen in the reductions, in comparison with pain-free controls in maximum voluntary contraction force and/or EMG activity

- In chronic TMD pain patients[53–55] and experimental pain patients[56,57]
- In maximum voluntary jaw opening in TMD patients[58]
- Following wisdom tooth removal or minor oral surgery[59,60]
- During empty open-close-clench cycles in the presence of pain induced by hypertonic saline into the left masseter muscle[2]
- During maximum voluntary contraction in the anterior tibialis and with hypertonic saline injection into the anterior tibialis[61]
- In chronic low-back pain patients[3,62]

In addition, continuous infusion of hypertonic saline into the masseter resulted in significantly reduced average EMG activity for the masseter and/or anterior temporalis muscles during the closing phase of the chewing cycle during mastication.[50,56] There is good evidence that the changes in EMG activity are due to central factors and not to changes in the peripheral sensorimotor apparatus.[2,63] There is also evidence for excitation of antagonist muscle activity (eg, increased jaw-closer activity during jaw opening) during maximum opening under experimental pain in comparison with pain-free controls,[2] and during the opening phase of the chewing cycle in TMD patients.[55,64]

Further evidence in support of the Pain Adaptation Model comes from the effect of experimental or clinical pain on jaw reflexes. The jaw-closing reflex appears to be facilitated by experimental jaw muscle pain.[34–39] It has been argued that, because of the lack of an effect of pain on the H-reflex amplitude,[23,24] the increased jaw-reflex activity is caused by increased fusimotor drive, which increases spindle discharge and thereby facilitates the reflex. This increased fusimotor drive may therefore lead to increased stiffness of the jaw sensorimotor system during pain to reduce mobility, which is consistent with the Pain Adaptation Model.[38] This increased fusimotor drive may also diminish the fidelity of spindle afferent transmission, as already mentioned, and may contribute to a deterioration in motor control.[47,65]

Despite the supporting literature, some data do not fit closely with the Pain Adaptation Model. Some studies of experimental jaw muscle pain at moderate[56] or intense[66] pain levels, as well as some studies of clinical orofacial pain,[64] have reported no significant limitation of jaw movement features during mastication in comparison with pain-free controls, and an increase in speed has been reported at intense experimental pain levels.[66] In 1 study,[56] limitations of the jaw tracking system were implicated as a possible reason for the lack of significant kinematic findings. In another study,[64] although the range of maximum gape in pain-free chewing cycles (9 to 23.1 mm) was not different from that in painful function (7.4 to 22.6 mm), the variability of the maximum gape and the number of discontinuities in the chewing cycles were significantly greater during pain than during pain-free function. Schwartz and Lund[51] also found that pain resulted in increased irregularity of electrically evoked rhythmical jaw movement cycles in the anesthetized decerebrate rabbit. The reason for this increased variability is unclear, although it is possible that nociceptive input could be modifying brainstem-mediated movements. Specifically it has been suggested that more frequent and careful reshaping and repositioning of the bolus in the presence of pain occurs to prevent unfavorable loading of the affected tissue.[64] The increased variability may be a reflection of the sensorimotor system "searching around" for a pattern of movement that minimizes pain and potential tissue damage during the movement. This could be viewed as an attempt to maintain homeostasis, which has been defined as a dynamic and ongoing process comprising many integrated mechanisms that maintain an optimal balance in the physiological condition of the body for the purpose of survival.[67] Recently, pain has been viewed as a homeostatic emotion reflecting an adverse condition in the body and requiring a behavioral response.[67] In this context, therefore, the sensorimotor changes observed in association with pain may be 1 of a number of behavioral responses directed toward the maintenance of homeostasis.

The reported effects of pain on EMG activity are also not always consistent with the Pain Adaptation Model. For example, in well-studied experimental rat models of TMJ and masseter muscle pain, short-duration, robust and concomitant increases in EMG activity in both jaw-opening and jaw-closing muscles are routinely observed following injection of algesic chemicals (eg, mustard oil, glutamate) into the TMJ[13,30–32] or jaw muscles.[29] It was proposed that these EMG

changes may result in a "splinting" effect that would limit movement and protect the masticatory system from further injury. Importantly, although the exact changes in EMG activity may not be entirely consistent with the Pain Adaptation Model, this proposed protective effect is consistent.

In a number of human studies of submaximal isometric contractions, pain-induced changes have not been identified in agonist and antagonist EMG parameters.[61,68] In addition, marked increases in resting EMG activity have been reported in pain,[33] and such increases at rest are essentially inconsistent with the model. In chewing and clinical pain, results have been mixed. One research team reported no significant differences observed in comparison with pain-free controls in the activity of agonists during jaw closing,[64] while another reported no effect in most agonists or antagonists during pain[50] and, in a later study, decreased agonist activity during pain.[56]

Findings from studies of the effects of pain on locomotor,[61] trunk,[3] or elbow[69] muscle activity are also not always consistent with the Pain Adaptation Model. For example, in the locomotor system during dynamic contractions at a standard gait speed,[61] experimentally induced pain resulted in increased activity in the medial gastrocnemius during 62% to 75% of the stride cycle. Since the medial gastrocnemius acts as an agonist for 45% to 70% of the stride cycle,[70] pain may result in an increased agonist activity during part of the locomotor cycle. In a recent, comprehensive, seminal review of the trunk muscle literature in chronic low-back pain in terms of the Vicious Cycle Theory and Pain Adaptation Model, van Dieën et al[3] identified many inconsistencies between studies and concluded that neither the Vicious Cycle Theory nor the Pain Adaptation Model adequately predicted the effects of back pain on trunk muscle activation. It was suggested that the muscle activity changes tended to be task-dependent (related to the individual problem) and therefore highly variable between and within individuals.[3]

What Are Possible Reasons for the Lack of Consistency Between Studies with Respect to the Effects of Pain on EMG Activity?

The apparent inconsistency between the EMG data from the animal studies (which find increased jaw-opener and jaw-closer activity) and from the human experimental studies (which find changes in openers, closers, or neither, but not increases in both) may reflect species differences, differences in the functional state of the jaw sensorimotor system between the anesthetized state (animals) and the awake state in the human experimental pain model, other methodologic differences between human and animal experiments (eg, the surgery associated with experiments on rats), and the fact that most of the studies involving human experimental or clinical pain have centered on recording from individuals with pain primarily in muscles, while the experimental rat models have focused mainly on TMJ pain. Although similar increases in jaw-closer and jaw-opener EMG activity have been observed with algesic chemical injections in the rat masseter, these changes were of shorter duration than following the algesic chemical injection into the TMJ.[29] It is possible therefore that articular pain has different motor effects than muscle pain. Indeed, Schaible and Grubb[60] reported that injured or inflamed joints are usually kept in mid-position and movements are minimized to lower nociceptive joint afferent activation. Coactivation of jaw openers and jaw closers could achieve such a pain minimization effect and might be more appropriate for reducing pain than increased antagonist and decreased agonist activity. Clinical research EMG activities in TMD patients with arthralgia to ascertain whether there are cocontractions of jaw openers and jaw closers consistent with the experimental rat pain models might be worthwhile.

