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Authors

Popovic, Tijana
Samaan, Michael A
Link, Thomas M
et al.

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Patients with Symptomatic Hip Osteoarthritis Have Altered Kinematics during Stair Ambulation

Tijana Popovic, MS,
Musculoskeletal Quantitative Imaging Research, Department of Radiology and Biomedical Imaging, University of California-San Francisco, San Francisco, CA.

Michael A. Samaan, PhD,
Musculoskeletal Quantitative Imaging Research, Department of Radiology and Biomedical Imaging, University of California-San Francisco, San Francisco, CA; and Department of Kinesiology and Health Promotion, University of Kentucky, Lexington, KY

Thomas M. Link, MD, PhD,
Musculoskeletal Quantitative Imaging Research, Department of Radiology and Biomedical Imaging, University of California-San Francisco, San Francisco, CA

Sharmila Majumdar, PhD,
Musculoskeletal Quantitative Imaging Research, Department of Radiology and Biomedical Imaging, University of California-San Francisco, San Francisco, CA

Richard B. Souza, PT, PhD
Musculoskeletal Quantitative Imaging Research, Department of Radiology and Biomedical Imaging, University of California-San Francisco, San Francisco, CA; and Department of Physical Therapy and Rehabilitation Science, University of California-San Francisco, San Francisco, CA

Abstract

Background: Osteoarthritis (OA) is a degenerative joint disease. Understanding contributing factors to slowing or stopping disease progression is crucial. There has been no research describing lower extremity kinematics of the hip, knee, and ankle during stair ambulation in individuals with hip OA.

Objective: To explore the differences in lower extremity kinematics between participants with clinical and morphological findings of hip OA and controls.

Design: A cross-sectional study.

Setting: Clinical research laboratory.

Participants: Participants with radiographic and symptomatic signs of hip OA (n = 42) and healthy controls (n = 30) were enrolled.

Address correspondence to: T.P.; tijana.popovic@ucsf.edu.

Disclosure

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Interventions: Participants underwent hip magnetic resonance imaging (MRI). The Scoring Hip Osteoarthritis with MRI (SHOMRI) method was used to assess cartilage abnormalities. Self-reported measures of hip pain and function were obtained using the Hip Disability and Osteoarthritis Outcome Score (HOOS). Participants were assigned into a symptomatic hip osteoarthritis group (HOA) with SHOMRI>0 and HOOS≤80, and a control group (CG) with SHOMRI = 0 and HOOS>90. Patients underwent 3D motion analysis during stair ascent/descent at self-selected speed.

Main Outcome Measures: The primary outcome measurements were peak hip, knee, and ankle kinematics. General Estimation Equations were used to compare kinematics between groups ($P \leq .05$).

Results: The HOA group ascended stairs with a more internally rotated hip (CG = 1.77 ± 6.3; HOA = 4.97 ± 4.2; $P = .02$), more abducted hip (CG = −5 ± 2.7, HOA = −3.5 ± 3; $P = .02$), and a more externally rotated knee (CG = −8.02 ± 3; HOA = −10.63 ± 6.3; $P = .02$) and ankle (CG = −11.8 ± 6.1; HOA = −16.3 ± 5.6; $P = .01$). Similarly, HOA participants descended stairs with a more extended knee (CG = −15.5 ± 4.9; HOA = −12 ± 4.9; $P = .01$), and more externally rotated knee (CG = −10.1 ± 4.4; HOA = −13.1 ± 6.6; $P = .04$) and ankle (CG = −13.5 ± 5.3; HOA = −17.9 ± 5.5; $P = .002$).

Conclusion: Participants with hip OA-related morphology and symptoms ambulate stairs utilizing abnormal lower extremity mechanics.

Introduction

Hip osteoarthritis (OA) is a common and progressive joint disease[1–3] that causes pain,[2,3] impaired mobility, and gait abnormalities reducing the quality of life for those affected by the disease.[3] More than 332 000 hip replacements are performed every year primarily because of severe hip OA, with the number affected by this disease expected to climb to more than a quarter of the U.S. adult population by 2030.[3–5] Radiography is the most commonly used method by radiologists and other clinicians to assess the severity of OA through use of the Kellgren-Lawrence (KL) grading system.[6] Radiographic assessment of hip OA is limited to joint space narrowing and osteophytes that occur at the later stages of disease, thereby making it difficult to assess early signs of hip joint degenerations.

