Experimental quadriceps muscle pain impairs knee joint control during walking

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Knee pain is a cardinal symptom in musculoskeletal diseases involving the knee joint, such as osteoarthritis and patellofemoral pain syndrome (8, 9, 34), and aberrant movement patterns and motor control strategies are often present in these patients (29, 39). Patients with osteoarthritis of the knee exhibit reduced knee extensor torque during walking (29) and altered muscle activation patterns (25), but it is unclear whether these changes in movement and motor control are a consequence of the disease per se or whether neuromuscular changes are causal factors in the development of musculoskeletal diseases (47).

The neuromuscular control of walking has been linked theoretically to the development of osteoarthritis of the knee (2, 18, 41). Failure to properly absorb joint loads during walking is suspected to provoke a biological response that potentiates the progression of articular cartilage damage in knee osteoarthritis (5, 17, 58), and bilateral proprioceptive deficits indicating generally altered motor control have been reported in unilateral knee osteoarthritis (48).

Other changes not directly connected to motor control occur in the wake of a musculoskeletal disease, such as osteoarthritis. These include joint contractures, deformities, instability, muscle wasting and weakness, reduced muscular oxidative capacity, inflammation, and joint effusion (35). Many of these changes are progressive with disease severity (e.g., 27), and because pain most often is uncorrelated to disease severity (7, 11), pain adaptations in the knee dynamics during early stages of a disease, could be a possible source for development of the changes. Therefore, there is a need for the investigation of how pain affects the knee joint function. However, the changes associated with knee pathology may introduce unwanted variability in movement and motor control analyses. By consequence, it is difficult to assess the isolated effects of pain on motor control in a patient population. Experimental techniques to induce pain in healthy subjects are thus advantageous in this respect. A harmless technique inducing reversible, localized pain in the muscles has been used in many experimental situations (19, 23). This allows an assessment of the effect of pain on movement and motor activity and control. Experimental muscle pain has been shown to impair the neuromuscular function (55), which is suggested to be a risk factor for the development of osteoarthritis of the knee (26, 46, 47, 52).

Strategies for treatment and prevention of musculoskeletal pain are mainly empirical, and a better understanding of underlying sensory-motor mechanisms would be one step forward toward optimizing therapy. In addition, there is a lack of studies combining sensory-motor evaluation of pain with biomechanical analyses of the effects of pain on movement. The purpose of this study was to investigate the effects of experimental muscle pain on the knee joint function during walking in healthy subjects, as assessed by three-dimensional gait analyses yielding knee joint kinematics and kinetics synchronized with surface electromyography (EMG).

MATERIALS AND METHODS

Subjects. Twenty healthy subjects (10 women and 10 men) with no history of lower extremity pathology, trauma, or pain were included.

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The average age was 26 (range: 19–42) yr, weight 70 (range: 54–85) kg, and height 1.75 (range: 1.65–1.98) m. All subjects gave their informed consent before inclusion, and the study was approved by the local ethics committee [J.no. (KF) 01-258645].

Study design. The study was designed as a crossover study with each subject tested on 2 days separated by at least 1 wk (Fig. 1). Each test day consisted of a total of five sequences: a baseline and four consecutive sequences. Each sequence consisted of four valid (acceptable) gait trials, with each trial containing the recording of one gait cycle. The baseline and sequences 1–3 were separated by 5-min pauses where the subjects rested on a chair. After the third sequence, there was a 20-min pause before the fourth sequence was completed. On each test day intramuscular saline injections into the vastus medialis (VM) muscle were given immediately before sequences 2 and 3. The saline was either hypertonic (5.8%; painful) or isotonic (0.9%; nonpainful) saline. The test day order of saline injections was decided by randomization, and only one solution was administered on each test day.

Experimental muscle pain. Muscle pain was induced by intramuscular bolus injections of 1 ml sterile hypertonic saline (5.8%) into the VM muscle ~5 cm proximal and 5 cm medial to the medial corner of the patellar base. Injections of isotonic saline (0.9%) were used as control. Manual injections over 20 s were accomplished by a 5-ml plastic syringe with a disposable needle (27 gauge, 40 mm) in a depth of 3.5 cm. The pain intensity was scored on a 100-mm visual analog scale (VAS) where 0 mm indicated “no pain” and 100 mm indicated “worst imaginable pain.”

