



Hip joint motion and gluteal muscle activation differences between healthy controls and those with varying degrees of hip osteoarthritis during walking



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ABSTRACT

Purpose: Compare gluteal muscle activation patterns and three-dimensional hip joint movements among those with severe hip osteoarthritis (OA), moderate OA and a healthy group during walking. **Scope:** 20 individuals with severe OA, 20 with moderate OA and 20 healthy individuals were recruited. Three-dimensional hip motion and surface electromyograms from gluteus maximus and medius were collected during treadmill walking at a self-selected speed. Angular displacement characteristics were calculated for three-dimensional hip motions. Principal component analysis extracted amplitude and temporal features from electromyographic waveforms. Analysis of Variance models and student *t*-tests using Bonferroni corrections determined between group differences in these gait features ($\alpha = 0.05$). **Conclusions:** Sagittal plane hip range of motion was significantly reduced with increasing severity of OA ($p < 0.05$) where as frontal and transverse plane range of motion was reduced in the severe OA group only ($p < 0.05$). Activation patterns of gluteus medius and maximus did not differ between the healthy group and those with moderate hip OA ($p > 0.05$). Individuals with severe OA walking with more prolonged gluteus maximus activation and prolonged and less dynamic gluteus medius activation compared to the other two groups ($p < 0.05$). This study highlights the changing function of the hip joint during walking with increasing hip OA severity.

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1. Introduction

Lower extremity osteoarthritis (OA) is a leading cause of impaired mobility in older adults. In Canada, joint replacement surgeries for hip OA have increased over 60% in the last decade (CIHI, 2013) as many see it as a way to reduce the pain associated with the disease, thus allowing them to remain active. Hip OA is thought to result from a failed repair of the hip joint, secondary to mechanical stressors (Felson, 2013). Quantifying hip joint function as it pertains to mobility in individuals with OA may provide an objective metric to aid understanding joint stresses during walking; a familiar activity that many individuals with OA struggle with.

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Individuals with hip OA typically walk with reduced sagittal plane hip motion (Hurwitz et al., 1997; Foucher et al., 2012; Eitzen et al., 2012). A brief reversal of hip extension during mid-stance has also been found in individuals with severe hip OA (Foucher et al., 2012). Eitzen et al. (2012) found that sagittal plane hip movement was dependent on radiographic severity; individuals with severe disease walked with less hip extension during late stance than those with mild disease or asymptomatic individuals and no motion reversals were reported. Despite evidence that hip joint function is altered with OA presence, it is still unclear how three-dimensional movements during walking change over the spectrum of increasing hip OA severity.

Kinematics provide an analysis of joint dysfunction during gait but do not aid our understanding of underlying mechanisms. Muscles can control joint motion, and thus may provide information on potential mechanisms for altered movement. Unfortunately, the inter-relationships between hip joint motion and gluteal muscle activation remain unclear. Dwyer et al. (2013) found greater hip

abductor activation throughout gait in 13 individuals with end-stage unilateral hip OA compared to healthy adults. Others have found that gluteal activation remains abnormal in individuals following hip arthroplasty (Agostini et al., 2014). While altered muscle activation is thought to be a mechanism to stabilize the deteriorating joint, greater muscle activity may increase hip joint compressive forces, ultimately affecting joint health (Neumann and Hase, 1994) and long-term function (Sims, 1999). However, to our knowledge, this has not been comprehensively studied during gait and it is unknown how gluteal muscle activation patterns are altered as a result of increasing hip OA severity.

At this time, there is a lack of understanding of hip joint motion and gluteal muscle activation patterns during gait when comparing individuals with severe hip OA to those with moderate hip OA or those with no symptoms of OA. The objectives of this investigation were to determine whether three-dimensional (3D) hip range of motion (ROM) and patterns of gluteal muscle activation are different between those with severe and moderate OA, and when compared to a healthy group. It is hypothesized that as severity increases, the hip joint will become stiffer, resulting in reduced 3D motion and less dynamic, more prolonged stance phase gluteal muscle activation.

