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**The Effect of Unilateral cam Femoroacetabular Impingement on the Three-dimensional
Kinematics of the Pelvis and Hip and the Kinetics of the Hip**

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**The Effect of Unilateral Cam Femoroacetabular Impingement on
the Three-dimensional Kinematics of the Pelvis and Hip and the
Kinetics of the Hip**

Matthew J. Kennedy

Thesis submitted to the
Faculty of Graduate and Postdoctoral Studies
In partial fulfillment of the requirements
For the MSc degree in Human Kinetics

School of Human Kinetics
Faculty of Health Sciences
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NOTE:

This thesis is assembled in article format with three independent articles. These articles are entitled: *The Effect of Cam Femoroacetabular Impingement on Hip Kinematics during Maximal Dynamic Range of Motion*; *Hip Biomechanics of Cam Femoroacetabular Impingement During Daily Activities*; *The Effect of Cam FAI on Hip and Pelvic Kinematics during Maximum Squat*. The first section consists of the general introduction, review of literature and methodology which encompass all three articles. The three independent articles are then inserted following this first section. Finally, there is a general discussion and summary section which briefly restates the discussion of results from each of the three articles, as well as mentioning any findings not mentioned in the articles.

The Effect of Cam FAI on Hip and Pelvic Kinematics during Maximum Squat has been accepted by Clinical Orthopaedics and Related Research with minor corrections, and the other two articles are also intended to be submitted for publication.

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Abstract

Cam femoroacetabular impingement (FAI) is caused by decreased concavity of the femoral head-neck junction which results in a jamming of the proximal femur into the acetabulum within normal range of motion (ROM). This condition is known to decrease hip ROM, and has been reported to cause hip pain in a variety of daily activities including sitting and walking, and activities requiring large hip mobility. It is important to determine the effect of cam FAI on self-generated dynamic ROM and common daily activities in order to ascertain its influence on hip functionality. This study measured the effect of cam FAI on total dynamic hip ROM, and the biomechanics of the hip and pelvis during walking, sitting, standing and maximal depth squats. A cam impingement group ($n = 17$) was compared to a matched control group ($n = 14$) using between-group one-way ANOVAs. The level of variability for sitting and standing strategies was also compared between the two groups using F-tests. For maximal dynamic hip mobility, the FAI group had a decreased flexed internal rotation, external rotation and total transverse ROM, as well as decreased hip abduction and total sagittal ROM compared to the matched control group. For walking, the FAI had significantly lower peak hip abduction, frontal ROM, and sagittal ROM than the controls, as well as attenuated pelvic frontal ROM. Reduced hip mobility during gait may be caused by soft tissue restriction, and FAI participants may have decreased pelvic mobility in the frontal plane. For the sit-to-stand and stand-to-sit manoeuvres there were no significant differences in peak kinematic or kinetic values of the hip and pelvis between the two groups. However, the cam FAI group had significantly higher variability in peak angles in the frontal plane, and peak hip extension moments of force during both sitting and standing. This likely resulted from the FAI group having varying sit and stand compensatory strategies. For the

maximal depth squat the FAI group had decreased sagittal pelvic range of motion and could not squat as deep as the control group. Limited pelvic ROM in FAI patients reduces their capacity to perform motions requiring large amplitudes at the hip joint such as maximum squat. Restricted pelvic sagittal ROM could represent a key factor in the pathomechanics of FAI.

Introduction

Femoroacetabular impingement (FAI) is an increasingly recognised cause of hip pain in young active adults (Wisniewski & Grogg, 2006). Although it is currently an elusive impairment to diagnose (Wisniewski & Grogg, 2006), it is gaining recognition in the medical community. Femoroacetabular impingement causes abnormal abrasion at the hip joint, and is a chronic pathology. It often increases in severity with continued wear as the damaged bone compensates by ossifying and accumulating bone volume at the site of contact (Ganz et al., 2003). Moreover, FAI is widely acknowledged as a cause of hip osteoarthritis (Beck et al., 2005; Beck et al., 2004; Ganz et al., 2003; Leunig et al., 2003; Tonnis & Heinecke, 1999). Osteoarthritis (OA) most commonly affects individuals over 65 years of age, but estimations predict that the number of 45-54 year olds diagnosed with OA will double between 1991 and 2031 (Badley, 2005). According to a study administered by the Arthritis Society (*Highlights on the Different Forms of Arthritis.*, 1995), 10 percent of all Canadians are affected by osteoarthritis, and it is estimated that 85 percent of all Canadians will have been affected by it by the age of 70. This high prevalence of osteoarthritis, the direct link of FAI to osteoarthritis (OA), and the increasing recognition of FAI as a common and important pathology makes it imperative to increase our understanding of this ailment.

The close relationship of FAI and OA impacts more than just the health of Canadians. There is also a huge financial burden placed on the healthcare system when expensive surgeries are required to replace osteoarthritic hip joints. According to the Canadian Institution for Health Information (CIHI), ("Total hip and total knee replacements in Canada (report)," 2005), more than 18 694 hip replacements were performed between April 2001 and

March 2002, and 81 percent of total hip replacement surgeries are a result of degenerative OA. The cost of these surgeries is approximately \$158 million, or \$8457 per intervention (Antoniou et al., 2004). Since FAI is a recognised cause of OA, detailed documentation of its physical symptoms to allow for early diagnosis and the refining of minimally invasive surgical techniques is essential to minimize healthcare costs.

As well as acquiring information on the causes and anatomical effects of femoroacetabular impingement, it is also important to learn how this pathology influences movement. Research has been conducted on the effect of FAI on maximal hip range of motion (ROM) using computer simulation based on inputting medical imaging of participants' hips into a computer model (Kubiak-Langer et al., 2007; Tannast et al., 2007). There have also been multiple studies which assessed the passive assisted ROM of the hip in patients with FAI (Eijer et al., 2001; Ito et al., 2004; Philippon et al., 2007; Wyss et al., 2007; Zebala et al., 2007), however, only the study by Philippon mentioned the measuring device used. To our knowledge there have been no direct quantitative biomechanical studies on FAI using the functional dynamic ROM of participants captured live. In order to measure true functional biomechanics, it is essential to capture data live, as opposed to in a virtual setting. Moreover, it is also important to measure the hip ROM which can be actively generated by FAI participants without an applied external force from a clinician. Determining how FAI affects the kinematics and kinetics of common daily activities and maximal dynamic range of motion is important to facilitate early diagnosis and to help develop conservative treatments for this condition.

Research Question

The purpose of this study was to determine whether there were differences in the maximal dynamic ROM and biomechanics of the hip and pelvis during simulated daily activities between participants diagnosed with FAI and a healthy control group. The maximum three-dimensional (3-D) dynamic ROM of the hip was measured, followed by the 3-D angular kinematics of the pelvis and the 3-D kinematics and kinetics of the hip joint during level walking, rising from a seated position, sitting from a standing position, and maximal depth squats. Angular kinematic measures consisted of maximal ROM and peak angular displacement in each plane, and kinetic measures consisted of peak moments of force and mechanical powers in each plane. These values were compared between participants suffering from FAI and a healthy control group.

Hypothesis

Previous research has found that FAI causes a decrease in maximal passive or simulated hip flexion, abduction and internal rotation while the hip is in 90° of flexion compared to a healthy control group (Ito et al., 2004; Kubiak-Langer et al., 2007; Tannast et al., 2007; Wyss et al., 2007). There have been conflicting results on the effect of FAI on simulated or passive external rotation with the knee flexed at 90°, with Kubiak-Langer et al. (2007) and Tannast et al. (2007) reporting no differences, and Ito et al. (2004) reporting a decrease in the FAI group compared to healthy controls. Since there was agreement in the literature on decreased hip flexion, abduction and flexed internal rotation, we hypothesized that that kinematic variables would be decreased in participants with FAI compared to the control group. The contradictory results on flexed external rotation lead us to believe that there would be no differences in this variable between groups. Finally, since there were no reported differences in any other kinematic variables in the passive ROM literature it was

further postulated that there would not be differences in all other dynamic ROM variables between the two groups.

Since the 3-D angular kinematics of the hip during walking is much lower than the maximum range (Al-Zahrani & Bakheit, 2002; Bejek, Paroczai, Illyes, & Kiss, 2006; Crowinshield, Brand, & Johnston, 1978; Judge, Davis, & Ounpuu, 1996; Lee et al., 2005; Mann & Hagy, 1980; Mills & Barrett, 2001; Miyoshi, Shirota, Yamamoto, Nakazawa, & Akai, 2004; Mockel, Perka, Labs, & Duda, 2003; Winter, 1983) it was postulated that there would be no kinematic differences in level walking between participants diagnosed with FAI, and the control group. This idea is supported by results from a qualitative visual gait appraisal of people with FAI (Wisniewski & Grogg, 2006), but according to our knowledge, has never been proven with quantitative data. Since the moments of force and mechanical powers at the hip define the production of hip kinematics, we further hypothesize that there will be no differences in the 3-D kinetics at the hip between the two groups during gait. Since no previous research has quantified the maximal pelvic ROM in FAI patients, no hypotheses were made concerning pelvic kinematics during gait, and an exploratory approach was taken for these variables.

According to the literature, the peak hip flexion required for sitting and standing was larger than during level gait (Kralj et al., 1990; Pai & Rogers, 1991; Shum et al., 2005; Tully et al., 2005), but was still within the reported peak hip flexion in participants with FAI (Philippon et al., 2007). Thus we postulated that there would be no differences in sagittal hip kinematics and kinetics during sitting and standing between the FAI and control group. Since there was no information on transverse and frontal hip mobility required for sitting and standing, we could not form educated hypotheses for these variables. We decided to use a purely exploratory approach for sit and stand frontal and transverse kinematics and kinetics.

Finally, since no literature reported the maximal pelvic mobility in participants with FAI, no hypotheses were formed on pelvic kinematics for the aforementioned activities of daily living (ADL), and this section was also purely exploratory.

The hip flexion angles in the squat literature (Flanagan et al., 2003; Hase et al., 2004; Isear et al., 1997; Salem et al., 2003) are larger than the sit-to-stand and stand-to-sit values but are still within the passive ROM in participants with FAI (Philippon et al., 2007). Interestingly, none of these squat studies tested squat depths lower than knee height. No literature could be found which assessed the hip kinematics of maximal depth squats, which would require higher hip flexion angles than the aforementioned squat studies. Therefore it was postulated that there would be significantly decreased peak hip flexion kinematics and kinetics for the squat manoeuvre in the FAI group as compared to the healthy control. It was further hypothesized that the control group would squat to a lower level than the FAI group. There was also a paucity of literature on the frontal and transverse hip kinematics and kinetics and 3-D pelvic kinematics during squatting. Thus, as with sitting and standing, we decided to take an exploratory approach on these variables.

Rationale

Since the hip and pelvis joints are so important to locomotion, standing upright, and performing many daily activities, it is important to determine how FAI impacts pelvic mobility and hip function. Understanding what types of movements are affected, and to what extent, will help in diagnosis of FAI, as well as in determining what movements should be avoided to prevent exacerbation. FAI is an elusive condition to diagnose (Wisniewski & Grogg, 2006), so increasing our understanding of how cam impingement effects movement will help in its detection. An early diagnosis and treatment of FAI is important since it may

prevent the development of OA (Guanche & Bare, 2006). Moreover, when biomechanical tests are coupled with medical imaging of the impingement, a relationship can be found between the level of cam impingement and the corresponding effect on movement. The dynamic ROM data collected from this study is being used in a joint study to develop a computer model to aid in surgical operative planning. Using Radiographs and CT scans or MRI, computer models will be able to produce a three-dimensional (3-D) representation of the patient's hip which can be moved and manipulated to indicate points of impingement, and to determine an appropriate surgical protocol.

Limitations

There are various errors inherent in all biomechanical studies dealing with living human participants. All internal measures must be calculated indirectly by minimally invasive means. Joint kinematics are determined by external markers attached to the skin or clothing of participants. These markers are used to represent bones which are assumed to be rigid bodies. Bone articulations at joints are further assumed to be frictionless. Any movements of the skin or clothing which are independent of the underlying bone result in skin or clothing artefacts (Leardini et al., 2005). These artefacts can produce relatively large errors, and vary widely between participants (Reinschmidt et al., 1997). Another limitation to the accuracy of kinematic data is the determination of joint centres. Joint centre locations are essential to determining angular kinematics, and are also calculated based on marker placements. Joint centres are dependant on personal anatomy, but general algorithms with personal anthropometric data inputs are standard in biomechanical research (Camomilla et al., 2006). As with any generalized equations dealing with human anatomy, there is necessarily a margin of error. Finally, any marker misplacements by the researchers

culminate these potential errors (Della Croce et al., 2005). All these factors also limit the accuracy of kinetic data which is determined using the calculated kinematic information coupled with general segment parameter assumptions, and ground reaction force data. These factors and assumptions are intrinsic to all optoelectronic kinematic and kinetic research which use external markers, and limit the accuracy of the data acquired. Many precautions were used to minimize the effect of these inherent limitations, which are mentioned in the 'Limitations of Kinematic data' section of the text.

Further limitations specific to this study were the relatively small sample size of 31 participants (17 with FAI, and 14 control), and the number of variables which could affect results. Age, sex, body mass index (BMI), flexibility, and level of impingement could have all influenced biomechanics. However, these variables were measured and controlled for during participant selection, by matching the control group to the unilateral cam FAI group.

Review of Literature

Femoroacetabular Impingement

FAI is an anatomical morphology of the acetabulum and/or the head-neck junction of the femur. It occurs when there is abnormal contact between the femur and the acetabulum. This atypical articulation occurs most often between the anterior-superior aspect of the acetabulum and the head-neck junction of the femur. There are three classifications of FAI: *Cam* type FAI is caused by an abnormal femoral head-neck junction, *Pincer* impingement is a result of over-coverage by the acetabulum, and the third classification is a combination of these two types (Beck et al., 2005; Ito et al., 2004; Ito et al., 2001; Lavigne et al., 2004; Mardones et al., 2005; Murphy et al., 2004).

Cam impingement is a result of jamming an abnormal, non-spherical femoral head-neck junction into the acetabulum (Ito et al., 2001). This impingement arises at the limits of hip motion, especially at high speeds or forces. In severe cases impingement can occur even within the normal range of motion (Ito et al., 2001). It occurs most often in younger athletic males ranging from adolescence to middle age, and it usually results in more serious damage than pincer type (Ganz et al., 2003; Murray & Duncan, 1971). Long-term cam impingement can lead to labral and chondral lesions in the anterosuperior region of the acetabulum.

Pincer impingement is caused by linear contact between the femoral head-neck junction and the acetabular rim. It is primarily a result of an abnormal acetabulum - either from general acetabular over-coverage (coxa profunda), or local anterior acetabular over-coverage. Pincer FAI is most common in middle-aged athletic women (Ganz et al., 2003). Pincer impingement is generally localized to a small section of the acetabular rim.

According to a study by Beck et al (2005), the majority of FAI cases are a result of a combination of pincer and cam impingements. Both the acetabulum and the femoral head-neck junction are abnormally formed to varying degrees resulting in impingement. This allows for the greatest range of aetiological variations.

