Increased joint loads during walking – A consequence of pain relief in knee osteoarthritis

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Abstract

Joint pain is a primary symptom in knee osteoarthritis (OA), but the effect of pain and pain relief on the knee joint mechanics of walking is not clear. In this study, the effects of local knee joint analgesia on knee joint loads during walking were studied in a group of knee osteoarthritis patients. A group of healthy subjects was included as a reference group. The joint loads were calculated from standard gait analysis data obtained with standardised walking speed (4 km/h). The gait analyses were performed before and after pain relief by intra-articular injections of 10 mL lidocaine (1%). Pre-injection measurements revealed lower joint loads in the OA group compared to the reference group. Following injections pain during walking decreased significantly and the joint loads increased in the OA group during the late single support phase to a level comparable to the reference group. Although the patients walked with less compressive knee joint forces compared to the reference group, the effects of pain relief may accelerate the degenerative changes.

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1. Introduction

Joint pain is the cardinal symptom of knee osteoarthritis (OA) [1,2] and consequently pain reduction is the primary objective in treatment of the disease. Pain can be considered a protective mechanism causing compensatory changes in movement in order to counteract potentially harmful mechanical loads during locomotion. In this case, pain relief may act negatively on the underlying disease by eliminating the protective signals, with a subsequent inappropriate alteration of tissue loads during movement. This could result in higher mechanical loads in the knee joint, which in turn, might accelerate degeneration.

Mechanical loading of the knee joint during everyday activities such as walking may be implicated in the progression of the disease [3,4]. Previous studies of knee OA patients show changes in walking patterns that could be attributed to the presence of pain [5–11]. However, the possible effects of pain and pain relief on knee joint loads during walking are not clear. We studied the effect of local knee joint analgesia on the knee joint loads during walking in patients suffering from painful knee joint OA.

2. Subjects and methods

2.1. Subjects

Patients for the study were recruited from the Rheumatology outpatient clinic at Frederiksberg Hospital if they had unilateral medial knee OA diagnosed according to the criteria defined by the American College of Rheumatology [12], including pain during walking and radiographic evidence of knee OA on a standard anterior–posterior weight-bearing radiogram. Patients included had mild joint space narrowing based on the radiographic atlas for osteoarthritis of the knee [13]. Ten patients were included in the study. However, one patient was excluded due to a misplacement of the injection.
walking on a 100 mm Visual Analogue Scale (VAS) with the extremes being expressed as a percentage of the subject’s bodyweight (Nm/kg*100). Negative values represent abduction moments. All joint moments were moments. In the frontal plane, positive values represent adduction moments and negative values represent flexor moments (hamstrings and gastrocnemius muscles). The hamstring and gastrocnemius complex constituted a flexor muscle group active when the net sagittal knee joint moment favoured the flexors (i.e. negative) and the quadriceps muscle represented an extensor muscle group active when the net moment favoured extensors (positive). 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 [18]. The axial cruciate ligament forces were estimated under the assumption that the cruciates only resist antero-posterior shear forces. The medio-lateral position of the tibio-femoral contact point is fixed at 25% of the knee joint diameter from the knee joint centre, whereas the antero-posterior contact point changes with flexion [17]. As long as the overall knee compression forces acting over the contact point resist the frontal plane moment, no tension in lateral soft tissue is required, otherwise appropriate lateral tissue tension is introduced to avoid lateral joint opening. Lateral soft tissue tension indicates that all joint forces are supported by the medial joint compartment and more than the estimated muscle force is needed to avoid lateral opening of the joint.

### 2.2. Ultrasound examination

Ultrasound examinations of the patients’ knees were performed before the experiment. The examination included a test for possible effusion by compressing the lateral and medial aspects of the suprapatellar bursa. None of the patients had signs of effusion (dry knee OA). Furthermore, the placement of the injection was documented real-time on the screen.

### 2.3. General procedure

All subjects (patients and reference) gave their informed consent to participate in the experiment, and the study was approved by the local ethics committee (J. No. 01-193/03).

All patients assessed their OA status during the 48 h before measurements, using Western Ontario McMaster Universities Osteoarthritis Index (WOMAC), with pain (5 items), stiffness (2 items) and physical function (17 items) subscales. The WOMAC was designed as a 5-point Likert scale for each item, with high scores indicating high degree of impairment. The total score was normalised to a 0–100 score [14].

