Master's thesis

The effect of delayed onset muscle soreness in quadriceps on habitual thigh muscle activity

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BEV3901 Master's thesis in Human Movement Science

Trondheim, Spring 2014

Preface

I would like to thank my supervisor Paul Jarle Mork at the Human Movement Science Program at the Department of Neuroscience (NTNU) for valuable advices and feedback throughout this year. Despite his tight schedule at times, I have always been welcome in his office. Moreover, I would like to thank some of my fellow students for discussions and support throughout the year. Finally, I am very thankful to everyone who participated in my study.

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List of abbreviations

DOMS = Delayed onset muscle soreness

MVC = Maximal voluntary contraction

PPT = Pressure pain threshold

sEMG = Surface electromyography

VAS = Visual analogue scale

Abstract

Objective: The main purpose of this study was to investigate the effect of acute quadriceps pain, caused by delayed onset muscle soreness (DOMS), on long-term habitual muscle activity in knee extensors and flexors. A second purpose was to investigate the effect of DOMS on controlled muscle activation in the laboratory, and to compare this with the habitual recordings.
Methods: Eighteen healthy subjects (10 females and 8 males, mean age 23 years, range 20-31) participated in the study. Surface electromyography (sEMG) was recorded bilaterally from vastus lateralis, rectus femoris, vastus medialis and biceps femoris on two weekdays with one day of rest between recordings. The laboratory tests (standing, walking in stairs, standing up from a seated posture, isometric contraction, and maximal voluntary contraction [MVC]) were performed before the long-term field recordings. Body posture (sitting, standing, and walking) was recorded with an accelerometer, and heart rate was recorded with electrocardiography (ECG). Immediately after the first long-term recording, the subjects performed an eccentric exercise (Barbell lunges) with use of the dominant leg only. Pressure pain threshold (PPT), pain scored on visual analogue scale (VAS) and maximum force during a maximal isometric contraction were used to assess symptoms of DOMS.
Results: There were no differences in time spent in different body postures or heart rate between the first and second long-term recording. DOMS was indicated by a significant reduction of PPT in the quadriceps muscle, a significant raised VAS-score, and a significant reduction in maximum knee extension force in the exercised leg. Habitual sEMG activity (median sEMG level, μV) of the antagonist in the exercised thigh increased from first to second long-term recording during seated posture, while sEMG activity in the exercised vastus medialis decreased from first to second long-term recording during periods with standing posture ($P<.05$ for both comparisons). Thigh sEMG activity remained unchanged for the untrained leg in all postures during the long-term recordings. During the laboratory tests, sEMG activity of the antagonist of the painful thigh increased during walking in stairs ($P=.003$), but remained unchanged in other controlled contractions. When comparing standing posture in the laboratory with standing posture during the long-term field recording, the same tendency was observed, i.e., decreased agonist and increased antagonist activity in the exercised leg.
Conclusion: The present findings indicate that DOMS has no or only moderate effect on muscle activity, and the results indicates that the responses to muscle pain is not so stereotypical as suggested by the pain adaption model. Thus, the current findings support the notion that pain models should include a task dependency aspect.

Key words: Delayed onset muscle soreness, electromyography, vastus lateralis, rectus femoris, vastus medialis, biceps femoris, posture, habitual activity patterns, vicious cycle theory, pain adaption model

Introduction

Because of the high prevalence and the socioeconomic burden of musculoskeletal pain upon society, the mechanisms behind chronic musculoskeletal pain have received much interest among researchers for several decades. Musculoskeletal pain is a major clinical problem and often insufficiently treated [1], and the strategies for treatment and prevention of musculoskeletal pain syndromes are still not optimal.[2] The mechanisms involved in muscle pain are often difficult to resolve from clinical studies because of the high variability between patients.[1] Human experimental pain models applied to healthy volunteers are a potential strategy to investigate aspects of the mechanisms involved in muscle pain, and makes it possible to investigate the temporal association between muscle activity and pain.[1] Several theories have been formulated to explain the effect of pain on muscle activity; however, the mechanisms that underlie the motor adaptions to pain are not adequately understood. Two major and commonly cited theories proposed to explain the effect of pain on muscle activity are the *vicious cycle theory* [3] and the *pain adaption model*.[4]

The vicious cycle theory suggest that an initiating factor like sustained stress or awkward posture induce "muscle hyperactivity" that leads to muscle pain which, in turn, leads to muscle fatigue, muscle spasm, and thereby further pain.[5] Although some studies have found increased muscle activity[6, 7] and muscle spindle discharge during pain,[8] this is not uniform[9] and many studies show no change in muscle activity.[9-11] The vicious cycle theory is not consistent with the majority of observations.[4, 12-14] Studies have shown that if surface electromyographic (sEMG) activity increases, it does not last the duration of the painful stimulus.[9] In addition, no muscle hyperactivity was found during rest and static contractions.[10]

In contrast to the vicious cycle theory, the pain-adaption model predicts that acute or chronic muscle pain will cause inhibition of agonistic muscle activity and increased antagonist activity. Similarities may exist between acute and chronic muscle pain, but they are usually considered as two different entities, where acute pain may serve as a warning of disease or a threat to the body. Chronic pain is usually considered as pain that outlast the time that healing would have occurred after trauma or injury. The pain adaption model suggest further that changes in muscle activation pattern make movements slower and reduce movement amplitude, thereby reducing the risk for further injury. At the same time, reduced movement is assumed to promote muscle repair.[4] The pain adaption model has gained support from studies investigating the effect of experimental muscle pain on muscle activity in different muscles and during various conditions. For instance, experimentally induced muscle

pain is shown to reduce sEMG activity during isometric voluntary contractions,[15] decrease maximal force,[16] and reduce time to exhaustion during sustained contractions.[10] Several studies have demonstrated a decreased sEMG activity of the painful agonistic muscle.[12-14, 17-23] Although this is consistent with the pain adaption model, only some of these studies did investigate the antagonist activity.[14, 18, 20-23]

During a concentric abduction and a dynamic external rotation of the shoulder, pain in the supraspinatus or into the subacromially space was associated with increased EMG activity in the antagonist latissimus dorsi, while decreased activity in the agonist was only reported during the concentric abduction.[14] During low precision work with a computer mouse, the painful extensor carpi ulnaris showed lower sEMG activity in specific phases of the work cycle, while the antagonist (flexor carpi radialis) remained unchanged.[21] During horizontal elbow flexion movements, mild and moderate pain in biceps brachii was associated with decreased sEMG activity in agonists, synergists, as well as antagonists.[20] During sustained submaximal elbow flexion movements, the same authors reported that pain in biceps brachii or triceps brachii was associated with increased sEMG activity in trapezius and a decreased sEMG activity in agonists, antagonist as well as synergistic muscles.[22] These results indicate that acute upper arm pain modulates coordination of the shoulder muscles during voluntary movements. During walking, pain in either gastrocnemius or tibialis anterior resulted in a combination of decreased activity in the muscle agonistic to the painful muscle, and an increased antagonist activity.[10] However, this altered co-ordination between the agonist and antagonist was not consistent, and depended on the position of the legs during the gait cycle. Furthermore, during walking, both reduced agonist and synergist activity were found in lower limb muscles, while the antagonists were unaffected.[18] Reduced muscle activity was also observed in the pain-afflicted sternocleidomastoid and splenius capitis when they were acting as an agonist, whereas modulation of antagonist activity was task dependent.[23]

Because of the high prevalence of pain in the shoulder and neck area, trapezius has been frequently investigated, but with conflicting results. Induced pain in descending parts of trapezius resulted in increased sEMG activity of the transverse and ascending parts of trapezius during computer work,[24] which can be linked to the motor system ability to recruit synergistic muscles in presence of pain, but do not explain how antagonists contribute. In contrast, long-term sEMG recordings of acute trapezius pain induced by delayed onset muscle soreness (DOMS) showed increased habitual trapezius activity of the clavicular and descending part of the painful trapezius during periods with seated postures, while trapezius sEMG activity remained unchanged for all other trapezius parts and postures.[6] The authors

concluded that acute muscle pain induces elevated low-level muscle activity. This result may relate to a neural rather than muscular mechanism by increased motor unit firing rate and synchronization, which raise further questions about the pain adaption model.