Tables 1 and 2 list some possible reasons for the lack of consistency between studies with respect to the effects of pain on EMG activity in terms of anatomic and functional complexity (jaw muscles, central neural control, nature of the task being performed) and the multidimensional nature of pain. In terms of anatomic and functional complexity (Table 1), it is argued that the complexity of the jaw sensorimotor system and its movements influence the effects that pain has on jaw muscle activity. Further, the considerable variability in the organization of sensorimotor systems between individuals means that pain may have different effects on jaw sensorimotor systems in different individuals. As these systems are subject to neuroplastic changes, the effects of pain on these systems may change over time.

In terms of the multidimensional nature of pain (Tables 2 and 3), it is suggested that the multidimensional experience of pain, which includes sensory-discriminative, cognitive-evaluative, and motivational-affective dimensions, has a significant and highly individual influence on the effect of pain on motor activity. This concept has some basis in the recent view of pain in humans as a

Table 1 Reasons for the Lack of Consistency Between Studies as to the Effects of Pain on EMG Activity: Anatomic and Functional Complexity

Evidence suggesting a role in influencing the effect of pain on the sensorimotor system	Implications
Jaw muscles	
Little effect on jaw opener activity despite significant reductions in jaw displacement with masseter hypertonic saline infusion.[50]	Changes may occur within muscles that are not reflected in surface EMG recordings but require more detailed EMG analyses.[123]
Recordings from different locations within the masseter muscle appear to be associated with different EMG activity patterns.[100]	Need for multiple recordings from the same functionally heterogeneous jaw muscle.[82,83]
Dual agonist/antagonist role in jaw protrusion for digastric muscle.	Difficulty in definition of agonist/antagonist*; see also van [3,81] Interindividual variability in definition depending on morphology.
Central neural control	
Considerable interindividual variation in skeletomotor systems. Interindividual differences in motor cortical maps[103,104] may influence the demonstrated effects of pain on motor cortex.[99,105–107]	Interindividual variation in the reaction of sensorimotor systems to pain which also may undergo neuroplastic changes.[124]
Changes in muscle recruitment patterns during pain vs. pain-free controls have been shown for jaw muscles[1,4,108–113] as well as trunk, limb, neck, back and abdominal muscles.[3,52,61,69,78,80,94,114–122]	Effects of pain on motor activity may involve complex changes in the recruitment pattern throughout a movement.
The nature of the task being performed	
Noxious stimulation has a different motor effect for different chewing patterns in rabbits.[51] Previous studies in the jaw[66] and trunk[3] sensorimotor systems suggested the important influence of task on the effect of pain on the human sensorimotor system.	The use of a range of standardized jaw tasks may help in defining the effects of pain on motor activity.

*An *agonist* can be defined as a muscle that is shortening or at least not changing its length while it is contracting, while an *antagonist* can be defined as a muscle that is lengthening during a contraction. Mechanically, this may equate to the sign of the moment produced by the muscle being consistent with the sign of the net moment.[3]

homeostatic emotion reflecting an adverse condition in the body that requires autonomic and motor behavioral responses.[67] Different types of pain are thought to engage specific behavioral responses.[67] Thus, for example, and in line with the evidence from the trunk muscle literature,[3] each pain pattern, in terms of its quality, location, intensity, and/or duration, may be associated with a particular pattern of change in EMG activity. Melzack has proposed an individualized neuromatrix[71] that may help explain the variation in pain response. The neuromatrix comprises a widely distributed neural network that subserves the multidimensional nature of pain. The existence of an underlying individual neurosignature output overlain by the varying sensory inputs as well as cognitive and emotional inputs has already been established. The output pattern evokes a unique pain experience for that individual,[71] and we propose that there is a unique motor response as well.

Can Changes in Muscle Coordination Lead to Further Disability/Pain?

Irrespective of the nature of the changes in EMG activity that occur in pain, the changes that do occur may be protective in nature (eg, guarding, bracing, limiting movement, limiting pain, protecting from further injury) or pathologic (changes that could lead to further disability). Both protective and pathologic contributions could occur in any 1 individual. Clinically, it has been thought for many years that patients with chronic low-back pain undergo protective guarding, bracing, splinting, and compensatory posturing to limit the range of motion.[72] In these patients there is evidence for inhibition of muscles in pain, and this leads to an alteration in the normal pattern of muscle activation among synergistic muscles across joints.[73] There is the possibility of "favoring" the use of certain muscles. Such a change in muscle activation pattern might be a source of further disability and pain; for example, muscle disuse may cause altered recruitment and overload injuries in mus-

Table 2 Reasons for the Lack of Consistency Between Studies as to the Effects of Pain on EMG Activity: Multidimensional Nature of Pain

Evidence suggesting these reasons play a role in influencing pain's effects on the sensorimotor system	Implications
Sensory-discriminative	
Quality	
Some evidence for differences in resting muscle activity between chronic pain patients with different diagnoses, eg, myofascial pain versus neuropathic pain versus tension-type headache.[33,125–129]	Pain quality may be important in the EMG effects observed.
Location	
Diagnostic heterogeneity in patients may influence pain effects on jaw[27,130] and trunk[3] sensorimotor systems. Orofacial pain may result in increased[25,27,28,33,108,130–135] or decreased[136,137] jaw muscle activity*; in some cases, there may be no change.[1,2,4,61,138–141] Increased variability in resting activity may reflect differences in pain location (Table 3).	The symptom distribution may influence the pattern of change in EMG activity.
The site of a noxious stimulus may influence EMG effects.[57]	The complex internal architecture[82,83,156] of jaw muscles may provide insight into the intersubject variation of the EMG effects of injections of hypertonic saline.
Deep-tissue pain has specific effects on motor activity.[1,18,20] The group III/IV nociceptive influence on γ-motoneurons may diminish the fidelity of the spindle afferent system.[20]	The possible disturbances in motor control may vary between muscles because of the different spindle distribution between jaw muscles.[102]
Intensity and/or duration	
In jaw[50,66] and arm[142] sensorimotor systems, pain intensity influences motor effects.	
Motivational-affective and cognitive-evaluative dimensions (ie, supraspinal influences)	
The drive to reduce damage and escape/alleviate the pain[143] can be modulated through supraspinal/suprabulbar influences.[13,144,145]	Factors affecting physical functioning include task importance and perceived ability to perform the task (eg, experience, role models, fear of further injury).[157]
How one chooses to cope with pain influences the behavioral response. Motivation to undertake and complete the task is changeable.[146,147]	This may explain some of the variation in the relationship between pain and motor function.
The motivational-affective system promotes goal-directed behavior by facilitating "on-task" behavior and minimizing "off-task" interference, eg, chronic pain.[148]	The motivational component of pain may influence the sensorimotor interaction.[1]
Recent forelimb study that required target acquisition both before and during pain found no significant kinematic or EMG changes.[149]	Motivation is relevant to orofacial pain patients who need to perform a task, eg, speaking with clear articulation, chewing unexpectedly hard foods.
The autonomic nervous system and hypothalmic-pituitary-adrenal axis contribute to the response to pain-related stress, which helps facilitate acceptable function and ultimately the re-establishment of homeostasis.[71,143] With extended exposure, pathologic responses may ensue.[143] Stress has been implicated in EMG effects in studies comparing subjects with pain to pain-free control subjects.[150–153]	Significant roles for the autonomic nervous system and hypothalmic-pituitary-adrenal axis in the motor response to pain.
Sincerity of effort,[154] unfamiliar testing environment, fear of pain/injury, depression, and anxiety may influence motor behavior. Higher-order influences have been implicated in the ability of patients and controls to attain similar gapes,[64] in changing pain levels during mastication,[155] and in sham pain effects.[25]	Higher central influences can have a dramatic influence on the effect of pain on EMG activity.

