

TITLE

Hip joint biomechanics during gait in people with and without symptomatic femoroacetabular impingement

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Highlights

- Symptomatic FAI is associated with minimal impairments in gait biomechanics.
- Individuals with symptomatic FAI demonstrate decreased sagittal plane hip ROM.
- The magnitude of the gait changes are small and of unknown clinical relevance.
- Gait assessment/training is unlikely to be beneficial in symptomatic FAI.
- More demanding functional tasks targeting impinging positions should be examined.

ABSTRACT

Femoroacetabular impingement (FAI) is a morphological hip condition that can cause hip/groin pain and impaired function in younger active adults, and may lead to stiffness, muscle weakness, structural damage, and hip osteoarthritis. Understanding the impairments associated with FAI is crucial to guide treatment and rehabilitation strategies. Evidence is limited and conflicting about whether hip biomechanics are impaired during walking in people with symptomatic FAI. The objective of this study was to determine whether kinematics and kinetics during gait differ between people with symptomatic FAI and control participants. Fifteen participants diagnosed with symptomatic cam-type or combined (cam plus pincer) FAI who were scheduled for arthroscopic surgery and 14 age-, and sex-matched disease-free controls underwent three-dimensional gait analysis. Tri-planar hip kinematics and kinetics were compared between the two groups. There were limited significant between-group differences with respect to spatiotemporal variables. Participants with FAI walked with less range of motion in the sagittal plane during a gait cycle, but did not exhibit any significant kinematic differences in the frontal or transverse planes. There were no systematic differences in kinetics between the groups in any plane. Findings suggest that individuals with symptomatic FAI have minimal impairments in gait biomechanics. Although these individuals demonstrate reduced hip joint motion in the sagittal plane, the size of the difference is small and its significance for symptoms and function is unclear. More pronounced deficits in hip kinetics and kinematics may be evident during functional tasks that challenge the hip towards the position of impingement.

Key Words: Femoroacetabular impingement (FAI); Biomechanics; Gait; Hip; Range of motion

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1. Introduction

Femoroacetabular impingement (FAI) is a morphological hip condition that can cause hip and/or groin pain and impaired function, often in younger active adults [1-3]. In addition, FAI can lead to stiffness, muscle weakness, and structural damage [2-4]. FAI results when the proximal femur abuts against the acetabular rim [2, 5]; although this description is straightforward, underlying mechanisms remain poorly understood. Impingement typically occurs with combined movements of hip flexion, adduction and internal rotation [1, 2], but a consistent definition of FAI is lacking [6]. Classifications of FAI include “cam” impingement, caused by abnormal morphology of the femoral head, “pincer” impingement characterized by excessive acetabular coverage of the femoral head, and “combined” impingement which is a combination of both cam and pincer abnormalities [2, 3, 5, 7]. A study of 1076 people with symptomatic FAI undergoing surgery, reported a prevalence of cam FAI of 47.6%, combined FAI of 44.5%, and pincer FAI of 7.9% [8]. The less common pincer-type typically presents in an older demographic (30-40 years) [9] and likely associates with unique biomechanical strategies in the absence of a bony deformity of the femoral head.

Not all FAI is associated with symptoms. The proportion of young adults with imaging features consistent with FAI is high. A recent review of 26 studies states the prevalence of asymptomatic cam deformity as 37% in a general healthy and pain-free population, and almost 55% in athletes [9]. Similarly, a study of over 2000 hip radiographs from healthy young adults showed that cam-type FAI was present in 35% of males and 10% of females [10]. Amongst all surgical hip conditions, 36% of elite athletes who underwent hip arthroscopic surgery over a five-year period were treated for FAI [11], which highlights the prevalence of the morphological abnormalities associated with FAI, regardless of symptoms.

This is particularly important, as it has been suggested that structural damage due to FAI may be a risk factor for the onset and development of hip osteoarthritis [2-4].