Because of the differences in the pathomechanics of these three FAI classifications, only cam impingement was analysed in this study in order to minimise the number of independent variables. Cam was chosen over mixed FAI because it has a smaller range of aetiological variations, minimizing the anatomical variability within the study group. Cam was chosen over pincer impingement based on the fact that it results in more serious damage, and since it was more available in the volunteers from the Ottawa Hospital.

FAI is a difficult condition to diagnose, and usually requires a combination of techniques. The standard procedure consists of an anteroposterior radiograph to test for pincer impingement, a cross-table lateral radiograph to test for cam impingement, and a physical examination (Siebenrock, Kalbermatten et al., 2003). The anteroposterior radiograph is used to test for acetabular abnormalities, and the cross-table lateral radiograph tests for an anatomical morphology of the femoral head-neck junction. MRI scans can be used instead of radiographs, allowing for internal slices of tissue to be viewed. A physical examination 'impingement test' assesses the limitations of hip internal rotation with the hip flexed to 90° with added adduction. A positive test produces sharp reproducible pain at the extremes of the range of motion (Ganz et al., 2003; MacDonald et al., 1997; Notzli et al., 2002; Philippon et al., 2007). This pain occurs when there is femoroacetabular contact with labral or chondral lesions which are a product of chronic impingement (MacDonald et al., 1997).

As well as causing labral and chondral lesions, FAI is also an important and widely accepted cause of hip osteoarthritis (Beck et al., 2004; Ganz et al., 2003; Laude et al., 2007; Murray, 1965; Tanzer & Noiseux, 2004; Wagner et al., 2003). In a healthy impingement free hip, the femoral head-neck offset prevents contact between the femoral neck and pelvis within a normal range of motion (ROM) (Tanzer & Noiseux, 2004). However, decreased femoral head-neck concavity causes a jamming of the femoral head into the acetabulum at the limits of motion (Ito et al., 2001). This contact produces repetitive low impact loading, with peak loads occurring during flexion and internal rotation which can cause reproducible hip pain (Leunig et al., 2005). This pain is thought to occur after the onset of joint damage, and is associated with contact between the proximal femur and labral or chondral lesions (MacDonald et al., 1997). Continual localized loading causes damaged bone to compensate by ossifying and accumulating bone volume at the site of contact, resulting in increased severity of impingement (Ganz et al., 2003). It is widely accepted that this degenerative cycle leads to the onset of hip osteoarthritis (Ganz et al., 2003; Harris, 1986; Laude et al., 2007; Leunig et al., 2000; Tanzer & Noiseux, 2004; Wagner et al., 2003).

There are various techniques to quantify the level and type of FAI, one of which is the α -angle. The α -angle (Figure 1) is a quantitative measure of the femoral head-neck junction designed by Notzli et al (2002). This procedure uses a 2 dimensional MRI frame along the length of the femoral neck for analysis. A line is drawn from the centre of the femoral head, down the centre of the femoral neck as a reference line. Assuming a cross section of the femoral head is a circle, point 'A' is defined by the point where the distance from the centre of the femoral head to the anterior edge of the bone periphery exceeds the radius of the femoral head (approximately at the femoral head-neck junction). The angle from the reference line to point 'A' is the α -angle. People suffering from cam FAI have a

larger α -angle than healthy control group. An α -angle of less than 50.5° is considered normal, and 50.5° or higher indicates cam type FAI (Notzli et al., 2002). The larger the α -angle the lower the free range of motion before impingement occurs.

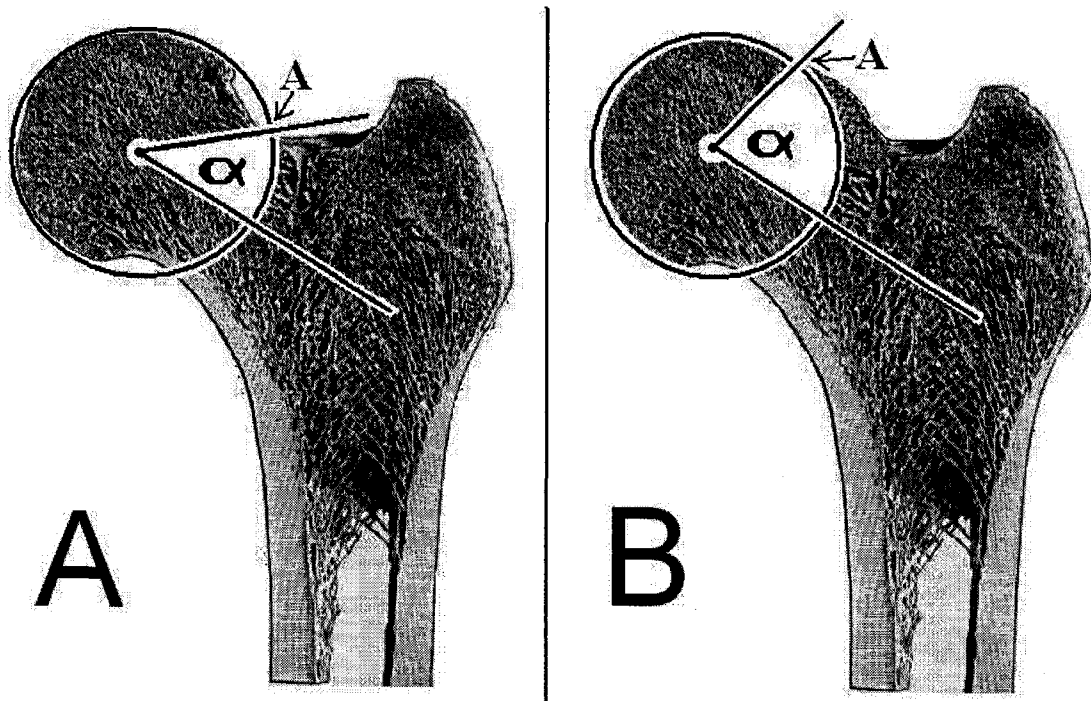


Figure 1. Healthy (A) and cam impingement (B) α -angle modified from Notzli (2002).

There are essentially two forms of treatment for FAI, the first being a conservative treatment consisting of activity modification reducing excessive motion and demand of the hip, and the other is surgical treatment (Lavigne et al., 2004). The conservative treatment helps to prevent advancement of FAI, and may alleviate symptoms, but does not remove the problem (Guanche & Bare, 2006). Moreover, it occurs at the expense of the lifestyle of the affected individual, which can be unrealistic with some people, especially the younger age range. It is suggested that initially a conservative treatment should be attempted for all patients (Guanche & Bare, 2006). Surgical treatment is the other option, and there are a variety of surgical measures. Two common procedures are the minimally invasive arthroscopic debridement, or the more invasive open debridement, both methods have their

advantages (Guanche & Bare, 2006; Lavigne et al., 2004). Arthroscopy helps decrease recovery time, but is a more complex procedure requiring multiple small incisions and indirect visualization through arthroscopes (Guanche & Bare, 2006). Whereas, surgical dislocation allows for a full 360° visual appraisal, making it easier for surgeons to remove all impinging tissue, and if done with proper respect for blood supply to the femoral head, it is deemed a safe procedure by orthopaedic surgeons (Ganz et al., 2003; Murphy et al., 2004). Within these two general categories is a large number of varying surgical procedures depending on individual cases. The primary goal of FAI resection hip surgery is to improve the clearance of hip motion by removing the bone or cartilage impeding movement (Lavigne et al., 2004). The surgical modality chosen depends on the type and level of impingement, the status of the acetabular articular cartilage, and the post-operative activities and hip demands of the patient (Lavigne et al., 2004).

The success of these surgical treatments is directly related to the level of degenerative transformations which have occurred by the time of intervention. It is imperative that FAI be diagnosed as early as possible to minimize the permanent damage to the hip joint, and the chance of OA development. According to a study by Beck et al (2004) on post-surgery results with the open debridement surgical technique using 19 patients with the average age of 36 years and average follow-up time of 4.7 years, surgery only produced good long lasting results with patients in the early degenerative stages of FAI, before extensive cartilage damage. This was found with arthroscopic debridement as well, according to the surgical results of 10 patients with the average age of 34 years who were treated arthroscopically. After an average follow-up time of 16 months, the eight patients with no intra-articular cartilage degenerative disease at the time of surgery were much better off than the two who had degenerative disease at the time of surgery (Guanche & Bare, 2006). These results show

that regardless of the surgical technique used, the success of the intervention depends on an early diagnosis and treatment of FAI. This reinforces the need to determine the biomechanical impact of FAI on movement, to increase our understanding of this deleterious condition, and to aid in early diagnosis.

Furthermore, due to the lack of long-term surgical results, it is important to determine how cam FAI affects movement, in order to develop proper conservative treatments. Defining how FAI affects functional dynamic hip mobility, and articulating which activities are altered or limited by this condition, will help formulate conservative solutions.

Kinematics

As Femoroacetabular impingement persists, it produces increasing labral and chondral damage. This can result in pain during a variety of common daily activities such as: low depth or prolonged sitting, stair climbing, excessive walking and athletic activities requiring a large ROM (Crawford & Villar, 2005; Ganz et al., 2003; Laude et al., 2007; Leunig et al., 2005; Wisniewski & Grogg, 2006). As mentioned before, chronic wear at the site of impingement can result in compensatory ossification which builds up the bone, increasing its size. This can substantially decrease the range of motion at the hip and adversely affect the performance of some movements. Although there have been studies measuring the passive hip ROM when assisted by a clinician (Eijer et al., 2001; Ito et al., 2004; Philippon et al., 2007; Wyss et al., 2007), to our knowledge there have been no studies which measured the effect of cam FAI on dynamic self-generated ROM of the hip, or on walking, sitting, standing and squatting hip kinematics. It is important to quantify how hip impingement affects the functional ROM of the hip in order to predict which activities may exacerbate the condition when developing conservative treatments. Furthermore, sitting

standing and walking are all common daily activities which are essential to independent living, and thus it is important to measure how they are affected by cam FAI. Finally, maximal depth squatting is a component of daily activities such as proper lifting technique, and bending down to tie shoes, as well as being a good indication of lower limb proficiency (Flanagan et al., 2003; Isear et al., 1997). This highlights the importance to determine how the maximal depth squat is affected by FAI.

Hip kinematics were measured according to the technique defined by Davis et al. (1991). The lower body was divided into a pelvis and left and right thighs, lower legs, and feet segments as defined by retroreflective marker placements (Appendix C). Each segment was connected to the adjacent segments by joint centres, each with three degrees of freedom. The ankle joint was defined as the midpoint between the lateral and medial ankle markers, the knee joint was defined as the midpoint between the lateral and medial knee markers and the hip joint was defined relative to the left and right anterior superior iliac spine (ASIS) markers (Figure 2). Hip joint centres were calculated based on the following variables and calculations (Davis et al., 1991):

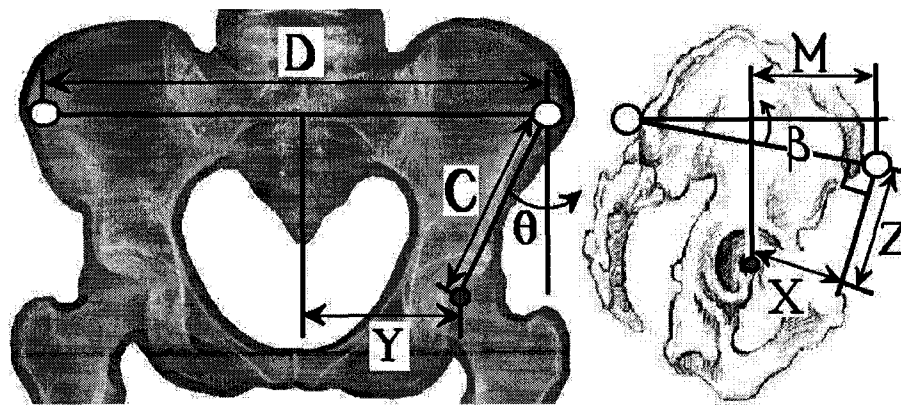


Figure 2. Hip joint center geometrical variables as modified from Davis et al. (1991)

$$\begin{aligned}
 C &= 0.115 L_{leg} - 0.0153 \\
 X &= [-M - r_{marker}] \cos(\beta) + C \cos(\theta) \sin(\beta) \\
 Y &= S [C \sin(\theta) - D/2]
 \end{aligned}$$

$$Z = [-M -r_{\text{marker}}] \sin(\beta) - C \cos(\theta) \cos(\beta)$$

Where:

D = inter-ASIS distance (in meters) measured during clinical examination
 C = calculation based on leg length (L_{leg}) (in meters)
 X = anterior/posterior component of ASIS hip joint center distance (in meters)
 r_{marker} = marker radius (in meters)
 S = +1 for right side, and -1 for left side

The angles at each joint were calculated based on the determination of Euler angles with a y-x-z axis rotation sequence (Kadaba et al., 1990). Transformation matrixes defined the orientation of the distal coordinate axes (e^D) relative to the proximal set of axes (e^P), which yield θ_y (flexion/extension), θ_x (adduction/ abduction) and θ_z (internal/ external rotation) as computed by the following equations (Davis et al., 1991):

$$\theta_y = \sin^{-1} \left[\frac{e_z^D \cdot e_x^P}{\cos(\theta_x)} \right]$$

$$\theta_x = -\sin^{-1} [e_z^D \cdot e_y^P]$$

$$\theta_z = \sin^{-1} \left[\frac{e_x^D \cdot e_y^P}{\cos(\theta_x)} \right]$$

Hip, knee and ankle angles are all relative to the proximal segment involved in the respective joints, but pelvic angles are absolute and are relative to the global coordinate system which was defined by the position of a static reference frame.

Range of Motion

Depending on the location of bone deformation, FAI patients with cam impingement show a decreased passive hip ROM as compared to healthy controls in flexion (Ito et al., 2004; Wyss et al., 2007), abduction (Ito et al., 2004), internal rotation with hip flexed at 90° (Ito et al., 2004; Notzli et al., 2002; Wyss et al., 2007) and external rotation with hip flexed at 90° (Ito et al., 2004). Similar results were also found in two computer-simulated ROM

studies based on the CT scans (Kubiak-Langer et al., 2007; Tannast et al., 2007) of participants with FAI, and matched controls. According to these studies, FAI patients had decreased simulated hip flexion, abduction and internal rotation. Other studies show a low passive ROM in patients with cam FAI, without comparing to a control group (Eijer et al., 2001; Reynolds et al., 1999; Zebala et al., 2007). Finally Philippon et al. (2007) found that unilateral FAI patients had decreased passive hip flexion, abduction, adduction, internal rotation and external rotation compared to their contralateral leg. However, there are limitations to these studies. There was no mention of the measuring device used to quantify passive ROM in any of the aforementioned non-simulated studies except for the study by Philippon et al. (2007) who used a goniometer. Furthermore, the computer-simulated ROM studies only accounted for bone on bone contact, greatly exaggerating hip mobility, and neglecting the influence of all soft tissues on hip mobility. Finally, these studies only assessed the influence of hip impingement on the simulated, passive or assisted, as oppose to the self-generated dynamic movement of the hip.

Generating maximal passive hip ROM in patients with FAI by the application of an external force by a clinician is needed to determine the bony limitation in these patients. However, it is also important to quantify how cam FAI affects functional hip mobility. It is the functional hip ROM that is used in the ADLs which exacerbate this condition. Thus, it is imperative to measure how self-generated dynamic hip ROM is affected by FAI in order to aid in developing conservative treatments for this pathology. Furthermore, determining the effect of FAI on self-generated dynamic hip ROM may aid in the diagnosis of this elusive condition.