*Note that some of these studies have suffered from improper analytical methods. EMG of the jaw muscles may have been contaminated by activity of the muscles of facial expression in some cases. There may also have been improper matching of controls with pain groups.

cles being used in an unusual manner.[74] The contribution of such disturbances in muscle function to the pain have been recently suggested as a possible "negative consequence" of the pain-related changes in muscular recruitment in chronic low-back pain,[3,52,75–78] arm muscle pain,[69] sacro-iliac joint dysfunction,[79] and neck pain.[80] For example, it has recently also been proposed that sacro-iliac joint dysfunction can result from an altered pattern of muscle recruitment of gluteus maximus motor units during weight bearing that may result in compensatory biceps femoris over-action. The resulting soft tissue strain and joint instability may manifest itself in low-back pain.[62,79] A review of the chronic low-back pain literature[3] also suggested that many of these functional changes may remain after their functional significance has disappeared, ie, after recovery.[81] Since these changes

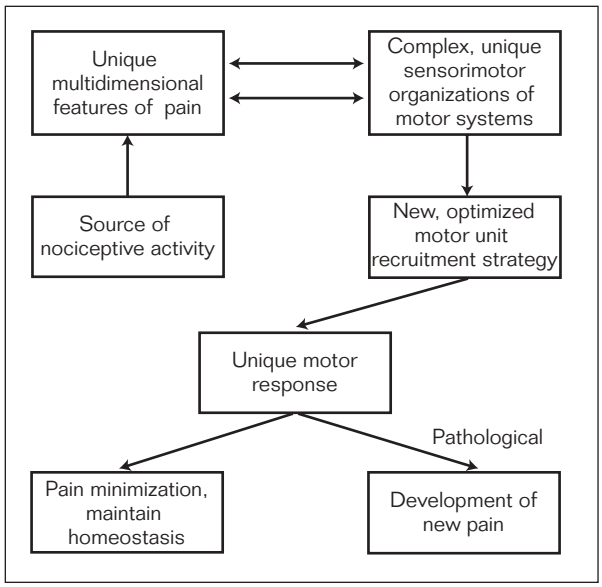

Fig 2 Diagram outlining essential components of the IPAM. The effect that pain has on motor activity depends on the interaction between pain, with its multidimensional nature, and the anatomically and functionally complex sensorimotor system. The net result is a new optimized motor recruitment strategy, which results in a unique motor response that aims to minimize pain. Sometimes, however, the new motor response is associated with the development of new pain or the worsening of existing pain.

in activity may represent compensatory mechanisms to stabilize the spine, the rehabilitation of chronic low-back pain patients with the sole purpose of restoring "normal" function has been cautioned.[3] It has also recently been proposed that muscle overloading may arise as a result of long-lasting, low-intensity work where the normally available protective mechanisms, such as "rotation" of active motor units, may fail; this may contribute to the development of chronic work-related myalgia.[47,65] Although there does not appear to be direct evidence for such negative effects in the jaw sensorimotor system (see Stohler[4]), Mongini et al[49] have pointed out that the contraction of the elevator muscles during opening, as suggested by the Pain Adaptation Model, is an eccentric contraction and that this elongation may be damaging beyond a certain level. Increased antagonist activity could induce muscle damage and could potentially contribute to further pain.

Revision to the Pain Adaptation Model: The Integrated Pain Adaptation Model

The Anatomic and Functional Complexity of the Jaw Sensorimotor System

Accumulating evidence suggests a high degree of functional heterogeneity within all muscles in the jaw sensorimotor system.[82,83] With this functional complexity in generating a movement, it is likely that the motor command is directed at motor units that are biomechanically optimized to generate the motor task rather than at the activation of specific muscles. Indeed, there may be changing combinations of motor units, or "rotations," throughout or on repetition of a movement.[65,84] In light of recent evidence,[85,86] the distribution of motor unit activity within the jaw sensorimotor system may reflect activation of the motor units in those jaw muscles that have the best mechanical advantage and the lowest metabolic cost for activation. With this in mind, the classification of muscles into agonists and antagonists in some tasks may become problematic.

We suggest that the motoneuron task group hypothesis[87] is the most appropriate way to view the organization of the jaw sensorimotor system. Here a *task group* is defined as an ensemble of elements (including extra- and intrafusal motoneurons, muscle fibers, and associated proprioceptive afferents) that work together in an orderly manner during task performance. A task group has no necessary correlate in the anatomy of individual muscles, nerves, or nuclei. Thus, during a protrusive jaw movement, for example, the brain might activate a subset of single motor units within regions of muscles, including the lateral pterygoid and posterior temporalis (to control the amount of protrusion), superficial masseter and medial pterygoid (to assist in the protrusion and to prevent jaw opening), digastric (to control the amount of protrusion and to open the jaw to disclude the anterior teeth), and anterior temporalis (to control jaw opening). The somatosensory feedback assisting in the performance of this movement would also derive from these different muscles and orofacial regions. With individual variability in skeletal form, muscle architecture, and muscle composition, it is most likely that different people will activate their motor units in unique ways to achieve characteristic movement patterns. Chewing cycles[88] and condylar movement patterns during the same tasks[89] are different in different individuals, just as gait is different from person to person. In the context of biomechanical optimization, variability in skeletal architecture and muscular anatomy

Table 3 Effects of Pain on Postural EMG Activity in Some Studies

Study	Year	Controls		TMD/Pain group	
		Mean (μV)	SD	Mean (μV)	SD
Glaros et al[27]	1997				
Left temporalis		3.72	1.80	5.71	6.08
Left masseter		2.26	0.72	3.31	2.35
Gervais et al[132]	1989				
Bilateral masseter and temporalis		1.8–2.5	1.0–1.3	4.7–6.1	3.7–5.1
Burdette and Gale[133]	1988				
Masseter		1.09	0.34	1.54	0.90
Anterior temporalis		0.81	0.62	1.76	1.72
Sherman[139]	1985				
Masseter					
No pain, no bruxism		4.5	1.7		
Bruxism but no pain		13.8	5.0		
TMD, no bruxism				5.9	3.1
TMD and bruxism				14.2	6.5

Mean ± SD or range of values shown.

between individuals will therefore influence how motor units are activated.