Surgery is a common treatment for FAI, and there is evidence for favourable short-term improvements in function and pain [2, 12]. The long-term clinical outcomes are largely unknown [12]. A clear understanding of the impact of FAI on hip function prior to surgery is critical to evaluate the effectiveness of surgery as a means to restore normal hip biomechanics [13]. Furthermore, understanding the impairments associated with FAI is likely to assist in the design of conservative treatments, including postoperative rehabilitation strategies and preoperative exercise programs that aim to improve surgical outcomes.

Most research consistently demonstrates that individuals with FAI (both symptomatic and asymptomatic) have reduced hip range of motion (ROM), particularly towards directions of impingement [14-16]. However, the literature is sparse regarding kinematic and kinetic impairments during dynamic functional activities. Gait is the most common repetitive voluntary movement of the lower limbs and an essential activity of daily living. There is limited and conflicting evidence regarding alterations to hip ROM during gait in individuals with symptomatic FAI [13, 17-20]. Interpretation of current evidence is limited by the use of narrow patient samples that render it difficult to generalize results to the broader FAI population, and the failure to use diagnostic imaging to ensure healthy controls in the comparison groups have no evidence of asymptomatic morphological FAI. The objective of the current study was to compare kinematics and kinetics during gait of individuals with symptomatic FAI (of mixed classifications) and asymptomatic controls with no evidence of morphological FAI confirmed by imaging.

2. Methods

2.1 Participants

Fifteen volunteers (18-33 years) diagnosed with cam or combined-type FAI, who were scheduled for arthroscopic surgery were recruited. All participants were recruited from the surgical records of the study orthopaedic surgeon (JO). Participants were included if they tested positive for a clinical impingement test (presence of hip pain during passive hip flexion to 90°, followed by force applied into adduction and internal rotation [7]), and had definitive signs of FAI on imaging [2, 3] (alpha angle $\geq 55^\circ$ (cam FAI), and centre edge angle $\geq 39^\circ$ and/or positive crossover sign (combined FAI)). Participants were scheduled for arthroscopic surgery for treatment of FAI, and were to have persistent groin pain and activity limitation for at least 3 months, with no substantial improvement following conservative treatment. Participants with bilateral FAI were evaluated on the more symptomatic side. Potential participants were excluded if they had only pincer-type FAI because the absence of bony deformity of the femoral head likely manifests with unique biomechanical strategies. Exclusion criteria included: (i) history of hip surgery; (ii) moderate or severe radiographic osteoarthritis, defined as Kellgren-Lawrence grade 3 or 4 [21]; (iii) lower limb injury/pain limiting function in the past month; (vi) other forms of arthritis, diabetes, cardiac circulatory conditions that limit everyday activities.

Fifteen healthy asymptomatic control participants with no history of hip and/or groin pain or hip joint surgery were recruited from the community. Participants were comparable to the FAI group with respect to age, sex, activity level, and leg dominance. Control participants underwent a standard hip MRI with a 3-Tesla MR scanner (Siemens Magnetom TrioTim syngo MR B17) and a 16-channel body coil (coupled with a Siemens Spine array) to ensure they did not have morphological FAI. The alpha angle was measured on the oblique sagittal plane MRI [22, 23]; the coronal plane was used to measure the lateral centre edge angle

(CEA). Where required, the localizer sequence was used to correct for pelvic obliquity [22]. Angles were measured using OsiriX imaging software (©Pixmeo SARL, Switzerland) and the Orthopaedic Studio v1.2 Plugin (Spectronic AB, Helsingborg, Sweden). Participants were eligible if their alpha angles $\leq 50^\circ$ and centre edge angles $\leq 40^\circ$, which are commonly considered to represent “normal” values [24, 25].

The institutional medical research ethics committee approved the study, and participants provided written informed consent. One control participant was excluded after they were unable to undergo MRI.