In particular, intramuscular injections of hypertonic saline has been a widely used experimental muscle pain because the induced pain mimics chronic muscle pain [25], and causes well defined local and referred pain.[26] However, injection of hypertonic saline makes it possible to maintain muscle pain for only 15-20 minutes, [27] and is therefore only suitable to use in laboratory settings.

When intending to study the effect of experimental pain on patterns of habitual muscle activity, a method that offers a long-lasting pain response is required. A widely used endogenous technique is induction of DOMS by eccentric muscle exercise. DOMS is characterized by muscle hyperalgesia, thereby mimicking one of the central features of chronic muscle pain. DOMS sets in several hours after the eccentric exercise, peaks at about 48 hours and last for 1-4 days.[28] A common feature is that the intensity of pain increases during muscle loading or movement.[29] It is widely accepted that muscle pain as a symptom of DOMS is associated with a decreased ability to generate force.[28] This may result from pathophysiological changes in the muscle fibers[12] such as an inflammatory reaction caused by disrupted sarcomeres in myofibrils and damage to the excitation-contraction coupling system[30], or by factors associated with fiber damage such as myoplasmic Ca^{2+} release[30], and insufficient energy supply[31]. In the quadriceps muscle, the pressure pain threshold (PPT) is suggested to reach it lowest about 24-48 hours after an eccentric exercise intervention, and pain receptors sensitive to local pressure (group III nociceptors and mechanoreceptors served by group III axons) are more affected in distal muscle regions after damage induced by eccentric exercise.[12]

A large number of studies have investigated the relation between muscle activity and muscle pain in controlled laboratory studies, but surprisingly few studies have investigated the effect of pain on unconstrained habitual muscle activity patterns. Use of long-term sEMG recordings makes it possible to study changes in habitual muscle activity patterns when exposed to DOMS.[32] The main purpose of this study was to investigate the effect of DOMS in the quadriceps muscle on muscle activity in thigh muscles during unconstrained daily activities. Quadriceps and biceps femoris sEMG activity was recorded on two days, before and after the presence of DOMS. A second purpose was to investigate the effect of DOMS on thigh muscle activity during a controlled laboratory setting and to compare this with the unconstrained habitual muscle activation.

Methods and materials

Subjects

Ten females and eight males (age, mean ± SD 22.9 ± 3.0, range 20-31 years) volunteered to participate in the study. Body mass ranged from 53.7 to 100 kg (72.8 ± 14.2), body mass index (BMI) ranged from 18.7 to 33.0 (23.1 ± 3.5). Fifteen subjects had a dominant right foot, and three subjects had a dominant left foot. The dominant foot was determined on the basis of which foot they preferred to use in various tasks (kicking a ball, jumping, hop on one leg). All subjects were without back/hip/knee pain the last year or diseases that prevent physical activity. None of the subjects were pregnant. The study protocol was approved by the Regional Committee for Ethics in Medical Research and all subjects signed an informed consent before inclusion. The study was carried out according to the Declaration of Helsinki.

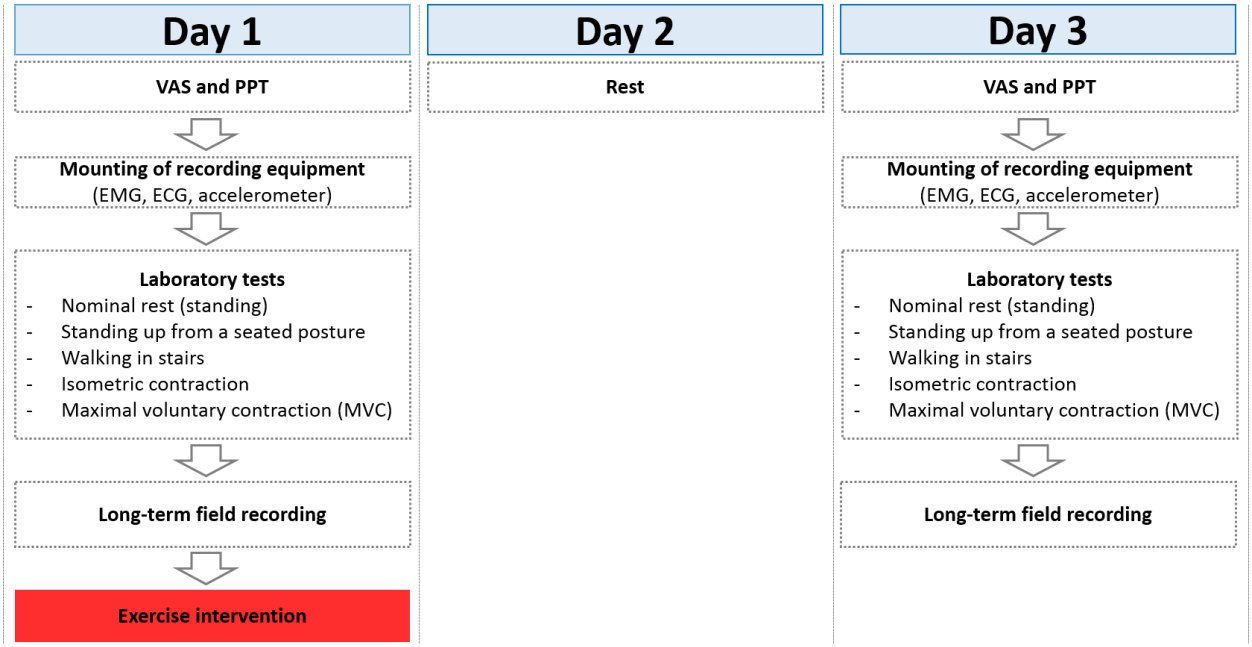

Figure 1. Schematic presentation of the study design. The procedure was identical on day 1 and day 3 except the eccentric muscle exercise at the end of day 1 to induce DOMS.

Experimental protocol

The study used an experimental within subject design in which each subject was tested on two different days (figure 1). To prevent recovery in the dominant exercised leg and to prevent DOMS in the non-dominant leg, the participants were recommended to not perform any physical activity between the two days of testing. First, measurements of body composition, subjective measurement of pain and PPT were performed. PPT was measured in the exactly

same site as the sEMG electrodes were applied. The position of the sEMG electrodes and accelerometer was marked by a waterproof pen to ensure similar recordings on both days. The laboratory tests consisted of nominal rest (quiet standing 60 seconds with the body weight distributed on both legs), standing up from a chair without use of arms, walking in stairs (40-60 seconds), isometric contraction (seated with 5 kg load hanging around the ankle with fixed leg), and MVC (isometric knee flexion and knee extension). The long-term field recording lasted for 2-5 hours. At the end of day 1 only, an exercise intervention was performed to induce DOMS in quadriceps in the dominant foot. During the long-term recording, the subjects were instructed to follow their daily routines and activities, but to avoid strenuous physical activity and lying down. The latter restriction was used to avoid misclassification of sitting and lying down. Beyond this, there were no restrictions concerning types of activities. After completion of the long-term recordings, the subjects met in the laboratory to remove the equipment.