The Effect of Pain on Motor Activity: The Integrated Pain Adaptation Model

We propose the Integrated Pain Adaptation Model (IPAM) to explain the motor effects of pain. In normal function, the brain will activate whatever motor units it needs to produce an appropriate movement. In the presence of pain, it is suggested that, in the individual, pain interacts in a unique way with the individual organization of the sensorimotor system (Fig 2). With the complex and individualized sensorimotor systems and pain experiences,[13,90,91] it is suggested that the interaction between pain and the sensorimotor system will also be unique between individuals (Fig 2). Indeed, there is evidence for considerable interindividual variability in the behavioral response to pain,[90,92] with both genetic and psychosocial variables playing crucial roles. In terms of the jaw sensorimotor system, the mechanical redundancy in this system[93] allows modification of the set of motor units activated so that a new optimized motor unit recruitment strategy can develop to help maintain homeostasis. The redundancy of the masticatory system means that many muscle recruitment strategies are possible to perform a task; therefore, individuals may develop unique recruitment strategies. One important aspect in maintaining homeostasis could be the need to minimize the generation of further pain at rest or during a subsequent movement. This hypothesis is consistent in general terms with the analysis of van Dieën et al[3] in that the changes in EMG activity in chronic low-back pain are aimed at avoiding noxious tensile stresses in injured structures, thereby avoiding or minimizing pain. This notion of pain minimization is also consistent with conclusions in neck-pain patients[94] and with the clinical observation that injured or inflamed joints are usually kept in midposition and that movements are prevented if possible to achieve pain minimization.[60] In addition, chronic pain patients frequently move more slowly, with smaller movements and with side preference to minimize the generation of further pain from their motor activities (eg, walking, chewing).[49]

The IPAM: A Possible Mechanism

The primary motor cerebral cortex (MI) may be 1 important site where this modification in recruitment strategy may occur, as recent evidence shows that noxious stimulation of the forelimb or trunk regions results in depression of MI excitability.[95–98] There is also recent evidence that algesic chemical injection into the tongue leads to selective depression of tongue MI excitability, but the excitability of other parts of the MI controlling other orofacial movements was not significantly affected.[99] Given the recent evidence for regional differences in masseter excitability in association with localized hypertonic saline injections into the masseter,[100] there might be selective depression of excitability of specific motor efferent zones within facial MI. It has also recently emerged that neuroplastic changes in sensorimotor cortical maps can occur in association with a variety of musculoskeletal pain conditions (for review, see van Vliet

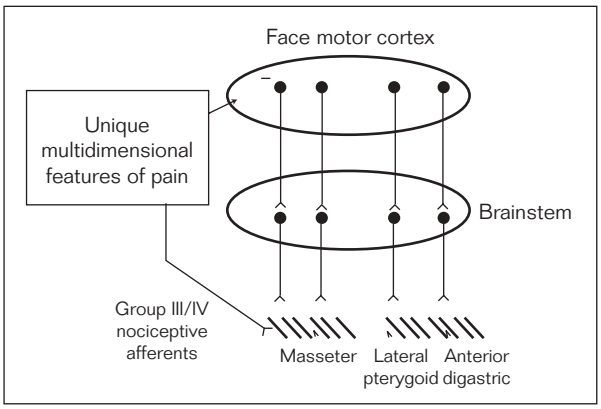

Fig 3 Detailed outline of 1 possible mechanism whereby the IPAM may operate. A noxious stimulus localized to 1 part of the masseter, for example, depresses the excitability of the somatotopically related part of the face MI and influences other efferent pathways. The set of motor units used to generate a movement is modified under these conditions so that the movement produced in the presence of pain is generated with the lowest pain possible.

and Heneghan[101]). The depression of MI activation could be also a possible reason for the reductions observed in maximum voluntary contraction and the altered sense of effort required to perform motor tasks[66] in the presence of pain. Where maximum voluntary contraction is required, there are simply fewer motor units available to be activated; therefore agonist activity must decrease. In addition to the MI, any level of the sensorimotor system, from local reflex modulatory influences[24,102] to supraspinal/suprabulbar processes involving either subconscious or conscious influences, could be involved in these motor responses to pain.

Therefore, 1 possible mechanism whereby the IPAM might operate is via effects of pain on MI excitability. Noxious input from, for example, a jaw muscle (portion of masseter in Fig 3), leads to a depression of excitability of the region of the MI driving the part of the muscle that is the site of the noxious stimulus (Fig 3). Given the redundancy in the jaw sensorimotor system,[93] the system can develop a new/modified motor activation pattern, which may involve the same and/or different motor unit task groups. The particular pattern of activation selected in any individual will be determined by the anatomic and functional complexity of the jaw sensorimotor system as well as the multidimensional nature of the pain experienced by that individual, that is, the sensory-discriminative, motivational-affective, and cognitive-evaluative aspects of the pain (Fig 3). The multidimensional nature of pain will influence the sensorimotor system through the connections that the limbic system, the hypothalamo-pituitary-adrenal axis, and the autonomic nervous system have with the peripheral and central components of the system. The specific sensory channels, in terms of a central homeostatic afferent pathway, and their interaction with sensory and motivational regions within the brain, have been recently summarized.[67] When strategies are developed to maintain homeostasis, objectives could include minimization of the pain generated in movement and/or the metabolic cost.[86]

The units that are activated under pain may require more energy, or may be less efficient, and in the absence of pain, would not necessarily be activated or would be activated in a different manner or sequence. Perhaps these patterns of recruitment were not adopted by the sensorimotor system during learning in the pain-free state because it was learned at an early stage that they were not the optimal methods of motor unit activation. The recruitment of units not normally recruited might help explain the increase in variability in the task actually performed, although direct effects of nociceptive afferents in interfering with the fidelity of spindle afferent encoding may also play a role here.[20] Under some circumstances, if some motor units are recruited in ways that they are not "used to," more pain may be generated (Fig 2).

The IPAM is not in conflict with the Pain Adaptation Model.[2] Rather, this new model is a reformulation of the Pain Adaptation Model in light of more recent data. The Pain Adaptation Model proposes that pain results in an inhibition of activity in a muscle when that muscle acts as an agonist and in an excitation of muscle activity when that muscle acts as an antagonist for the purpose of protecting the body from further injury and pain. The Pain Adaptation Model may be most clearly manifest at high force levels and/or for intense pain levels.[66] One reason for this could be the depression of primary motor cortical excitability associated with pain. The IPAM proposes that complex changes in activity occur in the entire sensorimotor system and that these changes are influenced by individual responses to pain and the complexity of the sensorimotor system. The resultant changes in muscle activity may involve

decreases in activity in that part of the muscle in pain and increases or decreases in other parts of the painful muscle or other muscles. Any changes in activity in muscles will occur irrespective of whether the muscle is being used as an agonist or an antagonist. The change in activity is brought about in an attempt to maintain homeostasis, which may have as a primary objective the minimization of further pain. It is possible, however, that in some individuals, these changes in muscle activity could lead to further pain, injury, and disability for reasons that have yet to be fully elucidated (Fig 2).[47] The complex association between muscle activity and pain proposed by the IPAM represents, in a sense, a unification of components of the Vicious Cycle Theory and the Pain Adaptation Model.

Implications

Does this new model help explain motor effects associated with different clinical pains, eg, acute versus chronic pain, local versus referred pain, jaw muscle versus TMJ pain versus dental pain? These pains are different and have different effects in different individuals. The IPAM would predict that the effects of these different pains on motor activity would be different. Just as an individual's experience of pain varies widely, we propose that so too will an individual's motor response to pain. It may therefore be necessary to define how each individual's sensorimotor system operates under pain to allow a tailoring of management strategies unique to that individual.