2.2 Procedures

Participants underwent three-dimensional (3D) gait analysis at a preferred, self-selected walking speed wearing standardised footwear (Dunlop Volley, Pacific Brands, Australia). Reflective markers were applied bilaterally to lower limb body segments in 3-marker triads according to Besier et al. [26]. Kinematic data were measured using a 12-camera motion analysis system (Vicon, MX, Oxford, UK) sampling at 120 Hz. Two force plates (AMTI Inc., Watertown, MA, USA) embedded in a 10-meter walkway collected ground reaction force data at 3000 Hz (to equate with electromyographic data not reported here). Six trials were obtained for the study limb. Hip joint centres were estimated using a dynamic approach where participants performed flexion, abduction, circumduction, and extension of the leg ensuring the foot remained elevated [26]. Knee joint centres and functional flexion/extension axes were defined from helical knee axes according to a previous approach [26] where participants performed squats through a range of approximately 60° of knee flexion motion.

A modified Tegner Activity Scale was administered to both group to assess physical activity level [27]. Physical function of participants with FAI was also assessed with the international Hip Outcome Tool (iHOT-33) [28] and the Copenhagen Hip and Groin Outcome Score

(HAGOS) [29]. An 11-point self-reported Numerical Rating Scale (NRS) anchored with “no pain” at 0 and “worst pain possible” at 10 was administered verbally to FAI participants immediately following the walking trials to quantify their pain during the task.

2.3 Data Analysis

Marker trajectories and ground reaction forces were low-pass filtered at 6 Hz using a 2nd order, dual-pass Butterworth filter. Spatiotemporal variables of stride length (m), step length (m), and cadence (strides/min) for the study limb, were measured for each trial along with walking speed (m/s) and hip joint kinematics. Inverse dynamics programmed in Vicon Body Builder [26] were used to calculate net external hip moments. Joint moment data were normalised to body weight (N) multiplied by body height (m) and expressed as a percentage (Nm/(BW.HT) (%)). Data analysis was undertaken using Microsoft Excel, version 14.4.1 (Microsoft Corporation, 2010) and Matlab, version 2013a (The Mathworks, Inc.).

Peak hip angles (measured as femur relative to pelvis) and ROM (measured as peak-to-peak excursion) in each plane (sagittal, frontal, transverse), and peak hip moments were averaged over the trials. All data were explored for normality. Between-group comparisons were made using independent t-tests and Mann-Whitney U tests where required. Spatiotemporal and demographic variables were examined for between-group differences using independent t-tests and Pearson’s chi-square as appropriate. Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS), version 22 (IBM, New York, USA). An alpha level of 0.05 was used to detect significance in all analyses.

3. Results

Demographic and clinical data for participants are summarized in Table 1. The FAI and control groups were comparable for age, BMI, sex, and dominant leg tested. Sporting activity level (Tegner activity scale) was significantly higher in the control than FAI group at the time of testing ($P < 0.05$). Symptom duration for the FAI participants averaged 30 months, and ranged from 5-48 months prior to testing. The iHOT-33 and HAGOS subcategory scores and pain scores during testing for FAI participants are reported in Table 1. Eight participants with FAI (53%) had bilateral symptoms and radiographic findings. In these cases the data are reported for the more symptomatic limb that was scheduled for imminent surgery.

There were no significant between-group differences for walking speed or other spatiotemporal variables of gait (Table 2). With respect to joint kinematics, participants with FAI walked with less ROM in the sagittal plane during a gait cycle (mean differences 4.2 [95% CI 0.3 to 8.0] degrees, $P=0.04$). Data for participants with FAI did not differ significantly from that of controls with respect to peak flexion angle during stance or swing, peak extension angle during swing, or any of the peak frontal or transverse plane angles. Although not compared statistically, an additional observation was that the between-participant variation in hip angle was substantially less for the FAI than control group at the maximal angles in the directions of impingement in the frontal (hip adduction) and transverse (internal rotation) planes (Figure 1).

There were no significant between-group differences for peak external joint moments in any plane. Visual inspection of ensemble averaged hip kinematic (Figure 1) and kinetic curves (Figure 2), time normalised over a gait cycle, reveals similar waveforms with comparable spatial and temporal characteristics.