Many studies have reported altered biomechanical patterns in people with hip OA during tasks of daily living such as level walking[7–9] and stair climbing.[10,11] Stair ascent/descent is described as a more challenging task that places a greater demand on the hip joint and may be associated with increased pain in individuals with hip OA.[11] Identifying biomechanical abnormalities in movement patterns in the early stages of the disease may be valuable in informing clinicians about how to develop better rehabilitation and treatment protocols to prevent disease progression.[12] Numerous studies have reported gait abnormalities of hip, knee, and ankle kinematics, in participants with varying severity of hip OA. It was reported that individuals with mild to moderate hip OA exhibited increased peak hip flexion and decreased hip extension during level walking when compared to asymptomatic controls.[7,13,14] Other studies reported increased hip and ankle abduction, increased knee flexion, and knee external rotation in individuals with hip OA.[14,15]

Despite the numerous studies that have assessed sagittal and frontal plane hip mechanics in individuals with hip OA, study findings related to transverse plane kinematics in individuals with hip OA are inconsistent and not well understood. Watelain et al[7] reported that individuals with moderate unilateral hip OA ambulated with twice as much hip internal rotation at push-off during level walking when compared to healthy controls. In contrast, Leigh et al[14] found that individuals with radiographic hip OA and pain have increased hip external rotation during level walking. Currently, only one study has investigated transverse plane kinematics during stair ascent and descent in individuals with symptomatic radiographic hip OA and in healthy controls without radiographic hip OA.[10] Although there was no significant difference between groups in hip external and internal rotation, this study reported decreased hip joint transverse plane range of motion (ROM) in individuals with hip OA. Lower extremity joint movement patterns in individuals with hip OA are not fully understood. In addition, use of magnetic resonance imaging (MRI) based methods to classify hip OA may provide insight into the potential biomechanical mechanisms involved in the early stages of hip OA.

To our knowledge, there has been no prior work performed to describe lower extremity transverse plane joint kinematics of the hip, knee, and ankle during stair ambulation in individuals with hip OA. Therefore, the purpose of our study was to explore the differences in lower extremity kinematic patterns between individuals with symptomatic hip OA, classified using patient reported outcomes and MR-based assessment of hip joint abnormalities and asymptomatic healthy controls during stair ascent and descent. We hypothesized that our hip OA group will have a more flexed and less extended hip and knee, more abducted hip and everted ankle, and more externally rotated hip, knee, and ankle joint when compared to the control group.

Methods

Participants

All participants provided written informed consent prior to participation and our institutional Committee on Human Research approved the project. Participants from two longitudinal studies on hip OA (N = 193) were selected, whereby participants underwent either a unilateral or bilateral hip joint MR examination depending on the study protocol. Participants in both of these longitudinal studies were recruited from the local community.[16,17] Each participant had an anterior-posterior pelvic radiograph to assess radiographic signs of hip OA using the Kellgren-Lawrence scale (KL).[6] A musculoskeletal radiologist with over 25 years of experience performed the KL grading. Participants were excluded from the study if they presented with any of the following: body mass index (BMI) >35 kg/m^2, previous hip surgery, hip joint KL grade > 3 (end stage of OA)[6] knee or ankle joint pain, presence of any neurological condition that may affect lower extremity function, MRI contraindications (ie, pacemaker, pregnancy, etc.), age < 18, and any other lower extremity condition that would prevent the participant from completing the MRI and biomechanics procedures.

Clinical Assessment

All participants completed the Hip disability and Osteoarthritis Outcome Scale (HOOS) to assess hip joint pain, symptoms, function during daily living, function in sport and recreation, and hip-related quality of life.[18] The HOOS uses a numerical scale from 0–100 to assess self-reported hip joint measures, where a score of 0 indicates severe hip-related pain and disability and a score of 100 indicates no hip-related pain or disability in any of the five categories. The HOOS was previously shown to be a reliable method to assess overall hip joint function and pain in individuals with hip OA.[18]

MR Imaging

All participants underwent MR imaging using a 3 Tesla scanner (GE Healthcare, Waukesha, WI) and an eight-channel cardiac coil (GE Healthcare, Waukesha, WI). Participants were positioned supine in the scanner with their lower extremities secured in an anatomically neutral position to prevent movement during the scan. Sagittal, oblique-coronal, and oblique-axial images were obtained using a previously published protocol.[19] The protocol included intermediate-weighted fat-suppressed, fast-spin echo (FSE) sequences with repetition times of 2400–3700 ms, slice thickness of 3–4 mm, field of view of 14–20 cm, matrix size 288 × 224 pixels, and echo time (TE) of 60 ms.

