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# Femoroacetabular impingement syndrome is associated with alterations in hindfoot mechanics: A three-dimensional gait analysis study☆

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ABSTRACT

*Background:* Gait analysis studies in patients with femoroacetabular impingement syndrome focused until today on alterations in pelvic and hip mechanics, but distal articulations in this syndrome were not explored. Viewing the inter-relationships between foot and hip mechanics and the importance of the subtalar joint in load attenuation at heel strike and during forward propulsion thereafter, alterations in hindfoot mechanics in this syndrome may have clinical significance.
*Methods:* Three-dimensional gait kinematics were explored with emphasis on hindfoot mechanics in a group of 15 men with cam-type femoroacetabular impingement and compared to 15 healthy men.
*Findings:* Subjects with femoroacetabular impingement had decreased pelvic internal rotation (effect size = 0.70) and hip abduction (effect size = 0.86) at heel strike, and increased sagittal pelvic range of motion during the stance (effect size = 0.81), compared to controls. At the hindfoot level, subjects with femoroacetabular impingement had inverted position at heel strike compared to neutral position in controls (effect size = 0.89), and reduced maximum hindfoot eversion during the stance (effect size = 0.72). Range of motion from heel strike to maximum eversion was not different between the groups (effect size = 0.21).
*Interpretation:* Young adult men with cam-type femoroacetabular impingement syndrome present excessively inverted hindfoot at the moment of heel strike and reduction in maximum eversion during the stance phase. Viewing the deleterious effects of hindfoot malalignment on load attenuation during the stance, custom-designed insoles may be a consideration in this population and this should be investigated further.


## 1. Introduction

Femoroacetabular impingement (FAI) syndrome is an increasingly recognized diagnosis in orthopedics. In this condition, deviation from a round, circular femoral head, or over-coverage of the anterior acetabular rim, results in abnormal contacts between these two parts of the joint at the extremes of hip motion. With repetitive motion, this can lead to chondro-labral injury, and subsequently cartilage wear and osteoarthritis of the hip (Ganz et al., 2008).

Several investigations provided insights into hip joint mechanics in patients with FAI syndrome during level walking. They observed limitations in hip internal rotation (Hunt et al., 2013), sagittal motion (Hunt et al., 2013; Kennedy et al., 2009; Brisson et al., 2013), and coronal plane motions (Hunt et al., 2013; Kennedy et al., 2009; Brisson et al., 2013). Less is known however, about the consequences this syndrome may have on the mechanics of distal articulations along the lower extremity. Since it was shown that abnormal gait mechanics in patients with FAI syndrome persisted after surgical correction of hip dysmorphism (Brisson et al., 2013), it is possible that such abnormalities are associated with mechanical alterations at other joints and segments of the lower limb, some of which indeed may not be corrected early after FAI hip surgery and play a role in the persistence of gait abnormalities. One example was reported recently when patients with FAI syndrome demonstrated abnormal pelvifemoral rhythm which was characterized by increase in pelvic sagittal motion during active hip flexion (Van Houcke et al., 2014). This finding was interpreted as compensational mechanical alteration in pelvic and lower lumbar spine motion aimed to decrease impacts between the femur neck and the acetabular rim during hip flexion. Information about other possible mechanical alterations in distal articulations in these patients is yet lacking. In this regard, the subtalar joint can be of substantial interest since it plays a key role in ground reaction forces attenuation at heel strike, during deceleration, and when preparing the foot for propulsion (Perry and Lafortune, 1995; Freychat et al., 1996). Minor alterations in subtalar motions, as represented by hindfoot eversion, were

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associated with chronic overuse injuries of the femur (Hetsroni et al., 2008), and changes in the orientation of this distal segment towards excessive varus had adverse effects on the peak resultant ground reaction forces during the stance phase (Perry and Lafortune, 1995). Moreover, some investigators showed how an increase in hindfoot eversion, induced by hindfoot wedges in healthy subjects, was associated with increase in femur internal rotation and in pelvic anterior tilt (Khamis and Yizhar, 2007), both of which can contribute to hip symptoms in patients with FAI syndrome. It seems therefore that deviations in hindfoot mechanics may be inter-related with some changes in hip mechanics in the immediate setting and that specific alterations at this level may affect ground reaction forces and resultant impacts encountered along the lower extremity. But, whether abnormal hip mechanics are inter-related with alterations in hindfoot mechanics in chronic conditions such as FAI syndrome is unknown. To the best of our knowledge, hindfoot mechanics in patients with FAI syndrome have never been investigated. The purpose of this study was therefore to test whether patients with FAI syndrome demonstrate abnormalities in hindfoot mechanics during the stance phase of the gait cycle.