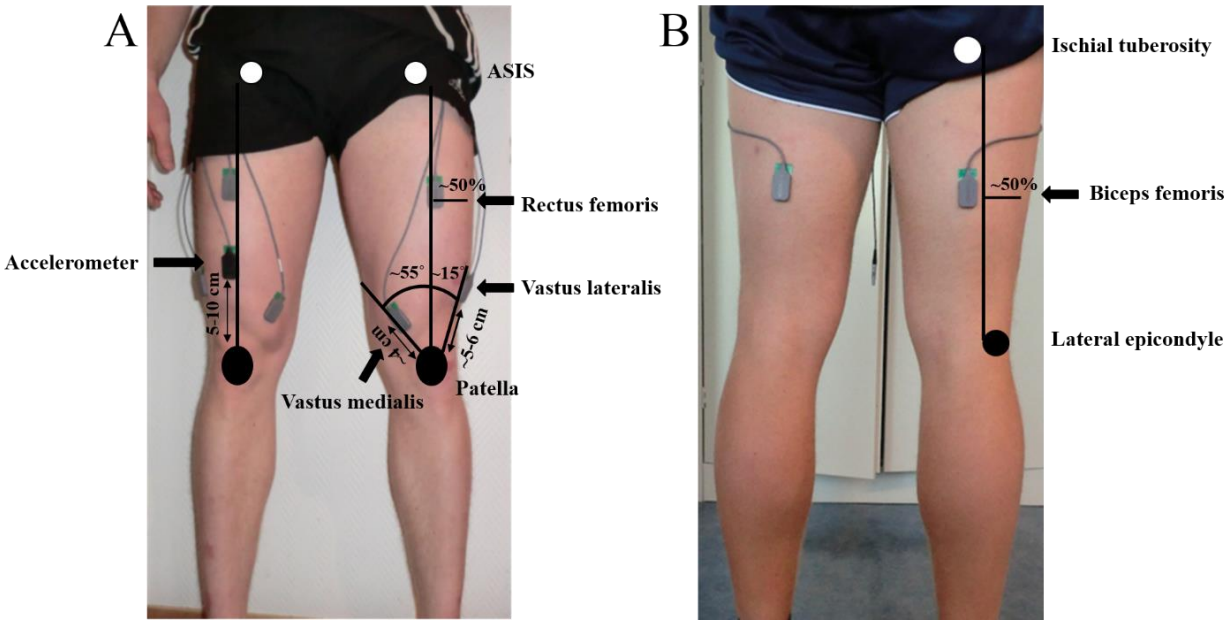

Figure 2. Illustration of the placement of (A) accelerometer, six quadriceps electrodes, and (B) two biceps femoris electrodes.

Physiological recordings

During the laboratory tests and long-term recordings, a portable Myomonitor IV EMG system (Delsys, Boston, MA) was used to record sEMG activity, accelerometer data from the thigh, heart rate, and knee extension and flexion force recorded by a force cell. All data was sampled at 1000Hz, and stored on a memory card for off-line analysis. The data logger was carried in a fanny pack during the field recording.

Bipolar sEMG was recorded bilaterally from vastus lateralis, rectus femoris, vastus medialis and biceps femoris using parallel bar sensors (Delsys inc., Boston, USA) with one electrode on each of the anatomical divisions of the muscle, and an inter-electrode distance of 10mm (bar length 10 mm, bar diameter 1 mm). Before mounting of the electrodes, the skin was shaved and washed with isopropanol. Adhesive skin interfaces (Delsys, Boston, MA) were attached to the electrodes, and the contact poles were greased with electrode gel (highly conductive multi-purpose electrolyte, Signa gel, Parker), and remained on throughout both laboratory and long-term field recording. The same person executed the electrode placement on all subjects.

The electrode for vastus lateralis was placed over the muscle belly ~5-6 cm above the superolateral corner of patella, the electrode for rectus femoris was placed at ~50% on the line from the anterior spina iliaca superior (ASIS) to the superior part of the patella, the electrode for vastus medialis was placed over the muscle belly ~4 cm above the superomedial corner of patella, and the electrode for biceps femoris was placed at the muscle belly ~50% on the line between the ischial tuberosity and lateral epicondyle of the tibia (Figure 2).

All electrodes were adjusted to the presumed muscle fiber direction. The reference electrode (Dermatrode reference electrode) was placed on the processus spinosus at C7.

Information of time in different postures were recorded by a 2D accelerometer (Delsys Inc., Boston, MA) The accelerometer was positioned so that one axis recorded thigh angle in the sagittal plane, thereby makes it possible to distinguish between sitting, standing and walking. The accelerometer was attached on the distal part of rectus femoris, just proximal to the knee joint (5-10 cm over patella).

Heart rate was recorded by electrocardiography (EKG Biosignal Sensor, range 5mV, resolution 4uV, bandwidth 0.5-30 Hz, Noise baseline 1.0 uV, Delsys, Boston, MA). The EKG sensor was placed at ~50% of the length of sternum, and to the left of the left breast, ~6^{th} rib.

Knee extension and flexion force was recorded during isometric MVC, and were carried out bilaterally for both quadriceps and biceps femoris. The MVCs were repeated three times, with 45 seconds break between each repetition. The contraction was held for ~3

seconds. The mean of the two most equal $sEMG_{max}$ values was used for normalization. During the execution of knee extension, the subjects were seated in an erect posture, knees 90 degrees flexed, holding both arms on handles laterally to the thighs. During the execution of knee flexion, the subjects were lying with the abdominal side towards the floor, knees 90 degrees flexed, and with the non-performing leg supportive towards an edge to prevent slippage during the execution. During both extension and flexion, a solid band was placed proximal to the ankle joint to provide resistance. A force transducer (Interface, MFG. in Scottsdale, Arizona, USA, 2000 N) were connected to the band in order to record force. The maximal values of sEMG were stored synchronously with the MVC. The force signal was digitally low pass filtered (Butterworth, 10Hz, 6^{th} order) and down sampled to 0.1 s time resolution before further analysis.

Exercise intervention

The exercise intervention consisted of 6 sets of 10 barbell lunges with 30 second rest between each set. External resistance (barbell on the subjects' shoulders) was 40% of the total body weight for women and 50% for men. The external resistance was based on previous studies[33] and results from pilot testing. Because of individual differences in strength, this was taken into consideration: if to light weight could be indicated visually by the instructor, or verbal by the subject, more weight was put on. If the subject could not finish all 6 sets with the pre-defined weight, the weight was slightly reduced until the subject was able to complete the described sets of exercise. Subjects were instructed how to execute the exercise. Because this study was dependent of pain in quadriceps in the dominant foot only, the execution was performed with a small step with the pressure on forefoot, because this isolates the quadriceps. In detail, subjects were told to start in an upright standing position with a straight body with the bar behind the neck resting on the back of the shoulders, and with the legs slightly apart (figure 3, A). The subjects took one step forward on their dominant foot while they kept the trunk as straight as possible, lunge until the front thigh was horizontal to the floor, just before the non-dominant knee touched the floor (figure 3, B), and returned to the initial position thereafter. The subjects were instructed to get back to an upright position by using only their dominant leg. After some trials without external resistance, the starting position and landing position with their dominant leg was marked with a tape to standardize the exercise. All subjects were allowed to practice a few repetitions before starting the exercise. To secure the best execution of the exercise, the subjects performed the lunges at their preferred speed, but markedly to slow or fast tempo was commented.