Image Analysis

The Scoring Hip OA with MRI (SHOMRI) method[19] was used to score articular cartilage abnormalities. Cartilage abnormalities were scored on a range from 0 to 2, where 0 represents no cartilage lesion, 1 a partial-thickness cartilage lesion, and 2 a full thickness lesion. The SHOMRI method has been validated with arthroscopic findings and was found to be a reliable and valid method to assess cartilage abnormalities.[20]

Gait Analysis

A 10-camera motion capture system (Vicon, Oxford, UK) and a force plate (AMTI, Watertown, MA) were used to obtain 3D marker position and ground reaction force (GRF) data at 250 and 1000 Hz, respectively. A marker set consisting of 41 reflective markers (29 tracking and 12 calibration) was used to track 3D segment position. The 12 calibration markers were placed on the greater trochanters, medial and lateral femoral epicondyles, medial and lateral malleoli, and the heads of the first metatarsal. Tracking markers included single retroreflective markers and clusters. Single markers were placed on the anterior superior iliac spines, iliac crests, L5/S1 joint, and the heads of the fifth metatarsal. Thigh and shank segment tracking was performed using clusters consisting of four markers each, and foot segment tracking was performed using clusters consisting of three markers each that were placed on the heel shoe counters.[13] A one-second standing calibration trial was obtained and then the calibration markers were removed.

Each participant was asked to ascend and descend a set of stairs at a self-selected, purposeful pace where the first step of the staircase consisted of a force plate (Figure 1). Each participant was provided with as many practice trials as needed to ensure comfort with the stair tasks. Upon completion of the practice trials, five successful trials were obtained for each participant. A successful trial was a trial where participants: (1) maintained their speed

within 5% of their self-selected speed; and (2) did not skip over any steps of the staircase or hold onto the railings while ascending or descending the staircase. The first two data collection trials were utilized to assess each participant's self-selected speed. If the speed of the second trial was not within 5% of the first trial, a new self-selected speed was determined utilizing the succeeding trials of the stair task until the participant's speed between two consecutive trials was within 5% of one another. We did not standardize the start and end points for stair negotiation.

The standing calibration trial was used to create a seven segment (ie, pelvis, bilateral thighs, shanks, and feet) kinematic model in Visual 3D (C-Motion, Germantown, MD). Local joint coordinate systems were created and an unweighted least squares optimization method was used to describe the position and orientation of each segment.[21] Joint rotations were solved using a Cardan sequence of X-Y′-Z″ representing the medial-lateral, anterior-posterior, and superior-inferior directions.[22] The hip joint centers were calculated as one-quarter of the distance from the ipsilateral to the contralateral greater trochanter.[23] Ankle[24] and knee[22] joint centers were calculated using the midpoint between malleoli (for ankle) and medial and lateral epicondyle (for knee). Lower extremity joint kinematics were assessed during the stance phase consisting of initial contact and toe-off. Initial contact was defined when the foot struck the force plate (the first stair) and the vertical ground reaction force exceeded 20 Newtons. The participants performed five successful trials bilaterally. The raw marker trajectory and GRF data were passed through fourth-order, zero-lag, low-pass Butterworth filters at 6 and 50 Hz, respectively.[8] Dependent variables assessed were hip, knee, and ankle sagittal, frontal, and transverse plane kinematics. Each trial was normalized to 101 frames. Average maximum and minimum values (peak angles) were calculated across all five trials for each participant and were used for statistical analysis. Hip flexion, hip adduction, knee extension, knee adduction, ankle dorsiflexion, and ankle inversion was considered positive. Hip, knee, and ankle internal rotation angles were considered positive.

Statistical Analysis

For grouping purposes, we selected only participants who had a HOOS score lower than 80 or higher than 90. The participants were stratified into two groups using the MR-based assessments of hip cartilage lesions and self-reported symptoms. The control group (CG) was defined as those participants without cartilage lesions (SHOMRI = 0) and scores greater than 90 in all five HOOS subscores.[25] Participants in the cartilage lesion + pain group (HOA) exhibited partial or full thickness lesions (SHOMRI = 1, 2) and poor self-reported outcomes in any of the five HOOS subscores (HOOS≤80). A total of 42 participants with morphological and symptomatic signs of hip OA and 30 participants without morphological or symptomatic signs of hip OA were enrolled in this study. We obtained bilateral data on 12 participants (seven from the OA group and five from the control group). The remaining participants provided unilateral data.

To assess differences in demographics (age, gender, and body mass index [BMI]), walking speed, and sagittal, frontal, and transverse plane kinematics between the CG and HOA groups, we used general estimating equations (GEE) in SPSS 16.0 (IBM Corporation, Armonk, NY). The GEE accounted for statistical dependency in the calculations within the

same participant, including accounting for bilateral data from some participants and unilateral data from others.[26] An alpha level was set a priori at 0.05.

Results

No significant differences were found between the HOA and CG groups for age, gender, BMI, or walking speed ($P > .05$) during stair ascent and stair descent (Table 1). Although not statistically significant, it should be noted that the average age in the HOA group was approximately 5 years older than in the CG.

