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Modulation of motor variability related to experimental muscle pain during elbow-flexion contractions

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ABSTRACT

Experimental muscle pain typically reorganizes the motor control. The pain effects may decrease when the three-dimensional force components are voluntarily adjusted, but it is not known if this could have negative consequences on other structures of the motor system. The present study assessed the effects of acute pain on the force variability during sustained elbow flexion when controlling task-related (one-dimensional) and all (three-dimensional) contraction force components via visual feedback. Experimental muscle pain was induced by bolus injection of hypertonic saline into m. biceps brachii, and isotonic saline was used as control. Twelve subjects performed sustained elbow flexion at different levels of the maximal voluntary contraction (5–30% MVC) before, during, and after the injections. Three-dimensional force components were measured simultaneously with surface electromyography (EMG) from elbow flexors and auxiliary muscles. Results showed that force variability was increased during pain compared to baseline for contractions using one-dimensional feedback ($P < .05$), but no significant differences were found for three-dimensional feedback. During painful contractions (1) EMG activity from m. trapezius was increased during contractions using both one-dimensional and three-dimensional feedback ($P < .05$), and (2) the complexity of EMG from m. triceps brachii and m. deltoid was higher for the three-dimensional feedback ($P < .05$). In conclusion, the three-

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dimensional feedback reduced the pain-related functional distortion at the cost of a more complex control of synergistic muscles.

1. Introduction

Musculoskeletal pain alters muscle activity and coordination leading to behavioral changes. In consequence, musculoskeletal pain patients are prone to have difficulties performing accurate contractions (Bandholm, Rasmussen, Aagaard, Diederichsen, & Jensen, 2008), affecting their quality of work and daily life function. Nevertheless, the assessment of pain effects is complex in pathological conditions, since musculoskeletal pain is related to diverse processes that finally result in motor dysfunction. Therefore, several experimental pain models have been proposed as potential strategies to mimic pathological conditions, reducing the intervening factors and facilitating the study of pain mechanisms (Graven-Nielsen, 2006).

Short term muscle adaptations to experimental muscle pain take place as an attempt to reduce pain and prevent further tissue damage (Hodges & Tucker, 2011). Modulation of muscle coordination during pain has been reported as increased and decreased activity in various muscle groups (Falla, Farina, Dahl, & Graven-Nielsen, 2007; Graven-Nielsen, Svensson, & Arendt-Nielsen, 1997; Schulte et al., 2004). Moreover, spatial redistribution of muscle activity within a muscle has also been observed as a response to experimental muscle pain (Falla, Arendt-Nielsen, & Farina, 2009; Madeleine, Leclerc, Arendt-Nielsen, Ravier, & Farina, 2006). In this regard, pain-related muscle adaptation can impair movement or decrease contraction performance (Hodges & Tucker, 2011), increasing force fluctuation during pain in isometric motor tasks (Bandholm et al., 2008; Salomoni & Graven-Nielsen, 2012a). Nonetheless, numerous reports have shown increasing or decreasing of muscle activity without affecting contraction quality, e.g. same force steadiness during isometric contractions (Bandholm, Rasmussen, Aagaard, Jensen, & Diederichsen, 2006) or same error and variability during dynamic motor tasks (Birch, Graven-Nielsen, Christensen, & Arendt-Nielsen, 2000; Ervilha, Farina, Arendt-Nielsen, & Graven-Nielsen, 2005; Willigenburg, Kingma, & van Dieën, 2012), suggesting an effective redundancy in the strategy of muscle recruitment and control during pain. However, pain-related muscle adaptations might result in a situation in which the load is increased across the structures of the motor system.

Previous studies inducing experimental muscle pain based their hypotheses on the pain adaptation theory, predicting reduced agonist muscle activity and increased antagonist muscle activity (Ervilha et al., 2005; Salomoni & Graven-Nielsen, 2012a; Schulte et al., 2004). However, several reports have found unchanged muscle activation during m. biceps brachii pain (Salomoni & Graven-Nielsen, 2012a; Schulte et al., 2004) and neuromuscular facilitation of both elbow flexor and extensor muscles, contrasting with the principles of the pain adaptation theory (Hodges & Tucker, 2011). Most studies have quantified the level of muscle activity based on linear analysis of dynamic changes of the muscle activity during a motor task. However, recent reports highlighted the relevance of quantifying the structure of the variability (sample entropy) of the motor output during pain (Madeleine, Mathiassen, & Arendt-Nielsen, 2008; Madeleine, Nielsen, & Arendt-Nielsen, 2011; Mathiassen, 2006). In line, low-back pain patients have shown higher complexity of the abdominal oblique muscle activity during trunk flexion (Kaufman, Zurcher, & Sung, 2007; Svendsen, Svarrer, Laessoe, Vollenbroek-Hutten, & Madeleine, 2013). Therefore, analyzing the complexity of the muscle activity could reveal changes not found by linear approaches during pain.

Pain adaptations on voluntary movements are less pronounced or even not detected when high precision motor task are required (Birch et al., 2000; Ervilha, Arendt-Nielsen, Duarte, & Graven-Nielsen, 2004; Willigenburg et al., 2012). Two approaches have been used to increase the demanded precision: increase the sensitivity of the visual feedback (Birch et al., 2000) or constrain the degrees of