
**Introduction**

Osteoarthritis (OA) is a progressive musculoskeletal joint disease that affects articular cartilage, bone, and periarticular soft tissues\(^1\). OA begins at a molecular level, affecting joint tissue metabolism that evolves into an anatomic and physiologic disruption in joint homeostasis\(^2\). This progressive disruption to joint function is a leading cause of chronic morbidity in older adults. In individuals with hip OA specifically, the pain and stiffness of this disease negatively impacts mobility and the ability to be physically active\(^3,4,5\).

Walking volume and intensity in individuals with hip OA is reduced compared to individuals without OA\(^6\). Physical disability is a common by-product of OA, and a number of factors have been proposed as possible explanations for an individual’s level of disability\(^3\) including loss of sagittal plane range of motion (ROM)\(^7,8,9\). Coincidentally, the largest ROM at the hip during gait occurs in the sagittal plane and reduced sagittal plane joint ROM during gait has been consistently reported in individuals with hip OA\(^7,10,11,12\). This reduction is primarily a result of attenuated hip extension during terminal stance where values of 4-8 degrees have been found\(^7,10\). Increased sagittal\(^13,14,15\) and frontal plane\(^15\) pelvic motion have been described in individuals with hip OA. Greater amounts of pelvic anterior pelvic tilt, peaking at toe-off are likely to compensate for this lack of hip extension\(^7\). This lack of extension may have implications for understanding altered knee mechanics\(^16\) or low back pain\(^17\) in this population associated with walking.
In contrast, healthy asymptomatic individuals walk with approximately 13-18 degrees of hip extension during terminal stance and approximately 26-30 degrees of hip flexion at initial contact\textsuperscript{7,11}. During walking, individuals more closely approach their limits of hip extension, than hip flexion\textsuperscript{7,18}. The periarticular ligament structure of the hip becomes tighter as the hip extends, abducts and externally rotates\textsuperscript{19,20}, which may explain the reduction in hip extension during this period of the gait cycle. In that the ligaments of the anterior hip joints are passive (non-contractile) structures, is it plausible that reduced hip extension during walking is a consequence of altered joint structures, correlated to the amount of passive ROM?

Many clinical approaches to hip OA management have focused on manual therapy to increase soft tissue extensibility\textsuperscript{21,22}. However, evidence of effectiveness has been of low to moderate quality\textsuperscript{23}. Currently, it is not clear whether these interventions improve gait mechanics and reduce the impact of OA on joint function. In rehabilitation settings, the evaluation of passive hip ROM is used to understand the impairments associated with OA\textsuperscript{24,25}, however, the relationship to walking dynamics has not been determined. Recently, Moreside and McGill found that improvements in passive hip mobility after a 6-week exercise program in healthy young adults\textsuperscript{26} did not translate to changes in dynamic range during functional activities which did not include walking\textsuperscript{27}. Whether a relationship exists between passive joint ROM and dynamic ROM during walking in individuals with hip OA remains unknown. In addition, it is thought that if a relationship does exist, OA severity will modify the relationship strength, given the influence of OA on the passive osteoligamentous structures of the joint. Despite hip flexibility exercises prescribed to improve hip mobility, there is no evidence to support the possible transfer to dynamic ROM during walking.
Thus, the main objective of this study was to determine the relationship between hip extension ROM during gait (dynamic) and passive hip extension ROM measurements in a healthy group, and in individuals with moderate and severe OA. We hypothesized there would be a positive correlation between dynamic and passive hip extension ROM in both moderate and severe OA groups, and no relationship in asymptomatic individuals. Secondly, we hypothesized that correlations will be greater in moderate compared to severe OA.

