



Experimental pain has a greater effect on single motor unit discharge during force-control than position-control tasks



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HIGHLIGHTS

- The manner that pain changes muscle activity during voluntary movements might not be appropriate for tasks with a more postural objective.
- Postural-control tasks involve different changes in activity of single motor units, the final common output of the motor system, than those commonly observed in force-control tasks.
- Unique responses of different tasks to nociceptive input challenge the veracity of simple theories of motor system adaption to pain.

ABSTRACT

Objective: When matching target force during pain, single motor unit (SMU) discharge is modified in a manner thought to redistribute load in painful tissue. This adaptation might not be appropriate when maintaining joint posture against an external load. We compared changes in SMU discharge rate of knee extensor muscles in a force-control and a position-control task during pain.

Methods: Thirteen healthy adults (31 ± 6 years) performed position- and force-control contractions using matched loads in non-pain and pain states. Pain was induced by injection of hypertonic saline into the infrapatellar fat pad. Intramuscular and surface electromyography of knee extensor and flexor muscles was recorded.

Results: When considering the discharge of a select population of SMUs that were recorded across all conditions performed on the same day, there was a decrease in mean discharge rate, and this was smaller in the position- than force-control task for the same SMUs. A similar tendency was observed for SMUs recorded on different days. However, gross agonist muscle activity (which incorporates SMUs that are not included in the discharge rate analysis because they were not present in all conditions) increased in both tasks during pain. Gross antagonist muscle EMG only increased in the force-control task.

Conclusion: The effect of pain on muscle activity appears unique to the contraction type, with less influence during position- than force-control tasks.

Significance: Simplistic theories of pain adaptation of movement during voluntary efforts cannot be extrapolated to more postural functions. This has implications for understanding movement changes that may underpin persistence/recurrence of pain and the management of musculoskeletal pain.

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1. Introduction

Muscle activity and kinematics underlying complex and simple motor tasks are altered during pain. Early pain theories explained these adaptations on the basis of generalized facilitation (Roland, 1986) and/or inhibition of muscle activity (Lund et al., 1991).

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Recent data provide evidence of more complex redistribution of muscle activity during pain in tasks where a participant is required to maintain a force output in pain free followed by painful trials (Madeleine et al., 2006; Falla et al., 2007; Hodges, 2011; Tucker et al., 2012). For example, single motor unit (SMU) discharge rate decreases or ceases for some units with concurrent recruitment of a new population of units (presumably to assist force/torque maintenance) when pain is induced in/near the muscle (Tucker and Hodges, 2009) and this has been argued to modify the direction of external force to adjust distribution of load applied to the painful tissue (Tucker et al., 2009; Tucker and Hodges, 2010).

Although consistent for a range of muscles (e.g., hand and leg (Tucker et al., 2009); jaw (Minami et al., 2013)), these observations are limited to contractions where a participant voluntarily exerts an isometric force against a resistance with feedback (force-controlled contractions) and small changes in force characteristics present no compromise to attainment of the task objective. Yet our movement repertoire involves many behaviors, including postural actions that aim to minimize disturbances and maintain the position of a body part against internal and external forces and are generally initiated/controlled subconsciously. Although early theorists argued that postural-type contractions may not be affected by pain (Lund et al., 1991), there is substantial evidence that this is not the case. For instance, gross activity of trunk muscles involved in anticipatory postural adjustments is altered in people with pain (e.g., onset of muscle activity can be delayed (Hodges and Richardson, 1996; Hodges et al., 2003) or augmented (Moseley and Hodges, 2005), and co-contraction of opposing muscle groups can increase (van Dieen et al., 2003)). Changes in discharge of SMUs during pain have not been explored in postural tasks. Pain adaptations in SMU activity in simple voluntary isometric contractions, which have consequences for force characteristics such as the force direction, might not be appropriate for contractions underlying postural adjustments where even small disturbances can have significant consequences and other types of adaptation may be favored. This could include increased joint stiffness by increased co-contraction of agonist and antagonist muscle pairs to simplify the maintenance of joint position (van Dieen et al., 2003).