Three-dimensional (3D) gait analyses were performed before and immediately after local knee joint analgesia. The reference group had no injections and only underwent one gait analysis. Local joint knee analgesia was induced by injecting 10 mL lidocaine (1%) with a lateral approach into the knee joint. The injections were ultrasound guided to ensure proper placement of the lidocaine bolus in the suprapatellar bursa [15].

The patients were asked to register their perceived knee joint pain during walking on a 100 mm Visual Analogue Scale (VAS) with the extremes being “no pain at all” (0 mm) and “worst imaginable pain” (100 mm).

### 2.4. Gait analyses

All subjects were instructed to walk at a speed of 4.0 km/h (~1.1 m/s). The speed was measured by a photocell system. The subjects practiced the walking speed of 4.0 km/h several times until they were able to walk naturally at the required walking speed.

Fifteen reflective markers were placed on the subjects according to the marker set-up described by Vaughan et al [16]. Five digital video cameras operating at 50 Hz were used to record the movements. Two force platforms (AMTI, OR6-5-1) measured the ground reaction forces, which were sampled at 1000 Hz.

The video sequences were digitised and stored on a computer. Three-dimensional coordinates were reconstructed by direct linear transformation using the Ariel Performance Analysis System (APAS) and 3D joint kinematics were calculated. Joint moments were calculated using an inverse dynamics approach by combining force plate and movement data [16]. In the sagittal plane, positive values represent extensor moments (quadriceps moments) and negative values represent flexor moments (hamstrings and gastrocnemius moments). In the frontal plane, positive values represent adduction moments and negative values represent abduction moments. All joint moments were expressed as a percentage of the subject’s bodyweight (Nm/kg*100).

Kinematic and kinetic data obtained from the leg with painful knee joint were analysed. In the reference group, the right leg was analysed in all subjects. Three valid gait trials were averaged for each subject, pre- and post-injection, respectively, with a valid trial defined as a trial with a walking speed of 4.0 km/h (±0.05 km/h). If a subject performed more than three valid trials, the three trials with walking speeds closest to the target speed (4.0 km/h) were selected for analysis.

To assess the knee joint compressive forces we applied a biomechanical model previously published [17]. The model assesses if the overall knee compression forces are sufficient to balance the net frontal plane moment, thereby keeping the joint closed laterally. The overall knee compression forces is calculated as the vector sum of a) the intersegmental reaction forces resolved along the long axis of the tibia, b) the compression components of the active muscle group forces and c) the axial cruciate ligament forces. The included muscles were the hamstrings, gastrocnemius and quadriceps muscles. The hamstring and gastrocnemius complex constituted a flexor muscle group active when the net sagittal knee joint moment favoured the flexors (i.e. negative) and the quadriceps muscle represented an extensor muscle group active when the net moment favoured extensors (positive). 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 [18].

### Table 1 Knee pain intensity during walking before and after intra-articular lidocaine injections in the OA group

<table>
<thead>
<tr>
<th>OA group (n=9)</th>
<th>Reference (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pre lidocaine</strong> [mean (S.E.M.)]</td>
<td><strong>Post lidocaine</strong> [mean (S.E.M.)]</td>
</tr>
<tr>
<td>Pain (mm)</td>
<td>37.3 (9.3)</td>
</tr>
<tr>
<td>Peak sagittal moment (Nm/kg*100)</td>
<td></td>
</tr>
<tr>
<td>First flexor (Ks1)</td>
<td>–33.9 (9.3)</td>
</tr>
<tr>
<td>First extensor (Ks2)</td>
<td>14.1 (4.4)</td>
</tr>
<tr>
<td>Second flexor (Ks3)</td>
<td>–9.9 (3.9)</td>
</tr>
<tr>
<td>Second extensor (Ks4)</td>
<td>33.3 (4.9)</td>
</tr>
<tr>
<td>Peak frontal moment (Nm/kg*100)</td>
<td></td>
</tr>
<tr>
<td>First adduction</td>
<td>38.2 (4.4)</td>
</tr>
<tr>
<td>Second adduction</td>
<td>24.5 (3.9)</td>
</tr>
<tr>
<td>Lateral tissue tension time (% stance)</td>
<td>36.3 (8.0)</td>
</tr>
</tbody>
</table>

Also group average values of peak sagittal and frontal plane moments during the stance phase of walking (see Fig. 1 for definitions) together with the predicted percentage of stance where lateral tissue tension were needed. The joint compression forces were calculated at the time of the first peak extensor and second peak flexor moment that occurred during the single support phase of the stance phase during walking.