During stair ascent, the HOA group ambulated with lower peak hip external rotation (CG = −7.49 ± 4; HOA = −5.19 ± 4.24; $P = .02$), lower peak hip abduction (CG = −4.98 ± 2.69 HOA = −3.45 ± 3.04; $P = .02$), higher peak knee external rotation (CG = −8.02 ± 3; HOA = −10.63 ± 6.29; $P = .02$), and higher peak ankle external rotation (CG = −11.8 ± 6.15; HOA = −16.24 ± 5.62; $P = .01$) when compared to the CG group. In addition, the HOA group exhibited a higher peak hip internal rotation (CG = 1.77 ± 6.29; HOA = 4.97 ± 4.15; $P = .02$), and a higher peak ankle external rotation (CG = −1.21 ± 6.85; HOA = −5.82 ± 6.21; $P = .01$), whereas there was no difference found in knee internal rotation angles (Figure 2).

During stair descent, the HOA group exhibited a higher peak knee external rotation (CG = −10.1 ± 4.38; HOA = −13.1 ± 6.61; $P = .04$), a higher peak knee extension (CG = −15.5 ± 4.9; HOA = −12 ± 4.9; $P = .01$), and a higher peak ankle external rotation (CG = −13.49 ± 5.32; HOA = −17.86 ± 5.49; $P = .002$) and plantar flexion (CG = −14.05 ± 4.07; HOA = −16.55 ± 5.2; $P = .037$), whereas peak hip external rotation was not different between the two groups. Furthermore, the HOA group had lower peak knee internal rotation (CG = 1.3 ± 5.55; HOA = −1.47 ± 6.33; $P = .049$) and peak ankle internal rotation (CG = −4.91 ± 5.99; HOA = −9.41 ± 6.04; $P = .004$), whereas peak hip internal rotation was similar between both groups (Figure 3). Means and SDs of the peak hip, knee, and ankle joint kinematics for both stair ascent and descent are shown in Tables 2 and 3, respectively.

Discussion

Our study showed that during stair ascent the HOA group ambulated with a more internally rotated and less abducted hip joint as compared to the control group. No differences in hip kinematics were found during stair descent. In addition, participants in the HOA group showed greater knee peak external rotation during both ascent and descent, along with lower internal rotation and lower peak extension during descent. Also, the HOA group ascended and descended stairs with higher peak ankle internal and external rotation angles.

Even though previous research in individuals with hip OA reported their most significant results in sagittal and frontal plane kinematics, our results showed significant differences in only three variables in the sagittal and frontal planes during both tasks. The HOA group ascended stairs with a lower peak hip abduction when compared to the control group. Our results differ from findings of Hall et al who did not find a significant difference in hip abduction while ascending and descending stairs.[10] Compared to participants in the Hall et al study, our study participants were on average 15–20 year younger, which could explain our different results. During stair descent, our HOA group had significantly lower knee

extension and higher plantar flexion when compared to the control group. However, it is important to note that some of the differences observed were quite small (~1.5°), which is similar to our reproducibility error (1–2°) and have an unknown clinical significance.

We speculate that differences in transverse plane kinematics are a combination of pathological movements related to cartilage abnormalities (hip OA) and pain (hip internal rotation), which may result in compensatory kinematics (knee and ankle external rotation). Our preliminary results suggest that abnormal transverse plane kinematics may represent possible therapeutic targets that can serve as intervention points for gait retraining in patients with symptomatic hip OA.

Our results showed that during stair ambulation, individuals with hip cartilage lesions and symptoms internally rotated their hip joint and externally rotated their knee and ankle joints when compared to healthy controls. These findings are in agreement with previous research from Watelain et al[7] who reported significantly greater peak hip internal rotation during walking in individuals with hip OA. Weak hip musculature correlates with increased severity of hip OA as documented in recent studies.[27,28] It is possible that our participants had weak hip external rotators and therefore their thigh rotated inward. However, this remains speculative as external rotation strength was not assessed in this cohort. It is also important to note that while the differences observed in the transverse plane were mostly outside of our reproducibility errors ranges (1.5–2.5°), the clinical significance of the observed group differences (2.2–4.6°) is unknown.

Similar findings were observed during stair descent where the HOA group exhibited significantly larger peak knee and ankle external rotation. Hip internal rotation was not significantly different between the two groups. Absence of significantly higher peak hip internal rotation during the stair descent may be explained by the relatively less flexed position of the hip joint during stair descent as compared to stair ascent.[10] Although we did not statistically analyze kinematic differences between stair ascent and descent, on average, all of the lower extremity joints were slightly more externally rotated during stair descent compared to stair ascent.

