Neural and Muscular Factors Associated with Motor Impairment in Neck Pain

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Clinical neck pain is associated with impairment of muscle performance, assessable at a functional level. Functional deficiencies reflect altered mechanisms of muscle control and changed muscle properties. The basic physiologic mechanisms of pain have been extensively investigated, and the functional impairments associated with neck pain are well documented. However, the cause–effect relationships between neck pain and motor control are poorly understood, due to difficulty translating basic physiologic findings into the complex scenario of clinical pain conditions. This article reviews current evidence of disturbances in neural control and muscle properties associated with neck pain and discusses their interrelationships. Although the links among pain, motor control, and muscle properties have been established, their relative significance for the perpetuation and recurrence of neck pain remains largely unexplored. Rehabilitation programs that include interventions for neuromuscular changes seem beneficial for restoring motor function and may prove effective for reducing neck pain recurrence.

Introduction

Neck pain is a common complaint, affecting up to 70% of individuals at some point in their lives [1], and its incidence is increasing [2]. Neck pain is associated with impairment of muscle function. Consistently, researchers have documented deficits in isometric cervical and craniocervical strength and endurance in people with neck disorders of both insidious and traumatic onset [3–7]. Electromyographic (EMG) studies have also shown altered muscle activity [8••,9–12] and greater myoelectric manifestations of cervical muscle fatigue [13–16] in people with chronic neck pain. Reduced proprioceptive acuity (eg, greater errors in positioning the head following voluntary movement) has been observed in people with neck pain of insidious [17] and traumatic onset [18–20], especially in those reporting higher pain and disability [21,22] and dizziness [23].

Impairment of neck muscle function reflects a combination of altered neural muscle control and muscle fiber properties that can potentially initiate or perpetuate neck pain (Fig. 1). This article reviews current evidence of disturbances in neural and muscular mechanisms associated with neck pain and discusses recent hypotheses on the cause–effect relationships between pain and changes in the neuromuscular system.

Altered Control Strategies

A common finding in people with neck pain is augmented activity of the superficial cervical muscles, which has been observed in both isometric contractions [22,24–27] and functional upper limb activities [10,28,29]. On the contrary, the level of activity of the deep cervical flexor muscles, longus colli and longus capitis, is reduced in neck pain patients [8••,9]. Activation of the deep cervical muscles is also impaired when people with neck pain perform a rapid arm movement that induces a perturbation to the body. Under these conditions, the onset of activity of the deep cervical muscles is delayed and adopts a direction-specific response, contrary to observations in healthy individuals [8••], reflecting a change in the strategy used by the central nervous system to control the cervical spine.

People with neck pain also demonstrate reduced ability to relax the anterior scalene, sternocleidomastoid, and upper trapezius muscles following activation [4,10,30], and they show reduced rest periods of the upper trapezius muscle during repetitive tasks [12,31,32].

Relationship between pain and altered control strategies

No consensus exists regarding the cause–effect relationship between clinical neck pain and changes in motor control strategies. However, in some cases, the degree of motor control deficits seems to be associated with perceived pain or disability. For example, the delay in