In terms of acute versus chronic pain, recent evidence points to substantial neuroplasticity within the central nervous system that may underlie/contribute to chronic pain states. The central neuroplastic changes accompanying such chronic pain states may lead to changes in the interaction between pain and motor control that would be different from the interaction occurring under acute pain. These neuroplastic changes are likely to occur not only in the somatosensory and sensorimotor systems, but also the autonomic nervous system, hypothalamo-pituitary-adrenal axis, and limbic systems.

Dao et al[155] reported that while most TMD patients (50%) showed increased pain (103%) with chewing, a subgroup of TMD patients (~30%) showed decreased pain (57% decrease). These patients had a significantly higher resting pain level than the other group of pain patients. Questions arise from such a study: Why are the patients experiencing decreased pain during movement? Is it possible that altered recruitment strategies alleviated the pain? Can motor strategies be developed to reduce pain? Might it be possible to identify those individuals who can move their jaws in such a way as to experience the minimum amount of pain while still maintaining acceptable function? Training strategies could be developed, based on these recruitment strategies. Such rehabilitation strategies could not only focus management on motor control strategies but also provide patients with self-management strategies, which would influence motivational and cognitive components of pain and the associated behaviors.

Possible Limitations of IPAM

Several possible limitations of the IPAM need to be kept in mind. First, the depressive effect of nociceptive activity on face motor cortical function has not been demonstrated in humans. However, the proposal that the motor cortex is involved is only 1 possible mechanism whereby nociceptive activity can influence motor activity. The existence of other nociceptive-motor interactions has been well demonstrated, eg, at the brainstem level,[102,145] and these could also influence motor activity. Second, the extent of the depression of motor cortical activity needs to be determined. Third, the connectivity and functionality of the connections between the neural representations of the multidimensional features of pain are not clear. Thus, it is unclear how the multidimensional nature of pain could affect the optimization of motor control during pain to ensure homeostasis.

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Critical Commentary 1

Orofacial Pain and Jaw Muscle Activity: A New Model

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The authors of the Focus Article[1] present a comprehensive literature overview of the mechanisms of temporomandibular disorders (TMD) and apply the data also to skeletal (locomotor and low-back) muscles. Starting from the Vicious Cycle Theory and the Pain Adaptation Model, the authors develop a new model that is supposed to correct many of the inconsistencies of these hypotheses. They weigh carefully the pros and cons of the Vicious Cycle Theory and the Pain Adaptation Model. Although the basic attitude of the authors toward both hypotheses is critical, they are fair enough to cite also observations that support these otherwise questionable models.

The article is an important and overdue contribution to the ongoing discussion about how a muscle or muscle group reacts to a painful lesion in that muscle or in tissues that are functionally connected with the muscle (eg, joints). One problem I see is the generalization of data obtained from jaw muscles to locomotor and low-back muscles, because the central wiring of jaw muscles differs from that of skeletal muscles.

The authors demonstrate that both the Vicious Cycle Theory and the Pain Adaptation Model are based on an oversimplified view of the central nervous connections of muscle afferents and efferents. This has long been known to neuroanatomists; nevertheless, it has been ignored by large parts of the scientific community. Actually, the simplicity of these models is probably 1 of the reasons why so many people find them attractive.

The diagram of the wiring of a spinal α-motoneuron in Fig 1 demonstrates the complexity of the connections. Only the pathway via the Ib interneuron pool is shown; the γ-motoneurons are omitted. The activity of the α-motoneurons is influenced by practically all sensory afferents from the body periphery and also by descending motor pathways. These influences are transmitted to the α-motoneurons through inhibitory interneurons of the Ib interneuron pool. The segmental and descending inputs may be active or silent; their activity varies with time. Most input sources to the Ib pool inhibit the motoneurons by activating the interneurons; increased activity of the motoneurons can be brought about by inhibition of the interneurons. The diagram clearly shows that all attempts to offer a simple model for the mechanisms of increased or decreased muscle tension and spasms are bound to fail. The contractile tension of a muscle depends on the balance between all these inputs.

Besides emphasizing the complexity of a patient's motor response to a painful lesion, the authors mention some other aspects of the motor reaction that are often overlooked in the literature:

1. Differences between acute and chronic muscle pain. Results obtained with intramuscular injections of algesic chemicals in subjects and experimental animals are valuable for the understanding of basic pain mechanisms but cannot be simply transferred to patients with chronic muscle pain. One example is that γ-motoneurons tend to respond to a painful stimulus with a certain time-course. For instance, the motoneurons may show increased activity in response to an acute painful stimulus in animal experiments but be inhibited during a longer-lasting lesion, such as an inflammation.[2]
2. Differences between muscles or even various compartments (subdivisions) of the same muscle. Recent behavioral experiments in rats have shown that the pressure pain threshold of the gastrocnemius muscle is lower than that of the multifidus muscle, a genuine low-back muscle (U. Hoheisel and S. Mense, 2007, unpublished data).
3. Interindividual differences
4. Time dependency in the same patient
5. Task dependency
6. Supraspinal influences. These include motor, sympathetic, and limbic inputs. The involuntary activation of pyramidal and extrapyramidal motor tracts is probably a more important factor