**Methods**

**Participants**

For this cross-sectional research study (Level III Evidence), participants with unilateral symptomatic hip OA were recruited from local orthopaedic clinics and diagnosed using American College of Rheumatology guidelines\(^{28}\). Individuals considered for total hip replacement (THR) were classified as severe OA (SOA). Individuals deemed not severe enough for a THR and treated conservatively were classified as moderate hip OA (MOA). In addition, individuals with MOA were required to self-report their ability to walk a city block, climb up and down stairs and jog 5 meters and would not be encumbered by their OA symptoms. Individuals with SOA were unable to complete one or more of these tasks. This functional criterion has been used in the knee OA literature to help define a moderate disease classification\(^{29}\). A healthy group was recruited from the general community using websites, email and poster advertisements and was considered a sample of convenience. Healthy participants demonstrated no symptoms or history of degenerative joint disease. All participants were required to have no history of a strain or sprain in the past year, have the ability to walk independently, exhibit no cardiovascular or neurological disease that impaired walking ability,
and be ≥ 50 years of age. The protocol was approved by the local institutional ethics review committee and participants provided written informed consent.

For descriptive purposes, standard anterior-posterior (AP) pelvis and frog leg radiographs were evaluated in individuals with hip OA. A single experienced reader, blinded to participant identification and gait analysis outcomes, graded radiographs using the Kellgren-Lawrence (KL) ordinal radiographic scale\(^{30,31}\). All participants with hip OA completed the Harris Hip Questionnaire and all participants completed the Hip Outcome Osteoarthritis Score (HOOS).

**Procedures**

Participants removed footwear and changed into tight fitting “Lycra” type shorts and T-shirt. Five walking trials across the GaitRITE™ pressure sensitive walkway (model P 401; CIR Systems, Sparta, NJ, USA) were completed at a self-selected speed. These trials were averaged and used to establish the speed of treadmill walking.

Four sets of rigid, retro-reflective markers were affixed level with the inferior scapular angles of the trunk, atop the sacrum, and on bilateral posterior femurs and tibias using Velcro straps and adhesive tape. Single retro-reflective markers were placed atop the lateral aspect of the shoulders below the acromion, spinous process of the 7\(^{th}\) cervical vertebra, greater trochanters, medial and lateral femoral and tibial epicondyles, medial and lateral malleoli, head of the 5\(^{th}\) metatarsals, and posterior heels. Marker motions were captured using 4 Qualisys® Pro-reflex motion analysis sensors (model MCU 240; Gothenburg, Sweden) at 50Hz. Lower limb electromyography was also collected as part of this protocol but will not be further discussed.

A kinematic model calibration was completed prior to gait analysis, including a standing calibration, virtual sternum, two virtual anterior superior iliac spine locations and two standing
hip joint center calculation trials that require the subject to move each leg through hip flexion, extension and abduction movements\textsuperscript{32}. After completing model calibrations, the reflective markers over the greater troCHANTers, medial tibial and femoral epicondyles, lateral tibial epicondyles and medial malleoli were removed.

Using the self-selected speed calculated from the GaitRITE\textsuperscript{TM} walkway, participants began treadmill walking with at least four minutes of accommodation/warm-up. Following accommodation, three 20-second data collections were completed with participants being blinded to collection intervals. At least one minute of walking was completed between recordings. After collection trials, treadmill speed was reduced by 15\% for a one-minute cool down period. All retro-reflective markers were removed.

Following walking trials, two experienced physiotherapists performed an assessment of passive hip joint ROM using standardized procedures. The physiotherapist manipulating the goniometer and the physiotherapist moving the limb were standardized for all participants, with the latter being blinded to all measurement outcomes. Hip flexion, extension, adduction, abduction, and internal and external rotation were measured twice in a random sequence and each pair of measurements was averaged. The participants were supine for all measurements, and a standard goniometer was used, affixed with a spirit level to increase accuracy\textsuperscript{33}. To test the hypothesis of this investigation, only flexion and extension range of motion were used and thus, these procedures are detailed below. To measure hip flexion, a physiotherapist passively flexed the hip to maximum allowable range before the lumbar spine began to flex. This was determined using therapist palpation; participant concurrence was encouraged when able. Non-sagittal plane motions were avoided. With the goniometer centroid placed over the greater trochanter, the spirit level was used to determine an axis parallel to the plinth (a horizontal axis), and the
moveable arm positioned towards the lateral femoral epicondyle. Hip extension was measured with the participant buttocks at the end of the plinth with knees flexed to the chest as per the modified Thomas test\textsuperscript{33}. The physiotherapist passively flexed both hips simultaneously to determine full hip and pelvis flexion; the point at which the lumbar spine began to also flex. One leg was stabilized in this position by the physiotherapist, while the limb of interest was allowed to slowly lower into maximum extension, while controlling for frontal and axial motion of the femur. Extension measurements represent the position of the extended limb relative to the horizontal\textsuperscript{33}; they do not include an additional 10 degrees representative of pelvis flexion, as is standard with the modified Thomas test in its original form\textsuperscript{34}.