* Significant difference (paired t-test, p<0.05) between pre and post lidocaine.
* Significant difference (unpaired t-test, p<0.05) between the OA group post lidocaine and the reference group.

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The model differed from the previously published, in that the knee joint diameter was obtained from each subject and used for calculation of the position of the tibio-femoral contact point. In the original model the knee joint diameter was fixed at 80 mm for all subjects. The predicted values are presented as a proportion of bodyweight.

2.5. Statistics

Unpaired t-tests were used to compare patients to healthy controls. Paired t-tests were used to compare the pre- and post-injection conditions in the patient group. The level of significance was set to 5%.

3. Results

Average WOMAC scores were 39.6 (S.D.=13.0) for pain, 41.1 (S.D.=14.5) for stiffness, 38.7 (S.D.=13.4) for physical function and 39.1 (S.D.=12.8) for total WOMAC, at the inclusion, i.e. during regular medication washout. These scores indicate mild OA in the patients which is supported by the radiographic classifications. Control subjects had no complaints in their knees equal to 0 in WOMAC.

In the OA group the average knee joint pain decreased significantly after the intra-articular injection of lidocaine ($p=0.005$, Table 1).

The time course pattern of the sagittal knee joint moment during the stance phase was characterized by initial flexor moment followed by oscillations between flexor and extensor moments (Fig. 1). Four peaks, two flexors (Ks1 and Ks3) and two extensors (Ks2 and Ks4), were identified at approximately 10%, 35%, 65% and 90% of the stance phase. The two midstance peaks (Ks2 and Ks3) occurred in the single support phase, whereas the peak moments occurring in the initial and terminal stance phase occurred during double support. In the frontal plane, the time course patterns are characterised by adduction moments throughout the stance phase, with two distinct peaks at approximately 20% and 70% of stance (Fig. 1). The overall shapes of the time course patterns in both sagittal and frontal planes were similar pre- and post-injection as well as between groups. As the joint moment changes occurred during single support, the analysis of joint loads was focused on this phase of the gait cycle.

Following pain relief, the OA group average peak extensor moment in early single support (Ks2 in Fig. 1) decreased significantly by approximately 8 Nm/kg*100 ($p=0.03$) while average peak flexor moment during late single support (Ks3) increased by of approximately 8 Nm/kg*100 in the OA group ($p=0.03$). A summary of the stance phase results is given in Table 1.

At the time of the early single support extensor moment (Ks2 in Fig. 1), the average knee joint angle was 4° more extended following pain relief ($p=0.02$) (Fig. 2), whereas no significant changes were observed in the adduction moment, total compressive force, medial and lateral compartment compression, or the lateral tissue tension. The compressive extensor muscle force was decreased by approximately 20% bodyweight, however, this change did not reach statistical significance ($p=0.055$).

At the time of the late single support flexor moment (Ks3 in Fig. 1), pain relief caused increased average knee joint extension angle ($p=0.007$) and increased total compressive force ($p=0.03$), medial compartment compression ($p=0.004$), and compressive flexor muscle forces ($p=0.04$). No changes were observed in the adduction moment, lateral compartment compression, or lateral tissue tension at the time of the late stance peak flexor moment.
A summary of the predicted joint loads during early and late single support is given in Table 2.

When comparing the OA group before pain relief to the reference group in early single support, the total joint compression and medial compartment compression forces were reduced ($p = 0.025$ and $0.007$, respectively) in the patient group, whereas the remaining variables were similar between groups (Table 2). Following pain relief, the peak extensor moment and the compressive extensor muscle force were decreased in the OA group compared to the reference group ($p = 0.03$ and $0.006$, respectively). The observed differences in total joint and medial compartment compression forces between the OA group before pain relief and the reference group persisted in the comparison between groups following pain relief (see Fig. 3).

In late single support, at the time of peak flexor moment (Ks3), the OA group had significantly more flexed knee joint angles ($p = 0.0005$), less medial compartment compression forces ($p = 0.04$), and lower lateral tissue tension ($p = 0.04$) before pain relief, compared to the reference group (Fig. 4). Following pain relief, no significant differences were observed between groups during late single support; only the knee joint angle and lateral tissue tension tended towards significance (both $p = 0.06$).

