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RESEARCH ARTICLES

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The effect of muscle pain on elbow flexion and coactivation tasks

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Abstract The effects of muscle pain on movement can easily be observed in daily life routines. However, the influence of muscle pain on motor control strategies has not been fully clarified. In this human experimental study it was hypothesized that muscle pain affects the motor control of elbow flexion movements, in different combinations of range of motion and target size, by decreased agonistic muscle activity and increased antagonistic muscle activity with consequent implications on kinematic parameters. The effects of experimentally induced muscle pain on movement strategy for: (1) small and large range of motion (ROM) elbow flexion movements towards a wide target, (2) large ROM flexion movements towards a narrow and wide target, and (3) subsequent coactivation of agonistic and antagonistic muscles to elbow flexion were assessed. Muscle pain induced by injections of hypertonic saline (1 ml, 5.8%) in either m. biceps brachii or m. triceps brachii caused similar effects on the movements. For low accurate movements the initial (100 ms) integrated electromyographic (EMG) activity of m. biceps brachii was decreased during muscle pain. In contrast, integrated EMG of the entire m. biceps brachii burst was decreased by muscle pain only for small ROM at a low accuracy, which also showed decreased EMG activity of m. triceps brachii and m. brachioradialis, together with increased activity of m. trapezius. Finally, high accurate movements and post-movement coactivation were generally not modulated by muscle pain. In summary, the present study shows that acute muscle pain can perturb the motor control strategy, which might be highly important in occupational settings where such a change may need compensatory actions from other muscles and thereby eventually contribute to the development of musculoskeletal pain problems.

Keywords Movement strategy · Electromyography · Pointing movement · Motor control · Single-joint movement

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Introduction

The effects of muscle pain on movement can easily be observed in daily life routines and in clinical practice. Several studies have reported interactions between muscle pain and voluntary as well as reflex motor function (Arendt-Nielsen et al. 1996; Graven-Nielsen et al. 1997; Madeleine et al. 1999a, 1999b; Zedka et al. 1999; Svensson et al. 1999; Wang et al. 2002). The electromyographic (EMG) amplitude and the force level of maximal voluntary contraction (MVC) are decreased during experimentally induced as well as non-experimentally induced muscle pain conditions (Graven-Nielsen et al. 1997; Backman et al. 1988; Suzuki and Endo 1983). However, submaximal isometric contractions during experimentally induced pain were found to cause no changes in either EMG activity or force, but reduced the endurance time compared to a non-painful condition (Ashton-Miller et al. 1990; Graven-Nielsen et al. 1997). During dynamic activity, it has been found that muscle pain modulates voluntary activation by either increasing EMG activity in phases where it is normally silent and decreasing electromyographic activity in phases where it is normally activated (Arendt-Nielsen et al. 1996; Graven-Nielsen et al. 1997; Svensson et al. 1998; Zedka et al. 1999). In addition, Birch et al. (2000) concluded that during low precision tasks the muscle activity was decreased in the muscle exposed to pain and muscle pain had no effect on high precision tasks. Most of the above effects of muscle pain follow the pain adaptation

model where muscle pain affects muscle activation by inhibition of agonistic muscle and excitation of antagonistic muscle that results in a reduced force production as well as a reduced range of motion and velocity (Lund et al. 1991).

Strategies used by the central nervous system in controlling human movement during non-pain conditions have been described in the literature. For example, elbow flexion movements (pointing) performed with two levels of loads or distances while the target size is kept the same have opposite effects on speed and movement time but similar effects on agonist muscle activity, especially in the integrated EMG of the initial agonistic burst (Corcos et al. 1989). This behavior suggests that the initial excitation to the motoneuron pool, once chosen, is constant and insensitive to the speed (speed-insensitive strategy) at which the subject performs this kind of movement (Gottlieb et al. 1989). If instead of changing the load or distance the target size is narrowed, demanding high accuracy in the task and as a consequence a lower speed, the initial slope of the agonist EMG bursts will decrease. In this case there is a different excitation pattern of the motoneuron pool, which means that the strategy chosen by the central nervous system is sensitive to changes in the movement speed (Corcos et al. 1989). In a previous study, muscle pain was shown to alter the internal representation used in the step initiation motor program (Madeleine et al. 1999b). It was revealed by increased reaction time, longer duration of the forward-oriented stepping movement, and decreased mechanical output during pain condition. These parameters together show a general inhibition, which leads to the use of a different step initiation strategy. Effects of muscle pain in the initial integrated EMG burst may indicate a modulation of the motor control strategies.

The activation of the antagonistic muscle at the same time as the agonistic, defined as co-contraction (Hammond et al. 1988), is a strategy that provides a way to adapt the limb to external perturbating forces and forces arising from multijoint dynamics (Gribble and Ostry 1998). How muscle pain perturbs this strategy is not exactly known.

In the present study it was hypothesized that muscle pain affects the control of elbow flexion movements by decreased agonistic muscle activity and increased antagonistic muscle activity with consequent implications on kinematic parameters. The effects of experimental muscle pain on movement strategy for horizontal elbow flexion were assessed during: (1) a small and large range of motion (ROM) elbow flexion movements towards a wide target, (2) large ROM flexion movements towards a narrow and wide target, and (3) subsequent coactivation of agonistic and antagonistic muscles.

Materials and methods

Subjects

Ten subjects [7 men, 3 women; mean age 26±4 (±SD) years, 173 ±8 cm, and 71±12 kg] participated in experiment 1 and 15 subjects (13 men, 2 women; mean age 27±5 years, 178±11 cm, and 78 ±17 kg) participated in experiment 2. All subjects had no known history of locomotor apparatus disorder or musculoskeletal pain problems. Volunteers received information about the experiment and subsequently written consents were obtained prior to inclusion. The study was conducted in accordance with the Helsinki Declaration and was approved by the local Ethics Committee.