Data Analysis

For biomechanical analysis, technical and local anatomical bone embedded trunk, pelvis, thigh and shank coordinate systems were derived from skin markers and virtual points. Joint angles were calculated using a six degree of freedom model through Cardan/Euler rotations, where hip flexion occurred about the z-axis\textsuperscript{35}. A flexion/extension – abduction/adduction – internal/external rotation sequence was utilised where positive angles were derived as flexion, adduction and internal rotation. Heel strike was determined with a kinematic method whereby the maximum forward trajectory of the heel marker with respect to the pelvis indicated heel strike\textsuperscript{36}. Hip joint motions were time normalized to 100\% of the gait cycle using a cubic spline interpolation technique, representing one complete gait cycle, beginning and ending with foot strike. Using custom script in MATLAB\textsuperscript{™} Ver. 2012 (The Mathworks Inc., Natick, Massachusetts, USA), all signal processing and analyses were completed.

Analysis
Motion data were ensemble averaged across the three walking trials. From motion data, joint excursion through 100% of the gait cycle was identified in the sagittal plane, including maximum hip flexion (dynamic flexion) and extension (dynamic extension). For the correlational analysis, hip extension was represented as a positive value.

**Statistical Analysis**

Paired t-tests were employed to determine if significant differences occurred between the right and left legs in the asymptomatic group. One-way ANOVAs were utilized to determine significance between groups in demographics and HOOS. For the Harris Hip Score (HHS), an independent t-test was used because no data were obtained from the asymptomatic group. Normality and equal variance of passive hip ROM, gait-based spatial and temporal characteristics collected on the GaitRITE™ walkway and 3D hip motion were tested utilizing a Kolmogorov-Smirnov and Levene’s test, respectively. A two-way mixed modeled ANOVA with Tukey post-hoc tests were used to determine differences between groups and between passive and dynamic ROM. Pearson’s correlations were used to determine the direction and strength of the relationships between independent variables. All statistical procedures were completed using Minitab™ Ver.17 (Minitab Inc. State College, PA, USA).

**Results**

Table 1 provides demographics, anthropometrics, gait velocity and maximal hip joint excursions for each group. KL grades were also included in the table for the MOA and SOA groups. No significant differences were found between the asymptomatic group’s right and left legs, thus the outcomes were averaged for the remaining statistical comparison.
No significant differences were found between participant age, mass, height and BMI in each group. Significant differences were found between the walking velocities of each group (p<0.001). Walking velocity decreased as OA severity progressed.

A significant group by ROM interaction was found for extension (p=0.004) measurements. The SOA group had less dynamic and passive extension than the MOA and ASYM groups (p<0.001), whereas no difference existed between the MOA and ASYM group for dynamic (p=0.206) or passive (p=1.000) extension. Dynamic extension ROM did not differ from passive extension ROM within the ASYM (p=0.999) and MOA (p=0.505) groups, however, the SOA group’s passive ROM was greater than the dynamic measurement (p<0.001).

A significant group by ROM interaction was also found for flexion (p<0.001) measurements. Passive flexion ROM significantly differed between the three groups (p<0.029). However, for dynamic flexion, no differences existed between any of the groups (p>0.05). Dynamic and passive flexion ROM were significantly different from each other within each of the three groups (p<0.001).

