
Abstract

**Objective**: To test the hypothesis that knee effusion presence in those with knee osteoarthritis alters knee joint muscle activation patterns and sagittal plane mechanics during gait. **Methods**: 35 patients with medial compartment knee osteoarthritis were assessed for the presence of effusion using a brush test. Based on the results, they were assigned to the knee effusion (n=17) and no knee effusion (n=18) groups. Electromyograms from seven lower extremity muscles (lateral and medial gastrocnemius, vastus lateralis and medialis, rectus femoris and the lateral and medial hamstrings), leg motion and ground reaction forces were recorded during self-selected walking. Isometric knee extensor, plantarflexor and knee flexor strength was measured. Discrete measures from angular knee motion and net external moment of force waveforms were identified. Principal component analysis extracted electromyographic waveform features. Analysis of variance models tested for main effects (group, muscle) and interactions (α=0.05). Bonferonni post hoc testing was employed. **Results**: No differences in age, body mass index, knee pain, Western Ontario McMaster Osteoarthritis Index scores, gait velocity and muscle strength were found between groups (p>0.05). Individuals with effusion had a greater overall quadriceps activation and prolonged hamstring activation into mid-stance (p<0.05). Knee joint flexion angles were higher (p<0.05) and net external knee extension moments in mid to late stance lower in the effusion group. **Conclusion**: Quadriceps and hamstrings activation during walking were altered when effusions were present. Increased knee flexion angles and decreased knee extension moment in mid-late stance
provides a mechanical explanation for the effect of joint effusion on muscle activation in those with knee OA.

**Key Words:**

Knee Osteoarthritis, Electromyography, Effusion, Biomechanics, Principal Component Analysis, Muscle activation, Muscle Strength
Introduction

Knee osteoarthritis (OA) is a major contributor to chronic morbidity in older adults causing significant activity limitations. Gait analysis has served as a good model to quantify these limitations where altered knee joint biomechanics and muscle activation differences are found in comparison to asymptomatic cohorts and with increasing OA severity.

A variety of factors have been suggested to explain altered muscle activation during gait in those with medial compartment knee OA compared to similarly aged asymptomatic individuals. Generally, mechanical factors including tibial adduction features during stance, laxity, osteophytosis, strength, gait velocity, and compressive loading have been provided. Knee joint effusion, a common clinical finding, has not been examined as a potential contributor. Knee effusions are easily measured with clinical tests, having moderate agreement with ultrasonography and good reliability. As well, effusions can be readily modifiable.

Much of our understanding about knee joint effusion and muscle activation alterations is based on acute effusion models in asymptomatic subjects. Static experimental studies have found that acute knee effusions reduce the Hoffmann reflex response amplitude, supporting a spinal mechanism of quadriceps alpha motor neuron inhibition. Pacinian corpuscles, golgi joint receptors and ruffini endings may provide an inhibitory signal in response to effusion related joint distension. Quadriceps force generating capacity was consistently reduced during acute experimental studies,
independent of pain stimulus, suggesting that knee joint mechanics are affected by this neurophysiological mechanism.

Supporting static experimental work, acute experimental effusions were found to alter knee joint mechanics and lower quadriceps activation during functional tasks in otherwise healthy individuals. During gait, Torry et al. reported a reduction in mean quadriceps and increased mean hamstrings activation during early to mid-stance. The higher hamstring activation amplitudes found by Torry et al. are consistent with the elevated activity reported in knee OA compared to asymptomatic individuals, but quadriceps activity tends to be higher in moderate knee OA. Altered sagittal plane motion, and net external knee moments were also reported and correspond with those reported for knee OA gait compared to an asymptomatic cohort.

The effect of effusion on quadriceps inhibition during static testing was more variable in those with chronic effusions; an effusion state more representative of that found in OA. While evidence supports that inhibition was associated with chronic effusion, results are equivocal. In addition, minimal inhibition has been found in individuals with moderate knee OA during maximal voluntary quadriceps activation compared to an asymptomatic cohort. In summary, most of what is known about knee joint effusion, joint mechanics and muscle inhibition is based on acute effusion studies. Whether effusion could provide a mechanism for altered mechanics and muscle activation in individuals with moderate knee OA is the fundamental question of the present study.