Gait analyses. A walkway (8 m long) with two force platforms (model OR6-5-1, AMTI) placed centrally was used for data acquisition. The subjects were instructed to walk at a speed of 4.5 km/h. The walking speed was measured by photocells, and a digital display provided the subjects with immediate visual feedback of the walking speed. On each test day, the subjects practiced the desired walking speed until they could walk comfortably several times before commencing the actual test sequences. This was done to accustomed the subjects to the target speed and to reduce the number of invalid trials. From each sequence four valid gait cycle trials were selected for analysis of each subject, with a valid trial defined as a trial with a walking speed of 4.5 km/h (± 0.05 km/h). The median number of trials needed to capture four acceptable trials in each sequence was seven. Pain was scored on the VAS scale after each walking trial.

Fifteen reflective markers were placed on the subjects according to the marker setup described by Vaughan et al. (57). Five digital video cameras operating at 50 Hz were used to record the movements. Two force platforms measured the ground reaction forces, which were sampled at 1,000 Hz. Synchronization between the video signals and the force plate signals was obtained by an audio signal, which was sampled at 1,000 Hz, preamplified (input impedance 1 GΩ), filtered (10–1,000 Hz), and digitalized. The video recordings were digitized and stored on a computer.

The video recordings were digitized and stored on a computer. Three-dimensional coordinates were constructed by direct linear transformation using the Ariel Performance Analysis System (Ariel Dynamics), and three-dimensional kinematics were calculated. Joint moments were calculated using an inverse dynamics approach by combining force plate and movement data (57). All joint moments were expressed as a percentage of the subject’s body mass (N·mkgs⁻¹·10⁻¹).

To assess the knee joint loadings, a statically determinate muscle model was applied (42). The knee compression force was calculated as the vector sum of 1) the intersegmental reaction force resolved along the long axis of the tibia, 2) the compression component of the active muscle group force, and 3) the axial component of the cruciate ligament tension. The included muscles were the hamstrings, gastrocnemius and quadriceps muscles. The hamstring and gastrocnemius complex constituted a flexor muscle group active when the sagittal knee joint moment favored the flexors, and the quadriceps muscle represented an extensor muscle group active when the net moment favored extensors. No antagonistic contractions were allowed in the model. The muscle forces were calculated by combining the net sagittal plane joint moments with the muscle moment arms derived from a third-order polynomial relating the knee joint angle to the muscle moment arms (12). The model was a modification of the method of Schipplein and Andriacchi (42) because the mediolateral knee joint diameter was obtained from each subject and used for calculation of the position of the tibiofemoral contact point. In the original model the knee joint diameter was fixed at 80 mm for all subjects. The predicted joint compression values were presented as a proportion of bodyweight.

Sagittal plane knee joint moments and frontal plane moments, and predicted joint compression forces in the right knee were all plotted against percent gait cycle, and the dependent variables from the gait analysis were stance phase peak values of the sagittal and frontal plane knee joint moment curves, predicted joint compression curves, and sagittal plane knee joint angle curves. An illustrative example of data from one subject is provided in Fig. 2.

EMG. Surface EMG was recorded from the VM, vastus lateralis (VL), semitendinosus (ST), and biceps femoris (BF) muscles using a bipolar surface electrode configuration (Blue-Sensor N-00-S, AMBU, Ballerup, Denmark) with a 2-cm interelectrode distance. Before mounting the electrodes the skin was carefully shaved and rinsed with pure alcohol. The EMG signals were band-pass filtered (10–1,000 Hz), preamplified (input impedance 1 GΩ) and sampled at 1,000 Hz using a lightweight (70 g) wireless EMG system with 12 bit analog-to-digital resolution (model MQ8, Marq-Medical, Farum, Denmark). After placement of the electrodes a measurement of resting EMG activity was made while the subject was sitting quietly on a chair and subsequently maximal EMG activity was measured during maximal isometric contractions performed in an isokinetic dynamometer (KinCom, Chattex, Chattanooga, TN).