Ensemble averaged sagittal plane hip range of motion for each of the groups is illustrated in Figure 1. Significant differences were found in total sagittal ROM between all three groups. The asymptomatic group utilized an average of 48°, MOA group 36° and SOA group 22° during the gait cycle (p<0.001).

Figure 2 illustrates the Pearson’s correlations found between dynamic and passive extension in (B) MOA (r=0.596, p=0.006) and (C) SOA (r=0.586, p=0.008) group. No correlation was found between dynamic and passive extension in the asymptomatic group (A) (r=0.139, p=0.559).
Discussion:

Many conservative and surgical treatments for hip OA are designed to influence the hip joint, yet it is often difficult to determine what effects these treatments may have directly on the function of the hip joint during important dynamic tasks such as walking. Previous studies have found that passive hip extension range of motion is limited in individuals with hip OA\(^7, 8, 9\). Coincidentally, the most common dynamic joint restriction recorded during gait in individuals with hip OA is a reduction in extension that occurs during late stance\(^7, 12\). It is plausible that passive ROM, as assessed in a clinical examination, does relate to dynamic ROM during walking although evidence of this association is lacking. The aim of this study was to investigate the relationship between these features and understand whether OA severity is implicated in these results.

Previously, gait studies have focused on only one severity of hip OA, either end stage\(^10\) or moderate disease\(^7\), limiting our understanding of how disease severity may impact hip function during walking. In the current study, the group with severe OA were candidates for THR and presented with a median KL grade of IV. These individuals demonstrated significantly reduced passive hip flexion and extension ROM (Table 1) compared to the other two groups. In fact, these individuals, on average, did not extend their hip beyond neutral during walking (Figure 1). This is consistent with other gait studies in those with end stage hip OA\(^10, 37\), and suggests that a significant restriction in hip motion affects both passive and active movement. Individuals with severe OA also walked slower than the other two groups, and similar to previously reported values for those receiving a THR\(^38\). HOOS and HHS in the severe group were similar to those previously reported for those awaiting THR\(^39, 40\). These similarities suggest the SOA group awaiting THR resemble functional (velocity) and self-reported
function/symptoms (HOOS, HHS) characteristic of a typical pre-operative THR population. In contrast to those with severe OA, the individuals with moderate hip OA in the current study walked faster than those with severe OA yet, slower than individuals with mild to moderate hip OA previously reported and presented with similar HOOS scores41. The HHS is often employed for perioperative function in total arthroplasty and thus direct comparisons to previous work in moderate hip OA was difficult. Individuals with moderate OA presented with a lower distribution of radiographic scores (median KL I) and significantly greater self-reported function than individuals with severe OA. HOOS scores were also lower in the moderate OA group compared to the asymptomatic group and while passive flexion ROM was different from the asymptomatic group, extension was not, partially contrasting previous work8, 9. Passive extension ROM values, in a population with moderate hip OA, were generally greater than those previously presented for individuals who were not candidates for THR8, 9 and may be explained by possible differences in measurement technique8, 9, as well as possible variations as to how the level of radiographic evidence of OA was graded. Together, these data suggest that two distinct clinical populations of individuals with hip OA were captured in this study, and populations are similar to those previously presented in the literature.

Many structures play different roles in the ROM of a joint42, which also means that many different processes could restrict joint movements. The hip joint is a capsular, ball-and-socket joint with a complex ligament structure that tightens as the hip extends, abducts and externally rotates27, 28. Hip extension and abduction occur naturally during terminal stance, suggesting that passive structures increasingly contribute to controlling range of motion at this stage of the gait cycle. In the healthy asymptomatic group, the difference in passive and dynamic extension was <1 degree; in flexion, dynamic values were approximately 27% of those obtained passively
(Table 1), suggesting restrictions to hip extension may have greater implications for hip motion during walking compared to flexion. In the asymptomatic group, no associations were found between dynamic and passive ROM, supporting previous work by Moreside and McGill\textsuperscript{26, 27}, in younger healthy adults. This null relationship in the asymptomatic group is thought to reflect the many possibilities in accomplishing hip extension during gait without restriction of joint pathology. As the joint becomes restricted, these restrictions will begin to govern the amount and directions of movement.