Protocol

Horizontal elbow flexion pointing movements were performed in two experiments. In the first, a small and a large range of motion were performed aiming at a large target, characterizing low accurate tasks. In the second experiment, one range of motion was used to reach a narrow and a large target. Pointing movements aiming at a narrow target demand high accuracy while low accuracy is necessary for large targets.

In both experiments, pain was induced by intramuscular injection of hypertonic saline. Injections were made randomly in m. biceps brachii (BB) or m. triceps brachii (TB) in two sessions with a time interval of a minimum of 7 days (in two subjects, 3 days). Elbow flexion movements were assessed in three conditions: pre-pain, during-pain, and post-pain. Angular position of the elbow joint and electromyography (EMG) from m. trapezius (TZ) (upper fibers), m. biceps brachii (long head), m. triceps brachii (lateral head), and m. brachioradialis (BR) were recorded. Biceps brachii muscle and triceps brachii muscle were chosen because they act as primary agonist and antagonist, respectively, to elbow flexion. Trapezius muscle and brachioradialis were chosen due to their synergistic role. The EMG intensity of each muscle was normalized by the respective peak (average of two trials) of maximal voluntary isometric contraction (MVC) recorded before the experimental-pain induction.

Apparatus and movement

Each subject was seated comfortably with the dominant arm in the semi-prone position, strapped to a high-adjustable support and fixed at 45° of shoulder horizontal flexion in 90° of abduction. Shoulder angles were defined relative to the coronal plane with 0° corresponding to the arm aligned with this plane. The forearm was strapped to a light manipulandum horizontally aligned with the arm support (Fig. 1). The elbow joint was positioned just above the fulcrum of the manipulandum so that only horizontal movements were permitted. The wrist was fixed in order to avoid unnecessary muscle activity during potential wrist flexion/extension.

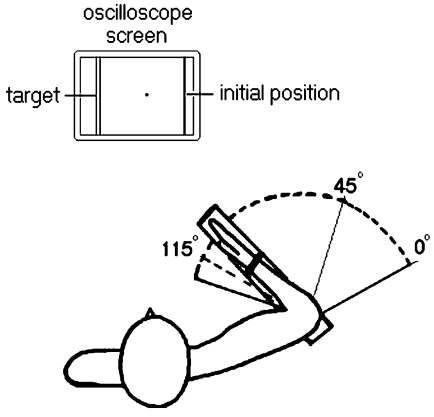

Fig. 1 Experimental setup. The oscilloscope was positioned in front of the subject, showing elbow movements based on electrogoniometer recordings. The final position was 115° in all experiments whereas the initial position was set according to the chosen range of motion

In experiment 1, two long sticks making a fixed angle of 12° were used as target. A third stick indicated the initial positioning marker. In experiment 2, the amplitude to be performed and the targets were shown on an oscilloscope. For every movement the final position was the same: upper limb in the horizontal plane at 45° of horizontal shoulder flexion and 115° of elbow flexion (full elbow extension equals 0°).

Subjects performed 12 trials of elbow flexion (the first and the last were not included in the analysis). An interval of 1 min was given between the two series of different amplitudes (30° and 90°) or target sizes (22° and 3°) and a 10-min interval between pre-pain and during-pain conditions. Thirty minutes after the pain had vanished, the post-pain trials were performed. Muscle activity was recorded during the set of trials for each combination of ROM and target size.

All subjects were instructed to "perform the movements as fast and as accurately as possible" (in experiment 2 the instruction to "strongly fix the upper arm to the target" was added). The subjects started the movement immediately after hearing a beep signal (300 Hz) and returned to the initial position after a second beep (600 Hz). The time between the first and second beeps was fixed at 1.9 s and the time between the second beep and the next go-beep was randomized in a range from 7 to 13 s.

Experimental muscle pain

One bolus of 1.0 ml sterile hypertonic saline (5.8%) was injected intramuscularly, at a rate of 90 ml/h, via a disposable stainless needle (27G, 40 mm) connected via a tube (IVAC, G30303) to an infusion pump (ALARIS Medical Systems, Asena, UK). A 10-cm electronic, visual analog scale (VAS) where 0 cm indicated "no pain" and 10 cm "intolerable pain" was used to score the pain intensity. The signal from the VAS was recorded continuously, allowing the subjects to adjust the values whenever needed. The adjustment was done with the contralateral hand not involved in the exercise. Subjects were asked to focus on the VAS in the intervals between the individual trials. The mean VAS scores obtained during and between the trials were calculated.

Kinematic and EMG recordings

An electrogoniometer (Biometrics SG110, Ladysmith, USA) was used to measure elbow angular position. A pair of surface electrodes (Medicotest 72001-k, Ølstykke, Denmark) was placed in the direction of the muscle fibers (2 cm apart) on shaved, abraded ethanol-cleaned skin, as follows: (1) trapezius (upper portion)—2 cm lateral to the midpoint of the lead line between the angle of the acromion and the spinal process of the seventh vertebra; (2) biceps brachii (long head)—on the lead line between the acromion and the fossa cubit at one-third the distance from the fossa cubit; (3) triceps brachii (lateral head)—1 cm lateral to the lead line just on the mid-point between the acromion and the olecranon process; and (4) brachioradialis—on the muscle belly, 5 cm distally from the elbow joint. The EMG signals were bandpass filtered (20–500 Hz), amplified (1,000–10,000; CounterPoint MK2, Dantec, Skovlunde, Denmark) times and sampled at 1 kHz.

Pain intensity (VAS), electrogoniometric, electromyographic, and beep signals were acquired in parallel by an analog/digital converter and stored on a personal computer. All parameters were averaged across ten trials.