Level Walking

The hip kinematics of level walking has been widely studied in healthy participants (Al-Zahrani & Bakheit, 2002; Bejek et al., 2006; Crowinshield et al., 1978; Judge et al., 1996; Lee et al., 2005; Mann & Hagy, 1980; Mills & Barrett, 2001; Miyoshi et al., 2004; Mockel et al., 2003; Winter, 1983). Ample research has been conducted on both the two and three dimensional kinematics of level gait. There is agreement that the ROM at the hip in the sagittal plane during level walking at a natural speed in healthy participants is between 15 and 40°, but there is an increase in hip ROM with increasing walking speeds (Bejek et al., 2006; Crowinshield et al., 1978; Miyoshi et al., 2004; Mockel et al., 2003). The maximal hip flexion angle during normal speed ranges from 15 to 30° (Miyoshi et al., 2004; Mockel et al., 2003; Winter, 1983), and the maximal hip extension ranges from 5 to 11° (Lee et al., 2005; Miyoshi et al., 2004; Mockel et al., 2003; Winter, 1983). The angular displacements in the frontal plane have a maximum adduction of approximately 5° and a maximal abduction of approximately 5° (Judge et al., 1996). Finally, the maximal hip external rotation is approximately 1°, and the maximal internal rotation is approximately 7° (Judge et al., 1996). These results proved as a useful comparison for the angular displacement of our control group.

The ROM in all three planes during level walking at a natural speed is much lower than the maximal hip ROM as described by Tannast (2007), making it likely that the effect of FAI on walking may be minimal, as compared to activities requiring a larger range of motion. This seems to coincide with the qualitative visually appraised report that people diagnosed with FAI have a normal gait by Wisniewski et al (2006). However, a quantitative kinematic analysis was still required to come to a definite conclusion about the effect of FAI on walking.

There have also been numerous articles which assess the 3-D pelvic ROM during level gait (Bejek et al., 2006; Smith et al., 2002; Taylor et al., 2004; Vink & Karssemeijer, 1988). There tends to be high variability in recorded pelvic mobility during gait (Apkarian et al., 1989) with values being closely related to gait speed (Bejek et al., 2006). According to the literature, the pelvic ROM values in healthy participants are as follows: frontal range between 2.9 to 10.2°; sagittal range between 2.1 to 6.2°; and transverse range between 4.7 to 10.4° (Bejek et al., 2006; Smith et al., 2002; Taylor et al., 2004; Vink & Karssemeijer, 1988). However, these values were recorded using varying techniques with gait speeds varying from 2km/h to 4km/h. Given the fact that no information on the maximal pelvic mobility of participants with cam FAI could be found, these values only served as a comparison for our control group.

The above-mentioned studies report level gait kinematic data for healthy participants, but give no insight into what is to be expected from participants diagnosed with FAI. As mentioned previously, femoroacetabular impingement is directly related to, and a significant cause of osteoarthritis. Unlike FAI, there have been biomechanical studies on the effect of OA on level walking. In a study by Bejek et al (2006), it was found that patients with severe coxarthrosis have lower walking speed, lower cadence, decreased step length, shorter single stance phase of the affected leg, a significantly decreased range of motion in the osteoarthritic hip, and a significantly increased oblique and sagittal range of motion at the pelvis during walking. The maximal sagittal ROM of the affected hip was significantly lower in the coxarthrosis group compared to the control at 2.0 km/h (the highest attainable speed by all OA participants). The coxarthrosis group had a sagittal hip ROM of $22.2 \pm 8.1^\circ$ compared to the control group of $37.3 \pm 8.9^\circ$. Moreover, the coxarthrosis group had a significantly higher frontal pelvic ROM than the control group of $8.2 \pm 1.3^\circ$ compared to 2.9

$\pm 1.2^\circ$ respectively. The same was true with sagittal pelvic ROM, with the coxarthrosis group having $16.9 \pm 2.7^\circ$ compared to $6.2 \pm 3.1^\circ$ in the control at 2.0 km/h. Since FAI is known as a cause and precursor to OA, and these results are from participants with advanced and severe hip OA, we expected less dramatic results in our study. Moreover, these alterations in level walking kinematics are likely caused by compensation to prevent hip pain (Hurwitz et al., 1997), rather than movement restriction caused by bone contact, which may make them even less comparable. However, the kinematic trends found in participants with severe OA were still a useful reference.

Sitting, Standing and Squatting Manoeuvres

There is a substantial database of information on the angular displacement of the hip joint in the sagittal plane during sitting, standing and squatting in healthy participants (Doorenbosch et al., 1994; Dubost et al., 2005; Flanagan et al., 2003; Hase et al., 2004; Khemlani et al., 1999; Kralj et al., 1990; Pai & Rogers, 1991; Rodosky et al., 1989; Schenkman et al., 1996; Tully et al., 2005; Yu et al., 2000). However, only one study could be found which quantified the coronal and transverse hip angles during sit-to-stand (Gilleard et al., 2008). No literature could be found which reports the coronal and transverse hip angles during stand-to-sit or maximal depth squat. There was substantially less research concerning the pelvic kinematics during these manoeuvres. Only one study was found which quantified the sagittal pelvic kinematics during sit-to-stand (Nuzik et al., 1986). No literature could be found which quantified the frontal or transverse pelvic angular displacement during sit-to-stand, and nothing could be found in any plane for stand-to-sit or maximal depth squats. Furthermore, there has been no previous research on the affect of FAI on the hip or pelvic kinematics of these three manoeuvres. Therefore, 3-D kinematic analyses of sitting,

standing and squatting manoeuvres were needed in both healthy participants, and those diagnosed with FAI.

Sitting, standing and squatting are all very closely related, but each has unique components. Sitting and standing have very similar maximal hip angular displacements, and range of motions in the sagittal plane, but it is important to note that these two movements are not merely reciprocals, and should be studied separately (Dubost et al., 2005; Hase et al., 2004). Squatting is essentially a combination of sitting and standing, but one very important difference between squatting and sitting or standing is that the centre of mass must remain between the feet for squatting, but moves backwards towards the seat, or forwards away from the seat in sitting and standing respectively (Hase et al., 2004). Furthermore, a maximal depth squat requires lowering the buttocks much further than sitting down to knee height, or standing up from a knee height seat, necessitating different joint kinematics.

Although there are many different methods of breaking up and classifying the sit-to-stand manoeuvre (Janssen et al., 2002), there are two general actions which are required for a successful chair rise. The first is moving the base of support from the seat to between the ankles (moving the centre of gravity forward), and the second is extending the body to an upright standing position. When in a seated position, the majority of bodyweight is concentrated on the chair, with relatively little found on the floor between the feet where it is concentrated during standing. Based on the 50th percentile of male anthropometry, the centre of gravity during sitting in an upright position, with the knee and hip joints at 90° angles is approximately 33 cm posterior to the ankles (Schultz et al., 1992). Thus, the first step of chair rising requires that the centre of gravity be shifted forward from above the seat to above the new base of support- the feet. After this has been accomplished, and the centre of gravity has moved above the midpoint between the ankles, the second step begins, and the body can

extend to an upright standing position. The sit-to-stand manoeuvre in our study was classified based on the two abovementioned steps. The first phase was defined by contact between the buttocks and the chair as defined by a force less than the weight of the participant on the force platforms beneath their feet. The second began once full bodyweight was on the platforms, and ended once the body had reached a stable upright position. For the stand-to-sit movement, the same two steps were used, but in reverse. The squat action was broken into descending and ascending classifications, separated by maximal attained squat depth. Maximal attained squat depth was defined as the smallest distance from the hip joint centers to the floor, divided by total leg length. No previous studies could be found on maximal squat depth without the participants holding weights.

Hip mobility is essential for rising from a seated position, in both shifting the centre of gravity towards the feet, and in extending the body (Yu et al., 2000). The range of motion in healthy individuals at the hip is substantially larger during both sit-to-stand and stand-to-sit manoeuvres than during level walking (Kralj et al., 1990; Pai & Rogers, 1991; Rodosky et al., 1989; Shum et al., 2005; Tully et al., 2005). The hip range of motion in the sagittal plane is between 80 and 100° during sit-to-stand (Kralj et al., 1990; Pai & Rogers, 1991; Rodosky et al., 1989; Shum et al., 2005; Tully et al., 2005), and is between 85 and 100° during stand-to-sit (Kralj et al., 1990; Shum et al., 2005). Maximal hip flexion angle during squatting depends directly on the depth of the squat, but was found to be approximately 90 to 100° in a study by Flanagan et al (2003) when squat depth was limited to approximately 80% of knee height. No studies could be found which assessed the hip kinematics in the frontal and transverse planes during sit-to-stand, stand-to-sit, or squatting. Furthermore, no studies have yet quantified the affect of FAI on the kinematics of these manoeuvres.

Sit stand and squat movement occur primarily in the sagittal plane. As a result, only sagittal pelvic angles have been quantified for sit-to-stand, stand-to-sit, or squat manoeuvres (Nuzik et al., 1986). However, it is important to quantify both frontal and transverse pelvic kinematics in participants with a unilateral hip pathology, since unilateral FAI may disrupt the frontal and transverse symmetry of these manoeuvres. When the data was zeroed to neutral standing values, Nuzik et al. (1986) found that during sit-to-stand, participants had approximately 25° of pelvic retroversion during sitting, and had a peak pelvic anteversion of approximately 10° at seat-off, which moved towards 0° at neutral stance. In total there was a sagittal pelvic ROM of approximately 35° during sit-to-stand. These values were useful references for our sagittal pelvic kinematics during sit-to-stand. The paucity of literature reporting the frontal and transverse pelvic kinematics during sitting, standing and squatting indicates the importance to quantify these values both in healthy participants, and in individuals diagnosed with cam FAI.

Kinetics

Net 3-D moments of force and mechanical powers at the hip were measured in this study. Moments of force are the net result of muscular, ligament and frictional forces acting to change the angular rotation of a joint (Winter, 1991a). Mechanical powers are the generation and absorption of the energy required to produce the net moments of force at each joint (Winter, 1991a). These two kinetic facets resulting primarily from muscle activation and gravity are responsible for generating joint kinematics during various movements. Mechanical power gives insight beyond the net moments of force acting on a joint, and allows for the determination of the net eccentric or concentric activities of the muscle group causing the net moment of force in each plane (Winter, 2005). These measures help

illuminate the differences in movement generation in FAI participants as compared to a healthy control group. As with kinematics, to our knowledge, there have not been any studies which measure the affect of FAI on moment of force and mechanical power generation at the hip.

The 3-D hip moments of force were calculated using Newtonian mechanics and Euler's equations of motion (Davis et al., 1991):

$$\begin{aligned} M_x &= I_{xx} \alpha_x + (I_{zz} - I_{yy}) \omega_y \omega_z \\ M_y &= I_{yy} \alpha_y + (I_{xx} - I_{zz}) \omega_z \omega_x \\ M_z &= I_{zz} \alpha_z + (I_{yy} - I_{xx}) \omega_x \omega_y \end{aligned}$$

Where:

- x, y, z = coordinate axes fixed to segment at the segment's centre of mass
- M_x, M_y, M_z = components of the three-dimensional external moments (about the segment center of gravity) applied to the specified segment
- $\alpha_x, \alpha_y, \alpha_z$ = components of the absolute angular acceleration of the specified segment
- $\omega_x, \omega_y, \omega_z$ = components of the absolute angular velocity of the specified segment
- I_{xx}, I_{yy}, I_{zz} = mass moments of inertia of specified segment

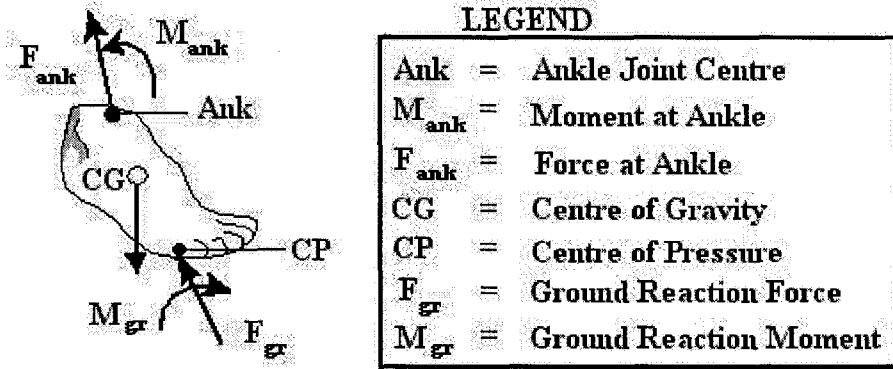


Figure 3. Free-body diagram of a foot segment, used to simplify the number of forces acting on the foot segment in order to solve the force and moment reactions acting at the ankle joint (Davis et al., 1991).

All segment parameters such as segmental mass, center of mass location and moments of inertia were based on anthropometric approximations by Dempster et al. (1959). All moments and power vectors were expressed relative to the local segment coordinate systems.

Only the moments of force and mechanical powers generated at the hip were analysed in this study.

Level Walking

The kinetics of level walking at a natural speed in healthy participants have been widely studied (Al-Zahrani & Bakheit, 2002; Crowinshield et al., 1978; Eng & Winter, 1995; Judge et al., 1996; Kerrigan et al., 1998; Kirkwood et al., 1999a; Mills & Barrett, 2001; Winter, 1983, 1989, 2005). The moment of force generated at the hip in the sagittal plane during level walking at a natural speed is quite variable, with the average peak extension moment of force being approximately 0.7 to 1.3 Nm/kg and occurring at the beginning of stance phase, and the peak flexion moment of force being approximately 0.5 to 1.2 Nm/kg and occurring during the latter half of stance phase (Al-Zahrani & Bakheit, 2002; Eng & Winter, 1995; Judge et al., 1996; Winter, 1983, 1989). Both the moments of force in the frontal and transverse planes also have high variability, with average findings being: abduction moment = 0.8 to 1.2 Nm/kg peaking at the beginning of stance phase with a second lesser peak at the end of stance phase, adduction moment = 0.1 to 0.2 Nm/kg occurring at the end of stance phase and during swing phase, internal rotation moment = 0.05 to 0.2 Nm/kg peaking at the end of stance phase, and external rotation = 0.05 to 0.2 Nm/kg occurring at the beginning of stance phase (Eng & Winter, 1995; Judge et al., 1996; Kirkwood et al., 1999a; Winter, 1983, 2005).

Like with the moments of force generated at the hip during walking, there is a lot of variability in the hip mechanical powers. The peak positive mechanical power generated in the sagittal plane at the hip joint during level walking at a natural speed was approximately 0.6 – 1.8 W/kg at or just before push-off, initiating swing phase. The maximal negative

power at the hip in the sagittal plane while walking at a natural speed was approximately -0.1 to -0.25 W/kg, and it occurred in the later half of stance phase. Maximal positive and negative power generation at the hip in the frontal and transverse plane are less studied than they are in the sagittal plane. The powers in the transverse and frontal planes are as follows: peak negative frontal power = -0.8 to -1.5 W/kg occurring at the beginning of stance phase, peak positive frontal power = 0.4 - 0.75 W/kg occurring at the end of stance phase, maximal negative transverse power = -0.025 to -0.35 W/kg occurring at the beginning of stance phase, and finally, peak positive transverse power = 0 - 0.08 W/kg occurring at the end of stance phase (Eng & Winter, 1995). These values were useful references for our control group for the walking condition.