4. Discussion

The aim of this exploratory cross-sectional study was to determine whether hip joint biomechanics differ between individuals with symptomatic morphologic FAI and individuals, who are confirmed by MRI to be free from this condition. The results show that gait biomechanics are only minimally affected in participants with FAI. Although participants with FAI used reduced hip ROM in the sagittal plane during gait, this was an average of 4° less ROM than that recorded for controls. There was no evidence of a systematic reduction in hip joint ROM in any other plane, or for altered hip joint moments during gait.

Although FAI is a relatively newly defined phenomenon [2], a growing research base has focused on impairments in hip ROM. These data demonstrate that people with symptomatic FAI have less available hip ROM, as defined by CT modelling towards positions of hip impingement (flexion/internal rotation in 90° flexion), relative to individuals without FAI [14-16]. There has been limited investigation of biomechanical impairments during dynamic functional activities such as gait in this population.

Five studies have compared gait biomechanics between people with and without symptomatic FAI. Our observed 4° reduction in sagittal plane ROM in those with symptomatic FAI is consistent with two previous studies that also reported deficits in sagittal plane motion of 4° [13, 19]. Although statistically different from the disorder-free participants, the clinical significance of this hip ROM impairment is unknown. This impairment in ROM might be too small to adversely impact important domains including symptoms and function. Although Hunt et al. [18] did not report total sagittal plane ROM, they found decreased maximum hip extension angle in participants with FAI. We did not find this difference in our data, but total ROM and maximum amplitude in the same plane are clearly not independent and the

difference in outcome between studies may depend on features such as the definition of reference position. Sagittal plane ROM alterations in FAI were not reported in the two remaining studies [17, 20] that specifically investigated participants with cam-type FAI.

Pain is a common symptom associated with FAI [7]. Thus it is feasible that impairments in ROM may be attributed to pain. However, given that individuals with asymptomatic FAI have also demonstrated reduced ROM compared to those without FAI morphology, it appears that factors other than pain also play a role in ROM reduction [14]. As participants in our study reported minimal pain (1/10 on the NRS) following gait testing, it is unlikely that pain during the task contributed to the reduced sagittal plane hip ROM we observed. However, this does not exclude the possibility that participants learned to modify ROM prior to testing due to pain, or experienced minimal pain during the task because of reduced ROM. Reduced hip ROM in participants with symptomatic FAI may be explained in part by bony deformity, though it is unclear whether the maximal hip angles measured in our cohort reached a range where mechanical restriction may occur. It is likely that cartilage lesions, which in combination with cam lesions have been previously associated with biomechanical movement abnormalities [20], damage to the surrounding soft tissue, abnormal joint contact forces, and altered neuromuscular function all contribute to a complex multifaceted mechanism for disruption to hip joint homeostasis in those with symptomatic FAI.

Similar to a study by Kumar et al. [20], our findings demonstrate no significant differences in frontal plane ROM during gait in people with symptomatic FAI. Although other studies have reported reduced ROM in this plane [13, 17-19], patient groups were older (4-10 years) than our cohort, and included individuals diagnosed with pincer FAI [18, 19] or were comprised of cam-type only patients [13, 17]. We found no evidence for reduced ROM in the transverse plane during gait. Although two studies have reported reduced peak hip internal rotation in

symptomatic FAI compared to controls with uncertain hip morphology [18, 19], differences were not found in other studies [13, 17, 20]. It is widely accepted that measurement of transverse plane kinematics is less reliable (curve r^2 between 0.28 and 0.47 [26]) at the hip than the knee and ankle. This may partially explain some discrepancies reported in the literature, especially considering the different marker models used.

An interesting observation was the tendency for less between-participant variation for the FAI than control group at peak hip adduction angle and peak hip internal rotation angle during stance. Although individuals with symptomatic FAI do not significantly reduce their movement into adduction or internal rotation during gait, they are less variable. One interpretation is that there is greater constraint within the FAI group with movements towards the impingement position in the frontal and transverse planes; however reasons for this constraint are currently unclear.