Data analysis

Angular position was digitally filtered (low-pass, fourth order, and zero-phase-lag Butterworth, filter with a 10 Hz cutoff frequency) and differentiated to obtain velocity and acceleration. Acceleration and EMG onset were automatically determined by a threshold procedure.

EMG signals were digitally band-pass filtered from 20 to 400 Hz (Butterworth), full-wave rectified, low-pass filtered (Butterworth) with a 50 Hz cut-off frequency and normalized by the maximal voluntary isometric contraction. The following variables were extracted from the data:

1. Integrated EMG amplitude over three epochs: epoch 1, the EMG integral of 100 ms before the biceps muscle EMG activity onset (pre-movement epoch); epoch 2, the EMG integral from the onset of biceps muscle EMG activity to the acceleration offset; epoch 3, the EMG integral of 100 ms after epoch 2 (post-movement epoch). Epoch 2 was time normalized (divided by duration and multiplied by 100) in order to be comparable with epoch 1 and epoch 3.
2. Integrated acceleration profiles
3. Movement time: the time interval from the acceleration onset to the acceleration offset
4. Time to peak velocity: time between acceleration onset and peak velocity
5. Effective amplitude: difference between the angular position when the acceleration offset and the acceleration onset occur
6. Reaction time: the time between the go-beep signal and the acceleration onset
7. Q_{100}: the integrated EMG profile (IEMG) from the onset to 100 ms
8. Coactivation index during epoch 3:

$$\text{Coactivation} = 2 \bullet \frac{\min\{\text{IEMG}\textit{biceps brachii}(t),\ \text{IEMG}\textit{triceps brachii}(t)\}}{\text{IEMG}\textit{biceps brachii}(t) + \text{IEMG}\textit{triceps brachii}(t)\ \mathrm{d}t} \bullet 100$$

where t is epoch 3 (100 ms).

Statistical analysis

Data are presented as means and standard errors of the mean (SEM). Three-way ANOVAs were used to examine the effects of ROM or target size, injected muscle (m. biceps brachii and m. triceps brachii), and condition (pre-, during and post-pain). When it was found to be significant, the Student-Newman-Keuls (SNK) post hoc test was used for multiple comparisons. A significance level of $P<0.05$ was accepted.

Results

In more than 99% of the trials the subjects were able to perform the task with the instructed accuracy. The number of rejected trials was equivalent for pain and no-pain conditions. The mean VAS scores during trials for each

Table 1 Mean and standard error of the mean of the VAS scores (acquired during each trial) according to the muscle injected and to the combination of ROM and target size

	M. biceps pain				M. triceps pain			
ROM (°)	30	90	70	70	30	90	70	70
Target size (°)	12	12	3	22	12	12	3	22
VAS (SEM) mm	36 (5)	16 (4)	25 (4)	20 (5)	31 (5)	13 (3)	24 (5)	27 (5)

Table 2 Mean and standard error of the mean of reaction time, movement time, time to velocity peak, and the effective amplitude (*pre* pre-pain, *dur* during muscle pain, *post* post-pain). Significant increase during pain compared to both pre- and post-pain is illustrated by [a]

	ROM 30°			ROM 90°			ROM 70°					
	Target size 12°			Target size 12°			Target size 3°			Target size 22°		
	Pre	Dur	Post	Pre	Dur	Post	Pre	Dur	Post	Pre	Dur	Post
Reaction time (ms)	155 (19)	179[a] (25)	150 (22)	162 (22)	163 (21)	156 (23)	194 (12)	209[a] (12)	189 (12)	209 (18)	226[a] (18)	218 (18)
Movement time (ms)	249 (27)	272[a] (33)	243 (29)	239 (39)	349 (47)	342 (47)	654 (68)	665 (67)	655 (57)	362 (15)	381 (18)	359 (15)
Time to peak velocity (ms)	177 (8)	184[a] (11)	177 (7)	242 (15)	262[a] (16)	246 (17)	304 (14)	324[a] (15)	302 (15)	233 (6)	251[a] (8)	236 (8)
Effective amplitude (°)	34 (1.3)	32 (1.4)	34 (1.3)	93 (3.0)	92 (2.9)	90 (2.8)	73 (1.8)	72 (2.0)	74 (1.9)	74 (1.6)	73 (1.6)	75 (1.7)
Coactivation index (%)	68 (7.0)	66 (6.3)	66 (7.7)	69 (4.0)	72 (4.6)	72 (4.8)	64 (5.3)	64 (5.4)	61 (6.0)	64 (4.8)	66 (4.4)	65 (5.3)

[a] *SNK* $P<0.05$

combination of target size and ROM are presented in Table 1. All subjects in experiment 1 performed the smallest range of motion trials (30°) first during the pain condition, which explains the slightly higher VAS values obtained compared with 90° of range of motion trials. In experiment 2, the order of the set of trials (according to the target size) to be performed was randomized. In general, there was no significant difference in any of the outcome parameters (EMG, kinematics, etc.) between injections into m. biceps brachii or m. triceps brachii ($P>0.08$ for all analyses). Therefore, if not otherwise mentioned, all parameters will be presented as pooled data from both injections.

The effect of pain was not the same for the two tasks related to ROM or the two tasks related to movement accuracy. Thus the results are presented in terms of tasks in which they are related.

Flexion movements with small ROM and low accuracy

Experimentally induced muscle pain during 30° ROM flexion movements to a 12° target significantly ($F_{(2,18)}=3.7$, $P<0.05$) increased movement time compared to pre-pain condition (Table 2). It also evoked a significantly ($F_{(2,18)}>3.6$, $P<0.05$) increased reaction time and time to peak velocity when compared to pre- and post-pain conditions (Table 2).