Altered mechanics and muscle activity associated with knee effusion could impact normal knee joint function while walking. Increased muscle activity could impact joint
loading \(^{21,37}\) and metabolic demand \(^{9}\), whereas reduced activity could affect active stabilization during gait. Reduced knee flexion angles and net external knee flexion moments in early stance, as shown for healthy individuals with an acute effusion \(^{43}\), may alter impact load attenuation.

Collectively the above literature provides the rationale for testing the main hypothesis that differences in knee joint muscle activation patterns and joint mechanics will exist in individuals with knee OA who have effusion compared to those without effusions during walking. The changes are expected to be consistent with those found for acute effusion models in asymptomatic individuals, and may help to explain some of the key differences in joint mechanics and muscle activation patterns reported for those with knee OA.

**Materials and Methods**

**Participants**

Participants with unilateral symptomatic knee OA (n=40) were prospectively recruited from the caseload of one orthopedic surgeon based on clinical diagnosis of knee OA made using American College of Rheumatology guidelines \(^1\). Knee radiographs were graded using the KL ordinal radiographic scale \(^{16}\) by a single, experienced reader (WDS) as recommended by Vignon et al., \(^{46}\). The reader was blinded to participant identification and gait analysis outcomes. Individuals with lateral compartment joint space narrowing greater than medial compartment joint space narrowing were excluded as the focus was
on predominant medial compartment OA. All participants were required to meet a radiographic (Kellgren-Lawrence (KL) I-III) and functional status criterion (required to safely walk one city block, reciprocally ascend and descend 10 stairs and jog five meters), consistent with a moderate OA classification \(^{14}\). Five individuals did not meet the KL I-III criterion. Participants were not scheduled for total knee arthroplasty at time of testing. All participants were required to have no fracture or previous lower extremity injury other than a sprain or strain (i.e. no ruptured anterior cruciate ligament).

Participants were required to have no cardiovascular/respiratory disease, neurological disorders, or musculoskeletal disorders other than their knee OA that affected their ability to safely complete the data collection protocol (i.e. stroke, Parkinson’s disease, myocardial infarct, arrhythmias). Written informed consent was obtained in accordance with the Institutional Research Ethics Board.

**Procedures**

The Western Ontario McMaster Osteoarthritis Index (WOMAC-LK3.1) was completed by all participants and height and mass were recorded. The most symptomatic knee was examined and prepared for testing. Current level of knee pain was quantified using a verbal scale (0 indicating no pain, 10 indicating extreme pain). Knee effusion was assessed by an experienced orthopaedic physiotherapist using the bulge test/sign \(^{23}\). The bulge sign has been shown to be highly reliable (\(R_c=0.97\)) \(^6\) for detecting effusion/no effusion and substantial agreement between raters to detect grade of effusion has been
found (Cohens Kappa = 0.61) \(^2\). Participants were assigned to effusion or no effusion group based on outcomes of this test prior to preparation for gait analysis.

Standard procedures previously reported for surface electromyography \(^4\) (EMG) were used to measure muscle activity during gait, consistent with current guidelines as suggested by the International Society of Electrophysiology and Kinesiology and SENIAM (Surface EMG for the Non-Invasive Assessment of Muscles) \(^36\). Skin preparation included light shaving and abrading with 70% alcohol wipes. Surface electrodes (Ag/AgCl, Medi-Trace 133 electrodes, Covidien, Mansfield, MA), were placed in a bipolar configuration over lateral (LG) and medial gastrocnemius (MG), vastus medialis (VM) and lateralis (VL), rectus femoris (RF), semitendinosis/membranosus (MH) and biceps femoris (LH). Muscle palpation and a series of isometric contractions for specific muscle groups were used for signal validation and gain adjustment. Signals were amplified using an AMT-8 (Bortec, Inc., Calgary, Alberta, Canada), eight-channel EMG system (Input Impedance: \(~10G\Omega\), CMRR:115dB at 60 Hz, Band-pass (10-1000 Hz)).

Infrared emitting diode (IRED) skin surface markers were affixed to the lateral lower extremity. Triangular sets of IRED markers were secured to the pelvis, femur, tibia and foot. Single IRED markers were placed on the lateral malleolus, lateral epicondyle and greater trochanter of the femur and lateral aspect of the shoulder. After a standing calibration trial, digitization of eight virtual points on predefined anatomical landmarks was completed, including right and left anterior superior iliac spines, medial epicondyle of the femur, fibular head, tibial tuberosity, medial malleolus, base of the second metatarsal and center of the posterior calcaneous \(^18\).
Data Acquisition

Three-dimensional lower extremity motion during gait was recorded at 100Hz using two optoelectronic motion analysis sensors (Optotrak 3020™, Northern Digital Inc., Waterloo, ON, Canada). Three-dimensional ground reaction forces were collected from a single force plate (AMTI™, Advanced Mechanical Technology Incorporation, Newton, MA, USA) that was aligned to global motion capture system coordinates.