Sitting, Standing and Squatting Manoeuvres

The kinetics of standing, sitting and squatting has also been widely studied in healthy participants (Doorenbosch et al., 1994; Flanagan et al., 2003; Khemlani et al., 1999; Kralj et al., 1990; Mathiyakom et al., 2005; Pai & Rogers, 1991; Schultz et al., 1992; Shum et al., 2007), but no studies were found which analyzed the moments of force or mechanical powers generated at the hip in people diagnosed with FAI. Data from these previous studies on healthy participants acted as a reference for our control group.

The peak weight normalized hip extensor moment of force in healthy participants during rising from a chair is approximately 1.0 - 2.0 Nm/kg (Flanagan et al., 2003; Fleckenstein et al., 1988; Pai & Rogers, 1991; Schultz et al., 1992), and is 0.94 ± 0.24 Nm/kg during sitting down (Flanagan et al., 2003). Peak extensor moment of force at the hip for chair rise occurred at seat-off, and gradually decreased to zero at full body extension (Flanagan et al., 2003; Fleckenstein et al., 1988; Pai & Rogers, 1991; Schultz et al., 1992).

For sitting down, the peak extensor moment of force occurred prior to seat contact, and gradually decreased to zero once fully seated (Flanagan et al., 2003). Maximal hip flexion moments of force generated during chair rise in healthy participants was approximately 0.6 - 0.94 Nm/kg, occurring midway between initiation and seat-off (Fleckenstein et al., 1988; Pai & Rogers, 1991). Data on the peak hip flexion moment of force produced during sitting down was not found.

The maximum weight normalized hip power in the sagittal plane during rising from a chair was 0.93 ± 0.32 W/kg compared to -0.78 ± 0.29 W/kg produced while sitting (Flanagan et al., 2003). The positive mechanical power generated during chair rise indicates a concentric contraction of the hip extensors as the body rises off the seat. The negative power produced while sitting down indicates an eccentric contraction of the hip extensors allowing for a controlled descent.

Only one study was found which analyzed the frontal and transverse plane kinetics of sitting and standing in healthy individuals (Shum et al., 2007). These results however are difficult to compare to the aforementioned studies, since they were normalized according to weight and lower limb length such that the units cancelled out, rather than just controlling for the participants' weight. The normalization calculations used were as follows:

$$\begin{aligned} \text{Normalized moment, } \mathbf{M} &= \mathbf{M} / m \cdot g \cdot l \\ \text{Normalized power, } \mathbf{P} &= \mathbf{P} / m \cdot g^{1/2} \cdot l^{3/2} \\ \text{Where } m &= \text{body mass (kg); } g = \text{gravity (m/s}^2\text{); and } l = \text{leg length (m)} \end{aligned}$$

The peak moments of force during standing were between -0.009 to -0.013 for adduction and between -0.014 to -0.016 for internal rotation. Peak concentric hip power during standing was from 0.112 to 0.121 in the frontal plane, and from 0.013 to 0.015 in the transverse plane. Maximal eccentric hip power during standing was from -0.080 to -0.079 in the frontal plane,

and from -0.041 to -0.039 in the transverse plane. For sitting down, the moments and powers generated at the hip were similar to the standing condition: peak hip adduction moment of force = 0.016 to 0.017, peak internal rotation moment of force = -0.007 to -0.006, maximal concentric hip power = 0.106 to 0.101 in the frontal plane, and 0.035 to 0.051 in the transverse plane, peak eccentric hip power in the frontal plane = -0.092 to -0.093, and -0.039 to -0.044 in the transverse plane (Shum et al., 2007).

A lower moment of force and mechanical power is generated at the hip during squatting down to knee height without the presence of a chair, than during sitting down onto a chair. The peak hip extensor moment of force value during this squatting manoeuvre was found to be 0.73 ± 0.21 Nm/kg during squat descent and 0.74 ± 0.22 Nm/kg during squat ascent, and the peak power at the hip was 0.52 ± 0.21 W/kg during squat descent and 0.60 ± 0.25 W/kg during squat ascent in healthy older adults (Flanagan et al., 2003). However, the decreased hip force and power was compensated for by an increase in knee and ankle moment of force and power generation. The squat values in our control group were expected to be substantially higher than these since they were performing a maximal depth squat down to a maximum of 1/3 of tibia height, rather than a chair of approximately knee height. No information on the kinetics in the frontal and transverse planes during squatting could be found.

Control Variables

Level Walking

There are various kinematic differences between the gait biomechanics of young and older participants. Older adults have a decreased stride length than younger participants (Judge et al., 1996; Winter, 1991b; Winter et al., 1990), they have a longer stance phase

(Winter et al., 1990), and decreased hip extension and ROM, especially at higher walking speeds (Crowinshield et al., 1978; Lee et al., 2005). There are conflicting results about the preferred walking speed of elderly and younger participants, with Judge (1996) finding a decreased preferred walking speed in the elderly, and Mills (2001) finding no difference. The only difference in age in terms of joint moments of force and powers was that older subjects had lower peak ankle plantarflexor power and moments of force (Judge et al., 1996; Winter, 1991b), but when step length was controlled for, the only significant difference was that the elderly group had a larger relative hip flexion power (Judge et al., 1996). The effect of age on gait biomechanics found in the abovementioned studies were between two samples with a large age discrepancy of 40 to 50 years. The age span in our study will be smaller than this, but age is a factor which should not be ignored. To control for age being a confounding factor in our study, age was controlled for by matching the FAI and control groups. The correlation between age and gait biomechanics was tested to ensure that biomechanical differences were not caused by age in either the FAI or control groups.

Gait speed also has some important implications on the biomechanics of level gait. Increasing walking speed produces a larger range of motion, and maximal angular displacements at the hip, knee and ankle in healthy participants in the sagittal plane (Bejek et al., 2006; Crowinshield et al., 1978; Mockel et al., 2003; Winter, 1983, 1991a). A linear relationship has also been found between gait velocity and peak hip resultant forces and resultant moments (Crowinshield et al., 1978; Winter, 1983). Moreover, the power produced at the hip increases significantly with an increase in gait speed (Winter, 1983, 1991a). In order to allow for comparisons between participants, and to avoid gait speed acting as a confounding factor the participants were asked to walk at their own natural out-door walking

speed. Furthermore, gait speed was compared between groups to ensure it did not differ significantly.

There is some debate as to the affect of sex on gait biomechanics. According to Kerrigan et al (1998), when data is both height and weight normalized, females have significantly higher cadence, hip flexion and relative stride length than males, however, Crowinshield et al. (1978) found no differences in hip kinematics or kinetics when body weight and height were controlled for. This difference is likely due to the increased statistical power in the study by Kerrigan as a result of having a large sample size ($N = 99$) compared to the small sample size in the study by Crowinshield ($N = 11$). Kerrigan et al. (1998) also found an increased relative hip power generation in females compared to males, which is likely a result of higher cadence. These differences indicate that sex needs to be accounted for in research design, and so both groups were matched according to sex. Moreover, the gait biomechanics of walking were compared between male and female participants to ensure that the mixed gender samples in both the control and FAI groups were homologous.

Sit, Stand and Squat

In order to measure for the effect of FAI on standing, sitting and squatting biomechanics, there can be only one independent variable (the presence or absence of FAI), and all other variables which influence biomechanical results must be controlled for. There are some important factors which are known to affect the biomechanics of these manoeuvres. These factors are foot placement, initial trunk orientation, the use of upper extremities, age and relative seat height. Each must be controlled for in order to ensure that the presence or absence of FAI, and not a confounding factor, is the cause of any biomechanical differences.

It was found by Schultz et al. (1992) that flexing the lower legs, and hence bringing the feet posterior during standing from a seated position is the most effective way to move the centre of gravity towards the base of support. Since the first stage of rising from a chair consists of moving the centre of gravity forward, placing the feet posteriorly can have a large impact on the biomechanics of the sit-to-stand manoeuvre. Moreover, a posterior positioning of the feet can decrease mean hip extension moments by over 110 Nm (Fleckenstein et al., 1988; Kawagoe et al., 2000). Also, a more anterior foot placement increases the duration and total displacement of the sit-to-stand movement, increases trunk flexion velocity, and alters the onset of hip, knee and ankle extensors, in young males (Khemlani et al., 1999). These findings make it clear how important initial foot placement is on the biomechanics of rising from a chair, and emphasized the need to control foot placement in order to produce relevant results. This was controlled for by participants sitting with their knees at approximately 90° of flexion, and their feet planted 10 cm in front of the height-adjustable bench at shoulder width apart.

Increasing initial trunk flexion causes an increase in the duration of the maximum support moment (Shepherd & Gentile, 1994). Moreover, the duration of the extension phase after the buttocks left the seat increased with a more flexed initial trunk position. These effects on the biomechanics of the sit-to-stand movement are relatively small, but are another influential factor which was easily controlled for by standardizing initial trunk position.

The majority of sit-to-stand or stand-to-sit studies did not allow the use of arms in the rise manoeuvre. Participants were instructed to fold their arms, keep them down at their sides, put them in their lap or place them on their knees in order to prevent them from altering the lower body biomechanics during standing (Janssen et al., 2002). The effect of upper limb use by pushing off of arm-rests on the biomechanics of the sit-to-stand movement

was studied by Schultz et al (1992), and was shown to have a significant kinetic impact. The same study by Schultz et al. (1992) showed that extending the arms forward had the potential to move the total body centre of gravity 4.2 cm forward, which would play a role in both kinematics and kinetics of the initial stage of moving the centre of gravity initially located over the seat towards the feet to allow for body extension. These results made it clear that the arms should be controlled for to avoid confounding factors.

Another variable which is known to have an effect on sit-to-stand biomechanics is age. In a study by Alexander et al in 1991 it was found that there were differences in chair-rising biomechanics between young (average age of 23 years) and healthy older participants (average age of 72 years). The older group produced significantly more maximal trunk flexion (Alexander et al., 1991; Papa & Cappozzo, 2000), spent significantly more time in the seated phase of the sit-to-stand manoeuvre, and both flexed and extended their legs significantly more than the younger group. However, the age spectrum in the aforementioned study was much larger than in ours, and our participants were matched according to age. The average and standard-deviation of age between the FAI and control groups were very similar. Furthermore, Pearson's correlations ($p = 0.05$) were run between age and sit, stand and squat biomechanics with no significant correlations found.

One of the most important factors in sitting, standing and squatting is relative seat height, and squat depth. It is important to note, that seat height must be adjusted to the individual depending on their knee height in order to not adversely affect biomechanics. A shorter chair elevation relative to knee height places more load on the lower limb, increasing the activity of the quadriceps and tibialis anterior (Arborelius et al., 1992; Yamada & Demura, 2004), and indicating a higher moment of force generation at the knee and ankle. There are also much larger moments of force produced at the hip with lower chair heights

than with higher chair heights (Doorenbosch et al., 1994; Fleckenstein et al., 1988). Another study by Hughes & Schenkman (1996) found a significant change in sit-to-stand strategy with changing seat height. For the sit-to-stand and stand-to-sit portions of this study, chair height was controlled to 100% knee height, and maximal squat depth was controlled to 1/3 leg length. Furthermore maximal squat depth was included as a covariate to ensure biomechanical differences were a result of FAI and not squat depth.

Limitations of Kinematics data

As mentioned briefly in the limitations section, one area of potential error is the placement of markers on the participants. Misplaced marker locations can have a substantial effect on measured kinematics. Markers can be misplaced by 0.7 – 18 mm (della Croce et al., 1999), resulting in 3-D hip angle variations ranging from 2.5 – 5.6° during simulated gait (Della Croce et al., 2005). This was corrected for in our study by having only one researcher (MK) place the key anatomical markers, which substantially reduces inter-researcher variability (Della Croce et al., 2005). Furthermore, any errors in marker placement can adversely affect the determination of joint centres which also affects kinematic calculations. However, the main errors caused by small marker misplacements are incorrect joint angular offsets in a neutral position, which was corrected in our study by zeroing all kinematic values based on a static neutral standing trial.

Another area of potential error in kinematic data collection occurs when markers on the skin or clothing surface moving independently of the underlying bone which are known as skin or clothing artefact respectively (Leardini et al., 2005). Clothing artefact can be very large if loose clothing is used, but this was minimized by wearing a skin-tight spandex suit for the pelvic and trunk markers. Skin artefact is largest with rapid ballistic movements, but

still produces a root mean square difference in angular kinematics of the knee of $1.5 - 5.3^\circ$ during walking (Leardini et al., 2005). Skin artefact was thus minimized due to the relatively slow controlled nature of dynamic ROM, walking, sitting, standing and squatting manoeuvres.

Kinematic data is also limited by potential errors in the generic algorithms used to calculate joint centers (Camomilla et al., 2006). However, since the same calculations were used for all participants and both groups were closely matched, any small errors in joint center determination were common to both groups. There are also some inherent errors in kinetic calculations, based on the assumptions essential for their determination such as rigid segments, frictionless joints, and generic centre of gravity locations. However, these same assumptions are common to both groups, and so their effects are minimized in a comparative study.

Methodology

Materials

The materials used for data collection in this study consisted of seven VICON MX-13 cameras (VICON, Los Angeles, CA USA) with associated retro-reflective markers and two AMTI force plates (one AMTI OR6-6-2000, and one AMTI OR6-6-1000, Watertown, MA USA). The VICON cameras were set-up around a raised walkway in the centre of the laboratory. The force plates were fastened to a metal track which was fixed to the concrete floor, and built into the walkway (Refer to Appendix A).

Three skin-tight suits of varying sizes (large, medium and small), consisting of a short sleeved shirt and shorts, with Velcro patches sewn to it where retro-reflective markers were attached (refer to Appendix B), were worn by participants. The location of the Velcro patches coincide with the marker positions as defined by a modified Plug-in Gait marker set (refer to Appendix C). Retro-reflective markers fixed to complimentary Velcro pieces were constructed to be attached to the Velcro patches on the skin-tight suit according to the anatomical landmarks defined by the Plug-in Gait marker set. The patches on the suit were large enough to allow for marker adjustment based on personal anatomy. Reflective markers were also attached to the skin which was not covered by the suits using double sided tape. The purpose of these skin-tight suits was to minimize the large errors caused by loose clothing.

A height adjustable stool was used for the sit-to-stand, the stand-to-sit, and the maximal squat depth protocols (refer to Appendix D). It was placed in the centre of the

walkway directly in front of both force-plates and in full view of the cameras, such that one foot was placed on each of the two force plates while the movements were being executed.

A support frame was also used to aid participants in performing dynamic hip range of motion trials (refer to Appendix E).

Finally, an adjustable sit and reach flexibility test (Model 01285A, Lafayette Instrument, Lafayette, IN) was used to quantify participant flexibility.

Participants

There were two groups in this study, the FAI group consisted of participants diagnosed with unilateral cam FAI, and the control group was age, sex and weight matched. The cam impingement participants were recruited after having a positive impingement test and visible cam morphology on anteroposterior (AP) and Dunn view radiographs (Eijer et al., 2001; Laude et al., 2007; Meyer et al., 2006; Siebenrock, Kalbermatten et al., 2003). Furthermore, each FAI participant had an alpha angle value greater than 50.5 degrees which is diagnostic of cam type FAI (Notzli et al., 2002). Matched control participants had no history of serious lower limb injury or surgery, and had spherical femoral heads which were assessed by an A/P radiograph (Meyer et al., 2006). Participants from both groups were excluded if hip OA was visible on the x-rays or if they had substantial hip joint space narrowing.