For each trial, EMG activity onsets and cessations were determined by a computer algorithm identifying the point where the mean of a rectified raw EMG sample lasting at least 50 ms exceeded the resting activity by 2 SDs (24). Subsequently, the raw EMG signals were digitally high- and low-pass filtered (Butterworth fourth-order zero-lag filter, cut-off frequencies 20 Hz and 450 Hz, respectively), full-wave rectified, and low-pass filtered (cutoff 15 Hz) to construct linear envelopes. The linear envelopes were normalized in amplitude to the EMG signal during the maximal isometric contraction (expressed as %maximal voluntary contraction) and from the normalized audio track of the audio and video interleaved file formats and used to synchronize the video signals. Additionally, the audio signal triggered the recordings from the force platforms, which ensured synchrony between the video and force signals. The synchronization signal was triggered when the subjects’ legs passed the first photocell. The 1,000-Hz force platform signals were down sampled to 50 Hz to fit the video signals.

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linear envelopes peak and mean amplitudes within the activity periods (onset to cessation) were calculated. The linear envelopes and the gait analysis data were time synchronized, using the force platform data and a foot switch placed under the right heel of the subjects. For all muscles, integrated EMG were calculated during the 50 ms before heel strike (iEMGpre), the eccentric contraction phase (iEMGecc; i.e., where the knee is flexing during load acceptance and initial single support) and the concentric contraction phase (iEMGcon; i.e., where the knee is extending during midstance and late single support). From the EMG analysis, the dependent variables were peak and mean EMG activity, iEMGpre, iEMGcon, iEMGecc, and onset and cessation times for each muscle. An illustrative example of rectified EMG signals from one subject before injection is provided in Fig. 2.

Statistical analysis. The extracted data (both gait analyses and EMG recordings) from the four valid trials were averaged within each sequence. Factorial repeated-measures ANOVAs with Bonferroni-adjusted confidence intervals were used. If the assumption of sphericity was violated for main effects of saline and sequence, the degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity and the corrected $P$ values are reported. All effects are reported as significant at $P < 0.05$. Any significant interactions between type of saline and sequence were broke down using contrast analyses comparing the effects of hypertonic saline to isotonic saline injections at sequences 1–4 to the baseline difference.

RESULTS

Muscle pain. There was a significant interaction between type of saline and sequence ($P < 0.0001$; Fig. 3). The contrast analysis revealed significant differences in pain scores, showing that hypertonic saline injections produced pain intensities significantly higher than the isotonic saline injections in sequences 2 and 3 ($P < 0.0001$). Pain scores following isotonic saline in sequences 2 and 3 were not significantly different from 0 ($P > 0.05$; Fig. 3).

Gait analyses. All gait analysis parameters were stable from the baseline measurement to sequence 1, i.e., before injections ($P > 0.05$; Fig. 4). There were significant interactions between type of saline and sequence in the knee extensor moment during the loading response in single support ($P = 0.017$). Contrast analyses showed significantly lower extensor moments following hypertonic saline at sequences 2, 3 (during pain), and 4 (after pain), ($P < 0.045$; Fig. 4). A detailed examination of the knee extensor moments revealed that four subjects walked with a moment pattern favoring the flexors throughout the stance phase during and following muscle pain (Fig. 5). All other subjects walked with a sagittal moment that alternated between flexor and extensor dominance (Fig. 2). The knee extensor moment data were analyzed without the observations from the four subjects, the statistical significant interactions persisted, and the contrast analyses showed significantly lower extensor moments following hypertonic saline at sequences 2 (during pain) and 4 (after pain) ($P > 0.014$). The contrast at sequence 3 was not statistically significant when excluding the four subjects ($P = 0.11$). No significant interaction was found in the total knee compressive force ($P > 0.05$; Fig. 4), a result that persisted when excluding the four subjects adopting quadriceps avoidance gait patterns from the analysis.

![Fig. 2. Representative time course patterns of gait analysis variables and rectified EMG during 1 gait cycle before experimental muscle pain induced in the vastus medialis muscle. From the top: sagittal plane knee joint moment, frontal plane knee joint moment, total knee joint compression force, sagittal knee joint angle, rectified EMG signals from vastus medialis (VM), vastus lateralis (VL), biceps femoris (BF), and semitendinosus (ST) muscles. BW, body weight (in kg).](image-url)

![Fig. 3. Mean visual analog (VAS) scores (± SE) during walking following injections of either isotonic (gray squares) or hypertonic (black squares) saline into the vastus medialis muscle. Injections were given between sequences 1 and 2, and between sequences 2 and 3. The pain intensity is reported on a 100-mm VAS. $P < 0.0001$.](image-url)
There were no effects of either type of saline injections on frontal plane knee moments or knee joint angles (both $P < 0.05$; Fig. 4).