After three familiarization trials, participants were instructed to complete at least five walking trials at a consistent self-selected velocity along a six-meter walkway. Trials were included if they were within 10% of their self-selected speed. Following the walking trials, muscle activity was recorded during supine lying (resting bias). For EMG amplitude normalization, participants completed at least one practice and two standardized three-second maximal voluntary isometric contraction (MVIC) trials of i) knee extension at 45°, ii) knee extension at 15°, iii) knee flexion at 55°, iv) knee flexion at 15°, v) knee extension-hip flexion at 45°, vi) prone knee flexion at 55°, vii) sitting plantarflexion viii) standing unilateral plantar flexion 14. Torque output during exercises one to four, six and seven were collected using a Cybex™ Isokinetic dynamometer (Lumex, NY, USA). A 60-second rest period separated each contraction, and standardized verbal encouragement was given 20. All force plate, EMG and torque signals were analog to digitally converted at 1000Hz (16bit, +/- 2V) using the analogue data capture feature of the Optotrak™ system and stored for processing.
Data Processing

Raw electromyographic signals were processed through custom MatLab™ Ver 7.1 programs (The Mathworks Inc., Natick, Massachusetts, USA). All signals (gait and MVIC trials) were corrected for resting bias and converted to micro-volts, full wave rectified, low-pass filtered (Butterworth, 4th order, Fc-6Hz). Maximum EMG amplitudes for each muscle during MVIC exercises were calculated by using a 100 ms moving-average window (99 ms overlap)\(^\text{14}\). Maximum EMG amplitudes, regardless of MVIC exercise in which it occurred, were used for amplitude normalization of gait EMG waveforms (% MVIC). Isometric torque values were corrected for gravity. Maximum torque for each exercise was identified using a 500 ms moving-average window (0ms overlap)\(^\text{14}\). The average of two trials was recorded as muscle strength in Newton-meters and normalized to body mass (Nm/kg).

Technical and local anatomical bone embedded coordinate systems for the pelvis, thigh, tibia and foot were derived from IRED markers and digitized points. Joint angles were specified through Cardan/Euler rotations (z-y-x sequence), with sagittal plane rotation of the tibia relative to femur occurring about the medial:lateral axis of the femur (z)\(^\text{18}\). Net external sagittal plane knee moments were calculated using an inverse dynamics model which combined ground reaction force and moment data, limb kinematics, limb anthropometrics and inertial properties\(^\text{45}\) and normalized to body mass (Nm/kg). Electromyograms, sagittal plane knee angles and net external moments were time normalized to 100% of the gait cycle using a cubic spline interpolation technique, representing one complete gait cycle. First heel strike was defined by a five Newton
increase in vertical ground reaction force, where second heel strike was determined using a kinematic event obtained from lateral malleolus\textsuperscript{18}. Ensemble averages were calculated from at least five walking trials. Four trials were used in one individual.

**Analysis**

WOMAC scores were tabulated. Discrete sagittal plane knee angles were determined for i) heel strike, ii) early stance maximum (0-30\% of gait cycle) and iii) mid to late stance minimum (30-60\% of gait cycle). From the net external sagittal plane knee moment waveforms, early stance maximum (0-30\% of gait cycle) and mid to late stance minimum (30-60\% of gait cycle) moments were determined.

Principal component analysis (PCA) was used to capture amplitude and temporal electromyographic waveform features using custom MatLab™ Ver.7.1 programs (The Mathworks Inc., Natick, Massachusetts, USA). This multivariate statistical technique has been previously described in detail\textsuperscript{14,33}. Briefly, three separate analyses were performed, one for each muscle grouping (quadriceps (VL, VM, RF), hamstrings (LH, MH) and gastrocnemii (LG, MG)). For each muscle grouping a matrix of original waveform data was generated [X]. An eigenvector decomposition of the cross product matrix ([S] = [X]\textsuperscript{t} x [X]) was performed, using standard notation U’SU= L, yielding predominant orthonormal components called eigenvectors. These eigenvectors (U) are refereed to as principal patterns (PP) in this manuscript. A percent trace was calculated to determine how much variability was contained in each principal pattern. Principal patterns explaining the greatest percent of variation (>90\% total variation) in the waveforms were
retained and referred to as PP1, PP2 etc. Principal pattern scores (PP-scores) were computed for individual gait waveforms in each separate analysis (PP-score =\([X]\times[PP]\)). PP-scores were utilized for statistical hypothesis testing.