2. Methods

2.1. Participants

Participants with unilateral symptomatic hip OA were recruited over 1 year (2013–14) from local orthopaedic clinics after consultation with an orthopaedic surgeon regarding hip arthroscopy for early disease management (moderate OA) and met a functional criteria previously used to define moderate knee OA (Rutherford et al., 2013) or were candidates for total hip replacement (severe OA). Participants were excluded from the moderate OA group if they were candidates for total hip replacement. Hip OA was determined using the American College of Rheumatology criteria (Altman, 1991). The healthy group was recruited from the general community using email and website based advertisements and considered a sample of convenience. These individuals had no pain in the ankles, knees or hips during testing and no symptoms of lower extremity OA. All participants were ≥ 50 years of age, had no fracture or injury other than a sprain or strain (within one year) or no previous knee/hip joint surgery. All had to be able to walk independently, with no neurological or cardiovascular disorders that would impair walking ability. The protocol was approved by the local institutional ethics review committee (NSHA-RS/2014-081) and participants provided written informed consent.

Standard A/P pelvis and lateral radiographs were used to describe hip OA radiographic severity. A single experienced reader (IW), who was blinded to participant identification and gait analysis outcomes at the time of scoring, graded radiographs using the Kellgren–Lawrence (KL) ordinal radiographic scale (Kellgren and Lawrence, 1957). Participants in the healthy group did not receive any radiographs.

2.2. Procedures

Participants changed into a T-shirt and fitted shorts, removed their footwear and completed at least five self-paced walking trials across the GaitRITE™ portable pressure sensitive walkway (CIR Systems, Clifton, NJ, USA) to determine average self-selected gait speed.

Following these trials, participants were prepared for surface electromyography (EMG); skin was lightly shaved and cleaned with 70% alcohol wipes. Consistent with guidelines (Hermens

et al., 2000) and standard procedures, Ag/AgCl surface electrodes (10 mm diameter, 30 mm inter-electrode distance, Red Dot, 3 M Health Care, St. Paul MN, USA) were placed in a bipolar configuration over the gluteus medius (GMd) – 50% of the distance from the iliac crest to the ipsilateral greater trochanter, and gluteus maximus (GMx) – 50% of the distance between the 2nd sacral vertebrae and the greater trochanters. Muscle palpation and a series of isometric contractions for specific muscle groups were used for signal validation and gain adjustment. Surface EMG was recorded at 2000 Hz using an AMT-8™ Bortec system (Bortec Inc. Calgary) (Input Impedance: $\sim 10\text{ G}\Omega$, CMRR: 115 dB at 60 Hz, Band-pass (10–1000 Hz), Gain Range 500–5000 $\times$) and custom LabVIEW™ 2013 programs (National Instruments Corporation, Austin, TX).

Rigid sets of four retro-reflective markers were affixed to the mid-dorsal trunk (level of the inferior scapular angles), the pelvis (atop the sacrum), posterior femur and tibia using Velcro straps and secured with adhesive tape. Single retro-reflective markers were placed over the lateral aspect of the shoulders (below acromion), atop the spinous process of the 7th cervical vertebra, greater trochanters, medial and lateral femoral and tibial epicondyles, medial and lateral malleoli, head of the 5th metatarsal, and posterior heel.

Prior to gait analysis, a kinematic model calibration was completed, including a standing calibration trial, a virtual sternum, two virtual anterior superior iliac spine location trials and two standing hip joint center calculation trials that required the subject to move each leg through hip flexion, abduction and extension (Camomilla et al., 2006). Markers over the greater trochanters, medial tibial and femoral epicondyles, lateral tibial epicondyles and medial malleolus were then removed. Retro-reflective skin marker motion was captured at 50 Hz using four Qualisys® Pro-reflex motion analysis sensors (Gothenburg, Sweden).

Participants began walking on the treadmill at the self-selected GaitRITE™ walkway speed for at least four minutes for accommodation/warm-up. Following this, three 20-s data collections were completed, with approximately one minute between collections, during which the participants continued walking, thus blinded to collection intervals. After completion, retro-reflective markers were removed and a resting muscle activity trial (EMG subject bias) was recorded with the participant lying supine. Electrodes were subsequently removed.

