

Review

Neuromuscular adaptation in experimental and clinical neck pain

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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.

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1. Introduction

The cervical spine is a dynamic structure which serves to support and orient the head in space and transmit forces

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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 various 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).

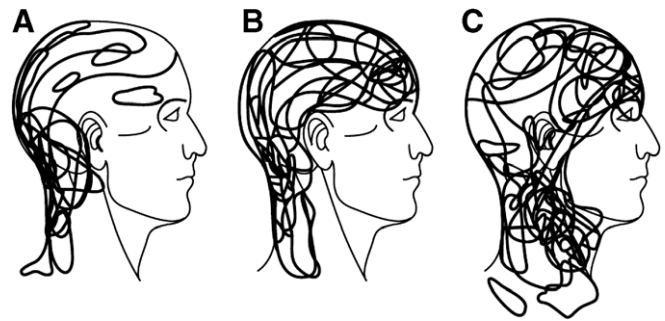


Fig. 1. Experimentally induced neck muscle pain: Area of perceived pain following infusion of 0.2 ml hypertonic saline into the (A) trapezius muscle in the middle of the muscle belly 6 cm below the external occipital protuberance; (B) splenius capitis muscle midway between the mastoid process and external occipital protuberance; and (C) sternomastoid muscle in the upper 1/3 of the muscle belly. Note the presence of both local and referred pain areas. Reprinted with permission from Schmidt-Hansen et al. (2006) with kind permission from Blackwell Publishing.

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

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