**Statistical Analysis**

Student t-tests were used to test for significant differences in pain, WOMAC, age, mass, height, body mass index (BMI), stride characteristics, strength and measures of sagittal plane knee joint angles and moments between effusion and no effusion groups. Differences in Kellgren-Lawrence radiographic grades between groups were determined using a Mann-Whitney U-test. Normality and equal variance of the PP-scores were determined from Kolmogorov-Smirnov and Levene’s tests, respectively. A two-factor mixed model ANOVA tested for significant group (between) and muscle (within) main effects and interactions (alpha=0.05). Participants were the only random factor in the ANOVA models. Post-hoc testing was employed for determining pair-wise significant findings using Bonferronni adjusted alpha levels. Statistical procedures were completed in Minitab™ Ver.16 (Minitab Inc. State College, PA, USA).

**Results**

No significant differences were found (p>0.05) between effusion and no effusion groups for characteristics in Table 1.
Table 1: Mean and standard deviation (SD) subject demographics, gait characteristics, knee joint strength and range of motion characteristics and WOMAC scores.

<table>
<thead>
<tr>
<th></th>
<th>No Effusion</th>
<th>Effusion</th>
<th>P-Value</th>
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<tbody>
<tr>
<td>N</td>
<td>18</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td># males</td>
<td>14</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>54 (8)</td>
<td>57 (8)</td>
<td>0.264</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>92.7 (17.9)</td>
<td>96.4 (16.9)</td>
<td>0.542</td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td>30.2 (5.0)</td>
<td>31.9 (5.3)</td>
<td>0.340</td>
</tr>
<tr>
<td>Pain (# / 10)</td>
<td>1.4 (1.9)</td>
<td>1.3 (1.8)</td>
<td>0.843</td>
</tr>
<tr>
<td>Gait Velocity (m/s)</td>
<td>1.26 (0.19)</td>
<td>1.28 (0.12)</td>
<td>0.758</td>
</tr>
<tr>
<td>Stride Length (m)</td>
<td>1.42 (0.18)</td>
<td>1.43 (0.11)</td>
<td>0.838</td>
</tr>
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</table>

**Strength (Nm)**

<table>
<thead>
<tr>
<th></th>
<th>No Effusion</th>
<th>Effusion</th>
<th>P-Value</th>
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<tbody>
<tr>
<td>KE45°</td>
<td>1.35 (0.48)</td>
<td>1.39 (0.36)</td>
<td>0.780</td>
</tr>
<tr>
<td>KE15°</td>
<td>0.90 (0.33)</td>
<td>0.86 (0.29)</td>
<td>0.659</td>
</tr>
<tr>
<td>KF55°</td>
<td>0.71 (0.29)</td>
<td>0.70 (0.24)</td>
<td>0.876</td>
</tr>
<tr>
<td>KF15°</td>
<td>0.60 (0.22)</td>
<td>0.51 (0.15)</td>
<td>0.212</td>
</tr>
<tr>
<td>PF</td>
<td>1.16 (0.38)</td>
<td>1.01 (0.34)</td>
<td>0.237</td>
</tr>
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</table>

**WOMAC**

<table>
<thead>
<tr>
<th></th>
<th>No Effusion</th>
<th>Effusion</th>
<th>P-Value</th>
</tr>
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<tbody>
<tr>
<td>Pain</td>
<td>6.2 (3.0)</td>
<td>5.4 (2.2)</td>
<td>0.405</td>
</tr>
<tr>
<td>Stiffness</td>
<td>3.4 (1.5)</td>
<td>3.4 (1.6)</td>
<td>0.978</td>
</tr>
<tr>
<td>Physical Function</td>
<td>19.7 (9.8)</td>
<td>17.2 (7.7)</td>
<td>0.422</td>
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KL-scores (median) ^

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^ Radiographs were not available for three subjects in the effusion group and two subjects in the no effusion group at the time of scoring. (KE – Knee extension, KF – Knee Flexion, PF – Plantar flexion)

Sagittal plane knee motion and net external moment of force waveforms are shown (Figure 1). Mean of the discrete measures extracted from these waveforms, 95% confidence intervals and p-values are found in Table 2. No significant differences were found for angles at heel strike (p=0.982). Early (~6° difference, p=0.016) and mid to late stance (~8° difference, p=0.001) knee flexion angles were greater in individuals with effusion. The mid to late stance net external extension moment was 51% less in individuals with effusion (p=0.017).
Table 2: Mean [95% CI] and standard deviation (SD) absolute angles and difference values for sagittal plane knee motion and moments of force.