We found no evidence of impaired hip joint kinetics during gait, which is consistent with most prior studies [13, 17, 19, 20]. Only one study has reported an impairment in kinetics, which involved decreased peak hip flexion and external rotation moments [18]. It is possible that abnormalities in kinetics are only evident during tasks that utilize the full hip ROM. It has been suggested that individuals with symptomatic FAI may activate muscles to protect the hip as it approaches end range to compensate for any muscle deficiencies [13]. It is thus likely that most studies (including our own) did not observe altered hip kinetics during gait because gait does not require movement to the end of available ROM. In this case, there may not be a sufficient trigger to initiate protective muscle activation that would alter joint forces.

On reflection, it appears unlikely that the demands of walking are sufficiently challenging to reveal impairments in hip joint biomechanics in people with symptomatic FAI. Motion in this task is generally pain free, and does not approach the end of available range, where painful

impingement might occur. More challenging functional tasks that target positions of impingement may be more likely to demonstrate alterations in hip ROM and kinetics. Of two studies that assessed squatting [20, 30] one reported significantly greater hip adduction angle and internal rotation moment in people with symptomatic FAI than controls without FAI [20]. In addition, Rylander et al. [19] reported abnormal sagittal and transverse plane kinematics during stair climbing in FAI, which suggests the impairments in FAI may be better identified in tasks requiring a greater range of hip motion. Such observations may be more likely to aid in optimisation of disease management.

The results of this exploratory cross-sectional study add to the emerging body of evidence of physical impairments related to symptomatic FAI. Our findings and those of others suggest that gait assessment is unlikely to provide information to guide treatment of symptomatic FAI. Future research using more provocative tasks may warrant consideration. Although we observed a 4° reduction in sagittal plane hip ROM during gait, its significance for symptoms and function is unclear. While abnormal hip function in FAI appears poorly identified through assessment of gait biomechanics, assessment of neuromuscular function might provide information of modifiable targets for treatment.

Interpretation of this study is limited by its small sample size but strengthened by the confirmation of absence of morphological abnormality in the controls. The cross-sectional design means we cannot determine whether the reduced sagittal plane hip ROM we observed in our participants with symptomatic FAI precedes or follows pathology development. Future research should aim to evaluate longitudinal changes in hip biomechanics, including the evaluation of more provocative functional and sporting tasks that place the hip in positions of impingement.

5. Conclusion

Individuals with symptomatic FAI have minimal impairments in gait biomechanics when compared to asymptomatic controls with no evidence of FAI on imaging. Although our FAI group demonstrated small differences in hip joint kinematics in the sagittal plane, the implications for symptoms and function is unclear. Evaluation of more demanding functional tasks placing the hip in positions of impingement might reveal greater insight into the nature of the biomechanical impairments in this population.

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Figure 1. Ensemble average and standard deviation (shown as absolute value of standard deviation relative to zero to emphasise change in amplitude of variation over gait cycle) hip angles for sagittal (top), frontal (middle) and transverse (lower) planes over a gait cycle for control and FAI participant groups. The vertical line indicates the time of peak angle towards the position of maximum impingement (flexion, adduction and internal rotation) for the FAI group.

Figure 2. Ensemble average (\pm standard deviation) hip kinetic patterns for sagittal (top), frontal (middle), and transverse (lower) planes over a gait cycle for control and FAI participant group.

Table 1. Demographic and clinical characteristics of the FAI and control groups (mean (standard deviation), unless otherwise stated).