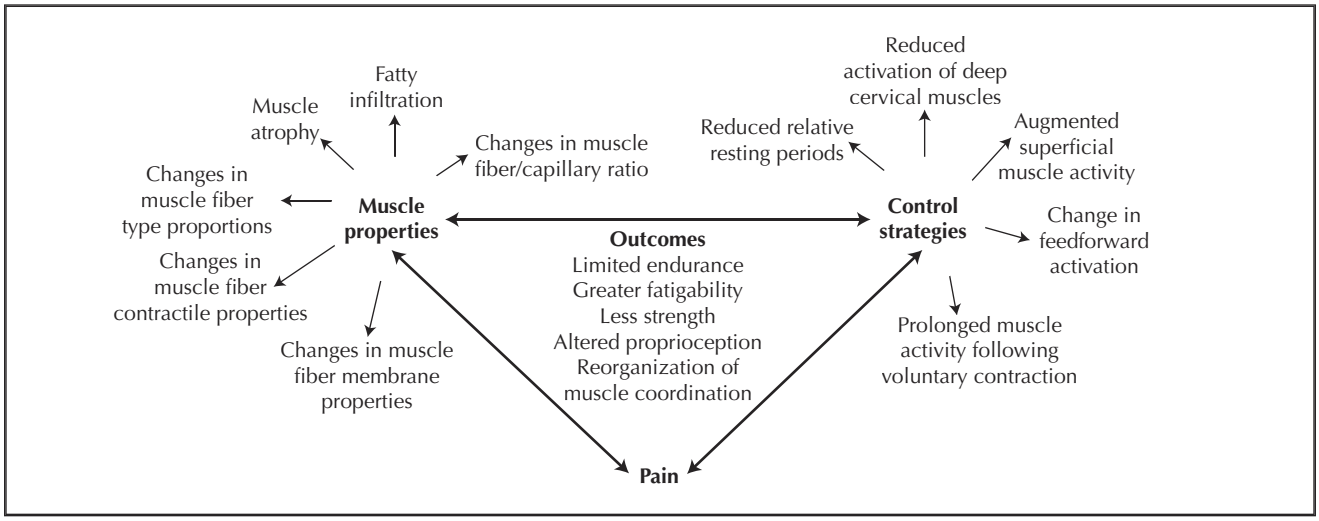

Figure 1. Inter-relationships between pain, altered control strategies, and peripheral changes of the cervical muscles.

onset of the deep cervical flexor muscles during a rapid arm movement is correlated with patient score on the Neck Disability Index, with a greater delay observed for patients with greater perceived disability (Falla et al., unpublished data). Moreover, greater perceived disability (on the Neck Disability Index) among patients with neck pain is associated with greater activity of the superficial cervical flexor muscles during a repetitive upper limb task [10], and higher frequency of pain has been associated with greater error in a head repositioning task [33]. Given the difficulties in interpreting the relationship between pain and motor control in clinical studies, experimental induction of neck pain in healthy individuals has been used to interpret clinical observations [34].

Elicitation of nociceptors has a profound effect on the spinal and supraspinal circuitries responsible for muscle control. Activation of small-diameter muscle afferents, which are sensitive to nociceptive stimuli, may affect motor neuron output through polysynaptic pathways, act on interneurons mediating larger diameter afferent input, and have an indirect effect on muscle spindle sensitivity through sympathetic activation [35••]. In addition, the descending drive from supraspinal centers is altered by pain. Given the complexity of projections following nociceptive stimuli, detailed mechanisms are difficult to study in vivo; however, common electrophysiologic techniques allow muscle coordination to be investigated, which is the final output of such complex interactions.

Experimental stimulation of cervical nociceptors by intramuscular injection of hypertonic saline has consistently shown reduced activity of the painful cervical muscle when acting as an agonist [36,37••]. Single motor unit studies in limb muscles have shown that the decreased muscle activity is due to a reflex-mediated adaptation of motor neuron discharges, which is modulated by the intensity of the painful stimulus without alteration of the muscle properties [38••,39].

Pain-induced inhibition of a cervical agonist is compensated by altered activity of antagonist or synergistic muscles when the motor output is maintained constant, so that the task can be performed in the same way in the presence of pain. For example, noxious stimulation of the sternocleidomastoid muscle during cervical flexion isometric contractions results in decreased sternocleidomastoid EMG activity with a concomitant bilateral reduction of splenius capitis and trapezius muscle activity [37••]. Similarly, splenius capitis muscle pain results in reduced EMG activity of splenius capitis during isometric cervical extension with increased activation of the trapezius muscle (synergist) [37••]. Thus, the compensatory strategy appears to depend on the biomechanical constraints dictated by the task performed. This suggests that in response to a reflex inhibition of motor neurons innervating the painful muscle, the central nervous system employs a compensatory strategy that allows the same task to be performed, taking advantage of the redundancy of the system. The observed change in motor strategy can be partially predicted from pure biomechanical considerations. Computer simulations predict reduced splenius capitis muscle activity in order for the same cervical flexion force output to be produced when inhibition of the sternocleidomastoid muscle is simulated [40]. This prediction is consistent with the strategy observed experimentally [37••]. Although this strategy is an optimal response to the painful condition in order to maintain the motor output, the overall activation of the cervical muscles is substantially altered with respect to the nonpainful condition.

