Abstract

The purpose of this brief review is to present evidence from experimental and clinical neck pain studies of pain-induced neuromuscular adaptations. It has been shown that clinical neck pain is associated with a substantial reorganization in the control strategies of cervical muscles during static and dynamic tasks. Experimental neck pain models allow local elicitation of nociceptive afferents, mimicking the sensory aspects of clinical pain, without major changes in muscle properties. These models may help understand the physiological mechanisms underlying the observations from clinical neck pain studies. The knowledge obtained from the interpretation of clinical findings with experimental pain models has relevance for the development of therapeutic interventions for the rehabilitation of patients with neck pain disorders.

Keywords: Neck pain; Motor control; Motor unit; Experimental muscle pain

1. Introduction

The cervical spine is a dynamic structure which serves to support and orient the head in space and transmit forces...
arising from the trunk that will influence the position of the head (Keshner, 2004). Control of cervical muscles allows three-dimensional movement of the head whilst maintaining mechanical stability. In addition to their role in movement and support, the cervical muscles are intimately related with reflex systems associated with stabilization of the head and the eyes, vestibular function and proprioceptive systems that serve general postural orientation and stability (Dutia, 1991; Keshner, 1990; Winters and Peles, 1990).

Given the complexity of the cervical spine, it is expected that alterations in afferent feedback from the muscles, for instance due to pain, induce major modifications of cervical motor control. Accordingly, altered muscle activity has been observed in individuals with neck pain during various tasks (e.g., Falla et al., 2004a; Jull et al., 2004; Mork and Westgaard, 2006). The mechanisms underlying these changes are not fully understood which poses limitations in the design of rehabilitation programs. One method to explore the pathophysiology associated with changes in neural control due to pain is the use of experimental paradigms to induce acute painful sensations, mimicking clinical pain, in healthy subjects (Arendt-Nielsen et al., 1996; Graven-Nielsen et al., 1997).

The purpose of this brief review is to present recent evidence on the effect of experimentally-induced muscle pain on the control of cervical muscles and to discuss the mechanisms of altered motor control in individuals with neck pain. The focus on both experimental and clinical pain manifestations shows that an experimental pain model may help understand the physiological mechanisms underlying alterations in neural control and muscle properties in people with neck pain disorders.

2. Nociception and motor control

2.1. Nociceptive input and motor neuron discharge

Group III and IV muscle afferents are sensitive to nociceptive stimuli (Mense and Meyer, 1985) and can be experimentally elicited in humans using exogenous or endogenous methods, including intramuscular injection of algogenic substances (Graven-Nielsen et al., 1997; Stohler and Lund, 1994), ischemia (Moore et al., 1979), exercise (Madeleine et al., 2006), repetitive mechanical pressure stimulation (Nie et al., 2005) and electrical stimulation (Marchettini et al., 1996). Nociceptive afferents in the neck–shoulder region are usually experimentally excited by intramuscular injection of chemical substances, such as hypertonic saline (e.g., Birch et al., 2001; Falla et al., in press-b; Schmidt-Hansen et al., 2006) (Fig. 1). This approach allows standardization of the intensity, location and duration of pain (Kellgren, 1938; Svensson and Arendt-Nielsen, 1995). In addition, injection of hypertonic saline produces a deep pain comparable in quality to clinical neck–shoulder muscle pain (Madeleine et al., 1998).

Muscle pain influences motor control via numerous reflex and central mechanisms (Le Pera et al., 2001, 2002). At rest, only a small and transient increase in postural electromyographic (EMG) activity has been observed in response to deep noxious stimulation of cervical muscles (Ashton Miller et al., 1990, 1943, 2004). On the contrary, during voluntary contraction, cervical muscles consistently demonstrate pain-induced inhibition when acting as agonists (Falla et al., in press-a, in press-b; Ge et al., 2005; Madeleine et al., 1999, 2006). For example, during cervical flexion contractions of linearly increasing force, injection of hypertonic saline in the sternomastoid muscle results in a force-dependent reduction of sternomastoid EMG amplitude ipsilateral to the side of pain (Falla et al., in press-b). Similarly, injection of hypertonic saline into the upper trapezius muscle reduces upper trapezius EMG activity in isometric (Ge et al., 2005; Madeleine et al., 2006) and dynamic tasks (Falla et al., in press-a; Madeleine et al., 1999). These results support the hypothesis of an inhibitory effect of pain on motor neurons (Lund et al., 1991).