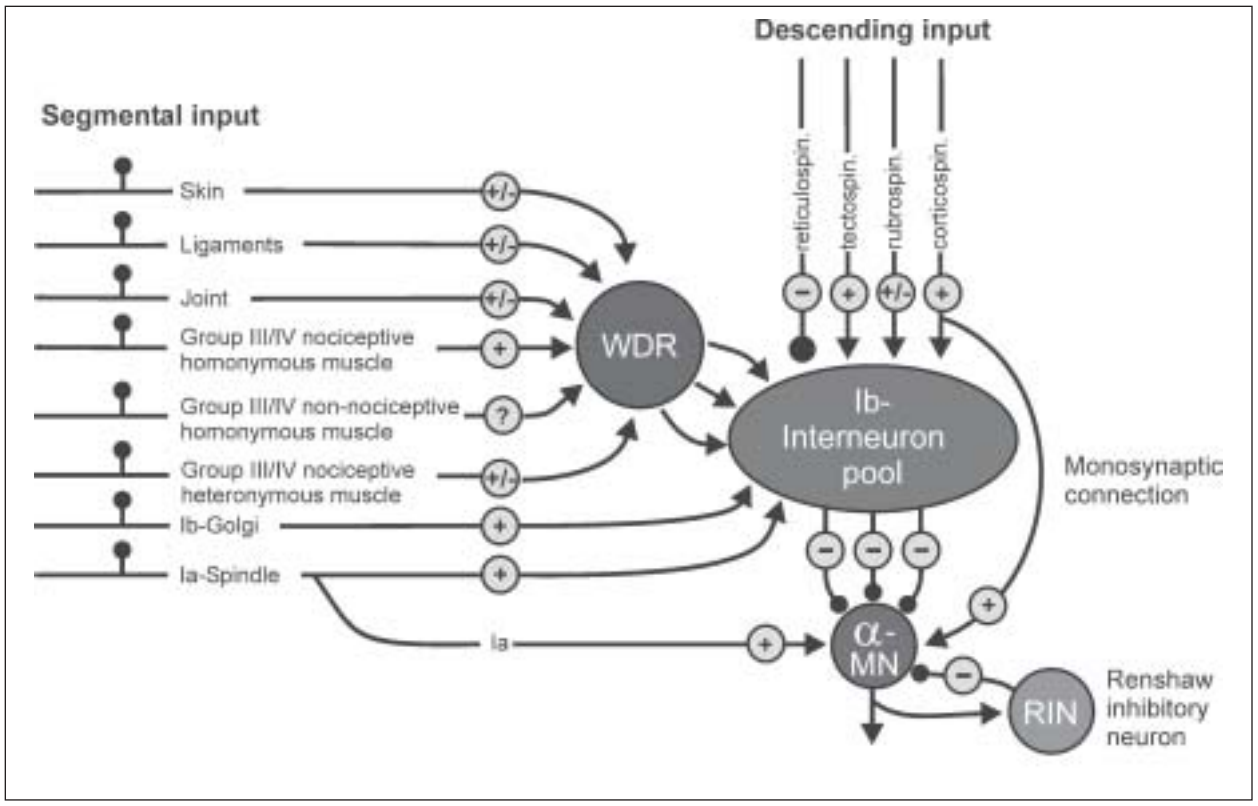

Fig 1 Simplified wiring diagram of a spinal α-motoneuron. Plus and minus signs mark excitatory or inhibitory influences. The question mark in the pathway from the group III/IV non-nociceptive muscle afferents indicates that the role of this input in pain-induced motor responses is obscure. Ib = afferent fibers from Golgi tendon organs; Ia = afferent fibers from primary endings of muscles spindles; WDR = wide-dynamic range neuron in the dorsal horn of the spinal cord; MN = motoneuron. The reticulospinal, tectospinal, and rubrospinal tracts originate in the brainstem and are part of the extrapyramidal system. "Corticospinal" refers to the pyramidal tract whose fibers contact the α-motoneurons both monosynaptically and through the Ib interneuron pool. Figure based on McCloskey and Mitchell.[8]

than generally thought. Particularly under time pressure and psychic stress, many people tonically activate a given muscle or parts of a muscle. This can lead to a chronic overload of that muscle or muscle fiber bundle, with ensuing pain. Moreover, activation of the sympathetic system in association with such a situation can increase the pain. An often-overlooked factor is that sympathetic efferent fibers use adenosine triphosphate (ATP) as a cotransmitter. ATP has been reported to be an effective stimulant for nociceptive group IV fibers in rat skeletal muscle.[3]

This list, together with the other factors included in the Focus Article, clearly demonstrates a basic problem with our understanding of chronic muscle pain syndromes: There are 2 choices, either simplifying the problem to an extent that it no longer reflects reality (this applies to the Vicious Cycle Model and to a lesser degree also to the Pain Adaptation Model) or including as many factors as possible, which may result in a model that is too complex to be clinically useful.

The problem becomes even greater if one considers the following factors, which were not addressed in the Focus Article:

1. The central wiring of the neck and jaw muscles differs markedly from that of locomotor and low-back muscles. In contrast to the left and right leg, the left and right half of the neck or jaw cannot be moved independently; therefore, the motor reflexes in these regions differ from those of skeletal muscles. For instance, an ipsilateral flexor reflex and a contralateral extensor reflex cannot occur simultaneously in jaw muscles.

2. Fascia should be included in all considerations concerning chronic muscle pain and changes in muscle tension. Fascia are contractile[4] and supplied with free nerve endings and other receptors.[5] They can apparently alter the kinematics of a muscle and can be the source of pains assumed to originate in muscle nociceptors.
3. Painful muscles can become atrophic due to a painful lesion in a neighboring joint. In 1987, Young and colleagues[6] put forward their hypothesis of reflex atrophy of muscle groups following a joint lesion. In many aspects, the essence of their work is just the opposite of the Vicious Cycle Theory, namely weakness and wasting of muscles as a sequel of joint input.
4. Not all small-diameter (group III and IV) fibers from muscle are nociceptive. Besides nociceptors (which are characterized by a high mechanical threshold in animal experiments), there are low-threshold mechanosensitive unmyelinated units[3] that do not appear to fulfill a nociceptive function. They may be involved in the adjustment of circulation and respiration during muscle work.[7] These fibers are excited by mechanical stimuli and algesic agents, but their influence on muscle activity during muscle or joint pain is largely unexplored.

Although I appreciate the thoughtful and meticulous work done by the authors, I am not convinced that the new model put forward by them will be helpful in the future. The main conclusion that can be drawn from their model is that the situation is so complex that it is not possible to predict the reaction of a given muscle to a painful lesion, not even in the same patient at different times. Further, a scientific hypothesis or model must contain statements that can be tested experimentally. The model proposed by the authors includes so many factors that it cannot be verified or falsified as a whole. The model appears to be a description of the situation rather than an explanation of the underlying mechanism.

It may be necessary to study 1 muscle group at a time under defined experimental conditions. This may lead to separate models for each muscle group, with submodels for each input source and other influences. At present, this is just a theoretical possibility; however, these data appear to be necessary to predict and understand the behavior of a given muscle in a given situation.

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Critical Commentary 2

Orofacial Pain and Jaw Muscle Activity: A New Model

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We are grateful for the opportunity to provide critical comments on the Focus Article[1] written by our Australian colleagues on the relationship between muscle activity and pain.

Our intent in carrying out the research that led to the introduction of the Pain Adaptation Model[2] was 4-fold: *(1)* to compile the evidence regarding the relationship between muscle activity and pain, *(2)* to determine the degree to which it was consistent with the prevailing thinking, such as the Vicious Cycle Theory,[3] *(3)* to develop a new model to explain current data if the Vicious Cycle Theory could not be supported, and *(4)* to address the implications of our findings for patients, as muscle hyperactivity was viewed as the cause of painful jaw dysfunction. The testability and generalizability of any explanatory model to both trigeminal and spinal systems was also a concern; thus, data from a number of chronic pain conditions, eg, low -back pain, fibromyalgia,[4] were used in the development of the model.

Since its introduction 15 years ago, many studies have supported the predictions of the originally proposed Pain Adaptation Model. Some studies have contradicted the model's predictions; all of these have been nicely compiled in the Focus Article. We agree that the time has come to re-evaluate the level of support for the Pain Adaptation Model, and like Drs Murray and Peck, we were most interested in those original data that show divergent results with respect to the model predictions.

The Vicious Cycle Theory (never more than an initial hypothesis) was an attempt to explain signs (limitation of movement, changes in gait) and symptoms (pain, difficulties in performing movements). It could not be tested until electromyographic (EMG) recording techniques were perfected. Since then, it has been tested many times and rejected almost always, as Drs Murray and Peck pointed out in their Focus Article. Consequently, we find it difficult to understand the current interest in the γ-loop and stretch reflexes, since this is just the Vicious Cycle in another form.

The Pain Adaptation Model has been tested in clinical studies and in human and animal experiments, and, as noted in the Focus Article, it has been validated many times. It includes the postulate that tonic activation of nociceptors does not cause a tonic increase in resting EMG activity in any muscle group, with the notable exception of activity in the muscles of facial expression, and this has been confirmed many times.