IEMG for m. biceps brachii was significantly ($F_{(2,18)}=5.7$, $P<0.01$) decreased during muscle pain compared to pre- and post-pain conditions in epoch 2. At the same epoch the IEMG for m. triceps brachii was significantly ($F_{(2,18)}=2.4$, $P<0.04$) decreased during muscle pain compared to pre-pain condition (Fig. 2). Biceps brachii and triceps brachii muscles in the post-movement epoch showed a significantly ($F_{(2,18)}>4.6$, $P<0.02$) decreased IEMG during muscle pain compared to the

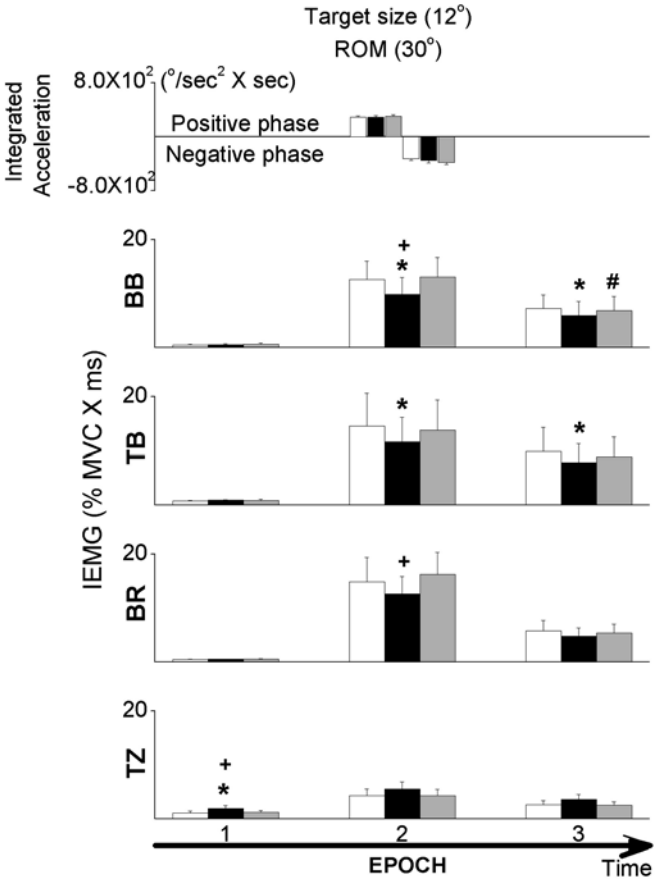

Fig. 2 Mean (+SEM) integrated acceleration profiles and integrated electromyogram (*IEMG*) from m. biceps brachialis (*BB*), m. triceps brachialis (lateral head; *TB*), m. brachioradialis (*BR*), and m. trapezius (*TZ*). Pooled data from both biceps brachii and triceps brachii muscle pain. Epoch 1: integrated 100 ms before the movement. Epoch 2: integrated EMG from the biceps muscle activity onset to the acceleration offset. The IEMG was normalized to the epoch duration. Epoch 3: integrated 100 ms after the movement. Pre-pain (*unfilled columns*), during-pain (*black columns*), and post-pain (*gray columns*). Flexion movements of a 30° range of motion (ROM) to a 12° target. Significant differences between during-pain and pre-pain (*), between during-pain and post-pain (+), and between post-pain and pre-pain conditions (#, SNK: $P<0.03$) are shown

pre-pain condition. The biceps brachii muscle also showed a significantly ($F_{(2,18)}$=4.6, P<0.02) decreased IEMG for post-pain compared to the pre-pain condition. However, the coactivation index was not significantly changed (Table 2). Thus, the post-movement muscle activation was significantly attenuated by muscle pain but the ratio between agonist and antagonist muscles was not. The synergistic brachioradialis muscle IEMG was significantly ($F_{(2,18)}$=4.3, P<0.03) decreased for the during-pain condition compared to post-pain condition. Trapezius IEMG was significantly ($F_{(2,18)}$=9.6, P<0.01) increased during muscle pain in epoch 1 compared to pre- and post-pain conditions. Integrated acceleration profiles were not significantly decreased by experimentally induced muscle pain (Fig. 2).

The initial part of the EMG burst (Q_{100}) of biceps brachii was significantly ($F_{(2,18)}$>10.04, P<0.001) decreased during muscle pain compared to pre-pain and post-pain conditions. Moreover, an interaction between muscle injected and condition showed that Q_{100} during pain condition was significantly ($F_{(2,18)}$>8.14, P<0.001) decreased compared to post-pain for both injected muscles