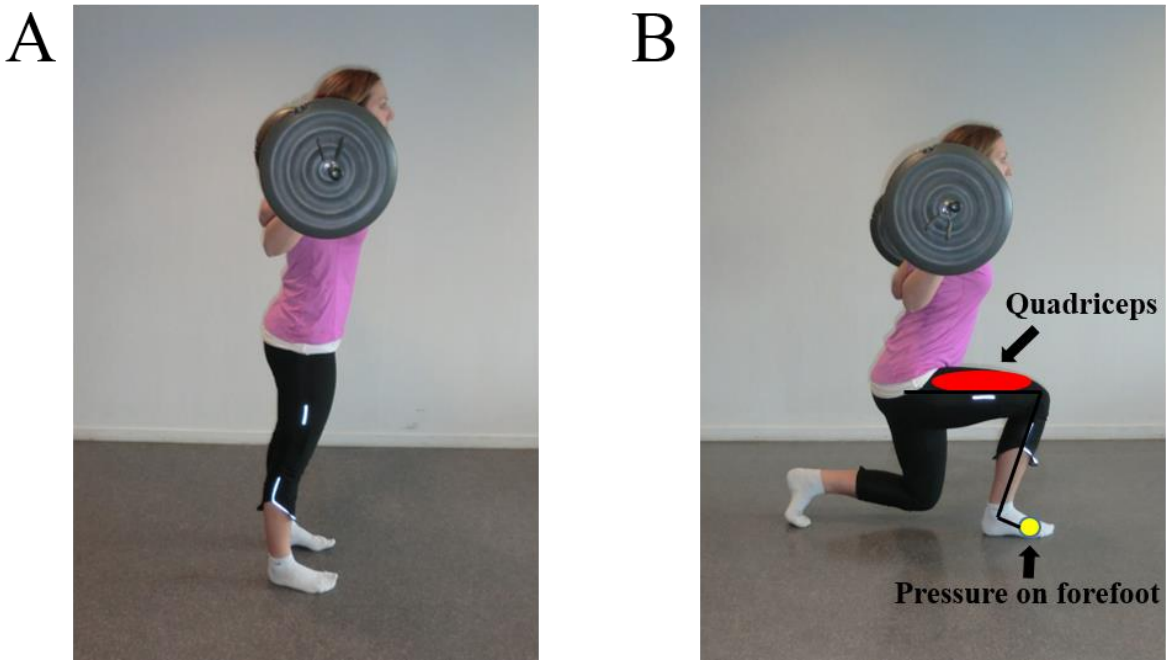

Figure 3. Barbell lunges

Pressure pain threshold

PPT was included in the study as a method to measure the effect of the exercise intervention. PPT was measured on both days, before the equipment was mounted. PPT was measured by a hand-held pressure algometer (Somedic Algometer type II, Sweden) with a probe diameter of 10 mm. A steady increase of 40 kPa/s was maintained during the execution of PPT. The same person performed all measurements. During PPT measurements on quadriceps, the subject sat in an erect seated posture, with arms resting in the lap without preventing the recordings. During PPT measurement on hamstrings, the subject was lying with the abdominal side towards the floor, fixated legs, with arms in a comfort position. The subjects were instructed to respond verbally when the applied pressure changed from pressure to pain. All PPT measurements on quadriceps started on the non-dominant side, and were performed in this order: vastus lateralis, rectus femoris and vastus medialis on non-dominant thigh first, vastus lateralis, rectus femoris and vastus medialis on dominant thigh second. PPT measurements on biceps femoris was carried out bilaterally, i.e., left biceps femoris first, right biceps femoris second, etc.

Subjective variables

Pain on the front of the thigh was scored on a subjectively 10 cm visual analogue scale (VAS) with the end points very low and very high. VAS scale was scored on each thigh independently of each other and on both days before the equipment was mounted.

sEMG analysis

The sEMG signal was digitally band-pass filtered (10-450 Hz, Butterworth, 6^{th} order), root mean-squared with a window width of 0.1 s with no overlap, and stored with a time resolution of 0.1 sec. The noise level was first made equal to the minimum level of sEMG activity during the long-term field recordings. When quantifying the sEMG responses, the sEMG noise level was subtracted from the RMS detected signal. sEMG activity during periods with sitting, standing and walking was quantified by the median and peak sEMG level, and the coactivation ratio was quantified by the median sEMG levels (defined as the 50^{th} and 90^{th} percentile of the cumulative distribution curve[34], i.e., the sEMG level that the sEMG activity is above for 50% and 10% of the recording time). sEMG is presented as µV In the main analysis. In supplementary analysis, the sEMG responses during the long-term recordings were normalized relative to the maximal sEMG response ($sEMG_{max}$) obtained during the isometric MVCs.

Long-term sEMG recordings failed for two subjects on exercised vastus medialis, and one subject for the rectus femoris on the exercised foot. During the laboratory tests, sEMG recordings failed for three subjects on exercised vastus medialis, and for three subjects during the force measurements. The cause of failure was defect electrodes and involuntary removal of the electrodes. The rest of the recorded channels were retained for further analysis. sEMG activity from different postures (sitting, standing, walking) was extracted from the long-term recordings.

Posture analysis

To obtain information of periods of sitting, standing and walking, an accelerometer (2-Axis Accelerometer, range ±2 g, resolution 0.037 g, bandwidth 0-2000 Hz) recorded the thigh angle simultaneous with sEMG. Figure 4 shows a visual inspection of an amplitude-time display of the thigh accelerometer- and sEMG recordings, which was done using AcqWin (Jacobus systems, UK). Posture recordings failed halfway for one subject. The cause of failure was technical problems of the accelerometer during the long-term recording (the first part of the recording were retained in the data material).

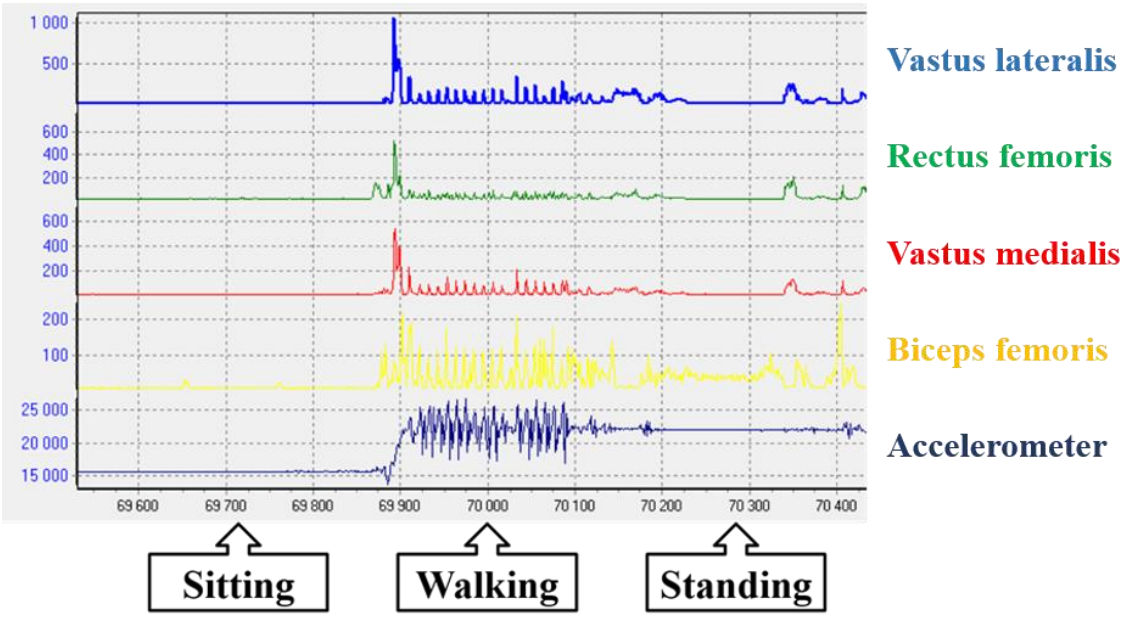

Figure 4. Illustration of the visual display during manual analysis (right leg)

Statistical analysis

The software package IBM SPSS Statistics for windows (version 21) was used for statistical analyses. In all tables, values are presented as mean ± SD. The Shapiro-Wilk W-test was used on all variables to assess normality. A paired samples t-test was used to test differences from first to second recording for normally distributed data (Maximal force, PPT and time in different postures). The Wilcoxon signed-rank test was used to test differences for data with non-normal distribution (all sEMG variables, and VAS scores). The Mann Whitney u-test was used to test differences for data between legs (VAS scores). Effect size estimates were calculated by converting t-values from the normally distributed data, and z-scores from the non-normally distributed data into Pearson's correlation coefficient (*r)*. A coefficient <.30 indicates a small effect, .30-.50 medium effect, and >.50 large effect [35]. A probability level of P<.05 was considered significant in all cases.

Results

Body posture and heart rate

The relative time (% of total recording time) spent in different postures and heart rate during the first and second long-term recording is presented in table 1. There was no difference in the time spent in different postures during first recording and second recording. About 75% of the time was spent in seated posture on both days, ~ 4% in standing posture and ~ 8% walking. About 6-7% of the total recording time could not be classified into any of the postures described above. Moreover, there was no difference in heart rate from first to second recording.

Table 1. Time (% of total recording time) with different postures and heart rate during the first and second long-term recording. Values are mean ± SD.