**EMG.** EMG parameters were similar in the baseline and sequence 1, i.e., before injections ($P > 0.05$; Fig. 6). Significant interactions between sequence and saline injections were found in both peak and mean EMG amplitudes of the VM muscle ($P = 0.03$ and $P = 0.009$, respectively). Contrast analyses revealed that both peak and mean EMG amplitudes were significantly reduced in the VM following hypertonic saline injections compared with isotonic saline ($P = 0.01$; Fig. 5), and iEMGpre in sequences 2 (during pain) and 4 (after pain) was significantly lower following hypertonic saline vs. isotonic saline ($P < 0.017$; Fig. 6). The significant interactions persisted after excluding the four subjects that adopted a quadriceps avoidance gait pattern from the analysis.

Analyses of the VL EMG parameters showed significant interactions between type of saline and sequence with respect to mean amplitude and iEMGpre ($P < 0.029$). Contrast analyses showed that the VL mean amplitude was significantly reduced following hypertonic saline in sequence 4 (after pain) compared with isotonic saline ($P = 0.01$; Fig. 5), and iEMGpre in sequences 2 (during pain) and 4 (after pain) was significantly lower following hypertonic saline vs. isotonic saline ($P < 0.017$; Fig. 6). The significant interactions persisted after excluding the four subjects that adopted a quadriceps avoidance gait pattern from the analysis. The EMG parameters of the hamstring muscles (BF and ST) did not change following either type of saline injections ($P > 0.05$). Similarly, there were no effects of either type of saline injections on the EMG onset or cessation times in either of the muscles (all $P > 0.05$).

![Fig. 4. Results of the three-dimensional gait analyses and the statically determinent muscle model showing the effects of muscle pain on knee joint function during the loading response in single support of the gait cycle. From the top: peak knee extensor moment, peak frontal plane moment, peak total knee joint compression force and peak knee joint angle. Mean ± SE is shown for effects of isotonic (gray squares) and hypertonic (black squares) saline injections into the VM muscle. The injections were made immediately before sequences 2 and 3. There were only observed significant effects ($P < 0.045$) of pain on the peak knee extensor moment (asterisks).](image)

![Fig. 5. An illustrative example of a quadriceps avoidance gait pattern developed by 4 of the 20 subjects following experimental VM muscle pain. From the top: sagittal plane knee joint moment, sagittal knee joint angle, rectified EMG signals from VM, VL, BF, and ST muscles. The quadriceps avoidance pattern is characterized by flexor moment during single support and a lack of oscillations between flexor and extensor moments throughout the stance phase. This subject locks the knee joint in near full extension during the first two-thirds of the stance phase as seen in the knee joint angle curve, together with reduced EMG activity in the VM and VL muscles.](image)
DISCUSSION

The present study shows that experimental vastus medialis pain attenuates the knee extensor moment during the loading response phase of walking. The attenuation is explained by reduced quadriceps activation observed as decreases in the EMG activities of the VM and VL muscles. The changes in knee joint moments and EMG activity persisted even when the pain was no longer present, suggesting a central inhibition persisting beyond the conscious pain perception. The experimental VM pain did not cause changes in knee joint compression forces, knee joint kinematics, adduction moments, knee flexor moments, hamstring EMG activity and EMG onset, and cessation times for either muscles. The present study is, to our knowledge, the first to assess the effects of experimental muscle pain on knee joint control and mechanics during walking.

Knee joint dynamics and muscle pain. The knee joint extensor moment pattern following experimental muscle pain resemble that of patients with knee pathology such as osteoarthritis (16, 29), anterior cruciate ligament (ACL) deficiency (10), and patellofemoral pain syndrome (39). In the case of osteoarthritis, reduced extensor moments is believed to be a compensation adopted to reduce the joint loading and thereby avoid pain (29). This is supported by studies where pain relief in knee osteoarthritis leads to increased knee extensor moments (28, 43), which is suggested to be detrimental to the joint, because knee extensor moments and joint loading are correlative. In the present study, we were not able to detect changes in the average joint compression force, although the extensor moments were reduced significantly.