## 2. Methods

### 2.1. Participants

Volunteers with FAI syndrome were recruited from a tertiary sports injuries hip clinic by one orthopedic surgeon (Iftach Hetsroni, M.D.). Inclusion criteria were (1) men only, (2) age 18 years and above, (3) insidious onset of hip pain or worsening pain after low-energy sports trauma, (4) positive hip impingement sign (Klaue et al., 1991) which was relieved after intra-articular injection of a local anesthetic, (5) Tonnis grade 0 to 1 (Tonnis, 1976), (6) magnetic resonance imaging (MRI) showing labral tear, (7) lateral center edge angle (CEA) between 25° and 40°, (8) anterior alpha angle 60° or above which is consistent with anterior cam-type FAI (Notzli et al., 2002), and (9) lack of other lower limb injuries which might interfere with gait pattern. Exclusion criteria were (1) history of fracture or surgery involving the femur or pelvis, and (2) anterior inferior iliac spine (AIIS) Type III variant (Hetsroni et al., 2013a) because of the concern this may represent a specific type of FAI with altered hip mechanics combining intra- and extra-articular sources of hip impingement (Hetsroni et al., 2012, 2013a). Healthy volunteers comprised a control group. Inclusion criteria in this group were (1) men only, (2) age 18 years and above, (4) the absence of any abnormality on examination of the foot and ankle, knee, and hip joints, with negative hip impingement sign, and (3) lack of lower limb injuries, acute or chronic, or any gross joint abnormality or limb malalignment which might interfere with gait pattern as evaluated by a biomechanical engineer (Moshe Ayalon, Ph.D.) and an orthopedic surgeon (Iftach Hetsroni, M.D.). All study participants underwent physical examination by one orthopedic surgeon (Iftach Hetsroni, M.D.). The examination included assessment of the following angular variables with the use of a Goniometer: (1) standing hindfoot angle (inter-observer reliability 0.86, and intra-observer reliability 0.88) (Jonson and Gross, 1997), (2) thigh–foot angle (inter-observer reliability 0.74) (Lee et al., 2009),and (3) range of hip flexion, and hip internal and external rotations with the hip flexed to 90° (intra-class coefficient 0.82, 0.90, and 0.90, respectively) (Holm et al., 2000). Clinical assessment was also used to confirm that there were no abnormal restrictions or asymmetry in subtalar, ankle, and knee range of motions (ROM), and that knee ligament laxity was normal and symmetric. Activity level was assessed with Tegner score (Tegner and Lysholm, 1985), and function was assessed with Hip Outcome Score (HOS) (Martin et al., 2006).

As there was no preliminary data on kinematic abnormalities at the foot level in populations with FAI syndrome, a sample size of 15 symptomatic limbs of 15 men with FAI syndrome and 30 control limbs of 15 healthy men was selected which corresponded to previous investigations' sample sizes that demonstrated significant kinematic variabilities in pelvis and hip motions in young adults with FAI syndrome and that were using similar gait testing procedures and motion analysis systems as the current investigation (Kennedy et al., 2009; Brisson et al., 2013). This sample size then resulted in sufficient power to detect significant differences between group means in hindfoot kinematics in the order of 0.65 of a standard deviation with moderate to large effect size (Cohen's D between 0.7 and 0.9). Consequently, volunteers' recruitment was terminated.