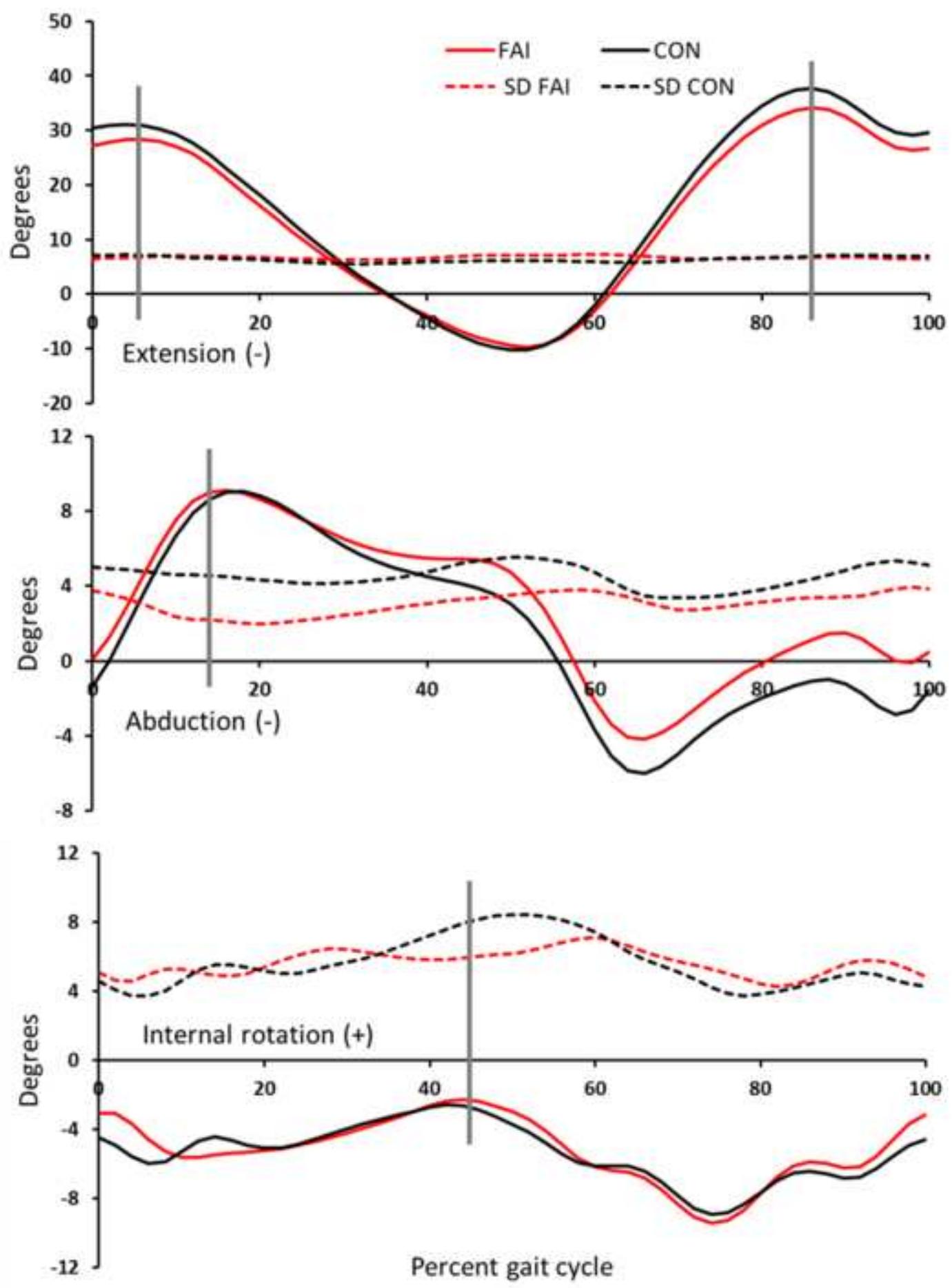
Characteristic	FAI group			Control group
	All	Cam	Combined (Cam + Pincer)	
	n=15	n=11	n=4	
Age (years)	24.7 (4.9)	24.3 (4.6)	25.8 (6.4)	27.1 (4.5)
Males, n (%)	11 (73%)	8 (73%)	3 (75%)	10 (71%)
Height (cm)	176.0 (8.7)	175.6 (9.6)	177.0 (6.8)	176.1 (8.3)
Body mass (kg)	76.0 (11.8)	75.5 (12.4)	77.5 (11.8)	72.6 (11.6)
Body mass index (kg/m ²)	24.4 (2.5)	24.3 (2.4)	24.7 (3.2)	23.2 (1.9)
Test hip (right:left)	9:6	7:4	2:2	10:4
Dominant side tested, n (%)	10 (67%)	8 (73%)	2 (50%)	9 (64%)
Bilateral FAI (yes:no)	8:7	7:4	1:3	-
Symptom duration (months)	29.9 (15.0)	25.6 (15.7)	34.5 (12.4)	-
Physical activity level (Modified Tegner Scale) [¶]	5.2 (2.1)	4.6 (2.1)	6.8 (1.7)	6.6 (1.0)*
The international Hip Outcome Tool (iHOT-33) [†]	51.9 (23.0)	50.8 (24.2)	54.8 (22.3)	-
Copenhagen Hip and Groin Outcome Score (HAGOS) [†]				
Symptoms	53.6 (21.0)	53.2 (21.2)	54.5 (23.9)	-
Pain	66.5 (18.2)	64.8 (19.5)	71.3 (15.6)	-
ADL	70.3 (22.8)	70.0 (25.8)	71.3 (14.4)	-
Sport	49.6 (19.5)	49.7 (20.8)	49.2 (18.1)	-
Participation	33.3 (26.2)	31.8 (25.8)	37.5 (30.6)	-
QOL	41.7 (20.1)	41.4 (22.4)	42.5 (14.4)	-
Pain during gait testing (Numerical Rating Scale) [§]	0.9 (1.8)	1.1 (2.1)	0.3 (0.5)	-