Inhibited activity of the painful muscle in experimental pain studies occurs without changes in electrophysiological membrane properties of the muscle fibers, as indirectly assessed in EMG studies (Farina et al., 2004). Thus, decreased electrical activity from painful muscles with the same exerted force at the joint, is not mediated by changes in muscle properties due to injection of the painful substance (Farina et al., 2004, 2005a). Accordingly, single motor unit studies have proven a reflex-mediated adaptation of motor neuron discharges to pain in the absence of modification of muscle properties (Farina et al., 2004; Sohn et al., 2000).

2.2. Nociceptive input and muscle coordination

In isometric contractions, pain-induced inhibition of cervical muscles has been observed with unchanged force (Falla et al., in press-b). Similarly, decreased EMG
amplitude of the painful muscle during movement occurs with unchanged motor output (Falla et al., in press-a). Since peripheral muscle properties are not likely to change with injection of hypertonic saline (Farina et al., 2004), maintenance of the motor output should be due to a redistribution of load in the muscles contributing to the task. This may result in a variety of control strategies leading to the same exerted force or kinematics, especially in complex biomechanical systems such as the cervical spine.

For example, during cervical flexion isometric contractions of increasing force, noxious stimulation of the sternomastoid muscle results in decreased sternomastoid EMG activity with a concomitant bilateral increase of splenius capitis and trapezius muscle activity (Falla et al., in press-a). Reduced sternomastoid muscle activity has also been reported during cervical rotation following hypertonic saline-evoked splenius capitis muscle pain (Svensson et al., 2004).

In dynamic tasks of the upper limb, hypertonic saline-evoked unilateral upper trapezius muscle pain induces a bilateral reorganization in the coordinated activity of the three subdivisions of the trapezius (Falla et al., in press-a) (Fig. 2A). Similarly, in low load repetitive upper limb tasks, experimental upper trapezius muscle pain results in reduced EMG in the upper trapezius, with a tendency for increased activity of the infraspinatus muscle (Madeleine et al., 1999). Overall, these results suggest a dynamic reorganization of the coordination among muscles in order to minimize the use of the painful muscle with minimal disruption to the task.

Reorganization of activity following local painful stimulation has also been reported within parts of the same muscle. Two-dimensional multi-channel surface EMG recordings of the upper trapezius demonstrated a shift in activity to the more caudal region of the upper trapezius muscle following local excitation of trapezius nociceptive afferents in the cranial region. Reprinted with permission from Madeleine et al. (2006) with kind permission from the International Federation of Clinical Neurophysiology.

Fig. 2. (A) Pain induced reorganization of trapezius muscle activity during a dynamic task: Mean and standard error of EMG average rectified value (ARV) are shown for the upper and lower trapezius ipsilateral to the side of injection of hypertonic saline and the contralateral upper trapezius during performance of a 2.5-min repetitive upper limb task. Hypertonic saline-evoked unilateral upper trapezius muscle pain induces inhibition of the painful muscle and a concomitant increase in EMG activity of the lower trapezius on the painful side and upper trapezius on the contralateral side. *: significant difference pre to post injection \((P < 0.05)\). Reprinted from Falla et al. (in press-a) with kind permission of Springer Science and Business Media. (B) Pain induced reorganization of activity within the upper trapezius muscle during sustained contraction: Representative two-dimensional root mean square maps obtained from the upper trapezius muscle during 90-s isometric shoulder abduction contractions before, during, 15 min and 30 min after intramuscular injection of hypertonic saline. Dashed circle indicates location of the injection. ●: represents the position of the center of gravity of the root mean square map. Note the shift of activity to the more caudal region of the upper trapezius muscle following local elicitation of nociceptive afferents in the cranial region. Reprinted with permission from Madeleine et al. (2006) with kind permission from the International Federation of Clinical Neurophysiology.