### 2.2. Gait testing procedures

Twenty one photo-reflective markers were placed at anatomical landmarks on each lower extremity from foot to pelvis level. Location of markers on the foot and shank was based on the Oxford Foot Model (Stebbins et al., 2006). The pelvis was represented by markers at the anterior superior iliac spine and the posterior superior iliac spine, and the femur was represented by markers at the lateral mid-thigh and at the lateral femoral epicondyle according to standard plug-in gait protocol (User Manual, Vicon Motion Systems, Ltd., Oxford, UK). Knee alignment device (KAD) (Motion Lab Systems, Inc., Baton Rouge, LA, USA) was mounted on each knee at the beginning of each examination for the purpose of segments alignment setup at the neutral standing position during a static trial and was then removed prior to beginning the dynamic gait analysis (Fig. 1). After setup, each participant was instructed to perform several walking trials until he landed with the foot on a force plate (Kistler Group, Winterthur, Switzerland) located at the mid-distance of a walkway, without altering natural walking pattern. Six-camera optical stereometric system (Vicon Motion Systems, Ltd., Oxford, UK), sampling at 120 Hz, was used to track lower extremity motions. Three gait cycle trials were recorded for each participant. Data was sampled using NEXUS 1.7.1 program with Woltring filter for filling gaps and Butterworth fourth order filter with cut off frequency of 6 Hz built in the program, and reports were processed with Polygon 3.5.1 software (Vicon Motion Systems, Ltd., Oxford, UK). Walking speed and cadence were calculated. Kinematic variables measured during the stance phase of gait included pelvis and hip motions in sagittal, coronal, and transverse planes, hindfoot mechanics in the coronal plane, and

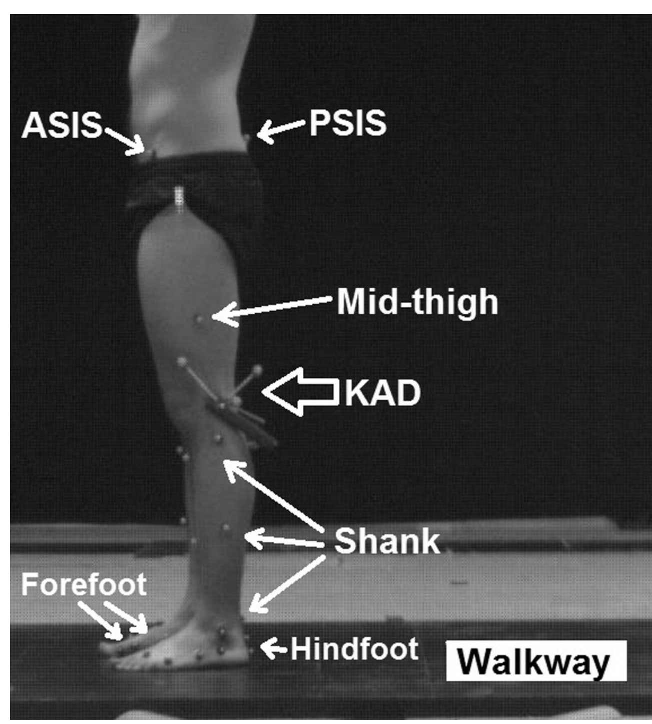


**Fig. 1.** Photoreflective markers locations with knee alignment device (KAD) mounted during a static trial. ASIS, anterior superior iliac spine; PSIS, posterior superior iliac spine.

foot motions in the transverse plane as represented by out-toeing motion.

### 2.3. Data analysis

Intra-class correlation (ICC) and standard errors of measurement (SEM) were used to calculate reliability and accuracy of the motion analysis system over the three walking trials for each subject. After confirming high reliability (ICC 0.95 or higher for most variables) and high accuracy (SEM < 1.0° for most variables) (Table 1), mean and standard deviations for each variable were calculated. The normality of the data distribution was evaluated for skewness and kurtosis. T-tests for independent samples were performed in order to evaluate mean differences in the kinematic variables that showed normal distribution between the 15 injured limbs of the 15 subjects with FAI syndrome and the 30 healthy limbs of the 15 healthy control subjects. In cases where the assumptions of normality were rejected, a non-parametric Mann–Whitney test was implemented. $P$ value < 0.05 was considered statistically significant. Effect size was calculated with Cohen's D procedure. Values above 0.2 were considered small, 0.5 considered medium, and 0.8 considered large. The study was approved by our institutional review board and all participants signed informed consent.