As well as the aforementioned screening or diagnosis x-rays, all FAI participants and five of the healthy controls underwent two orthogonal radiographs and either magnetic resonance imaging (MRI) or a computed tomography (CT) scan. For the orthogonal radiographs, radiopaque surface markers (electrocardiogram electrodes) were placed on anatomical landmarks. These radiopaque markers indicated the location for retro-reflective

marker placement for motion analysis. This was done in order to determine the relative distances from surface marker placement to the deep anatomical structures (Costigan et al., 2002; Kirkwood et al., 1999a, 1999b). This process was primarily to allow for accurate hip joint centre determination for the joint study focused on developing the surgical computer model. The MRI or CT scans were used to give the computer engineers from this joint study a 3-D model of each participant's pelvis and hips.

A total of 37 participants volunteered in this study. Eighteen participants diagnosed with cam FAI, and 19 age, gender and BMI matched control participants. The sample size was determined using two power analyses based on pilot data from the first 10 volunteers (five FAI and five control participants) to determine the effect size. The first power analysis was calculated using peak internal rotation with the hip flexed at 90° as the key dependent variable, and the second power analysis used maximal squat depth. Both power analyses were calculated using G*POWER version 3 (Faul, F. & Erdfelder, E., Bonn, Germany) with an alpha value set at 0.05, and a minimal power set at 80%. When the dependent variable was flexed internal rotation, a total sample size of 26 was calculated. When maximal squat depth was used, a total sample size of 22 participants was calculated. Because of the large number of variables being tested, two different power analyses were calculated using two different key dependent variables in order to better estimate the required sample size. These calculated sample sizes were the minimum number of participants required to achieve significant differences in flexed internal rotation and maximal squat depth with a power of 80% based on our pilot data. However, since we were looking for other variables as well, a larger sample size was chosen to maximize the statistical power of the study.

For the squat condition a total of only 32 participants (16 FAI, and 16 controls) were analyzed because of a tight article submission deadline from Clinical Orthopaedics and Related Research (CORR). This sample size was still substantially larger than the 22 predicted from our power analysis using maximal squat depth as the key dependent variable.

All FAI participants were recruited through the Ottawa Hospital, and all scans were administered by experimenters from the joint study at the Ottawa Hospital. The matched control participants were recruited from the Ottawa region using various forms of advertisement. Both the Ottawa Hospital Research Ethics Board and the University of Ottawa Health Sciences and Science Research Ethics Board approved the study protocol.

Procedure

All participants read and signed a consent form approved by the Ottawa Hospital Research Ethics Board before undergoing screening and diagnostic radiographs and MRI and CT scans which all took place at the Ottawa Hospital. Participants came to the University of Ottawa (120 University Private, room 125) for biomechanical analysis. After having the details of the research procedure explained to them, participants read and signed a second consent form approved by the University of Ottawa Health Sciences and Science Research Ethics Board. Participants then filled out the Western Ontario and McMaster University Osteoarthritis Index (WOMAC) questionnaire (Bellamy et al., 1988), answering all questions with respect to their hips. Participants then indicated whether they were right or left side dominant, and donned the abovementioned marker suit. Once wearing the suit, participants warmed-up by stretching their hamstrings, quadriceps and hip adductors, and then executed a maximal sit and stretch flexibility test. Further anthropometric measurements were then performed according to the Plug-in Gait participant measurements (Davis et al., 1991), and

then retro-reflective markers were attached to the suit and skin according to the modified Plug-in Gait marker-set (Appendix C).

After setup, each participant performed a static calibration trial, where they stood upright with their feet shoulder-width apart, parallel and facing anteriorly. This static trial was used for calculating segment lengths, and to determine neutral joint positions.

Participants were then instructed to perform a series of dynamic range of motions trials, with the researcher giving a demonstration of each manoeuvre. While standing upright and holding on to a support frame, participants maximally swung their left and right legs in each plane. Participants performed peak flexion, extension, adduction (with the mobile leg in front of the support leg), abduction, and internal and external rotation while the hip was flexed at 90°. Each motion was repeated five times for each leg.

For the second condition, each participant performed a few practice walking trials until they could land with their foot on a force-plate without altering their gait. Participants were asked to walk at a natural speed to help control for biomechanical differences caused by varying gait speed (Bejek et al., 2006; Crowinshield et al., 1978; Mockel et al., 2003; Winter, 1991a). Each participant performed ten level walking trials, five with their left foot and five with their right foot landing on the same force-plate. The force plates were positioned side-by-side to allow for the calculation of hip kinetics for each leg independently in the sit-to-stand and stand-to-sit conditions.

The third stage was a series of sit-to-stand and stand-to-sit conditions. For both sitting and standing trials, participants were asked to stand 10 cm in front of the height-adjustable bench (Fleckenstein et al., 1988; Kawagoe et al., 2000; Khemlani et al., 1999; Schultz et al., 1992) with both feet shoulder-width apart, parallel to each other and facing

anteriorly. Before any sitting or standing trials were collected, the bench was adjusted to the height of the participant's tibial plateau (Arborelius et al., 1992; Doorenbosch et al., 1994; Fleckenstein et al., 1988; Hughes & Schenkman, 1996; Yamada & Demura, 2004). Stand-to-sit and sit-to-stand were two connected movements and were collected in tandem, with participants being asked to sit down, pause, stand up, pause and so forth without moving their feet until five trials of each were collected. Starting from standing, participants were asked to cross their arms across their chest, (Janssen et al., 2002; Schultz et al., 1992) sit down on the height-adjusted stool, pause until notified, and then stand back up at a self selected and controlled pace (Pai & Rogers, 1991). Crossing the arms prevented the temptation to use them for support during the manoeuvres. All participants performed five sit-to-stand and five stand-to-sit trials.

Finally the last stage consisted of maximal depth squatting. The stool used in the previous stage was lowered to 1/3 of tibial plateau height. This indicated the maximum attainable squat depth, and the bench also acted as a safety mechanism in case the participant lost balance and fell back. Participants began in an upright standing position 10 cm in front of the stool, standing with one foot on each force plate, with their feet at shoulder width, and their arms extended anteriorly to increase balance and prevent temptation to use them during the squat (Janssen et al., 2002; Schultz et al., 1992). They then squatted down to their maximal depth without putting weight on the bench, and then ascended back to standing at a controlled pace (Pai & Rogers, 1991). Like in the previous sessions, this was repeated 5 times with 15 to 30 seconds of rest between trials to allow for software processing. In order for the participant to successfully squat to the maximum depth, they had to show controlled descent until their buttocks touch the stool, and extend back to standing position.

Participants' heels were required to remain on the ground, and their centre of mass had to remain between their feet (they could not put substantial weight on the bench). Peak squat depth was calculated as the distance of the hip joint centers from the floor, divided by total leg length which was defined as the averaged distance between the right and left anterior superior iliac spines to the corresponding medial malleoli.

Data Processing

Both analogue (force plate) and digital (motion capture) data were collected using VICON Workstation software. Workstation allowed the recording and integration of multiple channels during data collection. Using the information collected from each of the seven infrared cameras, Workstation used a mathematical process called direct linear transform to translate the two-dimensional images of the retro-reflective markers from each camera into 3-D coordinates. Once the video data from each individual camera had been integrated into a 3-D image, the spatial coordinates were labelled as specific body markers according to their position on the participant's body. Workstation used the labelled body markers to define body segments according to the segment definitions specified in the marker set (MKR file). It then calculated the kinematics for each trial based on the change in displacement of these body segments between camera frames (Kadaba et al., 1990). Euler angles were calculated at each joint using transformational matrices to determine the position of the distal segment relative to the proximal segment for each joint (Davis et al., 1991). This kinematic data was then coupled with the ground reaction forces measured by the force plates, the anthropometric data which was measured previously, and body segment parameters estimations (Dempster et al., 1959) to calculate joint kinetics (Davis et al., 1991). Before the kinetics and kinematics were calculated, all data was filtered. All marker

trajectories were filtered using a low pass Woltring filter with a predicted mean square error (MSE) of 15, and all raw ground reaction forces were filtered with a low-pass Butterworth filter with a cut-off frequency of 8 Hz. The MSE of 15 was determined through trial and error analysis of pilot data with the goal to remove noisy spikes in the data with minimal attenuation of the clear trends. The cut-off frequency of 8 Hz was chosen due to the slow controlled nature of the tested activities, in order to filter out the high level of noise in the raw ground reaction forces. After these calculations were completed for each participant, the data was transferred to VICON Polygone software which displayed and graphed the data. Finally, all data was exported and graphed in Excel (Microsoft Corporation, Redmond, Washington, USA) to allow for more customized grouping and averaging.

For all the conditions tested in this study, only the symptomatic hip of the unilateral cam impingement group was compared to the averaged left and right hips of the control group. This was because all participants in the FAI group had only one symptomatic hip, but the asymptomatic hip in some had the cam morphology, while the asymptomatic hip in others did not. Because of this, the asymptomatic hips of the cam FAI group were not a homologous sample, and so could not be properly compared with the control group. Furthermore, the sample size of the FAI group was not large enough to stratify and compare the asymptomatic hips with the cam morphology to the asymptomatic hips without the cam morphology. For the FAI group, there was not a strong relationship between the symptomatic hip and left or right side dominance, with the symptomatic hip occurring on the dominant side in approximately 60% of participants. However, approximately 90% of the control group was right side dominant. For this reason, both left and right hips in the control group

were averaged for each hip variable measured to prevent a dominance bias which would occur if only the right or left leg was compared to the FAI group.

Total maximal hip ROM in each plane, as well as peak hip flexion, extension, adduction, abduction and internal and external rotation with the hip flexed at 90° were measured for all participants for the dynamic ROM trials. Only the best three of five trials were averaged for each participant to prevent one or two poor trials from artificially attenuating peak hip mobility values. These averages for each participant were then ensemble averaged for the control and cam FAI groups and compared.

The same kinematic values were measured, as well as maximal 3D pelvic ROM, and peak positive and negative angles in all three planes for level walking. The peak flexion, extension, abduction, adduction and internal and external rotation moments of force generated at the hip were also measured for each participant, as well as the peak positive and negative hip mechanical powers during walking. All values were averaged for each of the five walking trials for each participant. The averaged participant values were then ensemble averaged for the control and FAI groups and compared.

The kinematic variables measured during sit-to-stand were the largest angular displacements in the hip and pelvis in each plane within 20% of the total sit cycle before and after seat-contact. The kinetic variables measured during sit-to-stand were the largest moments of force generated at the hip in each plane within the 20% of the total sit cycle after seat-off. This was because kinetic values could only be calculated when the full bodyweight of each participant was on the force plates. The peak positive and negative mechanical powers values were also measured, but were only limited temporally to the participants' full bodyweight being on the force plates. The same variables were measured for stand-to-sit in

the same manner, except the maximum hip moments of force were recorded in the 20% of sit cycle before seat-contact. Most peak values occurred within close temporal proximity to seat-off or seat-contact, since this was the most demanding aspect of the sit and stand manoeuvres. However there was some temporal variability of peak locations in the frontal and transverse planes, so temporal parameters were specified. Values were limited to the most demanding and relevant time-frame to prevent peak values occurring at the beginning of the cycle being averaged with peak values occurring at the end of the cycle. As with walking, each participant's five standing or sitting trials were averaged together, and then ensemble averaged for the control and cam FAI groups and compared.

Squat kinematic variables consisted of the peak hip angle in each plane at maximal squat depth, the peak 3-D pelvic angles during descent, ascent and at peak depth, the total pelvic ROM in each plane and the maximal attained squat depth. Three-dimensional hip angles were only assessed at maximal squat depth because they tended to only have one peak which occurred at peak squat depth. The 3-D pelvic angular displacements however tended to have a peak during descent, peak squat depth, and again during ascent. Peak squat depth was defined as the lowest point the hip joint centers reached during the squat divided by the participant's total leg height which was the averaged linear distance from the left and right medial malleoli to their respective anterior superior iliac spine. Kinetic variables were only measured for the hip joint and consisted of the peak 3-D moments of force and moment powers generated at the hip during both the ascending and descending phases of the squat. This was because there were clear peaks during ascent and descent, where the largest moments of force, and positive and negative moment powers were generated. All five trials were averaged for each participant, and ensemble averaged for each group.

Statistical Analysis

One-way between-subjects ANOVAs ($\alpha = 0.05$) were run to ascertain significant differences between the control group and the FAI group for all dependent variables. One-way between-subject ANOVAs ($\alpha = 0.05$) were also run for all dependent variables with sex as the independent variable. This was to ensure that there were no significant differences between males and females for each of the variables tested. Pearson correlations ($\alpha = 0.05$) were also run to ensure that age was not significantly correlated to any biomechanical parameters.

The dependent variables statistically analysed between the two groups for maximal dynamic ROM of the hip were the peak 3-D hip angles and the total ROM in each plane. The dynamic ROM dependent variables that were significantly different in between the two groups had additional ANCOVAs ($\alpha = 0.05$) run with flexibility as a covariate. Flexibility was determined by the sit-and-reach test score, and was added as a covariate to minimize the influence of muscular flexibility on dynamic ROM.

For the walking condition, the dependent variables statistically analysed were the peak 3-D hip and pelvic angles and total ROM in each plane, and the peak 3-D moments of force and mechanical powers generated at the hip. One-way between-subject ANOVAs ($\alpha = 0.05$) were also run on walking speed, and step length to ensure there were not significant differences between groups in these variables, as they may have interfered with other results.

For the sit-to-stand and stand-to-sit condition, the dependent variables statistically analysed were peak 3-D hip and pelvic kinematics within their aforementioned temporal parameters, and the peak 3-D hip moments of force and mechanical powers generated at the hip also within the specified temporal parameters. After finding large variability in frontal

and transverse kinematic and kinetic variables for sitting and standing, a series of F-tests ($\alpha = 0.05$) were run to compare the standard deviations between the cam FAI and control group using MedCalc software version 9.5.2.0 (Frank Schoonjans, Mariakerke, Belgium). This was tested to see if there were significant differences in variability between the two groups.

Finally, for the squat condition, the kinematic variables statistically analysed between the two groups were maximal hip angular displacement in each plane at peak squat depth, and peak pelvic angles during squat descent, ascent and maximal squat depth. The kinetic variables tested were the peak moments of force generated at the hip in each plane during squat descent and ascent as well as the maximum positive and negative hip powers throughout squat duration. Any significant differences in the aforementioned squat dependant variables between the two groups ($p < 0.05$) also had an ANCOVA run with squat depth included as a covariate. This was to ensure that the differences in joint kinematics or kinetics resulted from the presence or absence of cam FAI, rather than from discrepancies in squat depth. For all conditions, all ANOVAs and ANCOVAs were calculated using SPSS 15.0 software (SPSS Inc., Chicago, USA).