2.3. Potential implications of pain-induced alterations in the motor strategy

Experimental pain models demonstrate that pain-induced alterations of the motor strategy may be viewed as a compensatory mechanism to allow similar motor
output in painful and non-painful conditions. Evidence for an altered motor strategy has been provided at levels ranging from the motor unit (Farina et al., 2005b) and motor unit pool (Madeleine et al., 2006) to a reorganization of activity among agonist, synergists and antagonist muscles (Falla et al., in press-b). Reorganization of muscle control may be mediated by spinal (reflex) and supraspinal (cortical motor drive) mechanisms. Similar adaptations may also occur in neck pain patients and may have implications for the perpetuation and worsening of symptoms. Pain-induced altered neural control to the muscles may indeed dispose them to overload and as a consequence, injury (Hagg, 1991) or to reduced activity with consequent atrophy of specific fiber types (Uhlig et al., 1995). This may contribute to the development of chronic symptoms and provide an explanation for muscle morphological changes and motor control deficits in patients with chronic neck pain.

3. Clinical neck pain

3.1. Changes in cervical motor control strategies

Altered motor control strategies have been frequently observed in individuals with neck pain. Neck pain has been associated with inhibition of deep muscle activation concomitant with increased activation of the superficial muscles (Falla et al., 2004b; Jull et al., 2004) (Fig. 3A). Increased activity of superficial cervical flexor muscles in people with neck pain has also been observed during isometric cervical flexion contractions (Falla et al., 2004d) and during dynamic movement of the upper limb (Falla et al., 2004c). Furthermore, people with neck pain demonstrate reduced ability to relax the anterior scalene and sternomastoid muscles following activation (Barton and Hayes, 1996; Falla et al., 2004c). Upper trapezius also shows decreased ability to relax between (Fredin et al., 1997) and following (Falla et al., 2004c; Nederhand et al., 2002) repetitive arm movements, has reduced muscle rest periods during repetitive tasks (Hagg and Anstrom, 1997; Veiersted et al., 1990) and is generally susceptible to increased activity during tasks involving mental demand (Bansevicius and Sjaastad, 1996; Laursen et al., 2002; Westgaard, 1999).

Altered motor control of the cervical spine in individuals with neck pain is also evidenced by delayed activation of the deep and superficial cervical flexor muscles in association with rapid arm movements (Falla et al., 2004a) (Fig. 3B). Changes in the feed-forward control were observed in these conditions in both the deep and superficial cervical flexor muscles but were most evident in the deep longus colli and longus capitis. Onset of the deep cervical flexor muscles exceeds the criteria for feed-forward activation with movement, indicating a significant deficit in the automatic feed-forward control of the cervical spine (Falla et al., 2004a). Moreover, activation of the deep cervical flexor muscles is direction-specific in patients with neck pain, which is consistent with a change in the strategy

Fig. 3. (A) Reorganization of cervical flexor muscle activity during a prescribed task: Representative raw EMG data are shown for a control and person with neck pain during a task of staged cranio-cervical flexion. Data are shown for the deep cervical flexors (DCF) and left (L) and right (R) anterior scalene (AS) and sternomastoid (SM) muscles. Note the incremental increase in EMG activity for all muscles with increasing cranio-cervical flexion (recorded as an increase in pressure in a pressure sensor under the cervical spine) but with lesser activity in the deep cervical flexors and greater activity in the superficial muscles for the neck pain patient. EMG calibration: 0.5 mV. Reprinted with permission from Falla et al. (2004b). (B) Delayed activation of the cervical flexor muscles during a perturbation: Representative raw EMG data are shown for the anterior deltoid (AD), deep cervical flexors (DCF), left (L) and right (R) sternomastoid (SM) and the anterior scalene (AS) muscles for a control and person with neck pain during rapid upper limb flexion. Line indicates onset of the anterior deltoid. ▲ denotes onset of neck muscle activation. Note the delayed activation of the neck muscles for the neck pain patient. Reprinted from Falla et al. (2004a) with kind permission of Springer Science and Business Media.
used by the central nervous system to control the cervical spine (Falla et al., 2004a).