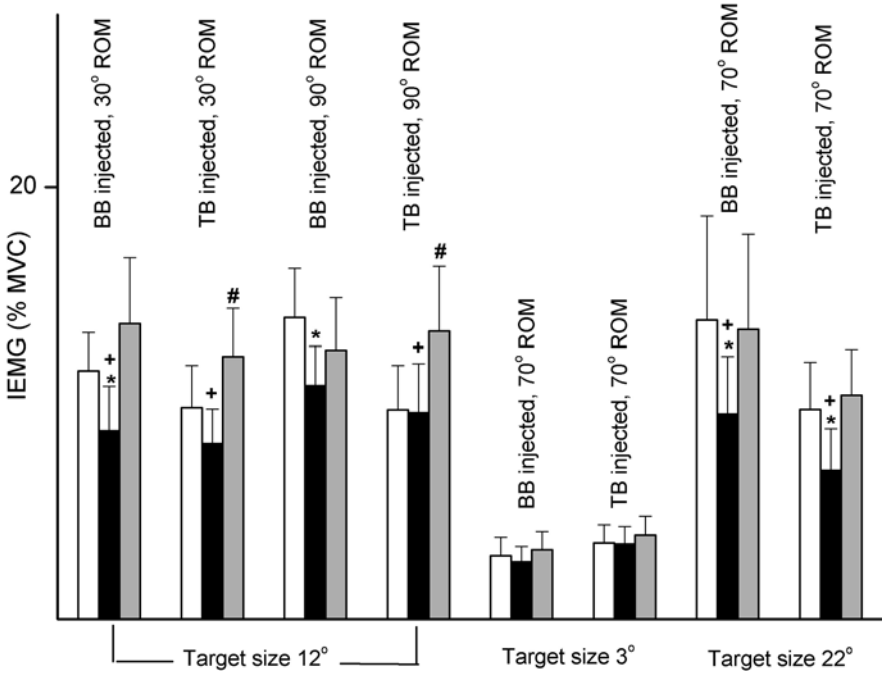

Fig. 3 The integral of the biceps brachii (*BB*) electromyogram (*EMG*) burst over 100 ms from the EMG onset (Q_{100}). The results are divided into two separate groups according to the muscle injected. Pre-pain (*unfilled columns*), during-pain (*black columns*), and post-pain (*gray columns*). Significant differences between muscle pain and pre-pain (*), between during-pain and post-pain conditions (+), and between pre- and post-pain (#, SNK: P<0.01) are shown

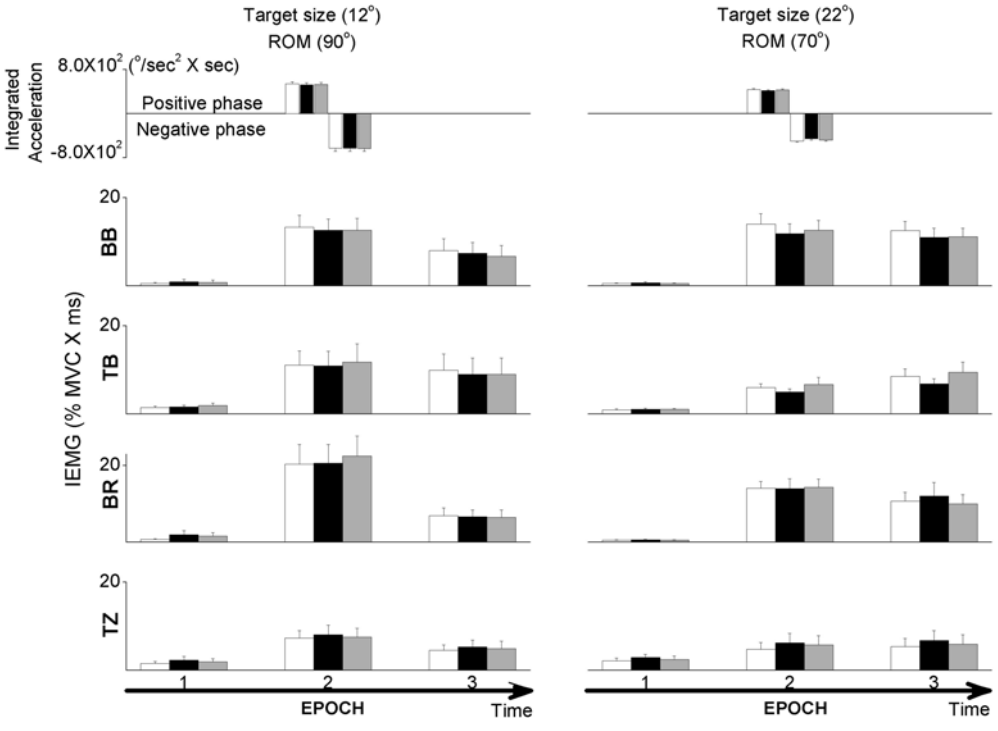

Fig. 4A, B Mean (+SEM) integrated acceleration profiles and integrated electromyogram (*IEMG*) from m. biceps brachialis (*BB*), m. triceps brachialis (lateral head) (*TB*), m. brachioradialis (*BR*), and m. trapezius (*TZ*). Pooled data from both biceps brachii and triceps brachii muscle pain. Epoch 1: integrated 100 ms before the movement. Epoch 2: integrated EMG from the biceps muscle activity onset to the acceleration offset. The IEMG was normalized to the epoch duration. Epoch 3: integrated 100 ms after the movement. Pre-pain (*unfilled columns*), during-pain (*black columns*), and post-pain (*gray columns*). Flexion movements of a 90° range of motion (ROM) to 12° (*left*) and 70° ROM to 22° (*right*) target

and significantly ($F_{(2,18)}$=11.50, P<0.04) decreased compared to pre-pain condition with pain in biceps brachii muscle (Fig. 3).

Flexion movements with large ROM and low accuracy

During flexion movements performed in a large ROM (90°) to a 12° target as well as 70° ROM to 22° target muscle pain there were no significant effects on the integrated acceleration profiles or on IEMG activity (Fig. 4).

Muscle pain evoked a significantly ($F_{(2,18)}$>4.6, P<0.02) decreased Q_{100} compared to pre-pain and post-pain conditions (Fig. 3). An interaction between injected muscle and condition showed that Q_{100} during post-pain condition was not significantly different from the pain condition with pain induced in biceps brachii muscle at 90° ROM. In addition, for the 90° ROM pain in the triceps brachii muscle did not show significant difference from the pre-pain condition (Fig. 3).

There was a significant increase in reaction time when subjects performed 70° ROM to a 22° target during muscle pain compared to pre- and post-pain conditions (Table 2). The time to peak velocity was significantly ($F_{(2,28)}$>3.76, P<0.04) increased for large ROM (90° and 70°; Table 2).