Articles

The Effect of Cam Femoroacetabular Impingement on Hip Kinematics during Maximal Dynamic Range of Motion

Short Title: Dynamic Hip Mobility of Cam FAI

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Abstract

Cam femoroacetabular impingement (FAI) is caused by decreased concavity of the femoral head-neck junction which results in a jamming of the proximal femur into the acetabulum within normal range of motion (ROM). This condition is known to decrease hip ROM, but only passive hip mobility has been quantitatively studied. It is important to determine the effect of cam FAI on self-generated dynamic ROM in order to ascertain its influence on hip functionality. This study quantified the affect of cam FAI on total dynamic hip ROM in each plane, and on peak angular displacement in flexion, extension, adduction, abduction and internal and external rotation with the hip flexed at 90°. A cam impingement group (n = 17) was compared to a matched control group (n = 14) using between-group one-way ANOVAs. The FAI group had a decreased flexed internal rotation, external rotation and total transverse ROM, as well as decreased hip abduction and total sagittal ROM compared to the matched control group.

INTRODUCTION

Femoroacetabular impingement (FAI) is a relatively new clinical diagnosis,¹ which is gaining recognition in the medical community as a cause of hip pain in young adults.^{2,3} Due to the fact that FAI is a relatively new diagnosis, a limited number of studies have been undertaken to define the multifaceted effects of this condition, contributing to its difficulty to diagnose with certainty.³ However, the database on FAI is rapidly growing due to its current popularity in research.¹ One reason for the increased attention generated by FAI is its potential as a cause of idiopathic hip osteoarthritis (OA).⁴⁻⁹ This relationship with coxa arthritis has increased the urgency to develop surgical strategies to correct FAI before the onset of hip OA.¹⁰⁻¹⁷ However, it is also imperative to increase our understanding of how FAI affects hip function in order to increase the diagnostic criteria for this evasive condition and to develop conservative treatments, especially with the lack of long-term surgical results.

1

FAI is defined by an anatomical morphology of the acetabulum and/or the proximal femur which causes abnormal contact at the hip joint. Pincer impingement is related to overcoverage of the acetabulum as with acetabular retroversion and coxa profunda. Cam impingement is caused by decreased concavity of the femoral head-neck junction, most commonly in the anterosuperior region.^{12,13,18,19} Pincer impingement is caused by a linear abutment of the acetabular labrum with the proximal femur.²⁰ Decreased femoral head-neck concavity causes a jamming of the femoral head into the acetabulum at the limits of range of motion (ROM) during assisted flexion and internal rotation.^{20,21} The pain generated by cam impingement is thought to occur after the onset of cartilage damage, caused by “outside-in” damage of the acetabular labral-chondral junction.¹⁸ Because of the different proposed

pathomechanism between cam and pincer FAI, it was necessary to only focus on one type of impingement to avoid having more than one independent variable. This study only assesses the dynamic ROM of cam FAI.

Depending on the location of bone deformation, FAI patients with *Cam* impingement show a decreased passive hip ROM as compared to healthy controls in flexion,^{19, 22} abduction,¹⁹ internal rotation with hip flexed at 90°^{19, 22, 23} and external rotation with hip flexed at 90°.¹⁹ Similar results were also found in two computer-simulated ROM studies based on the CT scans^{24, 25} of participants with FAI, and matched controls, with FAI patients having decreased simulated hip flexion, abduction and internal rotation. Other studies show a low passive ROM in patients with cam FAI, without comparing to a control group.^{17, 26, 27} Finally Philippon et al.²⁸ found that unilateral FAI patients had decreased passive hip flexion, abduction, adduction, internal rotation and external rotation compared to their contralateral leg. However, there are limitations to these studies. There was no mention of the measuring device used to quantify passive ROM in any of the aforementioned non-simulated studies except for the study by Philippon et al.²⁸ Furthermore, the computer-simulated ROM studies only accounted for bone on bone contact, greatly exaggerating hip mobility, and neglecting the influence of all soft tissues on hip mobility. Finally, these studies only assessed the influence of hip impingement on the simulated, passive or assisted, as oppose to the self-generated dynamic movement of the hip.

In order to predict which activities to avoid in developing a conservative treatment it is imperative to determine how FAI affects the functional mobility of the hip. There are some activities known to cause hip pain in people with FAI such as: squatting or prolonged sitting, stair climbing, excessive walking and athletic activities requiring a large ROM.^{3, 4, 6, 20, 29} However, in order to predict other exacerbating activities it is necessary to decipher which

hip movements are limited, and to quantify the maximum attainable ROM. Furthermore, determining the effect of FAI on self-generated dynamic hip ROM may aid in the diagnosis of this elusive condition.

The purpose of this study was to determine the effect of cam FAI on maximal total hip ROM in each plane, and on peak hip flexion, extension, adduction, abduction, and internal and external rotation with the hip flexed at 90°. According to the dominant trends in passive ROM limitations^{19, 22-25} it is hypothesized that cam FAI will have decreased self-generated dynamic flexion, internal rotation, and abduction.

MATERIALS AND METHODS

Participants

Two groups of participants were recruited: a group of patients diagnosed with cam hip impingement, and a matched control group. The cam impingement participants were recruited after having a positive impingement test and visible cam morphology on anteroposterior (A/P) and Dunn view radiographs.^{6, 26, 30, 31} The alpha angle of Notzli²³ was measured on each hip with a value greater than 50.5 degrees diagnostic of *Cam* type FAI. Matched control participants had no history of serious lower limb injury or surgery, and had spherical femoral heads which were assessed by an A/P radiograph.³¹ Participants from both groups were excluded if hip OA was visible on the x-rays or if they had substantial hip joint space narrowing.

A total of 37 participants volunteered in this study. Eighteen participants diagnosed with cam FAI, and 19 matched control participants by age, gender and BMI. A total sample size of 26 was calculated using G*POWER version 3 (Faul, F. & Erdfelder, E., 2007, Bonn, Germany) with an alpha value set at 0.05, and a minimal power set at 80%. This calculated sample size was the minimum required participants to achieve desired power based on our

pilot data of maximal dynamic internal rotation, however; since we were looking for other variables as well, a larger sample size was chosen to maximize the statistical power of the study. Before participating in the study, which was approved by the Ottawa Hospital Research Ethics Board, and the University of Ottawa Health Sciences and Science Research Ethics Board, all participants signed an informed written consent. Five of the original control participants were excluded. It was determined that three of them had at least one aspherical femoral head based on an A/P radiograph, and the other two could not have the necessary screening x-rays. Furthermore, one participant of the FAI group had to be excluded due to the presence of hip OA. Consequently, the remaining samples consisted of 14 control participants and 17 FAI participants (Table A1). Even with the diminished sample size, we still had more participants than the power analysis required. All FAI participants had *Cam* type deformities with only one symptomatic hip. All participants filled out a Western Ontario McMaster Osteoarthritis Index ³² (WOMAC) questionnaire to control for hip pain, function and stiffness.

Table A 1. Participant characteristics (mean $\pm$ standard deviation)

Condition	Number of Participants	Age (years)	BMI (kg/m ²)	WOMAC	Flexibility
Control	14 (8 males)	34.2 $\pm$ 9.5	23.0 $\pm$ 2.3	99.9 $\pm$ 0.3	27.2 $\pm$ 9.8
Cam FAI	17 (10 males)	35.5 $\pm$ 10.6	23.0 $\pm$ 2.3	77.3 $\pm$ 15.6	21.1 $\pm$ 7.5

Materials

Three-dimensional kinematics of dynamic ROM was collected using seven VICON MX-13 cameras (VICON, Los Angeles, CA USA) at 200 Hz with retro-reflective markers placed on anatomical landmarks according to a modified Helen-Hayes marker-set. ³³ A

support frame was constructed to give stability to the participants during dynamic hip ROM trials.

Procedure

Participants were asked to put on a skin-tight suit and performed a stretching warm-up routine. After the stretching warm-up, participants completed a sit and reach flexibility test, had retro-reflective markers placed on anatomical landmarks, and underwent anthropometric measurements. Thereafter participants performed a series of maximal dynamic hip ROM trials. Participants stood upright holding onto a support frame while they maximally swung their leg back and forth in five times in each plane at a self selected speed. Hip ROM movements consisted of maximal dynamic flexion and extension, adduction (with the mobile leg in front of the support leg) and abduction, and internal and external rotation (while flexed at 90 degrees) (Figure A1). Movements in each plane were completed 5 times for each participant.



Figure A 1. Instrumented participant performing dynamic ROM trial (external rotation at 90° of hip flexion) while holding on to support frame.

Data Analysis

Lower limb 3-D kinematics were calculated according to the methods described by Kadaba et al.^{33, 34} and Davis et al.,³⁵ using VICON Workstation (VICON, Los Angeles, CA USA) processing software. Hip joint angles were based on local Euler coordinate systems as defined by the aforementioned studies. Joint and segment angles were zeroed based on each participant's neutral position as determined by a standing static trial where feet were parallel and facing anteriorly approximately shoulder width apart. However, for the flexed internal and external rotation trials, the transverse hip angle was zeroed based on its transverse angle when the hip was flexed to 90° and the midline of the tibia was orthogonal to the line connecting the left and right anterior superior iliac spines of the pelvis when viewed down the axis of the femur (Figure A2).

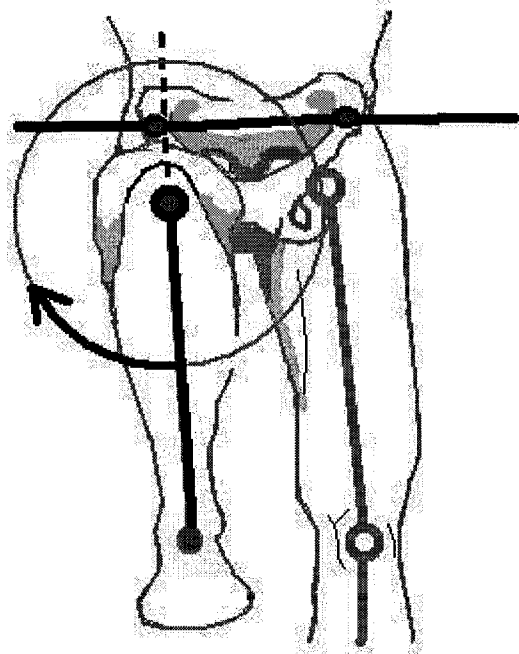


Figure A 2. The definition of neutral transverse hip angle with the angle flexed to 90°, with the arrow pointing in the positive direction (internal rotation)

The kinematic variables measured in the dynamic ROM trials consisted of the peak angular displacements and total ROM of the hip in each plane. Peak angular displacement was defined as the largest hip angle achieved in flexion, extension, abduction, adduction, and internal and external rotation. The maximum range was calculated based on the addition of the two opposite maximums in each plane. Of the five dynamic ROM trials for each participant, the three largest peak angular displacements and corresponding ranges were averaged to determine maximum angles and total range in each plane. This was done to ensure that the maximal values were not attenuated by a few poor trials. These were then ensemble averaged for both groups and compared.

Statistical Analysis

One-way between-subjects ANOVAs ($\alpha = 0.05$) were run for all dependent variables in order to ascertain significant differences between the control group and the FAI group. The dependent variables tested between groups were: total sagittal ROM, total frontal ROM, total transverse ROM (with the hip flexed at 90°), peak flexion, peak extension, peak adduction, peak abduction, flexed internal rotation and flexed external rotation. The dependent variables that were significantly different between the two groups had additional ANCOVAs ($\alpha = 0.05$) was run with flexibility as a covariate. Flexibility was determined by the sit-and-reach test score, and was added as a covariate to minimize the influence of muscular flexibility on dynamic ROM.

RESULTS

The FAI group had significantly lower peak hip internal rotation ($p = 0.005$) peak hip external rotation ($p = 0.009$) and total transverse hip ROM ($p = 0.002$) with 90° of hip flexion (figure A3), significantly lower peak hip abduction ($p = 0.003$) (figure A4), and significantly

lower total sagittal hip ROM ($p = 0.031$) (figure A5) than the control group. None of the other dynamic ROM variables were significantly different. However, there was a trend for the FAI group to display a lower peak hip extension ($p = 0.054$), and lower total hip frontal ROM ($p = 0.079$) compared to the control group. Refer to table 2 for the mean values of each finding.

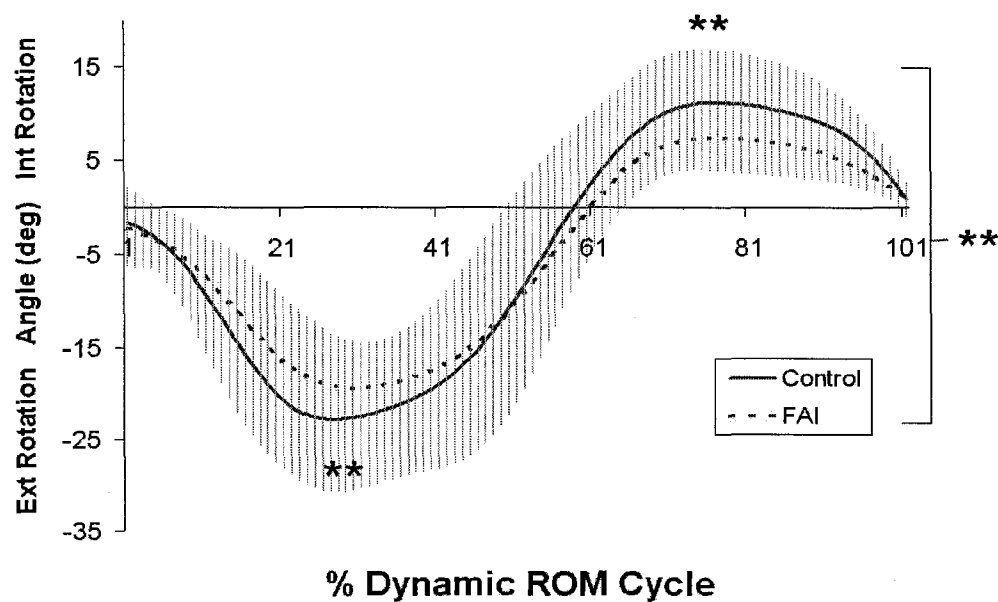


Figure A 3. Mean maximal dynamic ROM in transverse plane at 90° of hip flexion ($\pm$ standard deviation) for the symptomatic hip of cam FAI group, and for the averaged left and right hips of the matched control. Significant differences were found between the control and FAI group for peak internal rotation ($p = 0.005$), for peak external rotation ($p = 0.009$), and for total transverse ROM ($p = 0.002$).