Our findings are in disagreement with previous research in participants with hip OA during similar, but less demanding tasks. To our knowledge, there is only one previous study that assessed hip, knee, and ankle transverse plane kinematics in participants with hip OA.[14] Leigh et al[14] found that during level walking participants with mild to moderate hip OA have increased peak hip and knee external rotation, whereas ankle rotation was not different from the control group. HOA participants in our study exhibited greater peak hip internal rotation, whereas participants in the Leigh et al study[14] had a more externally rotated hip. The reason for these contradictory results could be the different nature of the task and greater demand on the lower extremity during stair ambulation or the differences in sample inclusion and exclusion criteria. Stair ambulation (especially during stair ascent) places greater demand on lower extremity joints and muscles responsible for stabilizing the leg during the stance phase.[29] During level walking, hip muscle strength in participants with hip OA may be sufficiently strong enough to keep the hip joint stable and to prevent it from collapsing inward. It was reported in earlier research that individuals tend to alter their

movement patterns to avoid pain.[30,31] It is possible that in our study, the HOA group increased both knee and ankle external rotation in order to prevent excessive hip internal rotation (due to weak hip musculature) and corresponding hip joint pain. This rotational misalignment in the hip, knee, and ankle joints could possibly increase the risk of developing hip OA and pain, or cause unknown consequences at the knee or ankle.[32,33] It is also important to note that our study included participants with both hip joint cartilage lesions and hip joint symptoms, whereas previous research used radiographic criteria alone as a means to define group assignment. Numerous studies have documented that hip OA has both structural and symptomatic consequences without a uniform presentation.[3,10,34] We therefore selected a control group that was both free of structural damage and without symptoms consistent with OA.

Our study has some limitations, which should be considered when interpreting these results. The cross-sectional nature of this study limits our ability to infer causation. Our findings suggest the need for longitudinal studies aimed at understanding the changes in and associations of transverse plane lower extremity joint kinematics with hip joint cartilage health. The soft tissue artifacts present during gait analysis, as well as potential errors in estimation of the hip joint centers, may affect the transverse plane kinematic data presented in the current study. Also, the lack of hip joint muscle strength and electromyography data in the current study limits our ability to understand the relationship between muscle strength, function, and lower extremity joint kinematics in the HOA group. Future studies should include electromyography to assess activity of the gluteal muscles that are responsible for hip joint rotation during stair ambulation. Given our approach to evaluate peak hip angles throughout the stance phase, we cannot ensure the peaks were temporally aligned across all participants. Future interventional studies should further explore where these peaks occur across individuals and populations. Another limitation of this study is that we excluded participants with HOOS scores in between 80 and 90, which means that our findings may not be representative for people with HOOS scores within that range.

Conclusion

In conclusion, our study identified abnormal joint kinematics in individuals with morphological signs and symptoms of hip OA during stair ascent and descent. The differences between groups were mostly observed in the transverse plane. Individuals with symptomatic hip OA ascend and descend stairs with more internally rotated hip joints and more externally rotated knee and ankle joints, possibly in an effort to avoid excessive hip joint loading and pain. In addition, the symptomatic hip OA group had a less abducted hip joint during stair ascent, possibly as a result of weak hip musculature. However, the effects of these altered joint mechanics on long-term hip joint health is unclear and requires further investigation. It is important that clinicians understand the abnormal movement patterns present in individuals with hip OA during stair ambulation, as it is possible that there might be clinical utility in targeting these biomechanical deviations in their rehabilitation programs. However, it remains unknown if excessive hip internal rotation during stair ambulation lies on the causal pathway to hip OA or if excessive hip internal rotation occurs after the onset of the hip OA disease. Future studies may consider a gait retraining

intervention to evaluate the response of joint damage and symptoms to reduced hip internal rotation during stair ambulation.