The amount of passive range of motion was positively correlated to the amount of active dynamic range of motion in individuals with hip OA (Figure 2). This correlation was similar in the MOA and SOA groups, refuting our secondary hypothesis. On Table 1, an approximate 30-degree restriction in flexion in the SOA group is shown; however, this still amounted to an assessed 60 degrees more than what was required during walking. Patients with less passive hip flexion were those with severe knee OA. Flexion occurs independent of extension, so the fact that the patients presenting with less dynamic hip extension were also those with less passive flexion is considered a factor of disease severity. Those presenting with less dynamic hip extension were also those presenting with less passive hip extension. Given the hip tightens through ligamentous constraint during terminal stance, joint space narrowing, osteophytosis and joint margin deformities\textsuperscript{30} will create a significant impediment to hip extension motion both passively and dynamically. In the current study, the absolute amount of extension was greater during passive testing, and the passive/dynamic differences were greater for the SOA group compared to those with moderate OA. The non-weight bearing nature of passive ROM testing may help explain these differences; in individuals with severe OA, active stabilization due to muscle contractions during gait, may limit motion in addition to passive structure restraint. These
results suggest that while a correlation exists, other mechanisms are working during dynamic walking to limit range of motion.

While it has been shown that hip extension range of motion is limited in individuals with hip OA during gait\textsuperscript{7,12}, there remains a poor understanding of the factors that might explain why these differences exist. During gait, a multidimensional integration exists between the passive, active and neurological subsystems\textsuperscript{30}, as previously described by Panjabi in relation to spine function, to preserve function and allow individuals to walk. During passive range of motion testing, participants were instructed to relax to the best of their ability while their range of motion was measured. From the correlations obtained in this work, we now understand that in individuals with moderate and severe OA, this passive range of motion measurement can explain between 36\% (MOA) and 34\% (SOA) of the dynamic extension utilized during gait. There remains however, approximately 60\% of unaccounted variability. The objective of this study was not to determine the best combination of non-collinear factors that could explain dynamic range of motion during gait in these groups. Further work is required to determine, for example, the implications of pain during walking and if the timing of pain (i.e. initial contact, mid stance and terminal stance), joint surface irregularities (e.g. narrowing and osteophytosis) and the influence of mass and leg diameter can explain, in addition to passive ROM, dynamic hip motion during gait. Given difficulty in quantifying these factors, and the finding of reduced extension ROM both passively and actively in individuals with hip OA, the focus on this paper was on ROM measures specifically. These data provide evidence to support the use of hip extension flexibility exercises to potentially improve hip motions in individuals with moderate and severe hip OA during walking. Reciprocally, if interventions are prescribed to alter hip motions, these should include those with a goal of improving passive hip extension range of motion. As
hypothesized, a relationship between passive and dynamic ROM was not found in the healthy group. Thus, in keeping with the work of Moreside and McGill\textsuperscript{26}, it is not expected that increasing passive hip extension ROM alone will alter dynamic hip extension ROM during gait in this group.