Much of the variation and confusion that the authors describe in the literature on head and neck pain is related to the fact that facial muscle activity is picked up by EMG electrodes placed over other muscles in the submandibular area, cheeks, jaws, scalp, and other parts of the skull and neck. Drs Murray and Peck cite a paper[5] that is supposed to have shown marked increases in resting EMG activity in jaw muscles that are inconsistent with the Pain Adaptation Model. However, in that study,[5] activity was recorded over both jaw and facial muscles, and resting activity levels for 2 groups of pain subjects (1 myofascial, 1 neuropathic) and a control group were compared. No difference was found between the pain groups, but both pain groups showed higher resting EMG activity than the controls, which indicates that pain, and not its source, was the important factor. When the pain was unilateral, there was no difference between sides. All this can be explained by the fact that pain was recorded from the facial muscles, which are responsible for the nonverbal expression of pain.

The Pain Adaptation Model was deliberately limited to the explanation of interactions of nociceptors and segmental motor circuits; it was not meant to account for all the changes in motor behavior that occur in the presence of chronic pain. However, it does explain much of what is

commonly observed—reduced range of motion, reduced ability to work against loads, reduced movement velocity, and reduced frequency of centrally programmed repetitive movements such as mastication and locomotion. It is to be expected that some experiments result in either nonsignificant data or result in data that are not entirely consistent with the Pain Adaptation Model. Variation is a feature of biology and of any form of measurement.

Although we are less concerned than Drs Murray and Peck, we agree that the Pain Adaptation Model does not take into account the multidimensional nature of pain, neuroplasticity, and individual variations in pain behavior. However, we are quite certain that the segmental mechanisms that make up the model are very robust, and that although they can be modified, they cannot be *consistently* overridden by any sort of "unique motor pattern." Hundreds of years of single-subject self-experiments on sports fields have proven that if your leg hurts, you will not be able to kick a ball as far as when it does not, and you will not be able to run as far and as fast. Similarly, if your jaw muscles hurt a lot, maximum jaw opening, maximum biting force and masticatory frequency go down, even if you are able to "individually" optimize your motor unit recruitment strategy.

No question, it would useful to know what sorts of adaptations to tonic nociceptive inputs are taking place at suprasegmental levels, and Drs Murray and Peck are right to draw our attention to the lack of thorough investigation of this topic. However, it is difficult to project how their proposed modification of the Pain Adaptation Model can be used to stimulate such research, because it is inherently untestable. If each pain input can evoke a unique motor response, then the model can only be formally disproved by showing that the response to a given stimulus is always exactly the same, a virtual impossibility.

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Critical Commentary 3

Orofacial Pain and Jaw Muscle Activity: A New Model

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From a clinical, diagnostic, and management perspective, it is of great importance to understand how pain and motor functions are interrelated. One possibility is that a dysfunction of the jaw motor system leads to an overloading of the musculoskeletal tissues, which sets up a condition capable of nociceptor activation, thereby leading to pain. The other possibility is that when pain occurs, the jaw motor system changes in response to nociceptive activity. In the first case, management would logically be directed toward correction of the dysfunction, which then should resolve the pain problem. In the second case, emphasis would be put on management of the pain, which should then normalize the jaw motor function. This controversy has for many years been discussed in relation to orofacial musculoskeletal pain conditions known as temporomandibular disorders (TMD). Different explanatory models have been discussed in the literature, such as the Vicious Cycle Theory[1] and the Pain Adaptation Model[2]; however, other models have been proposed.[3] The authors of the Focus Article[4] should be congratulated for their efforts in summarizing the available literature and attempting to integrate the current state-of-the-science into a new model, the Integrated Pain Adaptation Model (IPAM). There are many merits to this endeavor, and they correctly point out observations and research findings that were consistent with the Pain Adaptation Model but which may not be in accordance with the IPAM. Nevertheless, the present commentary is intended to point out some features that may challenge the impact of the IPAM.

Methodologic Aspects

As Murray and Peck obviously are aware, many methodologic concerns could have an impact on the interpretation of the papers included in their article. For example, many of the cited studies used injections or infusions of hypertonic saline to evoke pain in the masseter muscle. When hypertonic saline is infused or injected into the masseter and the subjects are asked to move their painful jaw, the rate at which the saline pool within the muscle is likely to be distributed and washed out is affected. Jaw movement may in fact reduce or "gate" the pain, an effect that is well known from other studies and one that may confound the effects of pain on movements.[5] Furthermore, many of the sensory-discriminative components of the acute muscle pain elicited by hypertonic saline are quite similar to clinical TMD pain, but hypertonic saline does not lead to a pronounced mechanical sensitization of the muscle tissues,[6] which is a key feature of clinical TMD pain conditions. Thus, nociceptive activity per se may not be the only important factor to consider in terms of jaw motor control. Mechanical sensitization without ongoing spontaneous pain is capable of influencing jaw motor function, eg, intramuscular injection of nerve growth factor causes a long-lasting mechanical sensitization without ongoing spontaneous pain but significantly affects normal functions, such as mastication and yawning.[7] Recently, associated phenomena from the jaw muscles, such as delayed onset muscle soreness and fatigue, have also begun to be studied in terms of their effect on jaw motor control.[8] Thus, the choice of specific experimental pain model may influence the impact on the regulation of jaw motor function, which should be taken into consideration in the development of explanatory sensory-motor integration models.

Another methodologic issue with human studies is that only 1 muscle site is usually stimulated. Although the perceived area of pain covers a relatively large aspect of the masseter and often the

temporal region and temporomandibular joint (TMJ),[6] this may be quite different from clinical TMD pain, where multiple pain sites are commonly found. This suggests that spatial summation mechanisms should be examined further and possibly be integrated into an explanatory model.

The vast majority of the cited studies have also used surface electromyographic (EMG) recordings to examine jaw muscle activity. Caution needs to be taken because of the possible cross-talk between facial (mimic) muscles and the jaw muscles. It is not likely that clinical studies will ever use more selective (intramuscular) EMG recordings on a large scale, as this technique often is painful or at least uncomfortable and may confound the study of pain and jaw motor function. One approach may be to use multichannel EMG recordings.[9] Such techniques may also address recruitment of other motor units, as suggested in the IPAM. So far the available studies on single masseter motor units during painful stimulation have not been able to show changes in recruitment order but rather a significant decrease in firing frequency when the same bite force is maintained.[10] Thus, further studies are needed in this area, combined with the physical properties of the single motor units, such as twitch responses.[10]

Finally, the jaw-tracking devices used in some of the cited studies in the Focus Article[4] may also have limitations, particularly in terms of sensitivity, especially if the physiologic changes in EMG activity and kinematics are relatively minor (eg, 10% to 15%) and the sample sizes are insufficient to detect a real difference. It is common sense that lack of significant difference is not proof of no effect but could be due to a small effect size or a lack of statistical power. Thus, the solution may be to use better jaw tracking systems and/or larger sample sizes to show the effects predicted by the Pain Adaptation Model. However, the existing studies do not necessarily prove that the Pain Adaptation Model is incorrect.