Prior to completing the study, all participants completed the Hip Outcome Osteoarthritis Score (HOOS), which was scored based on instructions provided at www.koos.nu. The International Hip Outcome Tool (iHOT-33) was scored according to Mohtadi et al. (2012). For both questionnaires, a higher score indicates a better outcome.

2.3. Data analysis

Raw EMG signals were first processed to minimize the effects of treadmill noise contamination. This included, (i) band pass filtering (4th order Butterworth) with a pass band from 20 to 500 Hz (De Luca et al., 2010) and (ii) band-stop filtering at 60 Hz (and harmonics) in the frequency domain (using Fast Fourier Transformation, FFT and following inverse FFT). All EMG signals were then corrected for resting bias, converted to micro-volts, full-wave rectified and filtered using a Butterworth, 6 Hz recursive, 4th order, low-pass filter. Compared to the 10 Hz high pass cut off recommended by the Journal of Electromyography and Kinesiology, the 20 Hz setting used in the current band pass filter settings did not influence the interpretation of the results in this manuscript. All EMG waveforms were amplitude normalized to the peak EMG amplitude obtained for each muscle during the gait cycle (Burden, 2010).

Technical and local anatomical bone embedded coordinate systems for the pelvis, thigh, and shank were derived from virtual

points and skin markers. The rigid plates were tracked during the walking trials. Joint angles were calculated using a 6-degree of freedom model through Cardan/Euler rotations (z - y - x sequence), where hip joint flexion occurred about the z -axis (Landry et al., 2007). A flexion/extension – adduction/abduction – internal/external rotation sequence was utilized, where flexion, adduction, and internal rotations were derived as a positive angle. For heel strike detection, a kinematic method was used (Zeni et al., 2008). Knee joint angles and EMG were time normalized to 100% of the gait cycle (i.e. 101 data points representing heel strike to heel strike). All signal processing and analyses were completed in MATLAB™ Ver. 2014 (The Mathworks Inc., Natick, Massachusetts, USA) using custom script.

Motion data, extracted from the affected limb of those with hip OA and a random limb of the healthy group was ensemble averaged across the three 20-s walking trials (≥ 40 strides/side). In each plane, discrete measures (maximum, minimum) and angular excursions were calculated.

Principal component analysis (PCA) (Chau, 2001) was used to capture temporal and amplitude based EMG waveform features using custom MATLAB™ 2014 script. This technique has been previously described in detail and has been used to identify quadriceps and hamstrings activation patterns in knee OA during gait (Rutherford et al., 2013), but has yet to be implemented in hip OA gait literature.

For each muscle (GMx and GMD), a matrix was formed from the linear enveloped EMG signal of each individual stride (≥ 40 strides/participant) $[X] = [X \text{ strides} \times 101]$. An eigenvector decomposition of the cross product matrix ($[S] = [X'] \times [X]$) was performed, using standard notation $U'SU = L$, yielding predominant orthonormal components called eigenvectors. These eigenvectors (U) are hereafter referred to as principal patterns (PP). A percent trace was calculated to determine how much variability was contained in each PP; those explaining the greatest percent of variation ($>90\%$ total variation) in the waveforms were retained and referred to as PP1, PP2, etc. Following PCA, EMG data were ensemble averaged across the 3 walking trials for each muscle within each group. Principal pattern scores (PP-scores) were computed for individual gait waveforms by multiplying the ensemble-averaged waveform by the principal pattern generated by the data set that included each individual trial.

2.4. Statistical analysis

A series of one-way Analysis of Variance (ANOVA) models were utilized to identify group differences in age, BMI, HOOS, iHOT-33, and WOMAC scores derived from the HOOS. Normality and equal variance of gait velocity collected on the GaitRITE™ walkway, three-dimensional hip joint motion characteristics and EMG PP-scores were tested using Kolmogorov–Smirnov and Levene's tests, respectively, followed by one-way ANOVAs to determine significant between group differences. Significance level was set at $\alpha = 0.05$. All statistical procedures were completed using SPSS V.21 (IBM, Armonk, New York).