## 3. Results

All study participants were young adult men with normal body mass index (Table 2). Patients in the FAI group were involved in similar levels of sports activities before their symptoms of FAI syndrome appeared as were the healthy control subjects (median Tegner score 7), but they were substantially limited in sports activities during the study period once they presented with symptoms of FAI syndrome (median Tegner score 3; mean HOS-sports subset 59). Activities of daily living were also affected in the FAI group (mean HOS-ADL subset 78). Physical examination revealed decrease of 20° in maximal hip flexion and 15° in maximal hip internal rotation in the FAI group compared to healthy controls ($P < 0.01$). No differences in hindfoot alignment or in thigh–foot angle were observed between the groups. Morphological evaluation on plain radiographs, MRIs, and Computerized Tomography scans confirmed that there were no cases of hip dysplasia or coxa profunda (mean CEA 33°, range 25°–40°; mean Tonnis angle 6°, range 3°–10°). Alpha angle measurements supported cam-type FAI categorization for all patients (mean alpha angle 72°, range 60°–85°), with four of them also showing some pincer component.

**Table 1**
Intra-class correlation coefficients (ICC) and standard error of measurements (SEM) [°].

| Variable | ICC | SEM |
|---|---|---|
| Pelvic kinematics | | |
| Anterior pelvic tilt — at heel strike | 0.97 | 0.82 |
| Anterior pelvic tilt — maximum | 0.98 | 0.71 |
| Pelvic tilt — ROM | 0.79 | 0.32 |
| Pelvic obliquity — at heel strike | 0.87 | 0.68 |
| Pelvic obliquity — maximum | 0.90 | 0.76 |
| Pelvic obliquity — ROM | 0.98 | 0.57 |
| Pelvic internal rotation — at heel strike | 0.92 | 0.82 |
| Pelvic internal rotation — maximum | 0.91 | 0.83 |
| Pelvic rotation — ROM | 0.93 | 1.21 |
| Hip kinematics | | |
| Hip flexion — at heel strike | 0.98 | 0.88 |
| Hip flexion — maximum | 0.98 | 0.89 |
| Hip sagittal ROM | 0.97 | 1.03 |
| Hip abduction — at heel strike | 0.96 | 0.55 |
| Hip abduction — maximum | 0.97 | 0.49 |
| Hip coronal ROM | 0.97 | 0.72 |
| Hip external rotation — at heel strike | 0.95 | 2.89 |
| Hip external rotation — maximum | 0.95 | 2.94 |
| Hip transverse ROM | 0.95 | 1.20 |
| Hindfoot kinematics (coronal plane) | | |
| Hindfoot eversion at static standing position | NA | NA |
| Hindfoot eversion — at heel strike | 0.89 | 1.41 |
| Hindfoot eversion — maximum | 0.95 | 0.99 |
| Hindfoot eversion ROM | 0.95 | 0.07 |
| Forefoot kinematics (transverse plane) | | |
| Out-toeing — at heel strike | 0.98 | 0.74 |
| Out-toeing — maximum | 0.98 | 0.85 |
| Out-toeing — ROM, range of motion | 0.79 | 1.19 |

NA, not applicable since only one measurement was performed at standing, prior to initiating walking trials.

**Table 2**
Mean (sd) demographic, function, physical exam, and hip morphology data.

| Variable | FAI group | Control group |
|---|---|---|
| Demographics | | |
| Age, years | 33 (6) | 27 (6) |
| Body mass index | 24.3 (2.8) | 24 (2.5) |
| Function | | |
| Tegner score, median (range) | 3 (1–7)<br>Pre-symptoms: 7 (5–10)* | 7 (4–9) |
| HOS — ADL subset | 78 (17) | 100 |
| HOS — sports subset | 59 (25) | 100 |
| Physical examination | | |
| Hindfoot valgus angle [°] | 4.2 (1.5) | 4.5 (1.4) |
| Thigh–foot angle [°] | 7.6 (6.5) | 8.7 (4.9) |
| Hip flexion [°] | 122 (14) | 139 (7) |
| Hip internal rotation [°] | 18 (9) | 34 (6) |
| Hip external rotation [°] | 41 (9) | 42 (8) |
| Hip morphology | | |
| Lateral CEA [°] | 33 (4) | |
| Tonnis angle [°] | 6 (3) | |
| Alpha angle [°] | 72 (8) | |
| Isolated cam FAI (n) | 11 | |
| Mixed cam + pincer FAI (n) | 4 | |

HOS, Hip Outcome Score.
CEA, Center-Edge Angle.
* Tegner score before symptoms first appeared.