Some considerations exist for the interpretation of our results. One of the limitations to treadmill gait protocols is selecting the speed of the treadmill belts. Our analysis was performed on a non-instrumented treadmill set to the speed of over-ground walking. This was a new task for many of the participants and some individuals reported that the treadmill felt faster than over ground walking. However, after an accommodation/warm-up period this sensation of a faster pace generally diminished. Gait speed selection has also been a topic of discussion when comparing disease states of increasing severity, as individuals with more severe OA often walk slower\textsuperscript{43}, as we found in this study. An experimental control of walking speed would have made at least one of the groups walk at an unnatural pace, stifling the generalizability to clinical populations who choose their walking speed for any number of reasons. A statistical control could have been implemented; however, as stated by Wilson 2012\textsuperscript{44}, this correction requires assumptions of the data and the questions being posed that would be violated in this situation. As others have done\textsuperscript{7,11,12}, speed was set to that self-selected by the participants and while it is agreed that individuals with OA walk slower; it is also part of the disease process. Secondly, despite studies supporting the use of passive testing to determine ROM in OA populations\textsuperscript{5,45}, passive sagittal plane range of motion can be difficult to measure in individuals with hip pathology. Research is lacking in regards to directly comparing clinical measures of ROM to motion capture methods. Both methods display limitations, with the goniometer measurement demonstrating high inter-tester variability\textsuperscript{46} and motion capture demonstrating sources of error
from skin motion artifacts\textsuperscript{47}. Moreside and McGill 2011, investigated motion capture and goniometric measurements of hip ROM, and found they are comparable\textsuperscript{33}. Moreside and McGill concluded, using the normative data, that the goniometer is a viable tool for measuring hip ROM and is highly comparable to a motion capture system ($R^2=0.8845$)\textsuperscript{33}. They did mention that when measuring hip extension, the understanding that goniometer measures can overstate extension by up to 4 degrees, should be considered. In the current study, significant differences approximated 10 degrees or more. As such, while some error in directly comparing passive to active ROM measures may exist, the size of this difference was large enough to suggest it was a true effect. Every procedural effort was made to standardize the collection of passive ROM measures. Special focus was placed on ensuring limited movement of the pelvis during hip flexion, and the lumbar spine during extension testing. Pelvic and lumbar motion palpation was used and when motion started, the measurement was recorded. To further minimize unwanted motion, participants were encouraged to provide verbal feedback to the physiotherapist as to their perception of pelvis rotation.

Dynamic ROM decreases when moving from a healthy control group to populations with moderate and severe OA. Individuals with SOA have significantly lower passive and dynamic extension ROM compared to the asymptomatic and moderate OA groups. Individuals with SOA also demonstrated less passive flexion compared to MOA and asymptomatic groups. Similarly, the MOA group demonstrated less passive flexion ROM than the asymptomatic group. A strong positive relationship between passive and dynamic hip extension was found in the MOA and SOA groups, however, this was not found in the asymptomatic group. This relationship demonstrated that as passive ROM decreased, so did dynamic range during gait. This research has clinical relevance for orthopaedic practice, yet further research is required to more fully
understand whether ROM treatment programs have the ability to slow or reverse the gradual loss of dynamic hip extension during gait.

Acknowledgements:

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References:


**Figure Legends:**

Figure 1: Ensemble averaged sagittal plane hip range of motion for the asymptomatic group (average of both legs), the moderate OA group and the severe OA group. Degrees of flexion (+ve) and extension (-ve) on the y-axis. Percent of gait cycle on the x-axis. The asymptomatic group is represented by the solid line, moderate OA group by the dashed line, and the severe OA group by the dotted line.

Figure 2: (A) Relationship between dynamic (x-axis) and passive (y-axis) extension of the asymptomatic group. (B) Relationship between dynamic (x-axis) and passive (y-axis) extension of the moderate OA group. (C) Relationship between dynamic (x-axis) and passive (y-axis) extension of the severe OA group.
Figure 1:

Figure 2:
Table 1: Mean (standard deviation) participant demographics, anthropometrics, and range of motion measurements.

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<th>Variable</th>
<th>Asymptomatic</th>
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\(^a\) = Significant difference between the SOA group and both MOA and ASYM groups  
\(^b\) = Significant difference between all three groups  
\(^c\) = Significant difference between the SOA group and the MOA group.  
\(^\text{†}\) = Significant differences between passive and dynamic measurements within a group.  
\(^*\) = Four individuals obtained magnetic resonance imaging to investigate hip OA.