	1st recording	2nd recording	*P*[a]
Body posture			
Sit	76 ± 13	75 ± 18	.86
Stand	3.7 ± 2.3	4.0 ± 2.7	.75
Walk	7.8 ± 5.9	8.4 ± 7.8	.70
Standing/walking	5.0 ± 5.8	6.7 ± 9.9	.53
Unclassified	7.1 ± 4.4	6.0 ± 3.0	.40
Heart rate			
Sit	84 ± 9.2	83 ± 11	.47
Stand	87 ± 15	88 ± 12	.90
Walk	92 ± 10	93 ± 12	.54

P[a] = Paired sample t-test

Pressure pain threshold and subjective pain scores

Table 2 presents PPT (kPa) for quadriceps and biceps femoris at the first and second recording. PPT was reduced for all parts of the exercised thigh ($P<.02$, effect size $\geq.13$ for all comparisons), and reduced for all parts of the non-exercised thigh ($P<.02$, effect size $\geq.21$ for all comparisons). The average reduction in PPT was more pronounced in the exercised thigh (vastus medialis -37%, rectus femoris -26%, vastus lateralis, -27%) compared to the non-exercised thigh (vastus medialis -9%, rectus femoris -9%, vastus lateralis -14%). In addition, the effects sizes clearly indicate that the reduction in PPT was substantially larger in the exercised thigh.

Table 2. Pressure pain threshold (kPa) on first recording and second recording for different parts of exercised and non-exercised thigh. Values are mean ± SD.

	1st recording	2nd recording	*P*[a]	*r*[b]
Exercised side				
Vastus lateralis	525 ± 217	381 ± 180	<.001	.50
Rectus femoris	612 ± 233	443 ± 172	<.001	.54
Vastus medialis	470 ± 165	297 ± 119	<.001	.98
Biceps femoris	607 ± 326	565 ± 332	<.02	.13
Non-exercised side				
Vastus lateralis	476 ± 162	416 ± 173	<.006	.36
Rectus femoris	601 ± 199	554 ± 222	<.003	.22
Vastus medialis	478 ± 162	441 ± 171	<.02	.22
Biceps femoris	548 ± 241	500 ± 208	<.02	.21

P[a] = Paired samples t-test
r[b]= Effect size

The VAS score on first recording and second recording is presented in table 3. There was a significant increase in VAS score on the front side of the exercised thigh (0.2 ± 0.5 vs. 3.9 ± 2.4, P<.001, r=.62) and on the front side of the non-exercised thigh (0.1 ± 0.4 vs 0.6 ± 0.6, P=.008, r=.44). There was no difference between the exercised thigh and the non-exercised thigh in the first recording (P=.63). In the second recording, there was a significant difference between the exercised thigh and the non-exercised thigh (P<.001). Moreover, the effect size indicate a more severe increase in pain in the exercised quadriceps (r=.62) compared to the non-exercised quadriceps (r=.44).

Table 3. Subjective variables recorded for quadriceps by VAS on first recording and second recording. Values are mean±SD.

	1st recording	2nd recording	Δ (pre-post)	P[a]	r[b]
Exercised side	0.2 ± 0.5	3.9 ± 2.4	3.7 ± 2.4	<.001	.62
Non-exercised side	0.1 ± 0.4	0.6 ± 0.6	0.5 ± 0.7	.008	.44
p-value*	.63	<.001	<.001		

P[a] = Wilcoxon signed rank test (within group)
* = Mann Whitney u-test (between legs)
r[b] = Effect size

Force and sEMGmax

Table 4 shows knee flexion and extension force during MVC, and table 5 shows $sEMG_{max}$ during the MVC. Knee extension force was significantly reduced from first recording to second recording in the exercised thigh (P<.001, r=.50) while knee flexion force tended to increase (P=.09, r=.17). There was no change in knee extension or flexion force in the non-exercised side (P≥ .54, r≤.05). $sEMG_{max}$ responses during MVCs tended to increase from first

recording to second recording for the exercised vastus lateralis (P=.07, r=.34), while $sEMG_{max}$ responses for the other thigh muscles remained unchanged from first to second recording.

Table 4. Knee flexion and knee extension force (N) during isometric voluntary contractions on first recording and second recording. Values are mean±SD.

Force	1st recording	2nd recording	P[a]	r[b]
Exercised side				
Flexion	196 ± 65	209 ± 78	.09	.17
Extension	507 ± 125	439 ± 146	<.001	.50
Non-exercised side				
Flexion	195 ± 59	198 ± 59	.54	.05
Extension	494 ± 115	494 ± 131	1.00	.00

P[a] = Paired samples t-test
r[b]= Effect size

Table 5. $sEMG_{max}$ during isometric voluntary contractions on first recording and second recording. Values are mean±SD.

$sEMG_{max}$	1st recording	2nd recording	P[a]	r[b]
Exercised side				
Vastus lateralis	865 ± 355	972 ± 344	.07	.34
Rectus femoris	772 ± 363	832 ± 384	.12	.29
Vastus medialis	1162 ± 594	1202 ± 612	.72	.08
Biceps femoris	915 ± 255	999 ± 355	.17	.24
Non-exercised side				
Vastus lateralis	819 ± 317	898 ± 382	.33	.18
Rectus femoris	783 ± 301	760 ± 308	.72	.06
Vastus medialis	914 ± 326	935 ± 382	.89	.03
Biceps femoris	1028 ± 490	1066 ± 410	.78	.05

P[a] = Wilcoxon signed rank test
r[b] = Effect size

Habitual muscle activity

Table 6 presents median trapezius sEMG level (µV) during periods with sitting, standing and walking posture during the first and second long-term recording. The median sEMG level increased for the biceps femoris in the exercised thigh from first to second recording during sitting (P< .01, r= .47) and decreased in the exercised vastus lateralis during standing (P<.03, r=.39). Moreover, sEMG tended to decrease during standing in the vastus lateralis (P=.08, r=.32), and biceps femoris in the exercised thigh (P=.06, r=.36). Median sEMG level remained unchanged during walking, but tended to increase in the biceps femoris in the exercised thigh (P=.08, r=.30).

For the peak sEMG level (data not shown) there was a significant increase during sitting in the biceps femoris on the exercised side (81 ± 32 vs. 122 ± 68, P= .02, r= .39). A significant decrease was observed during standing for the vastus lateralis (105±86 vs. 56±30,

P=.03, r=.39), rectus femoris (72±66 vs. 44±29, P=.002, r=.55) and vastus medialis (110±81 vs. 24±16, P= .02, r= .46) on the exercised side.

Overall, similar results were also observed when using normalized sEMG data (i.e., % EMG max) in the statistical analysis (data not shown).

Table 6. Median sEMG level (µV) during periods with sitting, standing, and walking during first and second long-term recording. Valus are mean ± SD.

	1st recording	2nd recording	P[a]	r[b]
Sitting				
Exercised side				
Vastus lateralis	18.3 ± 8.9	13.3 ± 2.1	.12	.30
Rectus femoris	11.8 ± 3.1	11.3 ± 2.1	.58	.11
Vastus medialis	15.7 ± 8.1	12.7 ± 4.2	.22	.22
Biceps femoris	15.5 ± 4.3	22.6 ± 8.8	<.01	.47
Non-exercised side				
Vastus lateralis	18.2 ± 10.1	18.9 ± 10.2	.28	.19
Rectus femoris	13.7 ± 9.4	13.6 ± 6.7	.81	.04
Vastus medialis	13.1 ± 4.9	12.2 ± 5.2	.46	.14
Biceps femoris	15.4 ± 7.3	15.2 ± 6.0	.91	.02
Standing				
Exercised side				
Vastus lateralis	19.6 ± 9.0	14.4 ± 4.8	.08	.32
Rectus femoris	13.4 ± 6.2	12.4 ± 4.8	.20	.23
Vastus medialis	18.3 ± 13.4	14.2 ± 11.0	<.03	.39
Biceps femoris	13.1 ± 3.5	21.6 ± 12.3	.06	.36
Non-exercised side				
Vastus lateralis	22.4 ± 13.4	20.5 ± 14.1	.58	.09
Rectus femoris	12.8 ± 4.5	12.1 ± 6.0	.49	.12
Vastus medialis	16.4 ± 10.3	14.5 ± 10.0	.55	.11
Biceps femoris	16.4 ± 10.3	14.8 ± 10.3	.75	.06
Walking				
Exercised side				
Vastus lateralis	20.0 ± 10.2	13.5 ± 2.7	.16	.27
Rectus femoris	12.8 ± 6.3	12.7 ± 4.5	.68	.07
Vastus medialis	14.6 ± 7.9	12.0 ± 4.3	.14	.28
Biceps femoris	13.7 ± 4.3	21.5 ± 14.3	.08	.30
Non-exercised side				
Vastus lateralis	19.3 ± 10.9	17.2 ± 8.1	.71	.07
Rectus femoris	11.7 ± 3.1	11.6 ± 5.4	.69	.07
Vastus medialis	13.7 ± 7.5	12.6 ± 7.0	.72	.13
Biceps femoris	14.6 ± 7.8	14.1 ± 6.1	.85	.03