The knee extensor moment has been shown to have a significant role in stabilizing the knee against varus deformations (36), and therefore it follows that the combination of unchanged adduction moment and reduced extensor moment, as observed in the present study (Fig. 3), may result in knee instability during the loading response phase of the gait cycle as a consequence of pain. Interestingly, the effects of the experimental muscle pain on the joint moments were sustained after the pain was no longer present. While the adduction moments were unaffected by pain, the sustained attenuation of the extensor moments after pain leaves the knee joint less stable (31, 33, 38) and thus prone to injury, although the protective pain sensation has ceased.

A detailed examination of the gait analysis data revealed that pain resulted in a complete quadriceps avoidance gait pattern in 4 of the 20 subjects (see Fig. 5 for an illustrative example), which could have affected the overall group results. However, when excluding the four subjects from the analysis, the reduced extensor moments and the lack of change in joint compression persisted. This implies that other factors than the extensor moments, such as the adduction moment, contribute significantly to the total joint compression. Quadriceps avoidance gait is not an unusual observation and has been reported in both healthy subjects as a preferred walking pattern (50, 59) and in individuals suffering from severe knee injuries such as ACL deficiency (1, 10). In the ACL example, the knee moment

Fig. 6. Effect of muscle pain on EMG activity in VM (left) and VL (right) during walking. Mean values (±SE) of peak and mean amplitudes together with iEMGpre, iEMGecc, and iEMGcon (see text for definitions) for both muscles are shown for effects of isotonic (gray squares) and hypertonic (black squares) saline injections into the vastus medialis muscle. MVC, maximal voluntary contraction. *Significant differences in EMG activity between types of saline, P < 0.04.
pattern showed only little response to 6 mo of rehabilitation (10) and the individual’s moment pattern seems to be quite robust to interventions. Nevertheless, induction of experimental muscle pain caused 4 of 20 subjects to switch to quadriceps avoidance (flexor dominance) during single support, showing that pain is a powerful sensation capable of modulating the movement pattern significantly. The results also show that some individuals adopt more radical compensatory movement patterns than others. Interestingly, the pain scores from the four subjects with the most dramatic compensations were not different from those of the rest of the study sample, indicating that the pain intensity was not the cause of the shift in joint moments.

The results from the present study are similar to a study of experimental knee joint effusion on gait by Torry et al. (56). In that study, reduced extensor moments and quadriceps EMG activity during the loading response were observed following experimental knee joint effusion. The effusion caused even more dramatic changes than in the present study, because also the kinematics and hamstring EMG were affected. However, Torry et al. could not discount possible influences of the nociceptive system caused by the effusions, whereas the present study provides data on the isolated effects of pain on the knee dynamics during walking. Nevertheless, both pain and effusions are relevant clinical conditions to investigate and the results support each other in describing the effects of common clinical manifestations of knee pathology on knee joint dynamics during gait.

Muscle activity and muscle pain. It has been proposed that the effects of muscle pain on muscle activity depend on the functional role of the painful muscle in the movement (55). Lund et al. (32) proposed in the pain adaptation model that pain reduces muscle activity in muscles with agonistic function (32), a theory that has gained support in a study of EMG activity of lower leg muscles during treadmill walking, where experimental muscle pain affected the muscle activity differently in various phases of the gait cycle (22). Furthermore, it has been shown that pain also causes reduced activity in nonpainful synergist muscles (6, 13, 44). In the present study, pain in VM reduced the EMG activity in both VM and in its synergist VL, during the loading response phase of the gait cycle where the muscles have agonistic function in eccentrically controlling the knee flexion. No changes in the EMG activity during the concentric quadriceps contraction in midstance were observed, although the muscle has agonistic function here as well. Synergistic inhibition, as observed in the present study, indicates that central mechanisms are involved in the altered muscle activity and movement pattern. This was also indicated in a study where experimental muscle pain caused reduced maximal voluntary isometric knee extensor torque without impairing the contractile apparatus (21). In the present study the antagonists, i.e., the hamstrings, were unaffected by the VM pain in any phase of the gait cycle. This concurs with the gait analyses, where no changes occurred in the peak knee flexor moments during the last half of the stance phase (Fig. 1).