In addition to a change in the coordination between muscles, local painful stimulation of a muscle may also induce a reorganization of activity within regions of the same muscle. Intramuscular injection of hypertonic saline into the cranial region of the upper trapezius results in a shift in the distribution of muscle activity toward more caudal muscle regions due to a relatively greater decrease

in activity in the cranial region [41]. The same change in activation has been observed following excitation of nociceptive afferents in the caudal region of the upper trapezius (Falla and Farina, unpublished data).

Experimental pain studies undoubtedly provide a link between pain and altered motor control, but less evidence supports the initiation of neck pain due to an altered motor control strategy. Deficits in motor control of the spine may lead to poor control of joint movement, repeated microtrauma, and thus, eventually pain [42,43]. For example, over time, augmented activity of upper trapezius and the levator scapulae muscles due to a poor working posture of the neck or awkward postures of the arms may increase compressive loads on the cervical segments and initiate a painful neck condition. Likewise, inhibition of the deep flexor or extensor muscles may affect the stability and posture of the cervical spine increasing the likelihood of neck pain. Accordingly, evidence exists that neck flexion [44], prolonged sedentary work [45], and working in upper limb elevation [46] are risk factors for the development of neck pain.

Altered Muscle Properties

Biochemical alterations in the upper trapezius muscle have been observed in people with neck pain, including increased interstitial levels of glutamate and serotonin in women with trapezius myalgia—which are positively correlated to pain intensity [47]—and higher interstitial interleukin and serotonin in people with chronic whiplash-associated disorders [48••]. Furthermore, muscle biopsies have shown evidence of disturbed oxidative metabolism of trapezius muscle fibers in people with myalgia [49,50], impaired trapezius intramuscular microcirculation in women with neck and arm pain [50,51], and a significant increase in the proportion of type-IIC fibers in the cervical flexor and extensor muscles in patients with chronic neck disorders, possibly indicating preferential atrophy of slow-twitch muscle fibers [52].

Researchers have also observed atrophy and connective-tissue infiltration of the extensor muscles—notably for the deeper muscles, the rectus capitis minor/major, and multifidi—in people with chronic neck pain [53–56].

Relationship between pain and altered muscle properties

Pain does not have a direct effect on muscle fibers, but it may activate the sympathetic nervous system as part of the defense reaction to noxious stimuli [57]. Accordingly, increased secretion of adrenaline occurs in response to acute pain stimuli such as the cold pressor test [58] and electrical stimulation of the skin [59].

Release of catecholamines, particularly adrenaline, due to sympathetic activation affects the mechanisms of release and reuptake of Ca^{++} ions from/into the sarcoplasmic reticulum (reviewed by Bowman [60]). Adrenaline and β_2-agonists slightly increase the amplitude of twitch force, especially for type II muscle fibers, and decreased twitch force duration for type I fibers only. Thus, the contractile mechanism of muscle fibers may be indirectly altered by pain.

Recently, this hypothesis was confirmed experimentally by the observation that low-threshold motor unit twitch force is shortened in the presence of pain [61]. Although the relevance of this mechanism in clinical pain conditions is unknown, a reduction in twitch duration of low-threshold motor units implies a lower force produced by motor units at a fixed discharge rate and thus impairment in force production as observed in people with neck pain. Additionally, impaired contractile properties of muscle fibers in part may explain lower neuromuscular efficiency observed in people with neck pain compared with healthy controls [24]. Finally, because the shortening effect of twitch force occurs for slow-twitch fibers [60], a larger proportion of higher threshold motor units would be required to perform the same motor task in painful conditions. This requirement may result in relatively larger activation of type II fibers in neck pain patients with respect to controls [13] and consequently a larger proportion of type II fibers in the long term [52].