P[a] = Wilcoxon signed rank test
r[b] = Effect size

Co-activation on first and second recording

Table 7 shows coactivation ratios during seated posture, standing posture and walking for the first and second long-term recording. On the exercised side, the vastus medialis/biceps femoris ratio decreased during seated posture (P=.009), while the vastus lateralis/biceps femoris ratio tended to decrease (P=.07). The vastus lateralis/biceps femoris ratio (P=.03) and vastus medialis/biceps femoris ratio (P=.02) decreased during standing posture. No differences in the coactivation ratio were found during walking. The coactivation ratio remained unchanged during all postures in the non-exercised leg.

Table 7. Co-activation ratio of the quadriceps and hamstring muscles during periods with sitting, standing, and walking during first and second long-term recording. Valus are mean ± SD.

	1st recording	2nd recording	P[a]	r[b]
Sitting				
Exercised side				
Vastus lateralis/Biceps femoris	1.1 ± 0.6	0.7 ± 0.3	.07	.36
Rectus femoris/Biceps femoris	0.8 ± 0.3	0.6 ± 0.3	.12	.32
Vastus medialis/Biceps femoris	1.1 ± 0.6	0.6 ± 0.3	.009	.48
Non-exercised side				
Vastus lateralis/Biceps femoris	1.0 ± 0.5	1.1 ± 0.6	.86	.03
Rectus femoris/Biceps femoris	0.8 ± 0.4	0.7 ± 0.3	.96	.16
Vastus medialis/Biceps femoris	0.7 ± 0.3	0.7 ± 0.2	.26	.26
Standing				
Exercised side				
Vastus lateralis/Biceps femoris	1.6 ± 0.7	0.8 ± 0.4	.03	.41
Rectus femoris/Biceps femoris	1.1 ± 0.7	0.8 ± 0.4	.11	.30
Vastus medialis/Biceps femoris	1.4 ± 0.9	0.8 ± 0.5	.02	.47
Non-exercised side				
Vastus lateralis/Biceps femoris	1.3 ± 1.0	1.4 ± 0.7	.43	.07
Rectus femoris/Biceps femoris	0.9 ± 0.3	0.9 ± 0.4	1.0	.01
Vastus medialis/Biceps femoris	1.0 0.4	0.9 ± 0.4	.45	.17
Walking				
Exercised side				
Vastus lateralis/Biceps femoris	1.3 ± 0.9	0.9 ± 0.4	.15	.28
Rectus femoris/Biceps femoris	1.0 ± 0.6	0.9 ± 0.3	.94	.09
Vastus medialis/Biceps femoris	1.4 ± 0.9	1.1 ± 0.4	.27	.27
Non-exercised side				
Vastus lateralis/Biceps femoris	1.2 ± 0.5	1.3 ± 0.5	.33	.22
Rectus femoris/Biceps femoris	0.7 ± 0.4	0.7 ± 0.3	.88	.01
Vastus medialis/Biceps femoris	1.1 ± 0.6	1.2 ± 0.5	.51	.03

P[a] = Wilcoxon signed rank test
r[b] = Effect size

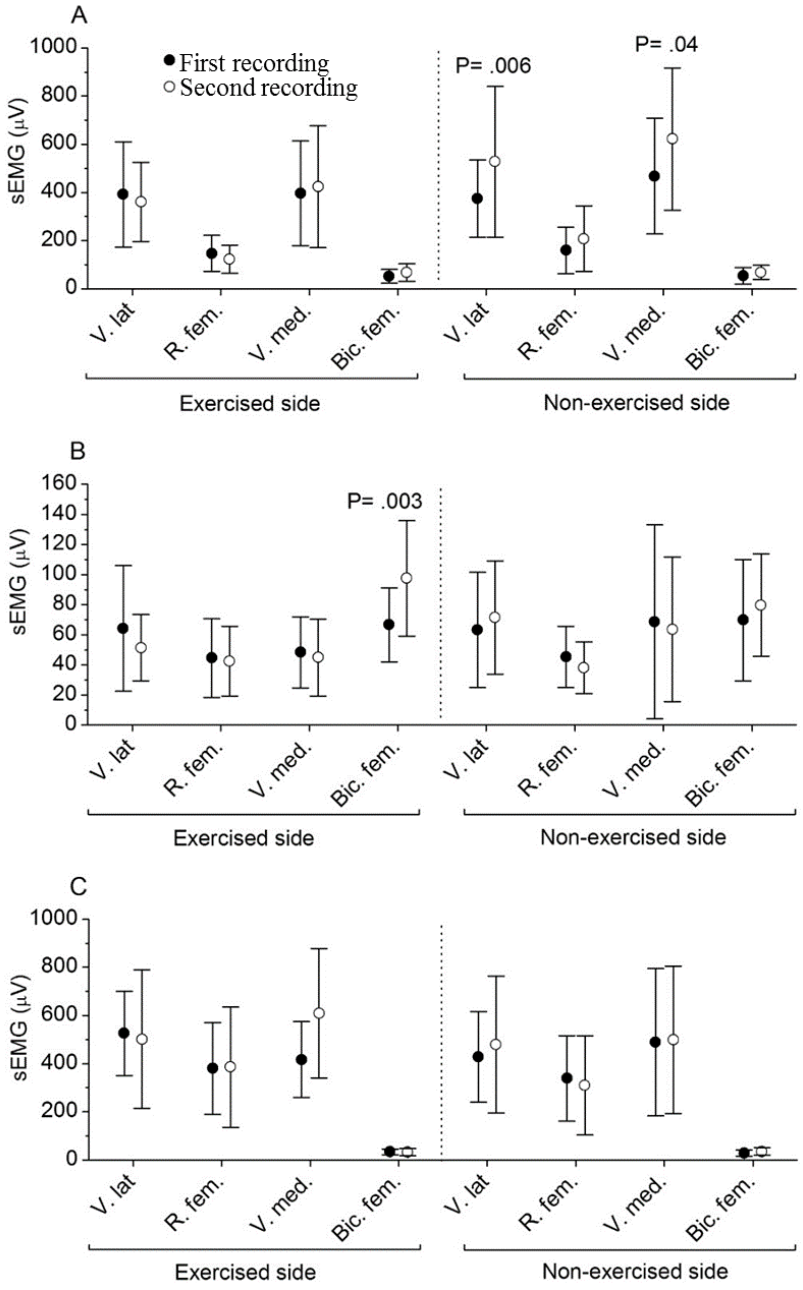

Figure 5. Median sEMG level (µV) for exercised and non-exercised vastus lateralis, rectus femoris, vastus medialis and biceps femoris during standing up from a chair (A), walking in stairs (B), and controlled loading (C) on first recording and second recording. Values are mean ± SD.