‘*’ : $p < 0.05$

‘**’ : $p < 0.01$

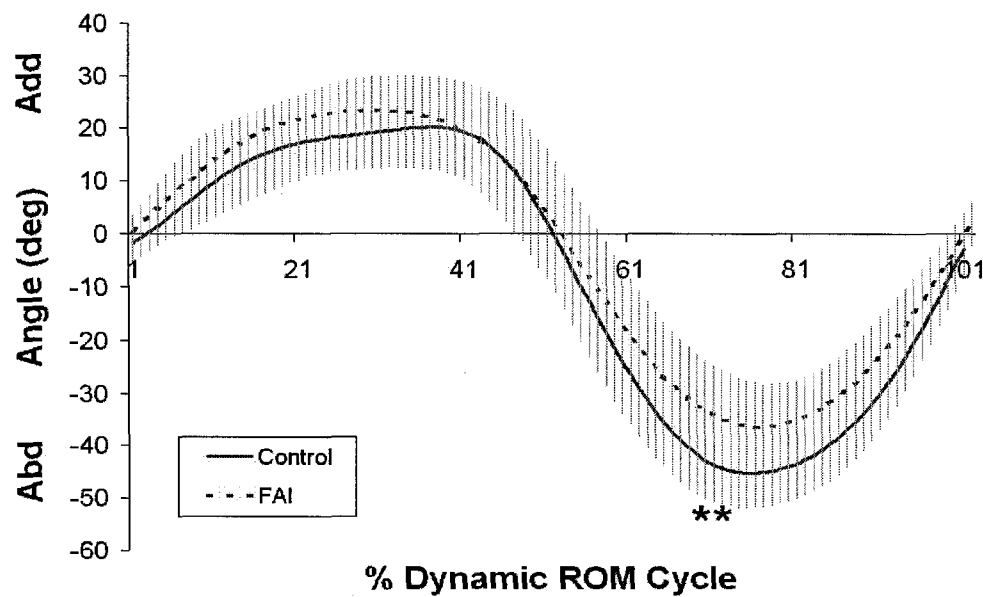


Figure A 4. Mean maximal dynamic ROM in the frontal plane ($\pm$ standard deviation). A significant differences was found between the control and FAI group for peak abduction ($p = 0.003$).

‘*’ : $p < 0.05$

‘**’ : $p < 0.01$

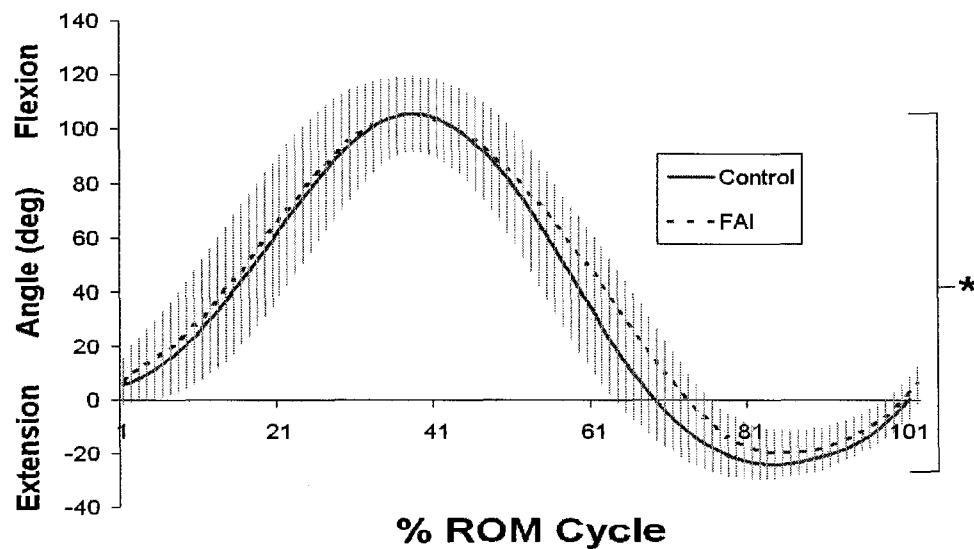


Figure A 5. Mean maximal dynamic ROM in the sagittal plane ($\pm$ standard deviation). A significant difference was found between the control and FAI group for total sagittal ROM ($p = 0.031$).

‘*’ : $p < 0.05$

‘**’ : $p < 0.01$

Table A 2. Average values from the dynamic Range of Motion significant findings. Mean $\pm$ standard deviation.

Dependent Variable	Group	Mean Value ($\pm$ SD)	F-Value	P-Value
Peak Flexion	Control	113.9 $\pm$ 9.4	1.467	0.236
	Cam FAI	109.8 $\pm$ 9.6		
Peak Extension	Control	26.6 $\pm$ 5.1	4.050	0.054
	Cam FAI	21.2 $\pm$ 9.0		
Total Sagittal ROM	Control	140.5 $\pm$ 9.0	5.163	0.031
	Cam FAI	130.9 $\pm$ 13.5		
Peak Abduction	Control	47.5 $\pm$ 5.7	10.773	0.003
	Cam FAI	38.3 $\pm$ 9.1		
Peak Adduction	Control	23.2 $\pm$ 7.5	0.235	0.632
	Cam FAI	24.4 $\pm$ 5.9		
Total Frontal ROM	Control	70.7 $\pm$ 12.2	3.307	0.079
	Cam FAI	62.7 $\pm$ 12.3		
Peak Internal Rotation (at 90° of hip flexion)	Control	12.4 $\pm$ 4.8	9.438	0.005
	Cam FAI	7.9 $\pm$ 3.4		
Peak External Rotation (at 90° of hip flexion)	Control	26.0 $\pm$ 7.0	7.722	0.009
	Cam FAI	20.3 $\pm$ 4.5		
Total Transverse ROM (at 90° of hip flexion)	Control	38.5 $\pm$ 9.7	12.077	0.002
	Cam FAI	28.2 $\pm$ 6.7		

DISCUSSION

Dynamic ROM

It has been well documented that cam FAI causes a decrease in passive hip ROM,^{17, 19, 22, 26-28} and produces bony limitations within the normal range of motion.^{24, 25}

Furthermore, it is common for patients to notice limited hip motion even before the onset of pain,²² indicating a loss of functional mobility. The limited mobility and joint damage characteristic of cam FAI is caused by premature abutment of the femoral head-neck junction with the acetabular rim, and is more dependent on extreme hip mobility than on axial loading.^{4, 19, 20} Since the jamming of the abnormal femoral head-neck junction into the acetabulum that causes joint damage occurs during self-generated dynamic hip movements,^{3, 4, 6, 20, 29} it is important to measure the extent to which active dynamic ROM is affected by

FAI. This information will allow for a deeper understanding of cam impingement, and is essential for predicting which movements will exacerbate the condition in developing conservative treatments and could add to diagnosis criteria for this evasive condition.

The results from our study on the impact of FAI on hip mobility have supported our hypotheses that this patient group would have decreased maximal dynamic self-generated internal rotation and abduction, but have refuted the hypothesis that the FAI group would have decreased maximal dynamic hip flexion. Moreover, there was also significant attenuation in maximal hip dynamic mobility in flexed external rotation, total hip transverse ROM and total hip sagittal ROM in the FAI group compared to the healthy matched control.

There are some limitations inherent to joint kinematic studies which may limit their accuracy and sensitivity. These limitations are caused by the use of generic calculations, marker misplacements³⁶ and skin or clothing artefacts.^{37,38} However, precautions were used to minimize the potential errors caused by these limitations. All marker placements were performed by the same investigator to minimize variability in placement locations. All angles were zeroed based on neutral static trials which removed any artificial neutral angles caused by marker misplacements. Finally, markers were either placed on the skin or tight spandex clothing to minimize clothing artefacts.

We found that there were similarities between the effect of FAI on passive hip ROM and dynamic hip ROM. The literature on passive hip ROM in participants with FAI indicated decreased maximal internal rotation at 90° of hip flexion^{19, 22-25} and abduction^{19, 24, 25, 28} compared to a matched control, both of which were supported by our dynamic mobility results. Both of these findings correspond well to the proposed pathomechanism of cam FAI which states that impingement results from the jamming of the abnormal ridge of the femoral head-neck junction into the anterosuperior aspect of the acetabulum.^{20, 21} Flexed internal

rotation^{4, 19, 28} and abduction^{24, 39} rotate the anterosuperior femoral head-neck junction towards the anterosuperior aspects of the acetabulum. Therefore, both dynamic flexed internal rotation and abduction of the hip are likely restricted by the proximal femur jamming into the acetabulum.

Conversely, reduced passive hip external rotation in FAI participants has conflicting findings in the literature. Our results showed that FAI participants had reduced self-generated dynamic external rotation compared to the control group. In accordance with our results, Ito et al.¹⁹ found a significant decrease in passive hip external rotation with the hip flexed at 90° in patients with FAI compared to healthy controls ($p = 0.02$). Another study by Phillipon et al.²⁸ found a decrease in passive external rotation with a fully extended hip in the symptomatic leg of unilateral FAI participants as compared to the contralateral leg. Despite the hip being in a different orientation with a straight legged rotation as compared to a flexed rotation, the movement is similar enough to be relevant to this study. Contrary to these findings, computer simulated hip ROM studies on the effect of FAI on hip mobility showed no significant differences between the FAI and control groups^{24, 25}. It should be noted that the aforementioned computer simulated studies examined only bony anatomy and neglected all soft tissues. This resulted in an exaggeration of hip mobility, with the predicted mobility in some planes being outside reasonable values. For example, the predicted maximum hip external rotation in 90° of flexion was 80-100°^{24, 25} compared to the results from live analysis in the literature of 20-30°^{19, 26} which are consistent with our results. The evident influence of soft tissue in hip external rotation makes these computer simulated studies, which neglected all soft tissue, irrelevant to our dynamic external rotation results, leaving only supportive passive external rotation findings in the literature. Previous studies have not tested for the effect of FAI on total transverse ROM. However, it can be assumed that since previous

research has documented decreased passive internal ^{19, 22-25} and external rotation at 90° of hip flexion ¹⁹ in participants with FAI, there would be decreased total hip passive ROM. This also supports our findings on dynamic hip transverse ROM.

Although there is support of our reduction of dynamic flexed external rotation results in the passive hip ROM literature,^{19, 28} this finding does not fit the abovementioned pathomechanism for cam FAI.^{20, 21} External rotation rotates the anteroposterior femoral head-neck ridge away from the acetabular labrum,^{39, 40} which also occurs while the hip is flexed as indicated by the large flexed external rotation predicted without the presence of soft tissue in the computer simulated studies.^{24, 25} This suggests that the soft-tissue of the hip likely contributes to the limited external rotation in the FAI group rather than just the bony structures and labrum. It is possible that the bulge on the anterosuperior head-neck junction in cam FAI causes increased tension on the iliofemoral ligament – which crosses the anterosuperior femoral neck, and limits external rotation ³⁹ – during flexed external rotation. This could contribute to the restricted hip mobility, however this is purely speculation and beyond the scope of this study.

Not all of our dynamic hip mobility findings were supported by the passive ROM literature. Most literature reported attenuated passive hip flexion in FAI patients,^{19, 22, 24, 25} yet our results showed no significant differences between the two groups in dynamic hip flexion ($p = 0.236$). The FAI group did have a slight decrease in hip flexion, but by only four degrees. The relatively high variability in peak hip flexion with a standard deviation of approximately nine degrees, coupled with a modest sample size, rendered the small difference between groups not significant. The convex anterosuperior head-neck junction is moved towards the anterosuperior acetabular labrum during hip flexion.^{4, 19, 28} Based on the proposed pathomechanism of cam FAI,^{20, 21} one would expect cam FAI to have decreased

hip flexion. A potential explanation is that self-generated dynamic hip flexion cannot achieve true peak hip flexion without external assistance.³⁹ The added applied pressure from a clinician in passive ROM trials may be required to push the hip to the point where bony limitation occurs, and therefore may be required in order to elicit differences in hip flexion between the FAI and control groups. This explanation is supported by the fact that our control group's peak mean flexion was only 113.9° compared to approximately 120-130° of flexion in the control groups of other studies,^{19, 24, 25, 28} indicating that the level of assisted hip flexion substantially exceeds self-generated peak hip flexion. Furthermore, this explanation is logical given the mechanical disadvantage hip flexor muscles are placed at when the hip approaches peak flexion, and since the hip flexor muscles are relaxed and can be compressed during passive hip flexion.³⁹ Although the literature reports decreased assisted maximal hip flexion, there is no significant decrease in self-generated hip flexion in participants with FAI compared to matched controls.

Considering the lack of expected differences in peak hip flexion between the two groups, it is surprising that the FAI group had decreased total sagittal hip mobility. This decreased level of sagittal hip ROM in participants with FAI was primarily a result of the impingement group having less (marginally significant) hip extension ($p = 0.054$) than the control group. No previous studies on passive hip mobility in patients with FAI report a decrease in hip extension. The proposed pathomechanism of cam impingement^{20, 21}, indicates hip mobility limitations are caused by contact between the femur and acetabular labrum. However, hip extension rotates the anterosuperior head-neck ridge away from the acetabular edge, avoiding contact with the acetabulum³⁹ indicating that it would not be affected by FAI. Furthermore, hip extension is solely limited by soft tissue such as the iliofemoral ligament,^{39, 41, 42} rather than bony contact. The decreased sagittal hip ROM in the

FAI group ($p = 0.031$), primarily resulting from hip extension ($p = 0.054$), indicates that the anatomical abnormality may influence the muscles and ligaments supporting the hip joint as well as the documented effect on bone and cartilage. Both the tendon of the iliopsoas muscle and the iliofemoral ligament become very tight during maximal hip extension, and may cause damage to the anterosuperior hip joint capsule and the development of herniation pits on the proximal femur^{42, 44} which are associated with cam FAI.²⁰ A bulge on the anterosuperior femoral head-neck junction could increase the distance these tissues travel to their distal attachments, causing this tension to occur earlier, and limiting extension. Our findings give no insight on the exact modality of limited hip sagittal ROM in FAI participants, but indicate that it is likely influenced by soft tissue.

In summary, participants diagnosed with cam FAI have significantly lower self-generated dynamic hip internal rotation at 90° of hip flexion and peak hip abduction compared to a matched control group in agreement with our hypotheses. However the postulated reduction in dynamic peak hip flexion in the FAI group as compared to the control group was not supported by our results. We also found that participants with cam FAI have a significantly lower dynamic flexed external rotation, and flexed total transverse ROM. Although not widely acknowledged, there is also supporting evidence of these findings in the passive hip mobility literature.¹⁹ Finally, despite the lack of differences in peak hip flexion, the FAI group had attenuated total sagittal hip ROM compared to the control. This unexpected finding was primarily a result of a large but non-significant decrease in peak hip extension in the FAI group. The differences in dynamic flexed external rotation and total sagittal ROM were likely a result of an abnormal soft tissue restriction in the impingement group. The effect of soft tissue on hip mobility in cam FAI should be investigated in future research.

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***Hip Biomechanics of Cam Femoroacetabular Impingement During
Daily Activities***

Short Title: Hip Biomechanics of FAI

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Abstract

Femoroacetabular impingement (FAI) has been reported to cause hip pain in a variety of daily activities including sitting and walking. However, it has not been determined whether there is a difference in how these activities are performed. This study quantified the affect of cam FAI on the three-dimensional (3-D) kinematics of the hip and pelvis, as well as the 3-D kinetics generated at the hip during walking, sit-to-stand and stand-to-sit. A unilateral cam impingement group ($n = 17$) was compared to a matched control group ($n = 14$) using between-group one-way ANOVAs, and level of variability was assessed using F-tests. For walking, the FAI had significantly lower peak hip abduction, frontal range of motion (ROM), and sagittal ROM than the controls, as well as attenuated pelvic frontal ROM. However, there were no kinetic differences between the two groups. For the sit-to-stand and stand-to-sit manoeuvres there were no significant differences in peak kinematic or kinetic values of the hip and pelvis between the two groups. However, the cam FAI group had significantly higher variability in peak angles in the frontal plane, and peak hip extension moments of force during both sitting and standing. It is likely that the higher variability in sit-to-stand and stand-to-sit strategy in FAI participants resulted from varying compensatory strategies. Attenuated hip abduction, frontal ROM and sagittal ROM during gait in FAI individuals may be caused by soft tissue restriction. Finally, the gait results may indicate limited frontal pelvic mobility in FAI participants.