A further consideration is that activation of the sympathetic nervous system may induce vasoconstriction [62], thus affecting the removal of metabolic byproducts (eg, lactic acid) during muscle contraction.

Relationship between Altered Control Strategies and Muscle Properties

Despite a lack of direct evidence in people with neck pain, altered motor control may affect the properties of muscle fibers and vice versa. For example, an altered motor strategy characterized by inhibition or continuous disuse of certain muscles may result in atrophic changes over time, due to an increase in proteolysis and a decrease in muscle protein synthesis. Likewise, continuous low-level muscle activity and overexertion of low-threshold motor units (Cinderella Hypothesis [63]) may result in peripheral adaptations including enlarged muscle fiber cross-sectional area, reduced capillary-to-fiber ratio of type I fibers, and mitochondrial disturbances, as seen in people with neck pain [49–51].

Muscle property alterations may also initiate or perpetuate an altered motor control strategy. For example, an altered metabolite concentration in the intercellular muscle interstitium due to reduced intramuscular circulation may activate chemosensitive group III and IV muscle afferents known to exert complex reflex actions on spinal neurons, thus leading to altered motor control strategies [35••]. Nevertheless, further research is required to examine the potential associations between motor control disturbances and alterations of muscle properties and the relevance of each factor in relation to the initiation, perpetuation, and recurrence of neck pain.

Furthermore, future studies must investigate the reversibility of neuromuscular changes with specific interventions and their relevance for symptoms of neck pain.

Preliminary evidence suggests that exercise intervention to improve peripheral or central neuromuscular adaptations in people with neck pain results in positive therapeutic benefits [64,65]. For example, a low-load exercise regimen that trains the deep craniocervical flexors, longus colli, and longus capitis, increased the activation of the deep cervical flexor muscles, enhanced the speed of their activation when challenged by a postural perturbation [65], and improved patient ability to maintain an upright posture of the cervical spine during prolonged sitting [66]. Similar outcomes were not achieved when patients with neck pain participated in 6 weeks of higher load strength and endurance training for the cervical muscles [65,66]. However, the strength and endurance training program for the neck flexor muscles was shown to reduce the fatigability of the sternocleidomastoid and anterior scalene muscles and improve the strength of the cervical muscles in people with neck pain [64], an outcome that did not occur following lower load craniocervical flexor training. However, neither strength and endurance training nor craniocervical flexion training changed the activation of the sternocleidomastoid muscle during a functional, repetitive upper limb task unrelated to the exercise protocols [67]. Following 6 weeks of training, both exercise groups showed higher levels of sternocleidomastoid muscle activity and a reduced ability to relax the sternocleidomastoid muscle compared to normative values, which were consistent with their preintervention scores [67]. This was observed even though both exercise groups had a significant reduction in pain and perceived disability over the training period. These findings suggest that improvements in muscle function are specific to the exercise and do not necessarily translate to changes in muscle function in tasks unrelated to the exercise performed. Additional research is required to corroborate this finding, but it implies that specific interventions are required to address both altered control strategies and muscle properties in people with neck pain.

Furthermore, current evidence suggests that a reduction in pain alone is not sufficient to reverse neuromuscular changes in patients with chronic neck pain conditions [64,67,68]. For example, manipulative therapy, which is effective for relieving neck pain [69], does not improve performance on the craniocervical flexion test, indicating no spontaneous improvement of muscle function despite the relief of pain [68]. Although the suggestion that alterations in neuromuscular function contribute to the high neck pain recurrence rate is tempting [70], the significance of persistent neuromuscular changes after resolution of pain is uncertain.

Conclusions

People with neck pain present with impaired motor function, which reflects a combination of altered neural control and muscle fiber properties. Experimental studies have clarified both the direct and indirect effects of neck pain on neuromuscular function and have assisted in interpreting clinical findings. Further studies are warranted to confirm the significance of neuromuscular changes on the perpetuation and recurrence of neck pain.

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