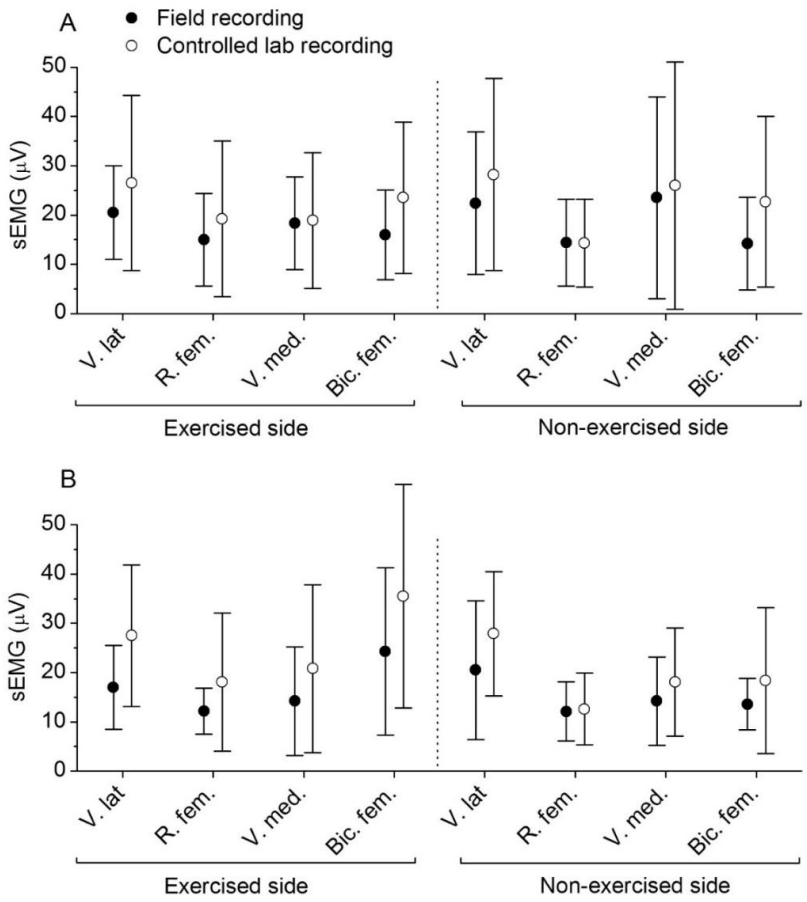

Figure 6. Field recording vs. controlled laboratory recording. Median sEMG level (µV) with SD for exercised and non-exercised vastus lateralis, rectus femoris, vastus medialis and biceps femoris during standing during first recording (A) and second recording (B).

Discussion

The primary aim of this study was to investigate the effect of acute quadriceps pain on habitual muscle activity in thigh muscles during unconstrained daily activities. A second aim was to investigate the effect of acute quadriceps pain on muscle activity in thigh muscles during a controlled laboratory setting, and to compare this with the unconstrained habitual muscle activation. Barbell lunges was used to induce DOMS in the dominant knee extensors. Achievement of DOMS was indicated by a reduction of PPT, raised VAS-score, and reduction in knee extension force on the exercised leg. Moreover, induction of DOMS in the dominant knee extensors was supported by an increased use of the non-exercised leg during standing up from a seated posture. An increased level of sEMG activity was observed in the antagonist in the exercised thigh during periods with seated posture, and a decreased level of sEMG activity in the exercised vastus medialis was observed during standing posture.

Furthermore, sEMG level tended to decrease during standing in the vastus lateralis, and tended to increase for biceps femoris in the exercised leg. Median sEMG level remained unchanged during walking, but tended to increase in the biceps femoris in the exercised leg. During walking in stairs, sEMG increased in biceps femoris in the exercised leg, while sEMG activity remained unchanged during the isometric contraction. When comparing standing posture in the laboratory with standing posture during the long-term field recording, the same tendency was observed, i.e., decreased agonist and increased antagonist activity in the exercised leg. There were no differences between the first and second recording in time spent in different body postures or heart rate during the long-term recording. These findings indicate that DOMS has no or only moderate effect on habitual muscle activity in the thigh muscles.

Tenderness, subjective variables and muscle performance

In this study, DOMS was chosen as a paradigm example to investigate the effect of muscle pain on habitual thigh muscle activity. It is well known that eccentric exercise leads to structural signs of muscle damage with initial manifestations including disrupted sarcomeres in myofibrils, and damage to the excitation-contraction coupling system, followed by an inflammatory response which promotes the breakdown, removal and resynthesis of the damaged muscle fibre.[30] This process sensitizes intramyofibril group IV afferents.[36] The present study measured sEMG activity in the distal regions of vastus medialis and vastus lateralis where the major part of group III nociceptors and mechanoreseptors served by group III axons are located.[37, 38] This makes the distal regions more sensitive to local pressure. On the other hand, to obtain measurement from the entire quadriceps, the electrode for rectus femoris was located half of the total muscle length. Moreover, type IIb fibers has been reported to be most evident in the distal regions compared to more proximal regions,[39] and type IIb fibers has been reported to be more sensitive to injury after eccentric contraction. This has been linked to their lack of oxidative capacity or increased strain and injury due to their shorter fiber length,[40] their higher tension,[41] and shorter optimum length for tension [42] compared to type I fibers. Contradictory to this, the Hennemans size principle [43, 44] claims that low threshold motor units are the main contributors to muscle activity in situations with low muscle force demands. The present study measured muscle activity only in situations of low muscle activity or nominal muscle rest, which means that the type II fibers most likely are not recruited in the present study. The fast-twitch fibers has been reported to not participate in the ordinary step cycle, and fast-twitch fibers only shows altered activity during rapid walking or

running.[45] Therefore, the observed alteration in muscle activity during the present study is not likely caused by alterations in the activation of fast-twitch fibers. One can argue that a more likely response due to decreased muscle activity in the present study might be due to muscle fibers that are not most injured, but still affected, by the eccentric exercise intervention.

DOMS is characterized by muscle hyperalgesia (tenderness) which mimic the most central feature of myofascial pain syndromes, and a typical feature is that pain intensity increases during loading or movement.[29, 46] Due to this, measurements of PPT is suggested to be a valid tool to quantify the extent of muscle tenderness.[47] PPT was markedly reduced in the exercised thigh 48 hours after the eccentric exercise, and most clearly reduced in the most distal part (vastus medialis) where it was a significant decrease in sEMG activity during standing posture, while the most proximal part (rectus femoris) demonstrated no change in sEMG activity during any postures. The greater distal PPT reduction is consistent with a previous study,[12] indicating that the site over the quadriceps where PPT decreased the most were also the area where the sEMG amplitude decreased after the eccentric exercise. Based on a more distinct change in the distal parts of vastus lateralis and vastus medialis compared to rectus femoris, the selection of measuring points can be of importance when intending to use DOMS as a model of pain. Furthermore, a lower peak force during MVC in the exercised knee extensors during second recording compared to first recording was clearly indicated in the present study. Moreover, averaged VAS scores of quadriceps pain increased by 3.7 units (i.e.,>3.7 cm) on the pain afflicted side from first to second long-term recording compared to 0.5 units (i.e., 0.5 cm) on the non-exercised side. Furthermore, an increased use of the non-exercised thigh was observed during standing up from a seated posture, which supports a pain induced activation difference between the legs. These results therefore indicate that DOMS was achieved.

The vicious cycle theory and the pain adaption model

According to the vicious cycle theory[3] a likely mechanism would be that an initiating factor (like abnormality in movement, posture, stress or injury) results in pain that further leads to muscle hyperactivity in a stereotypical manner. This is not supported by the present results because no hyperactivity was found at rest, during isometric contractions or during dynamic contractions. Moreover, the sEMG activity in the resting periods between each contraction during the MVCs demonstrated no hyperactivity. The vicious cycle theory was more an initial hypothesis and is not consistent with the majority of observations.[4, 9-14, 17-23] Still, two studies have observed increased motor drive restricted to the painful exercised part of trapezius