The sustenance of synergistic muscle inhibition after pain indicates that the immediate adaptive response to muscle pain in muscle activity and movement pattern is not as easily “unadapted” once the nociceptive input is vanished, but is sustained for at least 20 min after pain. This is in accordance with previous observations showing that pain inhibition of muscle function still occurs when the pain is no longer present (14, 45, 51). A possible explanation for this could be a central adaptation to nociceptive inputs (37) mediated by descending inhibition of the ascending nociceptive signals at spinal or supraspinal levels (54). This implies that the nociceptive signals from the periphery modulate motor circuits at spinal levels post pain without the nociceptive signals reaching consciousness. The sustained muscle inhibition beyond conscious pain perception could leave the knee joint instable and prone to injury and could contribute to the chronicity of knee problems.

Methodological considerations. The average pain intensity reported by the subjects during the experimental muscle pain was ~25 mm on a 0- to 100-mm VAS, which is lower than previous reports on experimental muscle pain. For example, in a similar study, where intramuscular injections of hypertonic saline into either the tibialis anterior or gastrocnemius muscles, the average pain intensity was 47 mm (22). Different muscle volumes between the studies may account for the differences in pain intensities. In addition, referred pain is usually reported during experimental muscle pain (20). However, in the present study only one subject reported referred pain to the medial and lateral aspects of the lower leg. This shows that the knee joint function is impaired during moderate pain, and it is possible that stronger pain sensations could cause even larger and/or additional effects on the knee joint function than those observed in the present study.

One limitation to the present study might be the choice of standardized walking speeds, as it is generally accepted that self-selected walking speeds produce more reliable results. However, different walking speeds between sequences could have caused unwanted variability in the data (30). The effort to maintain the target speed could be suspected to blur the effects of pain as the central nervous system must accommodate the demands of walking at a specified target speed and at the same time adapt the motor performance to the pain. Nevertheless, we were able to detect significant changes in the movement pattern and muscle activity during muscle pain. Standardized walking speed was used because it is likely that induction of pain would cause the walking speed to change. This is seen in clinical studies where patients adapt to pain therapy by increasing their walking speed (43, 49). Because gait parameters are highly influenced by differences in walking speeds (3, 30), standardized walking speeds are desirable when experimental studies are performed.

Another possible limitation of this study is that the applied knee joint model not allows antagonist contractions in the estimation of joint compression forces, and the estimated forces should be regarded as minimum boundaries. The quadriceps-hamstring balance often is disturbed in knee patients, such as osteoarthritis (25), which could question the validity of the model output. However, in the present study the experimental muscle pain caused reduced quadriceps activity and unchanged hamstring activity and thus, the agonist-antagonist balance was disturbed. Because only the agonist (quadriceps) activity was changed, the results from the applied model are valid, because any unknown contributions from the antagonists (hamstrings) are constant.

Clinical implications. It is important to recognize that the changes in movement patterns and muscle activity during walking caused by experimental muscle pain cannot be trans-
ferred directly to a clinical situation. However, in the present study, the use of experimental muscle pain demonstrated that basic knee joint control mechanisms are vulnerable to pain. Furthermore, the changes in movement pattern are similar to those observed in investigations of patients with knee pain, such as osteoarthritis (16, 29). The involvement of these mechanisms has been suggested to participate in both development and progression of the disease (15, 25, 26, 52, 53). In patients with knee pathology symptoms may flare over a period of time, and patients may even experience pain-free periods, which certainly is beneficial to the patients and the aim in most treatments. However, the results from this study suggest that reduced knee joint control may persist in these pain-free periods, preserving instability. Without the protective pain sensation, a pain-free knee joint is even more prone to further damage or injury, and pain relief may be a pathogenetic factor in disease progression. This has wide clinical implications. It has been shown that patients with knee osteoarthritis have increased consumption of pain medication during a period of exercise participation (4). By exercising and taking the medication at the same time, the knee joints of these patients might be exhibited to increased jeopardy. This could be one explanation for the indication of accelerated cartilage loss in pain medicated knee osteoarthritis patients (40).

In conclusion, the present study demonstrates that the function of the quadriceps muscle during walking is modulated by muscle pain leading to impaired knee joint control and instability during the loading response phase of walking and that these changes are sustained for at least 20 min after pain. The changes caused by experimental muscle pain are similar to those observed in patients with painful knee disorders. The loss of joint control, both during and after pain, may leave the knee prone to further injury and participate in the chronicity of musculoskeletal problems. The link between pain and muscle function demonstrated in this study, signifies the relevance of muscle function preservation and/or improvement as an important target in the treatment and rehabilitation of patients with painful knee joint diseases.

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