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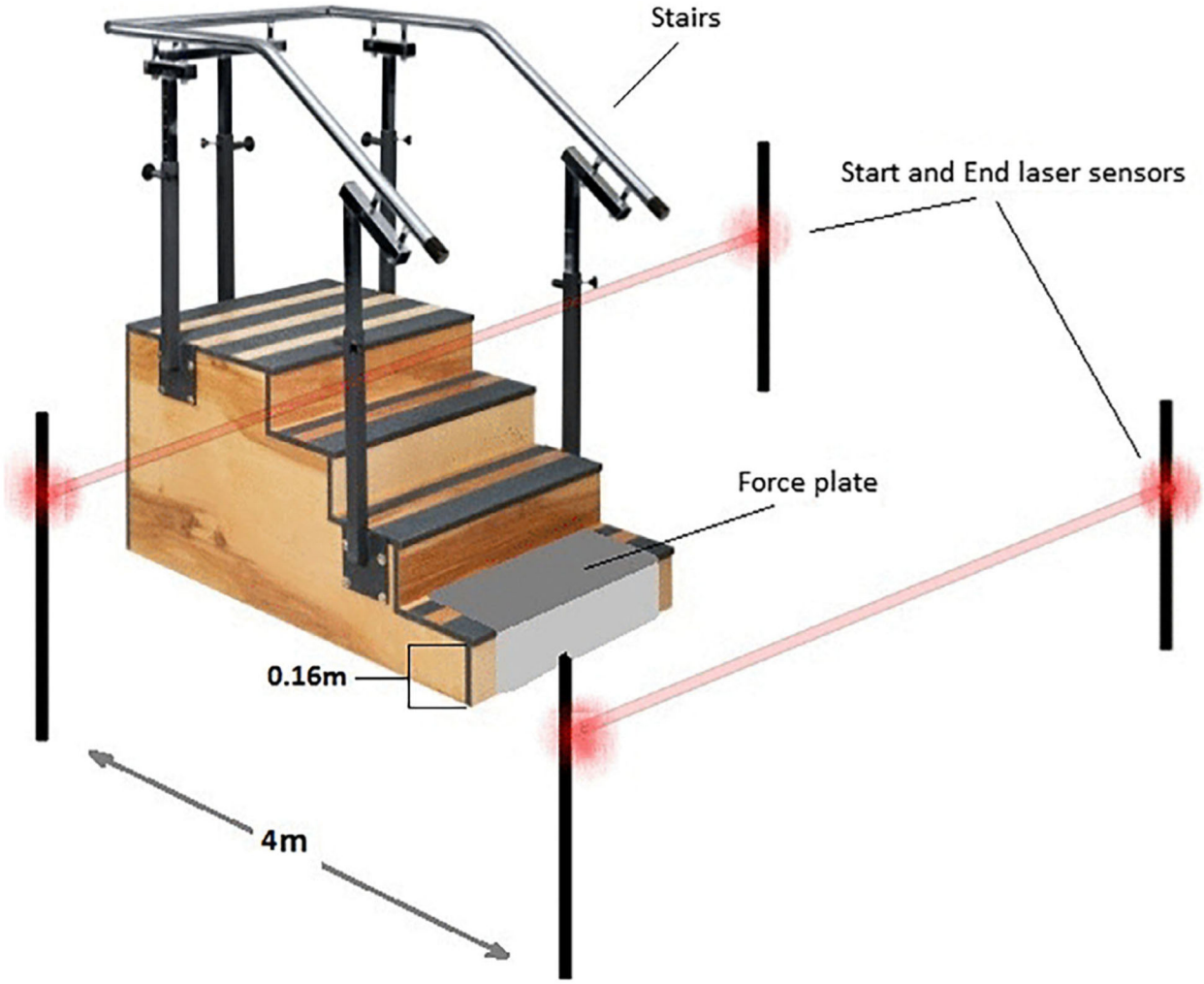

Figure 1.
Staircase setup and positions of the timing gates.

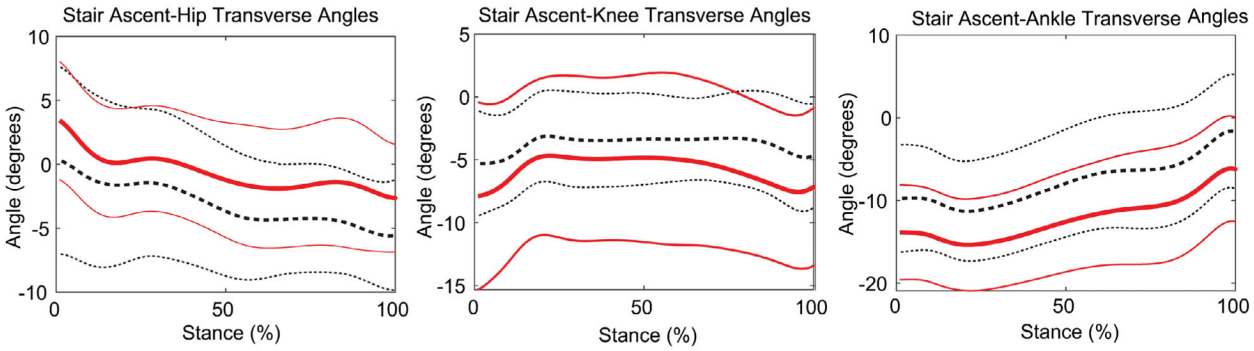

Figure 2.

Stair ascent kinematics. Mean values during stance phase in transverse plane kinematics at hip, knee, and ankle in control group (thicker dashed line) and hip osteoarthritis (HOA) group (thicker solid line); thinner lines represent ± one SD. Positive value = internal rotation; negative value = external rotation.