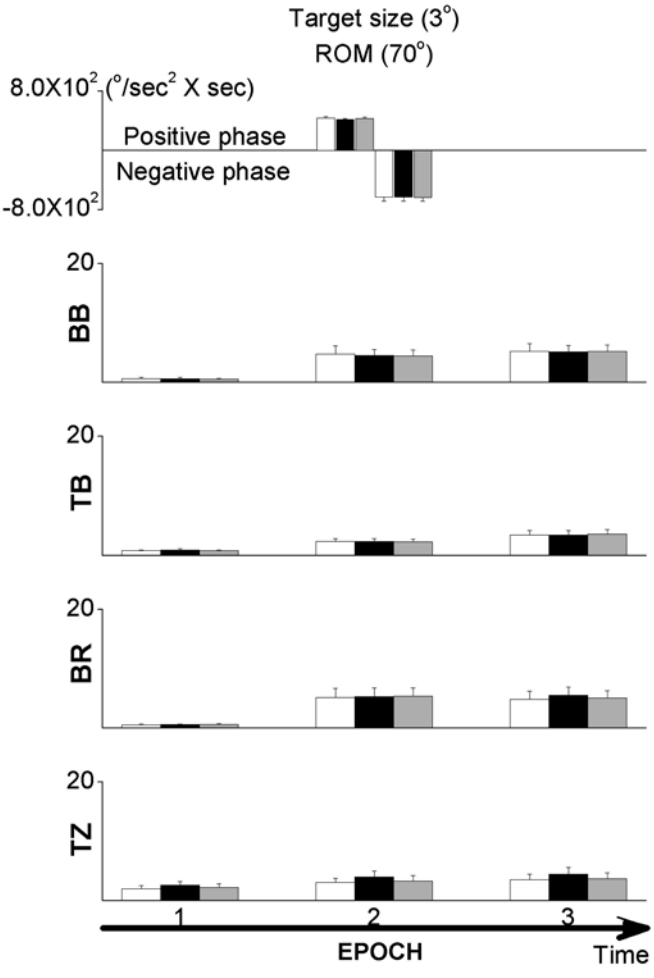

Fig. 5 Mean (+SEM) integrated acceleration profiles and integrated electromyogram (*IEMG*) from m. biceps brachialis (*BB*), m. triceps brachialis (*TB*), m. brachioradialis (*BR*), and m. trapezius (*TZ*). Pooled data from both biceps brachii and triceps brachii muscle pain. Epoch 1: integrated 100 ms before the movement. Epoch 2: integrated EMG from the biceps muscle activity onset to the acceleration offset. The IEMG was normalized to the epoch duration. Epoch 3: integrated 100 ms after the movement. Pre-pain (*unfilled columns*), during-pain (*black columns*), and post-pain (*gray columns*). Flexion movements of 70° range of motion to a 3° target

Flexion movements with large ROM and high accuracy

Experimentally induced muscle pain evoked no significant effects in integrated acceleration profiles or IEMG when 70° ROM was performed to a 3° target (Fig. 5). Similarly, there was no significant effect on Q_{100} due to muscle pain (Fig. 3). Reaction time and time to peak velocity were significantly ($F_{(2,28)}$>3.76, P<0.04) higher during muscle pain compared to pre- and post-pain conditions (Table 2).

Discussion

In this study, moderate pain in upper arm flexor/extensor muscles, during movements performed with a small (30°) range of motion, evoked changes in the reaction time, movement time and time to peak velocity as well as increased EMG activity of trapezius muscle in addition to decreased EMG activity of agonistic, antagonistic and distal synergistic muscles to elbow flexion. The overall EMG activity in m. biceps brachii, m. triceps brachii, m. brachioradialis, and m. trapezius during flexion movements performed with a large range of motion (70° and 90°) was generally not affected by muscle pain. However, the integral of the initial agonistic EMG burst (Q_{100}) was consistently reduced during flexion movements with a large as well as a small range of motion aiming at a wide target. Although the overall motor performance is only partly decreased by muscle pain, the initial motor control strategy is generally attenuated.

Hypertonic saline injection in biceps brachii or triceps brachii muscles showed the same effect. A general attenuation of the motor control mechanisms is possibly the most pronounced effect of muscle pain and can be seen as a protective mechanism. In the present study, this is seen as decreased initial agonistic EMG activity (Q_{100}) for pain induced both in the agonistic and antagonistic muscle.

Although the largest effect on EMG activity was observed in a condition with high pain scores (30° ROM), the pain intensities obtained during all the tasks were from low to moderate, which has been shown to evoke significant changes in motor control parameters (Birch et al. 2000; Matre et al. 1998).

Flexion movements with small ROM and low accuracy

During elbow flexion movements performed with 30° of ROM aiming at a 12° target-width, experimentally induced muscle pain had inhibitory effects in m. biceps brachii (agonist to elbow flexion) and in m. brachioradialis (synergistic to elbow flexion). Inhibition of agonistic muscles caused by muscle pain has been reported (Arendt-Nielsen et al. 1996; Graven-Nielsen et al. 1997; Backman et al. 1988; Birch et al. 2000) and some of these studies showed increased antagonistic muscle activity as well. The above-mentioned studies give support to the pain adaptation model by Lund et al. (1991) that proposes that pain reduces the activity of muscles when they act as agonists and increases the output when they act as antagonists. In the present study increased antagonistic activity by muscle pain was not a systematic finding. Nonetheless, if any change occurred in the antagonistic muscle, the muscle activity was decreased as for the agonistic muscle activity. This is in line with the pain adaptation model although only decreased agonistic activity was found. Interestingly, during muscle pain the overall trapezius muscle activity (IEMG) was significantly increased in the pre-movement epoch. Increased trapezius muscle activity is an important factor for development of musculoskeletal complaints in occupational settings (Veiersted et al. 1990; Larsson et al. 2000; Westgaard et al. 2001). The muscle activity in the pre-movement epoch (epoch 1) represents the period of time when muscle activity had to be just enough to keep the arm still at the initial position. Increased muscle activity in the pre-movement period might be a guarding response due to pain. Moreover, the increased trapezius muscle activity during the pre-movement epoch added to the decreased activity of m. biceps brachii and m. brachioradialis during the movement might produce a strategy to reduce limb movements, protecting the arm. Although the muscle activity inhibition was not large enough to modify the overall integrated acceleration profiles, the movement time, the time necessary to reach the peak velocity, as well as the reaction time were significantly increased, which are in line with the pain-adaptation model (Lund et al. 1991).