<table>
<thead>
<tr>
<th>Measure</th>
<th>No Effusion</th>
<th>Effusion</th>
<th>Diff.</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Absolute Angles</strong> (degrees of knee flexion)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Heel Strike</td>
<td>-5.3 [-8.5, -2.0]</td>
<td>-5.3 [-8.6, 2.1]</td>
<td>0.0</td>
<td>0.982</td>
</tr>
<tr>
<td></td>
<td>(6.4)</td>
<td>(5.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early Stance Maximum</td>
<td>12.7 [8.8, 16.7]</td>
<td>18.6 [15.9, 21.3]</td>
<td>5.9</td>
<td>0.016</td>
</tr>
<tr>
<td></td>
<td>(7.9)</td>
<td>(5.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid to Late Stance Minimum</td>
<td>2.5 [0.1, 4.8]</td>
<td>10.0 [6.3, 13.6]</td>
<td>7.5</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>(4.6)</td>
<td>(7.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Moments of Force</strong> (Nm/kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Flexion</td>
<td>0.24 [0.09, 0.39]</td>
<td>0.31 [0.19, 0.43]</td>
<td>0.07</td>
<td>0.449</td>
</tr>
<tr>
<td></td>
<td>(0.31)</td>
<td>(0.23)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Late Stance Extension</td>
<td>-0.37 [-0.46, -0.29]</td>
<td>-0.19 [-0.31, -0.08]</td>
<td>0.18</td>
<td>0.017</td>
</tr>
<tr>
<td></td>
<td>(0.19)</td>
<td>(0.22)</td>
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</table>

**Bolded** p-values are significant at p<0.05

Ensemble average electromyograms for the two groups are shown in Figure 2. Differences occurred throughout the gait cycle and were not consistent between or amongst muscles. For each muscle group, three features were identified, which together explained greater than 90% of the original waveform variability. Principal pattern descriptions and ANOVA statistical results are found in Table 3 along with \(PP\)-scores.

Three principal patterns captured over 95% of the gastrocnemius waveform variability. Unequal variance and non-normality were apparent in the \(PP2\)-scores and therefore data were transformed. No significant group differences (p>0.05) were identified for each of the three patterns. A significant muscle main effect for the phase shift pattern was found (p=0.002) where MG had greater scores than LG indicating an earlier increase in activation for MG compared to LG.
Ninety-six percent of the quadriceps waveform variability was accounted for by three principal patterns. Unequal variance and non-normality were apparent in the PP-scores and therefore data were transformed. A significant group x muscle interaction was found for the transformed PP1-scores (p=0.048) as indicated in Table 3. Individuals in the effusion group had greater vastus lateralis and medialis PP1-scores compared to those without, indicating higher overall amplitude as illustrated in Figure 2. No other group differences were found.

Three principal patterns captured over 95% of the hamstrings waveform variability. Unequal variance and non-normality were apparent in the PP1 and PP2-scores and data were transformed. A significant muscle main effect was found for the transformed PP1-scores (overall magnitude) and PP2-scores (prolonged stance phase activity) where, LH had greater amplitudes (p<0.001) and prolonged activation (p=0.023).
compared to MH (Table 3 and Figure 2). *PP2-scores* were greater in individuals with effusion compared to those without effusion (*p*=0.031) indicating prolonged hamstring activation during early to mid-stance for the effusion group as illustrated in Figure 2.

**Discussion**

Individuals with moderate knee OA and effusion walked with altered knee joint motion, moments of force and knee joint muscle activation characteristics but only specific features were significantly different. Individuals with effusion walked with a more flexed knee during stance with these absolute differences greater than previously found between asymptomatic individuals and individuals with moderate knee OA.\(^3\) Greater overall quadriceps activity, and prolonged hamstrings activity during mid-stance stance were also found in the effusion group. These results occurred despite group similarities in strength, WOMAC and verbal pain ratings during data collection, subject anthropometrics, KL–grades and walking velocity.

