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Alterations in central motor representation increase over time in individuals with rotator cuff tendinopathy

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HIGHLIGHTS

- Individuals with rotator cuff tendinopathy present inter-hemispheric asymmetry of infraspinatus active motor threshold.
- Chronicity of pain, but not its intensity, appears to be a factor related to lower excitability of infraspinatus representation.
- This study is the first to show central motor alterations in relation with rotator cuff tendinopathy which should be considered in the rehabilitation process of this population.

ABSTRACT

Objective: To investigate whether rotator cuff tendinopathy leads to changes in central motor representation of a rotator cuff muscle, and to assess whether such changes are related to pain intensity, pain duration, and physical disability.
Methods: Using transcranial magnetic stimulation, motor representation of infraspinatus muscle was assessed bilaterally in patients with unilateral rotator cuff tendinopathy.
Results: Active motor threshold is significantly larger for the affected shoulder comparatively to the unaffected shoulder ($n = 39$, $p = 0.01$), indicating decreased corticospinal excitability on the affected side compared to unaffected side. Further, results suggest that this asymmetry in corticospinal excitability is associated with duration of pain ($n = 39$; $r = 0.45$; $p = 0.005$), but not with pain intensity ($n = 39$; $r < 0.03$; $p > 0.43$). In contrast with findings in other populations with musculoskeletal pain, no significant inter-hemispheric asymmetry was observed in map location ($n = 16$; p-values $\geqslant 0.91$), or in the amplitude of motor responses obtained at various stimulation intensities ($n = 16$; $p = 0.83$).
Conclusion: Chronicity of pain, but not its intensity, appears to be a factor related to lower excitability of infraspinatus representation.
Significance: These results support the view that while cortical reorganization correlates with magnitude of pain in neuropathic pain syndromes, it could be more related to chronicity in the case of musculoskeletal disorders.
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1. Introduction

Shoulder pain affects about 20% of the population and is second only to low back pain in prevalence of musculoskeletal (MSK) conditions (Pope et al., 1997; Picavet and Schouten, 2003). Disorders of rotator cuff (RC) tendons is the most common pathology of the shoulder, with RC tendinopathy accounting for 35% to 50% of rendered diagnoses (Chard et al., 1991; van der Windt et al., 1995). Clinical trials suggest that long-term outcomes of patients with RC disorders receiving rehabilitation are comparable to that of patients treated with surgery (Brox et al., 1999; Haahr and Andersen, 2006; Seitz et al., 2011). Regardless of treatment, more than a third of patients do not have a positive outcome as they

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continue to present pain and disability after the intervention (Seitz et al., 2011).

It has been recently hypothesized that reorganization of the somatosensory and motor cortices could explain part of the deficits associated with RC tendinopathy (Myers et al., 2006; Roy et al., 2009; van Vliet and Heneghan, 2006). This reorganization could also explain the chronicity of the symptoms and the lack of treatment effectiveness for one third of patients. Central changes underlying movement deficits associated with RC tendinopathy remain unclear. To our knowledge, they have never been directly tested in this population. However, changes in the central nervous system (CNS) have been documented in patients with other MSK disorders, primarily regarding the functional organization of the primary somatosensory and motor cortices (van Vliet and Heneghan, 2006; Boudreau et al., 2010). Consequently, understanding the involvement of CNS in MSK disorders is now considered as a key aspect to improve the management of patients with such disorders (Tsao et al., 2010; Tsao et al., 2008).

Current interventions for RC tendinopathy mainly target deficits at the joint level, such as altered posture (Gumina et al., 2008; Finley and Lee, 2003; Kalra et al., 2010; Kebaetse et al., 1999), muscular deficit (weakness/lack of endurance) (Ludewig and Cook, 2000; Wadsworth and Bullock-Saxton, 1997; Cools et al., 2003; Cools et al., 2004; Cools et al., 2007) and soft tissue tightness (Tauro and Paulson, 2008). However, if central (neural) changes are present in individuals with RC tendinopathy, then specific interventions, such as sensorimotor training, should be performed in order to reverse these changes and thereby decrease pain during arm movement. Reversal of central changes related to the peripheral lesion could be an important step towards recovering a normal level of shoulder function.

Given the high prevalence of RC tendinopathy, and continued pain symptoms in one third of the patients despite treatment, research to better understand the factors that may explain the persistence of pain in individuals with RC tendinopathy is needed in order to guide the development of more effective treatment approaches. The first objective of the current study was to investigate whether individuals with RC tendinopathy exhibit changes in the excitability and location of the cortical motor representation of a RC muscle. The second objective was to determine whether such central changes are related to clinical variables such as pain intensity, pain duration, and physical disability.