Temporal variables of gait including walking speed, cadence, stride time, and stance time were not different between FAI subjects and healthy controls (Table 3). At heel strike, FAI subjects had decreased pelvic internal rotation (i.e. transverse plane) and hip abduction compared to controls ($P = 0.04$, and $P = 0.01$, respectively) (Table 4). Pelvic ROM in the sagittal plane was increased in FAI subjects compared to controls during the stance ($P = 0.01$). At the hindfoot, FAI subjects had inverted position at heel strike compared to neutral position in healthy controls ($P = 0.01$), and reduced maximum eversion during the stance ($P = 0.04$). Timing of maximum hindfoot eversion was not different between the groups which occurred at the second quarter of the stance phase. Hindfoot ROM from heel strike to maximum eversion was not different between the groups. Both groups were also similar with regard to eversion angle at static standing position. With regard to forefoot orientation at heel strike, FAI patients were excessively out-toeing compared to controls ($P = 0.05$). Means of peak vertical, peak anterior–posterior, and peak medio-lateral ground reaction forces [N/kg] were not different between FAI and control groups (11.5 ± 0.9 vs. 11.6 ± 1.2, 0.6 ± 0.2 vs. 0.6 ± 0.2, and 1.9 ± 0.3 vs. 2.0 ± 0.5, $P$ = NS, respectively).

## 4. Discussion

The main finding of this study was that young adults with FAI syndrome demonstrated excessively inverted hindfoot alignment at the

**Table 3**
Mean (sd) temporal variables data.

| Variable | FAI group | Control group | $P$ value | Effect size Cohen's D |
|---|---|---|---|---|
| Cadence [steps/min] | 110.8 (8.3) | 112.1 (9.0) | 0.65 | 0.15 |
| Stride time [seconds] | 1.1 (0.1) | 1.1 (0.1) | 0.68 | 0.13 |
| Walking speed [meters/second] | 1.3 (0.1) | 1.3 (0.2) | 0.24 | 0.35 |
| Stance time [seconds] | 0.7 (0.1) | 0.6 (0.1) | 0.78 | 0.09 |