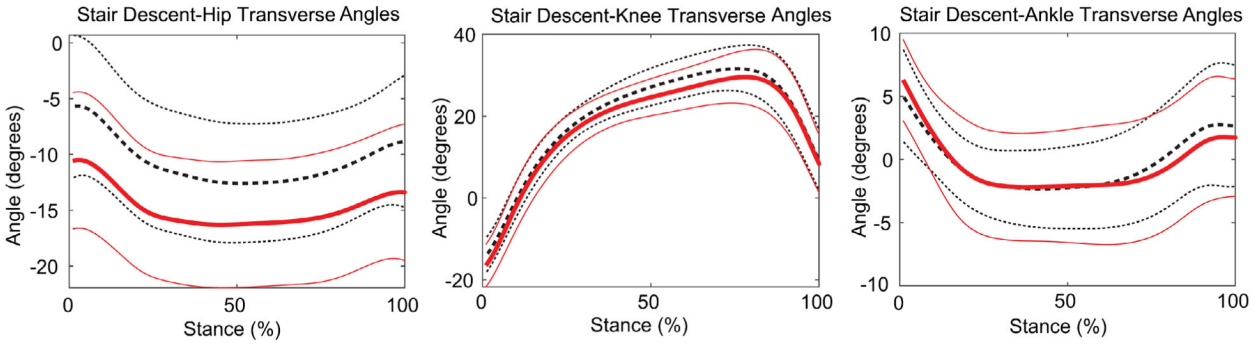

Figure 3.

Stair descent kinematics. Mean values during stance phase in transverse plane kinematics at hip, knee, and ankle in control group (thicker dashed line) and hip osteoarthritis (HOA) group (thicker solid line); thinner lines represent ± one SD. Positive value = internal rotation; negative value = external rotation.

Table 1

Participants' demographics

Variable	CG (n = 30) Mean (SD)	HOA (n = 42) Mean (SD)	β-value	*P* value
Gender (M:F)	14:16	19:23	−0.01	.92
Age (y)	44.7 (13.5)	49.6 (15.2)	−4.95	.19
BMI (kg/m^2)	23.7 (2.4)	24.5 (3.3)	−0.79	.29
Velocity stair ascent (m/s)	1.28 (0.2)	1.25 (0.2)	−0.04	.49
Velocity stair descent (m/s)	1.23 (0.2)	1.19 (0.2)	0.40	.40
HOOS	96.17 (11.1)	74.22 (16.3)		
Pain	97.25 (8.0)	77.98 (16.3)		
Symptoms	96.33 (7.4)	81.36 (18.4)		
ADL	96.81 (9.9)	72.66 (20.3)		
Sports	95.67 (15.0)	72.86 (20.8)		
QOL	94.79 (15.4)	66.22 (20.4)		

β and *P* values for group effects as determined by GEE test; GEE = generalized estimating equations; SD = standard deviation; HOA = experimental group; CG = control group; M = males; F = females; BMI = body mass index; HOOS = Hip disability and Osteoarthritis Outcome Score; ADL = Activity of Daily Living; QOL = Quality of Life.

^{s}Indicates statistically significant difference $P < .05$.

Table 2

Stair ascent kinematics

Joint	Peak Angles (degrees)	CG (n = 30) Mean (SD)	HOA (n = 42) Mean (SD)	Mean difference (95% CI)	β-value	*P* value
Hip	Flexion	47.05 (7.2)	48.3 (5.6)	−1.25 (−4.92, 2.43)	−1.25	.51
	Extension	2.93 (8.68)	4.18 (6.82)	−1.25 (−5.66, 3.17)	−1.25	.58
	Abduction	−4.98 (2.69)	−3.45 (3.04)	−1.53 (−2.81, −0.24)	−1.53	.02*
	Adduction	5.94 (4.1)	7.89 (4.62)	−1.95 (−3.96, 0.06)	−1.95	.057
	Internal rotation	1.77 (6.29)	4.97 (4.15)	−3.2 (−5.83, −0.57)	−3.2	.017*
	External rotation	−7.49 (4.01)	−5.19 (4.24)	2.3 (−4.23, −0.39)	−2.3	.019*
Knee	Flexion	−49.09 (5.82)	−48.58 (6.66)	0.51 (−3.64, 2.62)	−0.51	.75
	Extension	−11.61 (5.67)	−10.55 (4.68)	−1.06 (−3.77, 1.65)	−1.06	.44
	Abduction	−0.9 (4.03)	−0.88 (3.43)	−0.02 (−1.92, 1.88)	−0.02	.98
	Adduction	6.15 (5.85)	6.40 (5.28)	−0.25 (−3.08, 2.58)	−0.25	.86
	Internal rotation	−0.16 (3.35)	−2.38 (6.53)	2.22 (−0.12, 4.56)	2.22	.063
	External rotation	−8.02 (3)	−10.63 (6.29)	−2.61 (0.4, 4.83)	2.61	.021*
Ankle	Flexion	13 (3.6)	12.8 (4.42)	0.2 (−1.66, 2)	0.17	.85
	Extension	−13.32 (5.9)	−12.50 (5.7)	−0.82 (−3.86, 2.21)	−0.83	.059
	Inversion	−4.27 (3.4)	−5.56 (3.73)	1.29 (−2.87, 0.29)	−1.29	.11
	Eversion	−5.16 (2.78)	−6.19 (3.04)	1.03 (−0.35, 2.41)	1.03	.14
	Internal rotation	−1.21 (6.85)	−5.82 (6.21)	4.61 (1.08, 8.18)	4.61	.011*
	External rotation	−11.81 (6.14)	−16.24 (5.62)	4.43 (1.33, 7.54)	4.43	.005*