2. Methods

2.1. Participants

Thirty-nine participants (18 women, 21 men; mean age 46 ± 11 years) with unilateral RC tendinopathy were recruited (see Table 1 for participants' characteristics). Participants were considered eligible if they were aged between 18 and 65, presented pain in one shoulder, and had at least one positive finding in each of these categories: 1) painful arc of movement during flexion or abduction; 2) positive Neer or Kennedy-Hawkins impingement signs; 3) pain on resisted lateral rotation, abduction or Jobe test. The diagnosis accuracy of these tests for RC tendinopathy has been shown (sensitivity & specificity $\geqslant$ 0.74, Positive likelihood ratio = 3–5) (Michener et al., 2009).

Exclusion criteria were previous shoulder surgery, cervicobrachialgia or shoulder pain during neck movement, shoulder capsulitis ($\geqslant$ 30% restriction of passive glenohumeral movement for two or more directions), clinical signs of a full thickness RC tear (dramatic weakness on resisted movement or positive Lag signs), pain or movement limitation at the unimpaired shoulder, current use of steroidal anti-inflammatory or opioids drugs, as well as contraindications for magnetic resonance imaging (MRI) or transcranial magnetic stimulation (TMS) (e.g. metallic or electronic implants, pregnancy, history of epilepsy, etc.) (Rossi et al., 2009). None of the subjects had a reported history of neurological deficit or systemic disease. This study was approved by the Ethics Committee of the Quebec Rehabilitation Institute. All participants gave their written consent after being informed of the nature and purpose of the study.

2.2. Experimental design

Each participant took part in two evaluation sessions within 7 days. During the first session, after the evaluation of eligibility criteria, participants completed a questionnaire on sociodemographic, symptomatology, and comorbidity. Hand dominance was determined using the revised Edinburgh Handedness Inventory (Oldfield, 1971). Then, the level of pain and disability of the shoulder was evaluated using the French Canadian version of the Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire (Durand et al., 2005). The DASH is a 30-item self-reported questionnaire that addresses difficulty in performing various physical activities that require upper extremity function (21 items); symptoms of pain, activity-related pain, tingling, weakness, and stiffness (5 items); and impact of disability and symptoms on social activities, work, sleep, and psychological well-being (4 items) (Hudak et al., 1996). Response options are rated on a 5-item Likert scale. The final score range from 0 (no disability) to 100 (most severe disability).

Visual analog scales (VAS; 0 to 10) were also used to estimate participants' reported pain level during the 48 h preceding the test at rest, during daily activities, and during nighttime. For the first 16 participants included in the study, a structural MRI of the brain was obtained in order to use a frameless stereotaxy neuronavigation system (Brainsight, Rogue Research, Canada) for cortical mapping. This system allows accurate and reliable positioning of the TMS coil. For the other 23 participants, frameless stereotaxy neuronavigation system was also used, but participants' heads were coregistered with a standard MRI. In the following days, all the participants took part in a second evaluation session during which the motor cortex representation of a RC muscle was assessed bilaterally.

2.3. Cortical mapping

The infraspinatus has been selected as the target muscle for cortical mapping as it is a RC muscle for which the activation pattern has been shown to be altered during arm elevation in individuals with RC tendinopathy (Reddy et al., 2000). Furthermore, it is the only RC muscle for which the electromyographic (EMG) activity can be directly recorded using surface electrodes. Mapping was performed using a BiStim^2 stimulator (combined pulse mode) connected to a 70-mm figure-of-eight coil (Magstim Company Limited, United Kingdom). Stimuli were applied over grid sites spaced 1 cm apart and located over the upper limb representation of primary motor cortex (M1) in the contralateral hemisphere (using the neuronavigation system).

Motor evoked potentials (MEPs) were collected from the EMG recording of the infraspinatus muscle. After skin preparation, a pair of Ag/AgCl surface electrodes (1 cm^2 recording area) was placed over the infraspinatus muscle. The ground electrode was positioned over the ipsilateral acromion. Electrode placement over infraspinatus was based on Delagi & Perotto, i.e. 3–4 cm below and running parallel to the spine of the scapula, over the infraspinatus fossa (Delagi and Perotto, 1980). This arrangement has shown high levels of agreement between surface and intramuscular recordings (Johnson et al., 2011). EMG signals were amplified

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