**Table 4**
Mean (sd) pelvic, hip, and foot kinematic data during the stance [°].

| Variable | FAI group | Control group | *P* value | Effect size Cohen's D |
|---|---|---|---|---|
| Pelvic kinematics | | | | |
| Anterior pelvic tilt — at heel strike | 9.7 (5.2) | 11.0 (4.8) | 0.42 | 0.25 |
| Anterior pelvic tilt — maximum | 10.8 (4.9) | 11.6 (4.8) | 0.61 | 0.16 |
| **Pelvic tilt — ROM** | **3.1 (1.0)** | **2.4 (0.7)** | **0.01** | **0.81** |
| Pelvic obliquity — at heel strike | 2.6 (2.1) | 2.1 (1.9) | 0.46 | 0.23 |
| Pelvic obliquity — maximum | 5.3 (1.9) | 5.6 (2.4) | 0.70 | 0.14 |
| Pelvic obliquity — ROM | 9.5 (3.4) | 10.4 (3.7) | 0.42 | 0.26 |
| **Pelvic internal rotation — at heel strike** | **4.3 (2.6)** | **6.3 (2.9)** | **0.04** | **0.70** |
| Pelvic internal rotation — maximum | 5.0 (2.6) | 6.5 (2.8) | 0.09 | 0.56 |
| Pelvic rotation — ROM | 12.4 (3.5) | 12.5 (4.5) | 0.96 | 0.02 |
| Hip kinematics | | | | |
| Hip flexion — at Heel strike | 32.3 (6.2) | 34.3 (6.1) | 0.30 | 0.33 |
| Hip flexion — maximum | 32.3 (6.3) | 34.4 (6.1) | 0.30 | 0.33 |
| Hip sagittal ROM | 41.5 (4.1) | 43.4 (6.3) | 0.30 | 0.36 |
| **Hip abduction — at heel strike** | **0.5 (2.6)** | **2.8 (2.7)** | **0.01** | **0.86** |
| Hip abduction — maximum | 5.2 (2.7) | 4.4 (2.9) | 0.42 | 0.26 |
| Hip coronal ROM | 13.1 (3.8) | 12.6 (4.0) | 0.70 | 0.12 |
| Hip external rotation — at heel strike | 13.0 (10.0) | 12.5 (13.1) | 0.90 | 0.04 |
| Hip external rotation — maximum | 14.8 (8.9) | 15.1 (13.3) | 0.92 | 0.03 |
| Hip transverse ROM | 12.9 (3.2) | 11.8 (5.3) | 0.47 | 0.25 |
| Hindfoot kinematics (coronal plane) | | | | |
| Hindfoot eversion at static standing position | 1.5 (3.6) | 2.3 (3.8) | 0.49 | 0.22 |
| **Hindfoot eversion — at heel strike** | **(−)3.3 (3.1)** | **0.0 (4.2)** | **0.01** | **0.89** |
| **Hindfoot eversion — maximum** | **3.2 (3.2)** | **5.9 (4.4)** | **0.04** | **0.72** |
| Hindfoot eversion ROM | 6.5 (0.3) | 5.9 (0.3) | 0.55 | 0.21 |
| Timing of maximum hindfoot eversion [% stance] | 48.6 (14.5) | 47.8 (14.1) | 0.86 | 0.06 |
| Forefoot kinematics (transverse plane) | | | | |
| **Out-toeing — at heel strike** | **15.6 (7.9)** | **11.6 (5.4)** | **0.05** | **0.61** |
| Out-toeing — maximum | 17.8 (8.9) | 13.8 (5.6) | 0.07 | 0.56 |
| Out-toeing — ROM | 8.8 (2.5) | 8.7 (2.6) | 0.98 | 0.01 |

- Significant differences in bold
- Pelvic tilt, sagittal plane
- Pelvic obliquity, coronal plane
- Pelvic rotation, transverse plane
- ROM, range of motion
- Hindfoot eversion ROM, excursion from heel strike to maximum eversion.

moment of heel strike which was followed by reduction in maximum eversion during the stance phase. This observation can be attributed to several mechanisms. The most straightforward one would be that excessive hindfoot inversion at heel strike in patients with FAI syndrome was a direct consequence of decrease in hip abduction at that moment, both of which take place in the coronal plane. Decrease in hip abduction is in accordance with previous investigations (Kennedy et al., 2009; Brisson et al., 2013), and may be related to decreased electromyography activity of the tensor fascia lata (Casartelli et al., 2011) or weakness of hip abductors in general which characterizes patients with chronic hip pain (Harris-Hayes et al., 2014). Another potential mechanism which could be related to the excessively inverted hindfoot at the moment of heel strike and decrease in maximum eversion is based on the linkage between hindfoot eversion, internal tibia and femur rotation, and anterior pelvic tilt (Khamis and Yizhar, 2007; Tiberio, 1987, 1988; Wright et al., 1964). Since impingement between the femoral neck and the anteromedial acetabular rim is provoked by excessive femur internal rotation in this syndrome (Klaue et al., 1991; Bedi et al., 2011), reduction in hindfoot eversion which is linked to reduction in femur internal rotation may represent a protective mechanism these patients may have been acquired in order to reduce femoral neck abutment against the acetabular rim, and to eventually alleviate hip symptoms. Over-activating the tibialis posterior can assist in achieving such a hindfoot orientation during gait, although this was not tested in the current study. Of note, the excursion of hindfoot coronal motion in patients with FAI syndrome from the moment of heel strike to maximum eversion was not affected, nor hindfoot position at standing. Both findings are consistent with the absence of structural abnormalities at the hindfoot level and suggest that the reduction observed in maximum eversion was indeed most likely secondary to structural abnormalities originating within the hip joint.