3. Results

Twenty participants were recruited for each group. Data from 3 individuals in the severe OA group were removed due to corruption of the EMG data ($n = 2$) or inability to treadmill walk without using handrails ($n = 1$). Participants in the severe OA group had greater BMI than the healthy group ($p = 0.026$), while over-ground walking velocity and stride length differed among all groups ($p \leq 0.013$, $p \leq 0.012$, respectively) (Table 1). Similarly, there were significant differences between all groups for all aspects

Table 1

Mean (SD) group demographics, gait characteristics and questionnaire outcomes as well as radiographic grade counts (Kellgren–Lawrence) for each hip OA group.

	Healthy	Moderate OA	Severe OA
Age (years)	62 (6)	59 (8)	63 (8)
BMI ^a (kg/m ²)	25.6 (5.0)	28.7 (4.3)	30.0 (4.4)
HOOS			
Pain ^b	95 (8)	66 (16)	43 (15)
Symptoms ^b	92 (11)	68 (17)	44 (16)
ADL ^b	98 (4)	75 (20)	44 (20)
Sports and Rec ^b	96 (7)	54 (29)	25 (12)
Quality of life ^b	96 (8)	47 (22)	25 (12)
WOMAC			
Physical function ^b	1 (3)	17 (13)	38 (13)
Pain ^b	1 (1)	6 (3)	10 (3)
Stiffness ^b	1 (1)	3 (1)	5 (1)
iHOT-33 ^b	96 (3)	58 (21)	31 (18)
Walking speed ^b (m/s)	1.51 (0.16)	1.32 (0.22)	1.11 (0.27)
Stride length ^b (m)	1.48 (0.12)	1.34 (0.16)	1.16 (0.17)
Radiographic grade (n) ^A			
KL 0	–	0	0
KL I	–	9	0
KL II	–	5	0
KL III	–	2	4
KL IV	–	1	12

^a Significant difference between healthy and severe OA groups ($p < 0.05$).

^b Significant differences between all groups ($p < 0.05$).

^A Three individuals obtained magnetic resonance imaging to investigate Hip OA.

of the HOOS, WOMAC and iHOT-33 self-report questionnaires ($p < 0.0001$).

Fig. 1 illustrates ensemble averaged hip motion during gait. The severe OA group used significantly less total ROM in all 3 planes than the other 2 groups ($p < 0.026$) and the moderate and healthy groups also differed in the sagittal plane ($p = 0.002$) (Table 2).

Fig. 2A and B illustrates the ensemble averaged GMD activation for all groups and the three principal patterns that captured 98% of waveform variability. PP1 captures the overall waveform shape. High PP2-scores indicate a more distinct decrease in activation between early and mid-stance. PP3 captured a difference operator between mid and late stance. As shown in Table 3, there was significant difference between the severe OA group and both other groups for GMD PP1 and PP2 ($p < 0.001$).

The ensemble averaged GMx activation for the healthy group and the two hip OA groups are shown in Fig. 3A. Together, the three principal patterns (Fig. 3B), explain 98% of waveform variability. PP1 captured an overall shape of the GMx waveform. PP2 captured a difference between early and mid-stance, where higher scores indicated greater mid-stance activity compared to early stance. PP3 captured a difference between early, mid and late stance where greater scores indicated delayed peak activation and decreased activity during late stance/swing. PP-scores are displayed in Table 3. The only significant difference in GMx PP-scores is a higher PP2-score in the severe group compared to the asymptomatic ($p = 0.042$).

4. Discussion

While the literature describes altered gait kinematics in individuals with hip OA, the influence of disease severity on 3D hip motions and gluteal muscle activation during gait remains unclear. We hypothesized that joint motions and gluteal muscle activation would be altered during gait and depend on severity of hip OA.