Consistent with Torry *et al.*,\(^4\) the only other comparator gait study, the effusion group walked with more knee flexion during stance (Figure 1). This greater knee flexion was associated with a lower net external extension moment in mid to late stance in individuals with effusion, also similar to Torry *et al.*,\(^4\) In contrast to Torry *et al.*,\(^4\) the maximum early stance net knee flexion moment was not different between the two OA groups in the current study. They reported a decreased early stance flexion moment even for the lowest level of effusion (20cm\(^3\))\(^4\). This decreased moment was thought to reflect a quadriceps avoidance gait and was confirmed by a reduction in quadriceps activation
amplitudes. Others reported a more extended knee position, and quadriceps inhibition with acute effusions during drop jump landing. While Torry et al., reported that increasing levels of effusion did not generate a pain response, it was not clear whether walking velocity differed between conditions. Early stance net knee flexion moments and quadriceps activity amplitudes can be reduced by decreased walking velocity, making it difficult to ascertain the effect of knee joint effusion alone. In the current study, pain and walking velocity did not differ between the two OA groups, nor did early stance net knee flexion moments.

Higher quadriceps activity was found in individuals with effusion despite comparable strength, mass, gait velocity, symptoms and KL-grades between groups. In contrast to previous acute effusion work, where vastii activity was significantly reduced during the first 50% of stance, elevated activity seen in the current study could reflect the stability demands secondary to altered joint mechanics. Inhibitory influence on quadriceps muscle strength is more variable in non-acute effusion studies, supporting that quadriceps inhibition secondary to knee effusion does not completely explain the increased quadriceps activity.

Knee joint muscle forces, particularly the quadriceps, contribute to sagittal and frontal plane control of external moments during stance. In addition, it is estimated that hamstrings co-activation during stance assists to maintain knee joint stability. Cho et al., who found that joint effusion impairs knee joint proprioceptive function in individuals with OA. Simkin et al., provided a theoretical rational, based on available evidence, to support that effusions reduce stabilizing features of the sub-atmospheric intra-articular pressure in synovial joints. This may influence proprioception, explain
findings of Cho et al., 5 and provide a plausible explanation for elevated quadriceps activity found from early stance to late swing in the current study.

Elevated quadriceps activity continued into mid-stance, where prolonged hamstrings activation was found (Figure 2). This finding was consistent with previously reported differences between those with moderate knee OA compared to aged matched asymptomatic cohort 33,34. However, the no effusion group had patterns that were closer in shape and magnitude to asymptomatic controls than the effusion group 34. The current results suggest that an effusion effect is occurring. This prolonged hamstring activity partially corroborates the findings of Torry et al., 43 for acute effusion. Higher quadriceps activity and hamstrings co-activation in individuals with effusion during mid-stance can be partially explained as a response to increased joint stability demands associated with greater knee flexion. Reasons for this flexed position are not completely clear and have not been generally reported for individuals with knee OA 3. Higher intra-articular knee pressures were reported when the knee joint was extended in comparison to mid-range knee flexion angles of approximately 20-40 degrees 47. In addition, joint stiffness for both varus/valgus bending and tibial torsion was increased as the knee joint was extended 24 suggesting knee flexion can minimize the stabilization characteristics that ligamentous structures provide. Attempts to minimize higher intra-articular knee pressures provide a possible explanation for why individuals with effusion adopted a knee flexed position during stance in the current study and in response to an acute effusion 43.

Higher levels of muscle co-activity, while contributing to joint stability can however lead to altered joint loading 21,37, metabolic cost 9, and potentially increased intra-articular pressures 26 and muscular fatigue. It is estimated that healthy older adults
take up to 8500 steps/day where individuals with disabilities and chronic illnesses can take up to 5500 steps/day. Increases in muscle activation during walking might have implications for cumulative joint loading and fatigue.

In contrast to alterations in quadriceps and hamstrings activation, gastrocnemii were not affected by effusion in this cohort. Phase shifted activity found between medial and lateral gastrocnemii concurred with previous reports from age matched asymptomatic individuals and individuals with moderate knee OA. While facilitatory soleus activity has been shown to occur with acute distension of the knee joint, thought to be a possible compensation for inhibited quadriceps, knee joint mechanics and effusion have minimal effect on gastrocnemii activation during gait in those with moderate knee OA.