The finding that abnormal hip mechanics were linked to alterations in hindfoot eversion in patients with FAI syndrome is new to the best of our knowledge, but whether these alterations in hindfoot mechanics have clinical significance is yet unknown. Some investigators showed that inverted hindfoot at the moment of heel strike with prevention of normal eversion, a situation that resembles the observation in the current study, was associated with increases in peak resultant ground reaction forces (Perry and Lafortune, 1995). Such an effect may have impacts on the segments and joints of the lower extremity during heel strike, and this may have deleterious effects on an already affected hip joint in FAI syndrome. Although differences in peak ground reaction forces between FAI subjects and control subjects during walking were not observed in this study, it is possible that differences between the groups may have been masked during walking in the setup used, but could have been discovered during running, for example. This has some support in a previous observation that showed how abnormally inverted hindfoot with prevention of normal eversion was associated with increases in ground reaction impact loadings during running but not during walking (Perry and Lafortune, 1995). On the other hand, stresses absorbed through the segments and articulations of the lower extremity in such circumstances may not necessarily exceed the symptomatic threshold, and the clinical impact of such alterations in hindfoot mechanics remains vague at this point. Future comparisons of hindfoot mechanics between asymptomatic adults with FAI morphology (Hack et al., 2010) and symptomatic patients with FAI morphology (i.e. FAI syndrome) may be one way to explore whether alterations in hindfoot mechanics play a role in provoking symptoms in these cases. In respect to treating individuals with FAI syndrome, viewing the findings of decrease in hip abduction with the observation of excessively inverted hindfoot, several modifications may be considered. Strengthening of

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hip abductors is one alternative, but another adjunct for treatment can be considering using shock-attenuating custom-made insoles. Of note, attempting to improve shock attenuation by everting the excessively inverted hindfoot at heel strike with the use of lateral heel post should be considered with caution, as this may also be linked to increase in hip internal rotation and further provoke hip symptoms.

Additional kinematic alterations in FAI subjects in this study included: (1) increase in pelvic tilt (sagittal plane) range of motion which may represent a compensational motion as suggested by others (Van Houcke et al., 2014); (2) reduction in pelvic internal rotation (transverse plane) at the moment of heel strike which may represent a tendency to avoid transverse rotation-associated hip symptoms; and (3) increased out-toeing (transverse plane) at heel strike, which may be linked to decrease in pelvic internal rotation at that moment of the stance phase. Of note, despite all the kinematic alterations observed from pelvis to foot level, patients with FAI syndrome demonstrated effective adaptations in their walking profile, with maintenance of all temporal variables compared to healthy controls.

In this study, only men with FAI syndrome were included because men and women with cam-type FAI syndrome demonstrate substantial differences in hip morphology in relation to the extent and location of cam lesions on the femoral neck, as well as in relation to hip version characteristics (Hetsroni et al., 2013b). Moreover, increased peak hip flexion and greater frontal plane motions were observed in women during drop landings (Kernozek et al., 2005). Altogether, such data imply that gait mechanics should ideally be explored separately in men and in women with FAI syndrome and not in mixed groups as performed previously (Hunt et al., 2013; Kennedy et al., 2009; Brisson et al., 2013). In this regard, the uniqueness of this study is emphasized as a large men-only series of patients with FAI syndrome undergoing gait analysis. Moreover, Hip Outcome Score values recorded in this study support the generalizability of our findings to larger cohorts of young adult men with cam-type FAI syndrome and similarly disturbing symptoms (Hetsroni et al., 2013b). Limitations of this study include the descriptive design which does not permit to decide whether the kinematic variability observed reflected adaptive changes at the foot level, or whether this represented a primary normal variant that predisposed individuals to developing FAI syndrome. As to the methodology of using photoreflective skin markers, this bares the limitation that pure joint motions are not practically captured but rather a combination of joint motions with the concomitant motions of soft tissue interface (muscles, fat, and skin). Finally, performing the measurements at natural walking velocity may have been masking some alterations in gait mechanics that could appear in running or during other activities.

## 5. Conclusion

Young adult men with cam-type FAI syndrome present excessively inverted hindfoot at the moment of heel strike and reduction in maximum eversion during the stance phase. Viewing the deleterious effects of hindfoot malalignment on load attenuation during the stance, custom-designed insoles may be a consideration in this population and this should be investigated further.

## Conflict of interest

None of the authors have any financial or personal relationships with other people or organizations that could inappropriately influence this work.

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