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>OA group ($n=9$)</th>
<th>Reference group ($n=10$)</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre lidocaine [mean (S.E.M.)]</td>
<td>Post lidocaine [mean (S.E.M.)]</td>
<td></td>
</tr>
<tr>
<td><strong>Early single support phase (Ks2)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total joint compression (bodyweight)</td>
<td>1.8 (0.2)</td>
<td>1.7 (0.1)</td>
<td>2.4 (0.2)</td>
</tr>
<tr>
<td>Medial compartment compression (bodyweight)</td>
<td>1.7 (0.2)</td>
<td>1.7 (0.1)</td>
<td>2.5 (0.2)</td>
</tr>
<tr>
<td>Lateral compartment compression (bodyweight)</td>
<td>0.2 (0.1)</td>
<td>0.1 (0.1)</td>
<td>0.1 (0.1)</td>
</tr>
<tr>
<td>Extensor muscle compression (bodyweight)</td>
<td>0.5 (0.1)</td>
<td>0.3 (0.1)</td>
<td>0.8 (0.2)</td>
</tr>
<tr>
<td>Axial joint reaction force (bodyweight)</td>
<td>0.9 (0.1)</td>
<td>0.9 (0.1)</td>
<td>1.0 (0.1)</td>
</tr>
<tr>
<td>Lateral tissue tension (bodyweight)</td>
<td>0.1 (0.1)</td>
<td>0.1 (0.1)</td>
<td>0.2 (0.1)</td>
</tr>
<tr>
<td>Joint angle (deg)</td>
<td>20.7 (2.3)</td>
<td>16.7 (2.0)</td>
<td>19.2 (1.4)</td>
</tr>
<tr>
<td><strong>Late single support phase (Ks3)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total joint compression (bodyweight)</td>
<td>1.6 (0.2)</td>
<td>1.9 (0.2)</td>
<td>1.9 (0.3)</td>
</tr>
<tr>
<td>Medial compartment compression (bodyweight)</td>
<td>1.4 (0.1)</td>
<td>1.6 (0.2)</td>
<td>1.8 (0.2)</td>
</tr>
<tr>
<td>Lateral compartment compression (bodyweight)</td>
<td>0.2 (0.1)</td>
<td>0.3 (0.1)</td>
<td>0.2 (0.1)</td>
</tr>
<tr>
<td>Flexor muscle compression (bodyweight)</td>
<td>0.5 (0.2)</td>
<td>0.8 (0.2)</td>
<td>0.6 (0.3)</td>
</tr>
<tr>
<td>Axial joint reaction force (bodyweight)</td>
<td>0.9 (0.1)</td>
<td>0.9 (0.1)</td>
<td>0.9 (0.1)</td>
</tr>
<tr>
<td>Lateral tissue tension (bodyweight)</td>
<td>0.1 (0.1)</td>
<td>0.1 (0.1)</td>
<td>0.2 (0.1)</td>
</tr>
<tr>
<td>Joint angle (deg)</td>
<td>13.0 (1.9)</td>
<td>8.8 (2.4)</td>
<td>3.5 (1.2)</td>
</tr>
</tbody>
</table>

* Significant difference (unpaired t-test, $p<0.05$) between the OA group pre lidocaine and the reference group.

** Significant difference (unpaired t-test, $p<0.05$) between the OA group post lidocaine and the reference group.

*** Significant difference (paired t-test, $p<0.05$) between pre and post lidocaine.

### Early Single Support Knee Forces

![Early Single Support Knee Forces](image_url)

Fig. 3. Group averages ($±$1 S.E.M.) values of knee joint compressive forces during the early single support phase (at the time of Ks2 – see Fig. 1 – the peak sagittal plane extensor moment). Asterisks (*) denote significant differences ($p<0.05$).

### Late Single Support Knee Forces

![Late Single Support Knee Forces](image_url)

Fig. 4. Group averages ($±$1 S.E.M.) values of knee joint compressive forces during the late single support phase (at the time of Ks3 – see Fig. 1 – the peak sagittal plane flexor moment). Asterisks (*) denote significant differences ($p<0.05$).
4. Discussion

Local knee joint analgesia in the OA group resulted in a shift in the knee flexor–extensor joint moments during single support towards less extensor and more flexor moments, with subsequently increases in joint loads in late single support. Also, significantly less flexed knee joint angles were observed. No changes in the abduction–adduction moments occurred following pain relief. The changes in joint moments caused by pain relief, all occurred during single support and we focused our analysis to this part of the gait cycle. We chose to assess the predicted joint forces at the times of peak sagittal plane moments (Ks2 and Ks3) as the sagittal plane moments are determinants of joint loads. Additionally, it was assumed that the joint loads would be largest and pain relief would have a more profound effect on the loads in the single support. This is a valid assumption as no compensatory unloading strategies involving the swinging leg are possible.