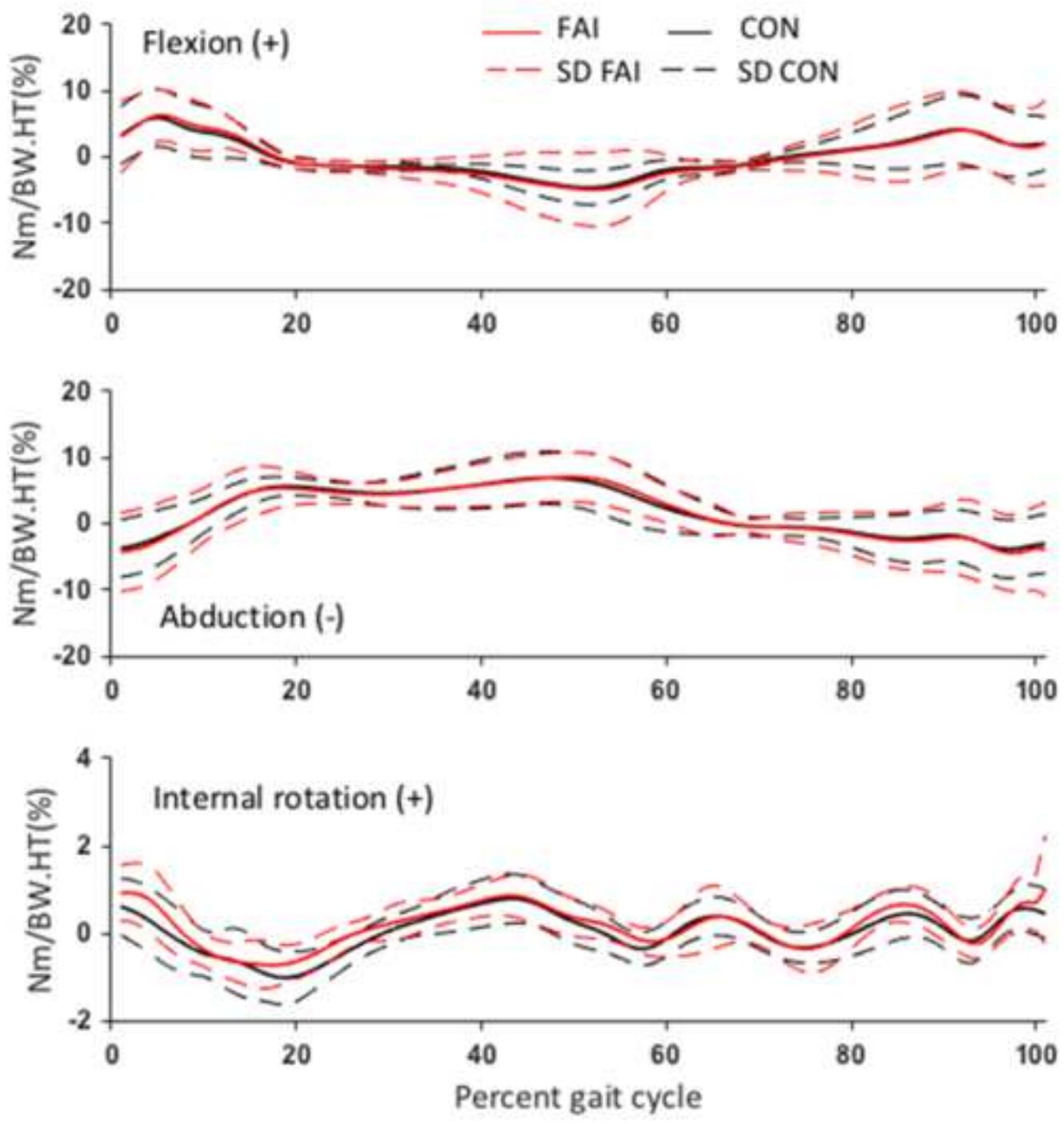
*significant difference $P < 0.05$ compared to all FAI; [¶]Tegner scale is 0-10 with zero representing disability and 10 representing competitive sport at the professional level; [†]iHOT-33 and HAGOS scales are 0-100 with zero representing extreme hip and/or groin problems and 100 representing no hip and/or groin problems; [§]Numerical Rating Scale is 0-10 with zero representing no pain and 10 representing worst pain possible; ADL, activity of daily living; QOL, quality of life.