3.2. Changes in the peripheral properties of the cervical muscles

Muscle biopsies on ventral (sternocleidomastoid, omohyoid, and longus colli) and dorsal neck muscles (rectus capitis posterior major, obliquus capitis inferior, splenius capitis) in individuals with neck pain have demonstrated a significant increase in the proportion of type IIC fibers with respect to control subjects (Uhlig et al., 1995). The observation was unrelated to the muscle investigated, patient diagnosis or presence of neurological symptoms (Uhlig et al., 1995). In addition, atrophy and connective-tissue infiltration of the deep suboccipital muscles have been documented in people with chronic neck pain (Andary et al., 1998; Hallgren et al., 1994; McPartland et al., 1997).

Muscle biopsies and laser Doppler flowmetry have also shown specific morphological and histological changes in the upper trapezius muscle in people with trapezius myalgia including, morphological signs of disturbed mitochondrial function (ragged red and cytochrome-c oxidase negative fibers, Kadi et al., 1998; Lindman et al., 1991b), reduced ATP content (Larsson et al., 1998; Lindman et al., 1991a) and increased cross-sectional area of type I muscle fibers despite a lower capillary to fiber area ratio (Kadi et al., 1998; Larsson et al., 1998; Lindman et al., 1991b). Such changes may be associated with overload of low-threshold motor units (Hagg, 1991) that may explain pain development in individuals performing repetitive tasks at low forces (e.g., Mork and Westgaard, 2006; Sjøgaard et al., 2000).

The observed greater proportion of type IIC fibers and lower capillary to fiber area ratio in people with neck pain is indirectly in agreement with the finding of larger changes in surface EMG signal features during sustained contraction (myoelectric manifestations of fatigue). The mean or median frequency of EMG were observed to vary more in neck pain patients than in controls during isometric contractions of the sternomastoid, anterior scalene (Falla et al., 2003) and splenius capitis (Gogia and Sabbahi, 1994) and upper trapezius muscle fiber conduction velocity demonstrated a greater decrease during repetitive shoulder elevation (Falla and Farina, 2005).

3.3. Potential relationships between altered motor control and peripheral adaptation

Results from experimental pain studies indicate that nociceptive stimulation, without damage to the muscle tissue, causes substantial changes in load sharing within and between muscles. These changes are also found in people with neck pain although, in the case of chronic neck pain, changes in motor control are probably associated with modifications in the muscle properties. For example, larger changes in upper trapezius muscle fiber conduction velocity observed in neck pain patients during repetitive shoulder elevation (Falla and Farina, 2005) cannot be reproduced in healthy subjects by experimental stimulation of the nociceptors (Falla et al., in press-a), as also confirmed at the single motor unit level (Farina et al., 2004). Therefore, findings from experimental pain studies indicate that pain by itself does not explain the electrophysiological observations in patients.

Modifications of muscle activity in people with neck pain must therefore be the result of a combination of altered neural input to muscles and changed muscle properties, in agreement with biopsy studies (Kadi et al., 1998; Lindman et al., 1991b; Uhlig et al., 1995). It can be hypothesized that perpetuation of an altered control strategy induced by nociceptive input contributes to muscle overload or disuse and thus induces additional adaptations at the muscle level. The large modification in relative muscle activation observed with experimental pain paradigms demonstrates that alterations in the level of muscle activity induced by nociception are sufficient to produce chronic changes in the long term (Falla et al., 2006).

4. Conclusion

Experimental pain models may provide a means to increase our understanding of the basic mechanisms underlying alterations of muscle activity in people with neck pain. Experimental pain research has convincingly demonstrated that nociception substantially alters the load sharing among muscles which may lead to subsequent muscle overuse or disuse in functional activities. It is also evident from experimental neck pain studies that the electrophysiological differences which have been observed in patients with neck pain cannot be solely explained by altered neural strategies. However, altered motor control strategies may enhance the changes in the peripheral properties of the cervical muscles. This knowledge has relevance for the design and implementation of therapeutic strategies for the rehabilitation of people with neck pain disorders.

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