Hip OA manifests in a heterogeneous clinical presentation despite previous literature focused on end-stage disease. Individuals with hip OA in the current study were grouped into moderate

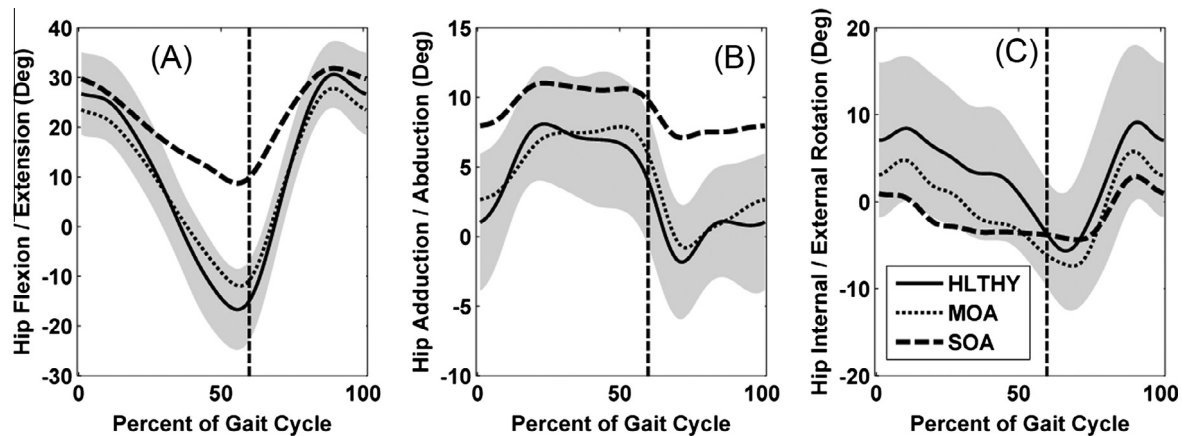


Fig. 1. (A) Sagittal, (B) frontal, and (C) transverse plane hip movements between healthy (solid), moderate (dotted) and severe hip OA (dashed) groups where flexion, adduction and internal rotation are positive angles. The shaded region represents 1-SD above and below the healthy group waveform and the vertical line represents toe off.

Table 2

Mean (SD) of maximum, minimum and total range of hip motion (maximum–minimum) in 3 planes of movement.

	Healthy	Moderate OA	Severe OA
<i>Sagittal</i>			
Maximum	31° (7°)	28° (6°)	32° (10°)
Minimum ^a	–17° (8°)	–12° (10°)	8° (15°)
Total ^{a,c}	48° (5°)	40° (7°)	24° (8°)
<i>Frontal</i>			
Maximum	9° (4°)	8° (5°)	12° (6°)
Minimum ^a	–2° (4°)	–1° (5°)	6° (6°)
Total ^a	11° (3°)	10° (3°)	6° (2°)
<i>Transverse</i>			
Maximum ^b	11° (8°)	7° (7°)	4° (8°)
Minimum	–7° (7°)	–9° (7°)	–7° (7°)
Total ^a	18° (4°)	16° (4°)	11° (4°)

^a Significant difference between the severe OA group and both other groups ($p < 0.05$).

^b Significant difference between severe OA group and healthy group ($p < 0.05$).

^c Significant difference between moderate OA and healthy group ($p < 0.05$).

and severe classifications based on clinical management and functional criteria used previously in knee OA literature (Rutherford et al., 2013). Differences were found among the groups pertaining to summary measures of function (gait velocity and questionnaires). As OA severity increased, individuals walked significantly

Table 3

Mean (SD) principal pattern scores for Gluteus Maximus (GMx) and Medius (GMd). Patterns are indicative of temporal and amplitude differences in average EMG waveforms between groups.

	Healthy	Moderate OA	Severe OA
<i>Glut Max</i>			
PP1	399.5 (84.0)	395.0 (118.2)	462.6 (68.5)
PP2 ^b	–42.3 (63.1)	–33.7 (102.3)	26.4 (90.2)
PP3	–10.6 (64.4)	–5.5 (66.6)	31.5 (80.9)
<i>Glut Med</i>			
PP1 ^a	410.6 (63.2)	436.6 (69.5)	521.2 (98.6)
PP2 ^a	62.0 (49.6)	30.7 (59.9)	–55.5 (106.9)
PP3	3.2 (60.2)	–3.1 (58.4)	–4.0 (57.6)

^a Significant difference between the severe OA group and both other groups ($p < 0.05$).