Table 2. Gait variables for FAI and control groups (mean (standard deviation)).

Variable	FAI	Control	P-value
Spatiotemporal			
Walking speed (m/s) *	1.3 (0.2)	1.4 (0.3)	0.53
Stride length (m)	1.5 (0.1)	1.5 (0.2)	0.32
Step length (m)	0.73 (0.07)	0.76 (0.10)	0.34
Cadence (strides/min)	55.4 (3.3)	56.1 (4.9)	0.66
Hip Joint Kinematics (°)			
<i>Sagittal Plane</i>			
Max flexion (stance)	28.6 (6.8)	31.4 (7.1)	0.30
Max flexion (swing)	34.3 (6.8)	37.8 (6.9)	0.18
Max extension (cycle)	9.8 (7.0)	10.5 (6.2)	0.80
Total ROM (cycle)	44.2 (5.5)	48.3 (4.6)	0.04
<i>Frontal Plane</i>			
Max adduction (stance)	9.7 (2.0)	9.7 (4.4)	0.98
Max abduction (swing)	5.1 (3.0)	6.9 (3.4)	0.14
Total ROM (cycle)	14.9 (3.3)	16.6 (3.7)	0.19
<i>Transverse Plane</i>			
Max internal rotation (stance)*	0.37 (5.3)	1.0 (6.0)	0.59
Max external rotation (swing)*	11.3 (4.8)	11.2 (3.9)	0.65
Total ROM (cycle)	12.2 (2.8)	13.0 (5.1)	0.64
Hip Joint Moments (Nm/BW.BH (%))			
Peak flexion moment*	7.1 (3.2)	6.4 (4.1)	1.00
Peak extension moment*	4.3 (2.2)	5.0 (2.3)	0.33
Peak adduction moment*	8.6 (1.7)	8.5 (2.2)	0.75
Peak abduction moment	6.0 (2.6)	5.2 (2.2)	0.39
Peak internal rotation moment	1.0 (0.4)	1.0 (0.3)	0.83
Peak external rotation moment*	1.2 (0.4)	1.4 (0.5)	0.16

*Mann-Whitney U tests; **bold indicates significance** P < 0.05





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