Madeleine et al. (1999b) reported increased reaction time on step initiation due to experimentally induced muscle pain, which together with kinematic and EMG parameters indicated changes related to the internal body representation used by the central nervous system and led to a reorganization of the strategy used during muscle pain. The initial EMG activity, defined in the present study as Q_{100}, was significantly decreased during muscle pain for 30° ROM aiming at a 12° wide target. According to the speed-strategy model proposed by Gottlieb et al. (1989), variation in the initial EMG activity shows that the central nervous system changed the excitation output in order to adapt to a new demand. As the external demands of the task (ROM and target width) were the same with and without muscle pain, central attenuation of the muscle control strategy is a possible consequence of muscle pain. Excitation of muscle nociceptors in animals significantly affects proprioceptive properties of jaw muscle spindles via central neural mechanisms (Ro and Capra 2001). In line with this, inhibition of maximal voluntary contraction force by injecting hypertonic saline in the muscle is centrally mediated in humans (Graven-Nielsen et al. 2002). Moreover, Le Pera et al. (2001) showed that tonic muscle pain induces inhibition of the motor system excitability at both a cortical and spinal level. Corcos et al. (1989) suggested that the initial EMG agonistic activity is a consequence of the initial excitation to the motoneuron pool, which is proportional to the speed that the subject wishes to perform the movement. Thus an inhibitory action on either the cortical or spinal cord level (or even in both) would potentially decrease the initial EMG agonistic activity (Q_{100}).

Flexion movements with large ROM and low accuracy

Fast movement with a large ROM aiming at a large target width leads to low precision tasks. Although not significant, muscle pain evoked a tendency for decreased IEMG and integrated acceleration profiles. There was also a significant increased time to peak velocity. This is in line with a study on tasks using a computer mouse, where Birch et al. (2000) showed that during low precision tasks muscle pain decreased the EMG activity. It is also in accordance with previous studies that showed decreased agonist muscle activity during muscle pain (Arendt-Nielsen et al. 1996; Graven-Nielsen et al. 1997; Zedka et al. 1999). Moreover, in the present study the initial EMG activity (Q_{100}) was decreased during muscle pain. The explanation for Q_{100} inhibition without a significant decrease in IEMG is not clear but it might be speculated that the subjects voluntarily compensate for the perturbation caused by the inhibition during the initial phase of the movement. Voluntary compensation for the pain effects might be dependent on pain intensity, the complexity level of the task, and muscle fatigue. Potentially, the initial EMG (strategy) activity might be attenuated by low to moderate pain intensity, whereas the overall IEMG might be attenuated at higher pain intensity.

Flexion movements with large ROM and high accuracy

During slow movements performed in large ROM (70° ROM to a 3° target-width) muscle pain did not change IEMG activity or acceleration profiles. This is comparable to other studies (Birch et al. 2000, 2001) in which muscle pain had a minor influence on performance during computer work and high precision tasks. It is also comparable with the study by Madeleine et al. (1999a), who showed that during controlled, low load, repetitive work, pain experimentally induced in trapezius muscle decreased EMG activity in that muscle whereas only the amplitude of the arm movement tended to increase during

muscle pain. Independently of the lack of changes in the overall IEMG and in Q_{100} during movement with large ROM, the time necessary to reach the peak velocity and the reaction time were significantly increased by pain.

The reaction time measures the time taken for mental events, such as stimulus processing, decision-making, and response programming (Matthews and Dorn 1989). Despite the target-width or the range of motion to be performed, the reaction time was enlarged by muscle pain in the present study. It seems that muscle pain enhances the reaction time in different situations as pointed out by Taimela and Kujala (1992), who showed that the reaction time for upper limb tasks is enlarged by pain in either the lower back or the lower extremity. Moreover, the prolonged reaction time found in the present study is comparable with the delay in the gait initiation showed by Madeleine et al. (1999b). This change together with the delay to reach the peak velocity might indicate a possible protective response.

In the present study, all high accurate tasks resulted in slow movements performed with low EMG activity, which was also the only task where Q_{100} was not affected by muscle pain. In a previous study decreased muscle activity caused by pain occurred in tasks where muscle activity was more than approximately 20% of the maximal voluntary contraction (Birch et al. 2000). Potentially the motor control strategy will be altered by muscle pain if the task demands more than a certain amount of muscle activation.

Coactivation

Coactivation after elbow flexion movements was not changed by experimentally induced muscle pain. In the only situation where post-movement muscle activity was affected, both the agonistic and the antagonistic muscle activity were proportionally decreased, resulting in an unchanged coactivation index. Although the subjects were asked to strongly contract the elbow joint muscles to fix the upper arm at the target after a 70° range of motion movement, the EMG intensity for the antagonist and also for the agonist muscle reached the maximal of 40% of the maximal voluntary contraction. Previous studies showed that muscle pain has inhibitory effects on isometric contractions when muscle activity reaches levels above 70% of the maximal voluntary contraction (Graven-Nielsen et al. 1997, 2002). Thus, the actual contraction level might explain the sporadic effects of muscle pain on post-movement muscle activity.