^b Significant difference between severe OA group and healthy group ($p < 0.05$).

slower where individuals with severe OA walked at 1.1 m/s and the healthy group at 1.5 m/s. Our severe group velocity was similar to previously reported values for those receiving a total hip replacement (Hurwitz et al., 1997) while faster than those in a group post-hip replacement (Agostini et al., 2014). Individuals with moderate hip OA walked slower than individuals with mild to moderate hip OA previously reported (Eitzen et al., 2012). HOOS and WOMAC scores in the severe group were similar to those previously reported for those awaiting total hip replacement

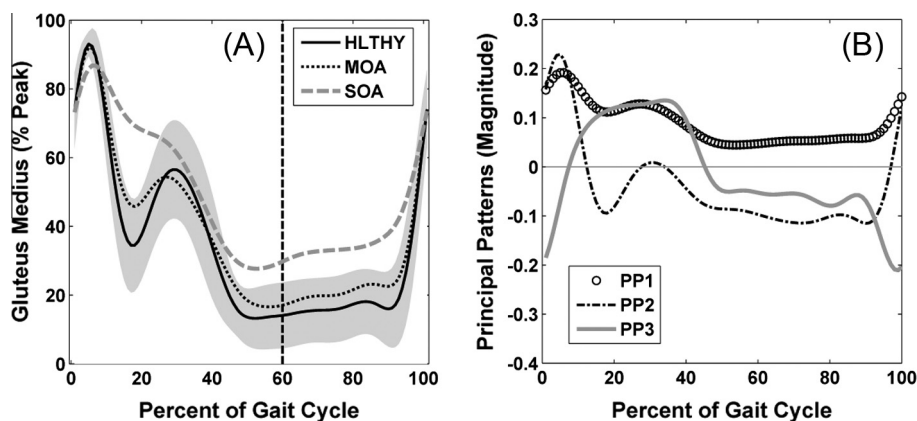


Fig. 2. (A) GMd electromyograms between healthy (solid), moderate (dotted) and severe hip OA (dashed) groups. Amplitude normalization to peak activation during the gait cycle. The shaded region represents 1-SD above and below the healthy group waveform. (B) Principal Patterns that together explained 98% of the waveform variance with circles (PP1), dash-dot (PP2) and solid (PP3) shown.

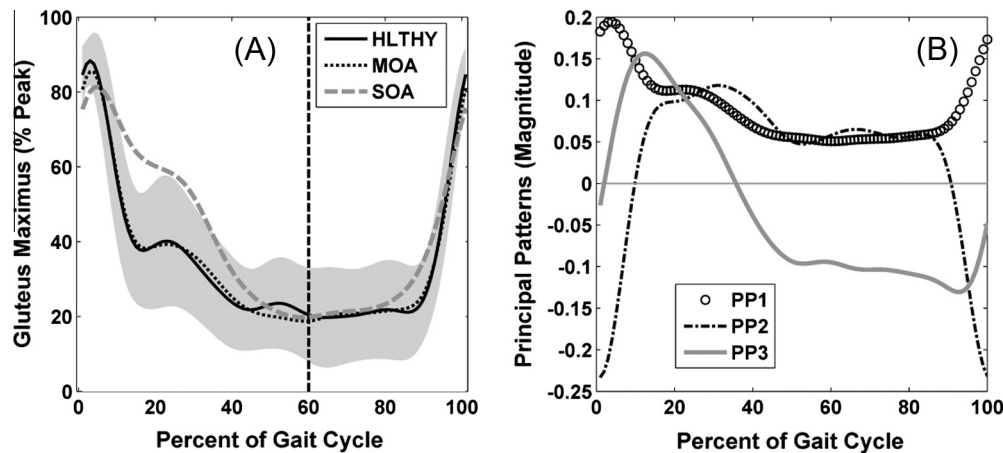


Fig. 3. (A) GMx electromyograms between healthy (solid), moderate (dotted) and severe hip OA (dashed) groups. Amplitude normalization to peak activation during the gait cycle. The shaded region represents 1-SD above and below the healthy group waveform. (B) Principal patterns that together explained 98% of the waveform variance with circles (PP1), dash-dot (PP2) and solid (PP3) shown.