Mean (SD) of minimum and maximum values (in degrees) for hip, knee, and ankle in sagittal, frontal, and transverse plane kinematics during stair ascent; β and P values for group effects as determined by GEE test; GEE = generalized estimating equations; SD = standard deviation; HOA = experimental group; CG = control group; CI = confidence interval.

*Indicates statistically significant difference $P < .05$; positive value-internal rotation, hip and ankle flexion, knee extension, adduction, and ankle inversion; negative value-external rotation, hip and ankle extension, knee flexion, abduction, and ankle eversion.

Table 3

Stair descent kinematics

Joint	Peak Angles (degrees)	CG (n = 30) Mean (SD)	HOA (n = 42) Mean (SD)	Mean difference (95% CI)	β-value	*P* value
Hip	Flexion	12.19 (7.96)	11.37 (5.3)	0.82 (−2.93, 4.57)	0.82	.67
	Extension	−11.36 (10.27)	−13.86 (7.9)	2.5 (−2.26, 7.26)	2.5	.3
	Abduction	−4.95 (3.47)	−3.51 (3.51)	−1.44 (−3.1, 0.21)	−1.44	.09
	Adduction	3.56 (3.93)	5.1 (3.55)	−1.54 (−3.45, 0.38)	−1.54	.12
	Internal rotation	−0.60 (4.91)	1.4 (4.17)	−2 (−4.35, 0.35)	−2	.095
	External rotation	−9.78 (4.8)	−7.99 (3.78)	−1.79 (−3.8, 0.23)	−1.79	.08
Knee	Flexion	−59.43 (10.33)	−57.7 (8.39)	−1.73 (−6.45, 2.99)	−1.73	.47
	Extension	−15.47 (4.87)	−12.01 (4.91)	−3.46 (−6.04, −0.88)	−3.46	.009*
	Abduction	−2.92 (4.84)	−1.94 (3.65)	−0.98 (−3.25, 1.28)	−0.99	.39
	Adduction	3.03 (5.03)	3.25 (3.96)	0.22 (−2.53, 2.1)	−0.21	.86
	Internal rotation	1.3 (5.5)	−1.43 (6.33)	2.73 (0.01, 5.45)	2.73	.049*
	External rotation	−10.06 (4.38)	−12.82 (6.64)	2.76 (0.19, 5.32)	2.76	.035*
Ankle	Flexion	−14.05 (4.07)	−16.55 (5.2)	2.5 (−0.55, 4.72)	2.49	.037*
	Extension	33.14 (5.32)	31.06 (5.61)	2.08 (0.15, 4.83)	2.08	.12
	Inversion	6.57 (3.54)	7.18 (3.16)	−0.61 (−2.23, 1)	−0.62	.46
	Eversion	−3.77 (2.85)	−4.66 (3.68)	0.89 (−0.71, 2.49)	0.89	.28
	Internal rotation	−4.91 (5.99)	−9.41 (6.04)	4.5 (1.33, 7.68)	4.51	.005*
	External rotation	−13.49 (5.32)	−17.86 (5.49)	4.37 (1.56, 7.17)	4.36	.002*

Mean (SD) of minimum and maximum values (in degrees) for hip, knee, and ankle in sagittal, frontal, and transverse plane kinematics during stair descent; β and *P* values for group effects as determined by GEE test; GEE = generalized estimating equations; SD = standard deviation; HOA = experimental group; CG = control group; CI = confidence interval.

*Indicates statistically significant difference $P < .05$; positive value-internal rotation, hip and ankle flexion, knee extension, adduction, and ankle inversion; negative value-external rotation, hip and ankle extension, knee flexion, abduction, and ankle eversion.