Conclusion

Experimental muscle pain: (1) modulated the motor control strategy, (2) attenuated the overall EMG activity in agonistic, antagonistic and synergistic muscles during flexions with a small range of motion, (3) did not affect the overall EMG burst activity during flexions performed with a large range of motion although decreased the initial agonistic EMG burst (Q_{100}) during flexions with a large range of motion aiming at a wide target, and (4) had no effects on post-movement coactivation of agonistic and antagonistic muscles.

The present study showed that acute muscle pain can perturb the motor control strategy. This might be highly important in occupational settings where such a change may need compensatory actions from other muscles and thereby eventually lead to development of musculoskeletal pain problems. Rehabilitation and pain prevention programs should take this observation into consideration.

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References

Arendt-Nielsen L, Graven-Nielsen T, Svanner H, Svensson P (1996) The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain 64:231–240

Ashton-Miller JA, McGlashen KM, Herzenberg JE, Stohler CS (1990) Cervical muscle myoelectric response to acute experimental sternocleidomastoid pain. Spine 15:1006–1012

Backman E, Bengtsson A, Bengtsson M, Lennmarken C, Henriksson KG (1988) Skeletal muscle function in primary fibromialgia: effect of regional sympathetic blockade with guanethidine. Acta Neurol Scand 77:187–191

Birch L, Graven-Nielsen T, Christensen H, Arendt-Nielsen L (2000) Experimental muscle pain modulates muscle activity and work performance differently during high and low precision use of a computer mouse. Eur J Appl Physiol 83:492–498

Birch, L, Arendt-Nielsen L, Graven-Nielsen T, Christensen H (2001) An investigation of how acute muscle pain modulates performance during computer work with digitizer and puck. Appl Ergon 32:281–286

Corcos DM, Gottlieb GL, Agarwal GC (1989) Organizing principles for single-joint movements II. A speed-sensitive strategy. J Neurophysiol 62:358–368

Gottlieb GL, Corcos DM, Agarwal GC (1989) Organizing principles for single-joint movements I. A speed-insensitive strategy. J Neurophysiol 62:342–247

Graven-Nielsen T, Svensson P, Arendt-Nielsen L (1997) Effects of experimental muscle pain on muscle activity and co-ordination during static and dynamic motor function. Electroencephalogr Clin Neurophysiol 105:156–164

Graven-Nielsen T, Lund H, Arendt-Nielsen L, Danneskiold-Samsøe B, Bliddal H (2002) Inhibition of maximal voluntary contraction force by experimental muscle pain: a centrally mediated mechanism. Muscle Nerve 26:708–712

Gribble PL, Ostry DJ (1998) Independent coactivation of shoulder and elbow muscles. Exp Brain Res 123:355–360

Hammond MC, Fitts SS, Kraft GH, Nutter PB, Trotte MJ, Robinson LM (1988) Co-contraction in the hemiparetic forearm: quantitative EMG evaluation. Arch Phys Med Rehabil 69:348–351

Larsson B, Bjork J, Elert J, Gerdle B (2000) Mechanical performance and electromyography during repeated maximal isokinetic shoulder forward flexions in female cleaners with and without myalgia of the trapezius muscle and in healthy controls. Eur J Appl Physiol 83:257–267

Le Pera D, Graven-Nielsen T, Valeriani M, Oliviero A, Di Lazzaro V, Tonali PA, Arendt-Nielsen L (2001) Inhibition of motor system excitability at cortical and spinal level by tonic muscle pain. Clin Neurophysiol 112:1633–1641

Lund JP, Donga R, Widner CG, Stohler CS (1991) The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 69:683–694

Madeleine P, Lundager B, Voigt M, Arendt-Nielsen L (1999a) Shoulder muscle co-ordination during chronic and acute experimental neck-shoulder pain: an occupational pain study. Eur J Appl Physiol 79:127–140

Madeleine P, Voigt M, Arendt-Nielsen L (1999b) Reorganization of human step initiation during acute experimental muscle pain. Gait Posture 10:240–247

Matre DA, Sinkjaer T, Svensson P, Arendt-Nielsen L (1998) Experimental muscle pain increases the human stretch reflex. Pain 75:331–339

Matthews G, Dorn L (1989) IQ and choice reaction time: an information processing analysis. Intelligence 13:299–317

Ro JY, Capra NF (2001) Modulation of jaw muscle spindle afferent activity following intramuscular injections with hypertonic saline. Pain 92:117–127

Suzuki N, Endo S (1983) A quantitative study of trunk muscle strength and fatigability in the low-back-pain syndrome. Spine 8:69–74

Svensson P, Graven-Nielsen T, Matre D, Arendt-Nielsen L (1998) Experimental muscle pain does not cause long-lasting increases in resting electromyographic activity. Muscle Nerve 21:1382–1389

Svensson P, McMillan AS, Graven-Nielsen T, Wang Kelun, Arendt-Nielsen L (1999) Modulation of an inhibitory reflex in single motor units in human masseter by tonic painful stimulation. Pain 83:441–446

Taimala S, Kujala UM (1992) Reaction times with reference to musculoskeletal complaints in adolescence. Percept Motor Skills 75:1075–1082

Veiersted KB, Westgaard Rh, Andersen P (1990) Pattern of muscle activity during stereotyped work and its relation to muscle pain. Int Arch Occup Environ Health 62:31–41

Wang K, Arendt-Nielsen L, Svensson P (2002) Capsaicin-induced muscle pain alters the excitability of the human jaw-stretch reflex. J Dent Res 81:650–654

Westgaard Rh, Vasseljen O, Holte KA (2001) Trapezius muscle activity as a risk indicator for shoulder and neck pain in female service workers with low biomechanical exposure. Ergonomics 44:339–353

Zedka M, Prochazka A, Knight B, Gillard D, Gauthier M (1999) Voluntary and reflex control of human back muscles during induced pain. J Physiol 520:591–604