(Nilsdotter et al., 2003; McHugh et al., 2013). The higher scores of the WOMAC and lower scores of the HOOS and iHOT-33 suggest participants with severe OA are self-reporting a significant deficit in physical function and increased symptoms compared to the other groups. HOOS and iHOT-33 scores for the moderate OA group were similar to those found for individuals with severe hip chondropathy, who were not yet candidates for arthroplasty (Kemp et al., 2014) and those with moderate knee OA (Rutherford et al., 2013). The differences between the groups for these measures were much greater than minimal clinical important differences previously reported (Mohtadi et al., 2012; Nilsdotter and Bremander, 2011) and suggest that these measures capture disease severity from an aspect of symptoms and self-reported function. From a biomechanical perspective, however, between group differences were not as clear and caution must be exercised when extrapolating joint function from these self-reported outcomes. Together, combined with median radiographic grades of each group, these data suggest that our classification captured distinct sub-populations of individuals with hip OA based on summary measures of function.

4.1. Kinematics during gait

Tri-planar hip motion was reduced in individuals with severe hip OA compared to both the healthy and moderate OA groups, whereas only sagittal plane motion was reduced between the healthy and moderate OA groups (Fig. 1). The reduced hip extension during mid to late stance (Fig. 1A and Table 2) was consistent with other studies (Hurwitz et al., 1997; Eitzen et al., 2012). However, individuals with severe OA in this study did not present with motion discontinuity during mid-stance, as previously described in end stage hip OA (Foucher et al., 2012). In a recent meta-analysis, Ewen et al. (2012) found that hip flexion/extension ROM during gait varied between 31° and 51° in control subjects and between 23° and 41° degrees in individuals post-THA, similar to those found in the current study. Stride length was also significantly reduced among the three groups (Table 1), which may explain the reduced sagittal motion as disease severity worsened. Frontal and transverse plane range of motion was significantly reduced in the severe OA group only. While numerical differences were small, they still reflect approximately a 50% decrease in functional ROM. Despite questionnaire and gait velocity being different among groups, ROM findings suggest that sagittal plane ROM begins to decrease early in the disease process. However, only in later stages of the

disease do significant alterations to frontal and transverse plane motions occur. This suggests that the hip joint is becoming stiffer and less dynamic in those with severe OA.

These kinematic alterations provide an outcome of the underlying mechanisms that control movement in this population. While novel in the study of hip OA gait mechanics, additional understanding of joint dynamics can be derived from the investigation of muscle activation patterns given the muscles role to initiate, maintain and terminate movements during gait and their contribution to active joint stiffness.

4.2. EMG during gait

To our knowledge, this is the first study to quantify gluteal muscle activation patterns during gait in a cross sectional group of individuals with hip OA. We hypothesized that altered GMd and GMx activation patterns would exist, given their role in controlling hip function during gait. While greater muscle activity may assist in maintaining joint function in the presence of the arthritic change, it will also result in increased joint compression forces (Neumann and Hase, 1994; Correa et al., 2010) and potentially reduce functional ROM during gait.

GMx and GMd activation were altered in individuals with severe hip OA compared to the other two groups, partially supporting our original hypothesis. Similar to previously reported data, GMd in our healthy group demonstrated two distinct activation peaks: initial contact and mid-stance (Fig. 2A) (Rutherford and Hubley-Kozey, 2009). This pattern was also found in the moderate OA group, suggesting a similar pattern of demand. In contrast, the associated reduction in GMd activation between these two peaks did not occur in individuals with severe hip OA, as was identified by the high PP1 and low PP2 scores (Fig. 2B and Table 3). Greater GMd activity has been reported during the stance phase in individuals with OA compared to healthy individuals (Dwyer et al., 2013). However, activity was averaged across the stance phase, thus extracting where the increase was most dominant was not possible. In contrast, Horstmann et al. (2013) presented GMd waveforms for individuals prior to total hip arthroplasty, showing the same general pattern as the current study although the analysis techniques did not allow identification of specific pattern differences (Horstmann et al., 2013). These changes have implications for fatigue and joint loading (Sims, 1999; Neumann and Hase, 1994). Previous literature has described GMd muscle atrophy and reduced strength in individuals with advanced hip OA (Grimaldi

et al., 2009; Rasch et al., 2007). While hip strength was not measured in the current study, van der Krogt et al. (2012) found through modeling, that GMd weakness can significantly affect gait: as GMd weakened, the simulated force curve became less dynamic. Weakness may also be a contributing factor to muscle activation patterns found in the current study but further *in vivo* testing is required to confirm this hypothesis.