Complexity of Jaw Motor Tasks

Jaw muscles are involved in a wide range of complex and highly integrated functions, such as mastication, speech, and swallowing; however, the influence of orofacial pain has mainly focused on mastication. Previous reviews[11] have pointed out the importance of distinguishing between the different tasks and a simple division into postural, static, dynamic, and reflex responses. An often-neglected fact is that the human mandible trembles at about 3 to 8 Hz when it is in its rest position with the jaw muscles relaxed, due to fluctuating activity in central neural pulse generators that activate both the jaw-opening and jaw-closing α-motoneurons.[12] Hypertonic saline-evoked pain in the masseter muscle can lead to a reduction in the power of the resting jaw tremor,[13] indicating that jaw muscle pain is capable of tonically modulating the amplitude of the outputs from the central "pulsatile control" generators producing jaw tremor at rest and during jaw movements. This mechanism may not be identical to the influence of pain and the circuitries described in the Pain Adaptation Model and suggest that more research is needed to fully understand the complexity of the brainstem responses to nociceptive afferent inputs.

Murray and Peck suggest that orofacial pain will influence jaw motor tasks in a highly individual manner depending on the unique motor programs. This could mean that the same external stimulus (eg, a given amount of hypertonic saline) could change mastication in opposite ways, eg, increasing the speed of chewing in 1 subject while slowing it down in another. In addition, the same subject could react with speeded-up or slowed chewing depending on internal factors (eg, being nervous, angry, stressed, or aroused) or context. For example, extra-trigeminal painful stimuli may increase EMG activity during mastication, whereas painful pressure applied to the TMJ has decreased the EMG activity.[14] Recent findings also support the view that types of pain other than musculoskeletal pain can influence the EMG activity during tooth clenching, eg, postoperative pain following third molar removal is associated with significant decreases in EMG activity in both the jaw-closing muscles and the jaw-opening muscles during maximal voluntary contractions.[15] However, the present IPAM is not able to predict the unique type of response in relation to specific motor tasks and therefore seems to contain a number of "holes." Thus, it might prove useful to examine other standardized jaw motor tasks, such as protrusion or laterotrusion, and hold-and-split types of tasks, as well as the influence of pain on other craniofacial muscle groups, such as the tongue muscles, facial muscles, and ocular muscles.

A central feature of the suggested IPAM is the cerebral cortical control and influence on the selection of appropriate motor responses. Few would argue against the suggestion that the brain is important for pain and can influence brainstem circuitries as described in the Pain Adaptation Model; however, to what extent is the cortex necessary for such changes? One study attempted to

measure the cortical excitability of the jaw motor cortex in the presence of muscle pain and found no significant effects,[16] although other cited studies from the trigeminal and spinal system have shown a decrease in cortical excitability.[4] Brain imaging studies suggest that painful stimulation also is associated with activation of the premotor cortex, supplementary motor area, and cerebellum, as well as other areas involved in motor planning and execution.[17] However, this still does not prove that the cortex is needed to change (adapt) jaw motor function in the presence of pain.

The IPAM highlights the need to view pain in a biopsychosocial context and as a multidimensional experience. Some would probably also mention pain genetics and its importance for the highly individual perception and expression of pain. The IPAM is therefore a natural progression of views emphasizing the individual motor responses. The challenge will be to close up some of the "holes" in this model (ie, areas where no prediction can be made). Otherwise it will be equal to the statement that all persons are unique or all persons have unique jaw motor responses to orofacial pain.

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We thank Drs Mense, Lund, Stohler, and Svensson for their stimulating and thoughtful comments[1–3] on our Focus Article.[4] We were encouraged by the positive comments and challenged by the critical insights. We are grateful for the opportunity of responding to some of the main issues raised.

Timeliness of Review

Our main purpose in this paper was to re-evaluate the level of support for the Vicious Cycle Theory and especially the Pain Adaptation Model. All commentators agreed that such a review is timely. As we pointed out, the Pain Adaptation Model does appear to provide a rational explanation for the effects of pain on motor activity in many instances. All commentators also agreed to some extent with our argumentthat the Pain Adaptation Model does not explain all of these interactions, and we summarized some relevant data sets in the trigeminal and spinal systems for and against the Pain Adaptation Model and the Vicious Cycle Theory.

Methodologic Issues

Svensson[3] rightly raised a number of methodologic issues (eg, data interpretation in relation to choice of experimental model, role of mechanical sensitization/muscle soreness/fatigue in influencing motor activity, complexity of temporomandibular disorder (TMD) pain spatial locations) that could challenge the impact of the Integrated Pain Adaptation Model (IPAM) proposal. The complexity of the TMD pain spatial locations is part of the sensory-discriminative dimension experienced by TMD patients that, as the IPAM proposes, would influence the relation between pain and motor activity. The role of other factors in influencing motor activity needs to be considered in relation to the model.

General Nature of IPAM

All commentators indicated that the IPAM is very general. We agree and it is, at best, a phenomenological model, that is, a description of the situation, as Mense[1] indicated. While he pointed out that many factors have to be considered in understanding the relation between pain and motor activity, he suggested that the IPAM contains so many factors that it is difficult to see how the model can be verified or falsified. He also identified the difficulty in generalizing data sets between trigeminal and spinal systems. Further, Stohler and Lund[2] indicated that the IPAM is inherently untestable. However, we submit that the IPAM provides a framework for the formulation of new hypotheses.

Specific Hypotheses

We cited evidence (Tables 1 and 2) indicating that the anatomic and functional complexity of motor systems and the multidimensional nature of pain influence the effects of pain on motor activity. The following hypotheses are examples of testable statements within an IPAM framework:

Hypothesis 1: The complex organization of the jaw motor system influences the effects of pain on jaw motor activity.

Working hypotheses: The effects of orofacial pain on the jaw motor system will vary:

- Depending on the kinematics of the specific jaw motor task being performed.
- In different functionally distinct units within a muscle.

Hypothesis 2: The biopsychosocial dimensions of pain (ie, sensory aspects, mood, beliefs, coping repertoire, environmental factors) interact with the complex motor system organization to produce a motor response.

Working hypotheses: The effects of experimental or clinical orofacial pain on the jaw motor system vary depending on

- The intensity and location of pain.
- Pain-related cognitions and mood.

For example, according to the IPAM, changing the intensity or location of the pain or changing the level of motivation to perform a task should significantly, clearly, and consistently change the relation between pain and motor activity. Indeed,

Svensson[3] cited his earlier paper[5] providing evidence that different pain locations (trigeminal versus extratrigeminal) could have markedly different effects on jaw muscle electromyographic activity. There is also recent evidence that different tasks influence the pain/movement interaction.[6]

Given the large number of variables in the IPAM, Mense[1] suggested studying 1 muscle group at a time under defined experimental conditions to reduce the number of variables. This approach would help to "fill in" some of the "holes" identified by Svensson[3] in the IPAM.

A Way Forward?

The aforementioned hypotheses are still somewhat vague; for example, they do not specify the direction of an effect. But if indeed the IPAM, or some variant of it, is not operative, then none of the aforementioned factors should have a major and consistent impact on the relation between pain and motor activity most of the time. We would be the first to acknowledge this in the process of testing the IPAM.

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