during static shoulder flexion,[7] and during sitting posture in a long-term field recording.[6] However, the latter study observed increased sEMG activity only during seated posture, a situation with low biomechanical loading of the shoulder and neck muscles. These results question the pain adaption model during very low levels of muscle activation or nominal muscle rest. However, this is in contrast to the present study that shows a tendency of decreased activation of the pain afflicted muscles, but an increased activity in the antagonist during seated posture, which is supporting the pain adaption model. The pain adaption model was developed to explain changes in voluntary movement and argues that a painful muscle will be uniformly inhibited, whereas antagonist activity will be facilitated [4]. The outcome of this would be decreased displacement, velocity and force to reduce pain provocation and further injury.[48] Reduced agonist muscle activity has been reported during pain in back and neck,[13, 17, 23] shoulder and upper limbs,[14, 20-22] and lower limbs.[12, 18, 19] One study showed that when pain was induced in vastus medialis, the muscle activity was also reduced in the synergist vastus lateralis, while the antagonist demonstrated no change.[18] Furthermore, a combination of decreased activity in the muscle agonistic to the painful muscle, and increased antagonist activity has been reported during dynamic movements of the legs.[10] This is consistent with the results of this study, which found a decreased muscle activity in the pain afflicted muscle during standing posture. However, a simultaneous antagonist increase and agonist decrease was not found during the different postures. Nevertheless, when a decreased agonist muscle activity was found during standing posture, the antagonist tended to increase although this was not significant, and when an increased antagonist activation was found during seated posture, the painful agonist vastus lateralis tended to decrease. One reason why no difference were found during gait could be due to the complexity of gait and a possible compensation of motor function by other leg muscles.[10, 49] Still, compared to the non-exercised thigh muscles during gait, the antagonist activity tended to increase, while the activity in the two most distal areas tended to decrease in the exercised leg. In contrast to the pain adaption model[4], we found no reduction in $sEMG_{max}$ for the exercised quadriceps during the MVC.

Coactivation during long-term recording

The coactivation ratio showed that in the exercised leg, the vastus medialis/biceps femoris coactivation ratio decreased during seated posture, and the vastus lateralis/biceps femoris and vastus medialis coactivation ratio decreased during standing posture. No changes in the coactivation ratio were found during walking, or in the non-exercised leg during any postures.

Moreover, during seated posture, the vastus lateralis/biceps femoris ratio tended to decrease. Increased coactivation has been associated with lower strength, higher pain levels and disability.[50] Moreover, altered coactivation may reflect reorganization of the motor output to enhance joint stability[51] For example, knee osteoarthritis is a condition with pain and impaired strength in quadriceps, which is assumed to reduce the knee joint stability.[52] Consistent with the present study, subjects with knee osteoarthritis generate compensatory muscle activity, by increased antagonist activity, probably to reduce the instability.[53] Moreover, in patients with chronic neck pain, coactivation of the neck flexor and extensor muscles has been proposed as a strategy to increase the stiffness of the cervical spine.[50] In the present study, the altered coactivation was caused by reduced quadriceps activity and an increased hamstring activity. These results show that acute muscle pain affect the coactivation ratio in some selected muscles during seated posture and standing posture, while again, no significant changes was found during walking.

Current findings and relativism to pain models

Interestingly, previous studies have suggested that the pain adaption mechanism does not apply to very low levels of muscle activation or nominal muscle rest.[6] In the present study, the antagonist tended to increase during all postures, but it was only a significant increase during seated posture, i.e., a posture with no or minimal biomechanical loading of the legs. During gait and standing, the changed antagonist activity could indicate a redistribution of load in the muscle groups involved. Decreased quadriceps force during gait (quadriceps avoidance) which promote the use of flexors, is probably an effective method to stabilize the knee, and has been reported in subjects suffering from knee-injury [54] and during experimental muscle pain.[18] According to the pain adaption model,[4] one can argue that this could be a conscious act because loading of quadriceps is painful. Still, this does not explain the increased antagonist activity during seated posture.

The present study demonstrates that motor patterns can be altered by pain, but the mechanisms behind this are not clear. The present study cannot distinguish between peripheral and central phenomena. The vicious cycle theory implies that an increase in muscle activity is based upon the idea that group III and IV afferents activate the γ-motoneurons projecting to both agonists and synergists, which further increase the firing in the primary muscle spindle afferents.[55] As mentioned, this is not supported by the present study. A possible neurophysiological mechanism related to the pain adaption model[4] is that the activity of thin nociceptive muscle afferents facilitates inhibitory pathways when the muscle act as an

agonist and facilitates excitatory pathways during antagonist activity.[15] According to the present results, a likely response due to the decreased muscle activity in the pain afflicted area may be an inhibition of the motor systems excitability both at the cortical and the spinal levels.[56] This is in agreement with the present study which showed a larger decrease in EMG amplitude in the distal parts where the PPT reductions where more evident compared to the more proximal part. This findings is consistent with a previous study.[12] There is some controversy regarding the effect of pain on excitability of the motor pathways. Some possible explanation may be that tissue injury can influence primary afferents of muscle spindles at superficial layers of the dorsal horn of the spinal cord after eccentric exercise[57] and input from nociceptive afferents may inhibit the input from muscle spindles by presynaptic inhibition in the injury muscle. This may reduce the motor unit discharge rate which results in a decreased drive to the painful muscle.[58] Studies that shows reduced synergistic muscles activity in presence of pain[18, 59] strengthens the assumption that central mechanisms are involved in the altered muscle activity during muscle pain. Thus, the exact mechanism leading to decreased quadriceps, and increased biceps femoris sEMG activity after eccentric exercise remains to be explored.

Both the vicious cycle theory[3] and the pain adaption model[4] predict relatively stereotypical changes in muscle activity. Based on the present study, one can argue that pain not necessarily affect muscle activity in a simply stereotypical manner. As mentioned, observations on the lower limbs,[12, 18, 19] and upper limbs[20 21, 22] support that painful muscles reduce their activity. Still, just a few observations supports the notion that antagonist activity increases.[10, 14] Moreover, some studies shows no change in antagonist activity,[18, 21] and one study even shows decreased antagonist activity in presence of pain.[20] Moreover, studies on more complex muscle groups such as trunk muscles and back muscles shows both increased agonist[6, 7] and decreased agonist activity.[13, 17, 23] Further, an increased activity in the back muscles has been observed when the muscles normally are inactive, and decreased activity when the muscles normally are active,[60] which shows effects depending on the muscular function. This is supported by an occupational work-study, where experimental muscle pain seemed to modulate motor control differently depending on the precision level of the task,[21] supporting the statement that pain models should include a task dependency aspect.

Limitations

The present results indicate that the time spent in different postures was unchanged before and after the presence of DOMS. However, the present study did not assess if differences occurred

in knee-angle (flexed and/or fixated). Another limitation is the lack of a control group. As an alternative, the non-exercised thigh was used as control. The findings were consistent, i.e, decreased agonist and increased antagonist activity in the exercised thigh, and unchanged agonist and antagonist activity in the non-exercised thigh. Based on this, inclusion of a control group would probably not change the overall conclusion of the study. However, some subjects reported a tendency of muscle soreness in the non-exercised leg, which might be due to a not optimal execution of the exercise intervention. Another possible explanation may be a carry-over effect from the exercised to the non-exercised side. Nevertheless, the reduction in PPT was substantially larger in the exercised thigh, and the VAS measurements showed a marked difference between the exercised and non-exercised leg. Moreover, knee extension force was only reduced on the exercised side. An additional limitation to the exercise intervention is the use of the gluteal muscles during barbell lunges. Some of the subjects reported muscle soreness in the gluteal muscles on the exercised side. Anyway, this did not affect biceps femoris noteworthy because the PPT measurements did not decrease more on the biceps femoris on the exercised side compared to the non-exercised side.

Conclusion

While most of the previous studies have used a short lasting exogenous technique (hypertonic saline), the present study used an endogenous technique (eccentric exercise) to obtain a long-lasting pain response. During the long-term recording, the increased antagonistic muscle activity was restricted to periods with seated posture; a situation with low biomechanical loading of the legs. A decreased agonistic muscle activity was restricted to periods with standing posture. Overall, there was a tendency of decreased agonist and increased antagonist activity in the exercised leg during the long-term field recording. During the laboratory tests, an increased antagonistic muscle activity was observed during walking in stairs, while the muscle activity remained unchanged during the isometric contraction. When comparing standing posture in the laboratory with standing posture during the long-term field recording, the same tendency was observed, i.e., decreased agonist and increased antagonist activity in the exercised leg. The present findings indicate that DOMS has no or only moderate effect on muscle activity, and the results indicate that the response to muscle pain is not so stereotypical as suggested by the pain adaption model, supporting the notion that pain models should include a task dependency aspect.

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