Comparison of adaptations in muscle activity between tasks that require maintenance of force (force-control) or maintenance of a joint angle (position-control) with equivalent net muscle torque (Hunter et al., 2002), present a well-controlled experimental paradigm to determine whether maintained contractions of a postural nature follow the principles identified for simple voluntary force maintenance tasks. The position- and force-control tasks have the same mechanical requirements, involve identical muscle torques, and demonstrate similar reductions of maximal force amplitude as a result of fatigue at the time of task failure (although the time to failure differs). The similarity between tasks is highlighted by the identical behavior at the start of the contractions, which includes similar amplitude of EMG of the prime mover muscles. However, neuromotor control during these static tasks differs in several respects and these become apparent when task demands increase during the sustained contractions. Most notably, development of fatigue and the time task failure occur earlier in the postural-type task (position-control) (Maluf et al., 2005; Hunter et al., 2008; Baudry et al., 2009; Rudroff et al., 2010b; Poortvliet et al., 2013). This is thought to be explained by a combination of central mechanisms such as a difference in descending drive and motor output from the spinal cord to the primary muscles (i.e., greater recruitment (Mottram et al., 2005; Poortvliet et al., 2013), and more rapid decline in discharge rate of SMUs in the agonist muscle (Mottram et al., 2005; Baudry et al., 2009; Rudroff et al., 2010a), greater activity of antagonist and more proximal muscles to increase stability (Hunter et al., 2004; Maluf and Enoka, 2005;

Rudroff et al., 2007; Poortvliet et al., 2013)), and peripheral mechanisms such as differences in stretch reflex sensitivity (Maluf and Enoka, 2005).

This study compared changes in SMU discharge rate of the knee extensor muscles in a task that required voluntary matching of a target force against a visual target (force-control) and a task that involved maintenance of a joint posture/angle against an external force (position-control) during pain. We hypothesized that changes in SMU discharge rate during pain may be less profound for tasks that are postural in nature than the changes commonly observed in simple voluntary force maintenance tasks. Other strategies of adaptation such as increased co-contraction to stiffen the segment might be more apparent when the task goal is to maintain a joint posture.

2. Materials and methods

2.1. Participants

Thirteen healthy adults (31 ± 6 years, 8 men and 5 women) volunteered to participate in this study. None of the participants had a history of significant knee or leg pain, any known neurological conditions or previous surgery to the knee or leg. All procedures conformed to the Declaration of Helsinki, and were approved by the Institutional Medical Research Ethics Committee. Participants signed informed consent before taking part in this study.

2.2. Electromyography (EMG)

Recordings of myoelectric activity of knee and hip muscles were made using fine-wire and surface electrodes. Discharge properties of SMUs and global amplitude measures of activity of the agonist knee extensor muscles (vastus lateralis (VL) and vastus medialis (VM)) were evaluated using electromyography recordings made with intramuscular fine-wire electrodes (two pairs of two Teflon-coated 100 μ m stainless-steel wires with 0.5 mm insulation removed, and bent ~ 1 –2 mm from the tip) that were inserted into the muscles using a hypodermic needle ($\sim 25\text{G} \times 25$ mm). The electrodes were inserted into the muscle belly approximately 2 cm proximal to the distal border of the muscle (determined by palpation of a muscle contraction) and once in place, the needle was removed, leaving the wire electrodes in place. The wires were secured to the skin surface using tape to minimize electrode movement between different parts of the tasks. Surface electrodes (bipolar, AgCl discs, 8 mm diameter, 20 mm inter-electrode distance, Noraxon, USA) were placed over the proximal (tensor fasciae latae (TFL)) and antagonist (biceps femoris (BF) and semitendinosus (ST)) muscles.

Before insertion of fine wires, participants performed a series of maximal voluntary contractions (MVC) that targeted each of the recorded muscles for normalization of surface EMG recordings. Three contractions were performed for each muscle against manual resistance with the participant in the same position as that used for the experimental tasks to ensure muscle lengths were maintained. Participants were verbally encouraged, to gradually increase their isometric force to maximum over 3 s, hold for 3 s, and return to rest. A minimum of three MVCs was performed separated by a recovery time of at least 120 s. MVCs were repeated until the two highest rectified (and smoothed: time constant: 0.1 s) EMG values of each muscle differed by less than 5%. The maximal EMG amplitude was determined as peak rectified EMG produced during any of these contractions.

EMG data were pre-amplified 1000–10,000 times, bandpass filtered (20 Hz to 1 kHz for surface EMG and 20 Hz to 10 kHz for intramuscular EMG), notch filtered at 50 Hz, and sampled at 2 or

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