GMx also showed greater mid-stance activation compared to early stance (PP2-score) in the severe hip OA group (Fig. 3), differing significantly from the healthy group. This increase in activation relative to early stance, would suggest that individuals awaiting total joint replacement require a greater contribution from GMx for hip joint support, as GMx activity in the healthy group reduces after loading response as the hip extends toward terminal stance. This activation pattern may also be a response to reduced hip extension in the hip OA group (Fig. 1), although further work is required to ascertain the factors that might explain hip joint kinematics in this population.

In summary, self-reported questionnaires, gait velocity and sagittal plane hip ROM during gait were significantly different among the three groups studied. Those with severe OA walked with different 3D motions and gluteal activation patterns than both other groups, but only sagittal plane motion differed between the healthy and moderate OA group. These results highlight the importance of clearly determining hip OA severity before conducting gait analysis involving measurements of hip joint function, to more fully understand the implications of hip OA on gait.

While this investigation provided a comprehensive assessment of hip function in individuals with hip OA, there are some limitations. First, while all OA participants reported only one symptomatic hip, we cannot rule out the potential for asymptomatic radiographic hip OA in the contra lateral hip and thus cannot be considered non-arthritis and may influence gait dynamics. Further work on a larger scale is required to determine whether bilateral radiographic OA can explain the differences seen in this study. Similarly, the healthy group did not receive radiographs of the hips, thus it cannot be assumed that they are completely free of hip OA. Secondly, the treadmill used for this investigation was not instrumented. We were, therefore, unable to calculate joint moments and compare with previous works, who have reported increased frontal plane joint moments in individuals with severe hip OA (Foucher et al., 2012). Thirdly, in that EMG amplitudes were normalized to the peak amplitude obtained, absolute amplitude comparisons between groups could not be done (Burden, 2010). This method was chosen to avoid maximum muscle contractions in this population and remove the possibility of the raw EMG processing, required as a result of treadmill noise artifact, to influence amplitude comparisons. Instead, PCA was applied to quantify muscle activation temporal patterns, as previously performed in a knee OA population (Rutherford et al., 2013). Thus, only those patterns that were difference operators were interpreted (PP2 and PP3). This analysis technique limits conclusions based on absolute muscle activation magnitudes, such as overall compressive joint loading, however provides the statistical ability to understand waveform dynamics and relative loading (as a result of muscle activation) across the gait cycle. To our knowledge, this is the first use of PCA to analyze activation patterns in the muscles surrounding the hip in a group of participants with hip OA. Finally, treadmill speed was set to the self-selected walking speed as determined from the GaitRITE™ walkway. The assumption was made that participants would adopt similar spatial/temporal gait characteristics on the treadmill after a period of accommodation. While GaitRITE™ validity and reliability have been shown (McDonough et al., 2001), some participants commented that the treadmill seemed faster despite the same velocity, although this sensation tended to reduce after 2–3 min of initial treadmill walking.

5. Conclusion

The understanding of OA hip joint mechanics and concurrent muscle activation characteristics during gait provide a framework for understanding the outcomes of the disease, as well as how the body attempts to preserve walking ability with declining joint function. 3D joint motions were decreased in individuals with severe OA, and gluteal muscle activation patterns were generally higher, less dynamic than either the moderate OA or healthy groups. Only sagittal plane motion was reduced in the moderate OA group, when compared to the healthy group. These data have implications for understanding hip OA pathomechanics and considerations for joint function during gait as hip OA disease severity increases.

Competing interest statement

Authors have no competing interests pertaining to this manuscript.

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