A number of limitations need to be considered when interpreting the findings of this cross-sectional study. Lower activity during MVIC exercises could explain overall higher quadriceps activity in individuals with effusion during walking. While plausible, a number of reasons challenge this explanation. For these standardized exercises, comparable quadriceps strength was found (Table 1) between effusion and no effusion groups. Maximal levels of activity for normalization were obtained from exercises that positioned the leg differently from earlier studies on inhibition and pain levels were not increased during testing and were similar between groups. Furthermore, while acknowledging limitations of reporting raw EMG amplitudes, these values were similar between the two groups for MVIC exercises (not shown). This was consistent with studies that have shown minimal inhibition in individuals with moderate knee OA during maximal voluntary quadriceps activation compared to an asymptomatic cohort. Furthermore, minimal inhibition was reported to occur as a result of effusions in
individuals with arthritis \cite{15,26}, so a different mechanism from the acute effusion group is plausible. While the possibility still exists that a maximal effort contraction during normalization exercises was not achieved in the effusion group because of inhibition, this was not likely the main mechanism for increased quadriceps activity during gait.

Secondly, the bulge sign provided a dichotomous evaluation of knee effusion and previous work demonstrated reliability of this test and moderate agreement with ultrasonography \cite{6,11,42}. However, there is potential for false negative tests. Individuals classified as not having effusion may have had a small or conversely a very large effusion not detectable using the bulge sign. Current findings would thus be considered a conservative estimate of the difference. Using an instrument with improved sensitivity and specificity could determine whether a dose response exists.

As with any cross-sectional comparative study, there are limitations with drawing conclusions regarding a causal effect of the independent variable. Other factors could confound these results that we did not investigate during gait including knee joint laxity and/or foot and trunk mechanics. Future longitudinal studies or studies where effusion is evoked or aspirated may provide further insight. However, the strength of the present study is the similarity between groups for variables known to alter joint mechanics and muscle activation. Structural impairment similarities were particularly important since effusion was more prevalent in those with higher KL-grades \cite{17} and muscle activation pattern differences have been related to knee OA severity difference \cite{4,34}. Furthermore, while impairments to other joint structures not detected using the KL-grade (i.e. meniscus) could be present, possibly influencing gait mechanics and muscle activation independently of knee effusion, the exclusion criteria minimized that potential.
In summary, this was the first study to examine whether individuals with moderate knee OA and knee joint effusion walk with altered gait mechanics and muscle activation patterns. Effusion duration could not be confirmed, but radiographic evidence suggests long-standing knee OA and hence the assumption that effusion was not the result of a single episode. Some of the gait mechanics and muscle activation effects differed from acute effusion models. While the latter has more experimental control, differences suggest that the acute effusion model does not provide an accurate picture of the alterations found in those with knee OA.

**Conclusion**

Differences in sagittal plane knee mechanics and muscle activation patterns were found in individuals with knee OA and knee joint effusion compared to those with no knee effusion. The knee remained more flexed during stance. The mid to late stance net external knee extension moment was less in individuals with effusion. Higher quadriceps and prolonged hamstrings activity during mid-stance were found with no effect on gastrocnemii. These alterations to joint mechanics and muscle activations have implications for long-term joint function in individuals with moderate knee OA and support that clinical management should address effusion.

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**Contributions**
All authors made substantial contributions to conception and study design, analysis and interpretation of data, drafting the manuscript, editing for important intellectual content and preparing for submission to Osteoarthritis and Cartilage.

Conflict of Interest

The authors acknowledge that there are no conflicts of interest pertaining to this manuscript.

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Figures

**Figure 1:** A) Ensemble-averaged sagittal plane knee angles. A positive value indicates knee flexion. B) Net external sagittal plane moments of force for individuals. A positive value indicates a net external knee flexion moment and a negative value indicates a net external extension moment. Knee effusion (dotted) and without knee effusion (solid).
Figure 2: Ensemble-averaged electromyograms of A) lateral gastrocnemius, B) medial gastrocnemius, C) vastus lateralis, D) vastus medialis, E) rectus femoris, F) lateral hamstrings, G) medial hamstrings for individuals with effusion (dotted) and without knee effusion (solid). Percent MVIC is on the y-axis and percent of gait cycle on the x-axis.