Following pain relief, the patients walked with knee joint angles comparable to those of the reference group in late single support (Fig. 4). At heel strike and during early single support, the patients walked with more extended knees following pain relief. The knee joint angles can at least partially explain the reduced extensor moments, as it has been shown that at more extended knee joint angles, the extensor muscles can exert less extensor moments [19].

Reduced knee extensor moments are traditionally interpreted as unloading of the knee joint, as higher extensor moments contribute to higher overall compressive forces in the knee joint [17]. However, this study demonstrates that both total joint and medial compartment compressive forces were unaffected by the decreases in the extensor moments and the muscular compressive forces. There are two possible sources for this paradoxical observation: It could either be a result of antagonistic activity of the knee flexors or increased reaction forces. As the axial reaction forces were unchanged following pain relief, antagonistic muscle activity must account for the unchanged joint loads in early single support. However, the model applied in this study does not allow antagonistic muscle activity and therefore we cannot determine antagonist muscle compression forces. Both total joint and medial compartment compression forces increased in late single support following pain relief. This is primarily caused by the increased flexor muscle moments and forces.

Increased knee joint loads are believed to contribute to the development and progression of knee OA [3,4] and increased adduction moments are determinant of loads in the medial compartment of the knee [17]. Additionally, it has been shown that increases in the adduction moment are associated with increased risks of loosing joint space [3]. Although we did not observe changes in the adduction moment, the medial compartment loads increased following pain relief. It is therefore indicated that pain does exert a protective action and pain relief can have potentially detrimental consequences.

In a study of 4 weeks of NSAID pain treatment of knee OA patients, increased extensor moments and knee joint flexion angles were observed [20]. This is in contrast to the present study, however, the NSAID treatment also brought about overall increases in walking speeds, which per se will result in increased knee joint extensor moments and flexion angles [21]. The present study has the advantage of having equal walking speeds between groups and pain conditions. The prolonged pain relief caused by NSAID treatment differs from the short lasting effect of the lidocaine injections: the lidocaine abolishes the pain in practically all subjects, while NSAID has a more varying effect across subjects. Also, the variation of OA during a month is rather unpredictable, while in our patients the pain relief of lidocaine was confined to a few hours. In a study of combined lidocaine and steroid injected intra-articularly, no changes in the extensor moments or joint angles but increased adduction moments and walking speeds were reported [22] and it seems that selection of the patients may have great importance for the outcome.

Knee joint effusion affects the neuromuscular function of the knee joint. This has been shown in a study of experimental joint effusion in healthy subjects where reduced peak extensor moments during walking were observed [23]. This is in line with the present results. Similarly, knee joint effusion inhibits the maximal voluntary quadriceps contraction (MVC) capacity [24,25]. However, our patients had no effusion in the knees, as confirmed by the ultrasound examination. Introducing lidocaine intra-articularly could cause effusion derived quadriceps inhibition. However, it has been shown that quadriceps inhibition caused by experimental joint effusion disappears when injecting lidocaine prior to saline infusions [25]. Thus, quadriceps muscle function seems to be preserved following intra-articular lidocaine injections and the observed effects in the present study were, presumably, caused by the pain relieving effect – and not a general nerve block – of lidocaine. Furthermore, the increased joint load was caused by increased flexor muscle forces and it is unknown whether hamstring and gastrocnemius are influenced by joint effusions.

While we tested only small sample sizes, our results indicate that possible differences in joint loads are of clinical relevance. We calculated the sample size needed to detect the present results with a power of β=90% and α=5%, and found that 8 pairs of observations would be sufficient. Therefore it is concluded that the results from the present study are valid.

5. Conclusion

We conclude that pain relief in painful mild medial knee OA resulted in increased joint loads during the late single support phase during walking. Although the patients walked with less compressive knee joint forces compared to the reference group, the effects of pain relief caused increased joint loads which may facilitate the progression of the disease.

Acknowledgments

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