INTRODUCTION

Femoroacetabular impingement (FAI) is a morphological hip condition associated with hip pain in young active adults,¹ which is gaining momentum in the research community due to its potential causal relationship with idiopathic hip osteoarthritis.²⁻⁷ Despite the increasing number of studies being undertaken about FAI, the condition is still often misdiagnosed and is difficult to identify with certainty.^{1,2} There are two variations of FAI which are defined by an anatomical abnormality of the acetabulum and/or the proximal femur resulting in abnormal contact at the hip joint. Cam impingement is caused by decreased concavity of the femoral head-neck junction most commonly in the anterosuperior region.⁸⁻¹¹ Pincer impingement is a result of local or general over-coverage of the femoral head by the acetabulum,⁸⁻¹³ which results in a linear abutment between the femoral neck and the labrum.² Because of the differences between cam and pincer impingement, it was necessary to only assess one type in order to limit the study focus to one independent variable.

In a healthy impingement free hip, the femoral head-neck offset prevents contact between the femoral neck and pelvis within a normal range of motion (ROM).⁶ However, decreased femoral head-neck concavity causes a jamming of the femoral head into the acetabulum at the limits of motion.¹² This contact produces repetitive low impact loading, with peak loads occurring during flexion and internal rotation.¹⁴ A variety of activities of daily living (ADL) produce hip pain in people with FAI such as: low depth or prolonged sitting, stair climbing and excessive walking.^{1,2,4,14,15} This pain is thought to occur after the onset of joint damage, and is associated with contact between the proximal femur and labral or chondral lesions.¹⁶ This study assesses the biomechanics of ADLs in participants with cam FAI.

Sit-to-stand, stand-to-sit and level gait are three of the most common and essential activities to independent living. Although they are known to cause hip pain, there is a paucity of quantitative research concerning differences in sit-to-stand, stand-to-sit and level gait performance between individuals with FAI and the healthy population. However, a computer simulated motion analysis study by Rab¹⁷ analyzed stand-to-sit and walking in hips with slipped capital femoral epiphysis (SCFE), which is associated with cam FAI and has a closely related abnormal morphology.¹⁸⁻²⁰ This study predicted increased external rotation in the SCFE group during sitting, to avoid femoroacetabular contact. This prediction is interesting and should be tested with live data collection on participants with cam FAI. Rab¹⁷ also reported that SCFE commonly causes increased hip adduction and external rotation during gait, and the model predicted increased hip external rotation and decreased hip flexion during gait compared to normal.¹⁷ It should also be noted that these results were derived using a computer simulation which only accounted for bone tissue, neglecting the labrum and all soft tissue. Furthermore, internal and external rotation were defined as rotation about the long axis of the femoral neck, rather than the long axis connecting the hip joint centre to the knee joint centre, making these results more difficult to interpret. In contrast to these walking findings, Zebala et al.²¹ reported that patients with FAI had a normal gait, but that an antalgic gait may occur with FAI progression. However, this study was not quantitative, and was based on a visual appraisal of gait which is only capable to discern relatively large deviations from normal gait. Since these results are in contradiction, and both have limited relevance to the cam FAI population, it is important to properly quantify the true effect of cam FAI on gait using live capture. Assessing and quantifying how ADL's are affected by cam FAI will lead to a deeper understanding of this condition, as well as potentially identifying additional diagnostic criteria for this evasive pathology.

Although Rab¹⁷ predicted differences in hip kinematics during walking in patients with an abnormal femoral head-neck junction, there were contradictory results²¹ in FAI patients. Because there was so little previous research on the affect of FAI on walking, and due to the discontinuity between the two studies, we decided to base our walking hypotheses on comparisons between FAI passive hip ROM²² and the required hip mobility for walking in the literature.²³⁻²⁷ Only hip kinematics were assessed because no literature on the influence of hip moment of force or mechanical power generation on eliciting impingement in individuals with FAI could be found. We based our kinetic postulations on our kinematic hypotheses, founded on the idea that any differences in joint kinematics must be a result of altered kinetics. Cam impingement produces abnormal contact between the proximal femur and the acetabular edge at the limits of hip ROM.¹² Due to the low level of hip mobility required for level walking,²³⁻²⁷ which is within the limits of FAI hip mobility,²² we postulated that there would be no differences in hip kinematics or kinetics during level gait.

The fact that the only relevant study we found in the literature which assessed the influence of a femoral head-neck junction abnormality on standing kinematics was a computer simulation which neglected all soft tissue including the acetabular labrum, we decided to use a similar strategy for determining our standing and sitting hypotheses as we did for walking. There were numerous studies which quantified the sagittal kinematics of the hip²⁸⁻³¹ during sitting and standing, but no previous research could be found which reported the frontal and transverse kinematics of sit-to-stand and stand-to-sit manoeuvres. The peak hip flexion required for sitting and standing²⁸⁻³¹ was within the reported peak hip flexion in participants with FAI,²² so we postulated that there would be no differences in sagittal hip kinematics and kinetics during sitting and standing between the FAI and control group. Since there was no information on transverse and frontal hip mobility required for sitting and

standing, we could not form educated hypotheses for these variables. We decided to use a purely exploratory approach for sit and stand frontal and transverse kinematics and kinetics. Finally, there was nothing in the literature on maximal pelvic mobility in participants with cam FAI, so no hypotheses were formed on pelvic kinematics for the aforementioned ADLs, and this section was also purely exploratory.

MATERIALS AND METHODS

Participants

Two groups of participants were recruited: a group of patients diagnosed with unilateral cam hip impingement ($n = 17$ with 10 men), and a matched control group ($n = 14$ with 8 men). The cam impingement participants were recruited after having a positive impingement test and visible cam morphology on anteroposterior (AP) and Dunn view radiographs.^{4, 32-34} Matched control participants had no history of serious lower limb injury or surgery, and had spherical femoral heads which were assessed by an A/P radiograph³⁴. Participants from both groups were excluded if hip OA was visible on the x-rays or if they had substantial hip joint space narrowing.

A total of 37 participants volunteered in this study. Eighteen participants diagnosed with cam FAI, and 19 age, sex and BMI matched control participants. Before participating in the study which was approved by the University of Ottawa Health Sciences and Science Research Ethics Board, all participants signed an informed written consent. However, three of the control participants were excluded after it was determined that they had at least one aspherical femoral head based on an A/P radiograph, and two other could not have the necessary screening x-rays. Furthermore, one of the FAI group had to be excluded due to the presence of hip OA. The remaining samples consisted of 14 control participants and 17 FAI participants; refer to table B1 for sample characteristics.

Table B 1. Participant characteristics. Mean $\pm$ standard deviation.

Condition	Number of Participants	Age (years)	BMI (kg/m²)	WOMAC	Flexibility
Control	14 (8 males)	34.2 $\pm$ 9.5	23.0 $\pm$ 2.3	99.9 $\pm$ 0.3	27.2 $\pm$ 9.8
Cam FAI	17 (10 males)	35.5 $\pm$ 10.6	23.0 $\pm$ 2.3	77.3 $\pm$ 15.6	21.1 $\pm$ 7.5

Materials

Three dimensional kinematics of sit-to-stand and stand-to-sit motion and walking were collected using seven VICON MX-13 cameras (VICON, Los Angeles, CA USA) at 200 Hz with retro-reflective markers placed on anatomical landmarks according to a modified Helen-Hayes marker-set³⁵. Three-dimensional ground reaction forces were measured using two AMTI force plates (one AMTI OR6-6-2000, and one AMTI OR6-6-1000, Watertown, MA USA) captured at 1000 Hz, which were placed side-by-side and fixed to the floor in the middle of a raised walkway. A height adjustable bench was used to control seat height.

Procedure

Participants filled out a WOMAC questionnaire with instructions to answer each question according to their hip joint. They were then asked to put on a skin-tight suit and performed a stretching warm-up routine. After the stretching warm-up, participants completed a sit and reach flexibility test, had retro-reflective markers placed on anatomical landmarks, and underwent anthropometric measurements. Participants then performed a static trial where they stood in a neutral position with their feet shoulder-width apart and facing forward, their legs straight, and their arms at their sides. This static trial was used for segment length calculations and to define neutral joint angles.

After set-up, each participant performed a few practice walking trials until they could land with their foot on a force-plate which was fixed to the floor in the middle of the walkway without altering their gait. Participants were asked to walk at a natural speed to help

control for biomechanical differences caused by varying gait speed.³⁶⁻³⁹ Each participant performed ten level walking trials, five with their left foot and five with their right foot landing on the same force-plate (Figure B1). The force plates were positioned side-by-side to allow for the calculation of hip kinetics for both legs independently in the sit-to-stand and stand-to-sit conditions.

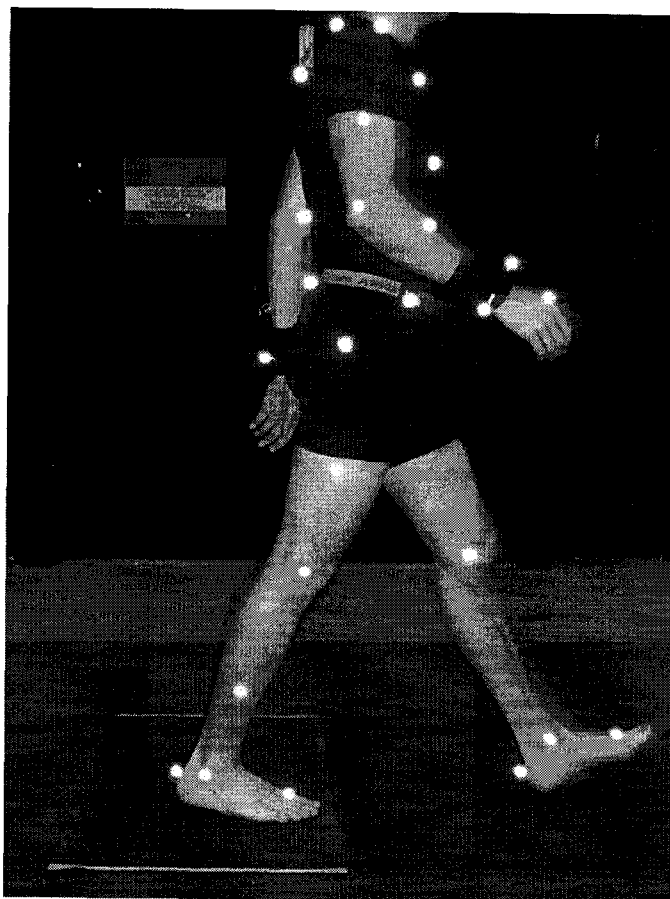


Figure B 1. Instrumented participant performing level walking trial with right foot landing on a force-plate which is fixed into the walkway.

For both sitting and standing trials, participants were asked to stand 10 cm in front of the height-adjustable bench⁴⁰⁻⁴³ with both feet shoulder-width apart, parallel to each other and facing anteriorly. Before any sitting or standing trials were collected, the bench was adjusted to the height of the participant's tibial plateau.^{40, 44-47} Stand-to-sit and sit-to-stand were two connected movements and were collected in tandem, with participants being asked

to sit down, pause, stand up, pause and so forth without moving their feet until five trials of each were collected. Starting from standing, participants were asked to cross their arms across their chest,^{43, 48} sit down on the height-adjusted stool, pause until notified, and then stand back up at a self selected and controlled pace.²⁹ Crossing the arms prevented the temptation to use them for support during the manoeuvres (Figure B2). All participants performed five sit-to-stand and five stand-to-sit trials, and the hip and pelvis kinematics and hip kinetics were calculated and averaged for each.

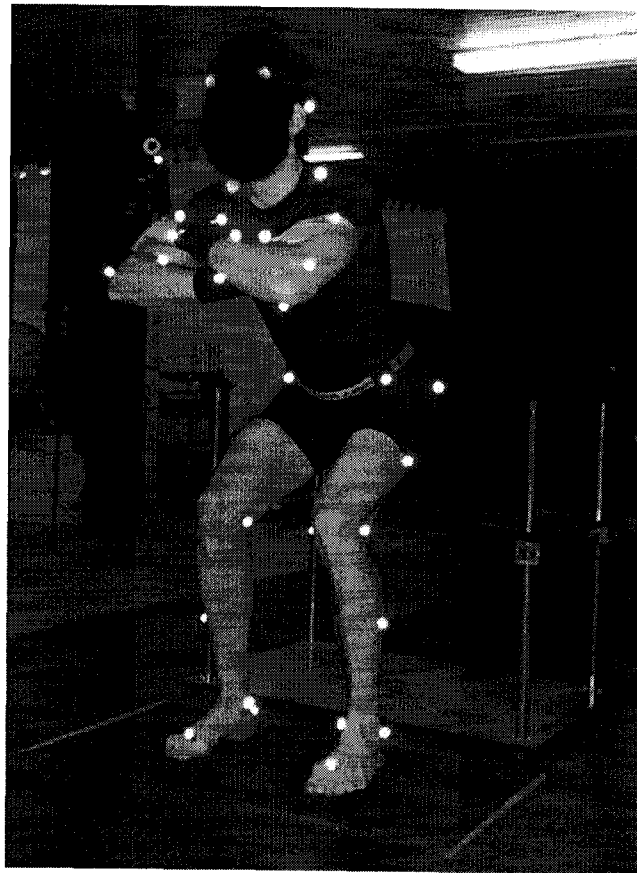


Figure B 2. Instrumented participant performing a sitting and standing trial with a height-adjustable bench adjusted to the height of the tibial plateau.

Data Analysis

Lower limb 3-D kinematics and kinetics were calculated according to the methods explained by Kadaba et al.^{35, 49} and Davis et al.⁵⁰, using VICON Workstation (VICON, Los

Angeles, CA USA) processing software. Walking speed and step length were also measured using VICON Workstation software. Hip joint angles were based on local Euler coordinate systems as defined by the aforementioned studies, and pelvic angles were measured relative to a global coordinate system. Joint and segment angles were zeroed based on each participant's neutral standing position.

The hip kinematic variables measured in the walking trials consisted of the peak hip flexion, extension, total sagittal ROM, adduction, abduction, total frontal ROM, internal and external rotation, and total transverse ROM. The pelvis angles measured were the peak positive and negative displacements and total ROM in each plane. The kinetic variables tested were the peak flexion, extension, adduction, abduction and internal and external moments of force generated at the hip in each plane, and the peak positive and negative hip powers. Peak 3-D kinetic and kinematic variables were measured without temporal parameters due to the relatively homogenous 3-D kinematic and kinetic patterns, with peak values occurring at the same time in the cycle. Winter reported variability in hip extensor moments during level gait⁵¹ however, the location of peak flexion and extension moments seem to be consistent throughout the literature with peak extension occurring at the beginning of stance phase, and peak flexion occurring near toe-off.^{23, 24, 27, 51, 52} Although there is larger variability in the literature for transverse and frontal hip kinematics and kinetics, peak values also tend to occur within the same general time-frame.^{23, 24} For each participant all five walking trials were averaged, and these values were ensemble averaged for the control and cam FAI groups and compared. Walking speed and step length were also calculated and compared between the FAI and control group to ensure that they were properly controlled.

The kinematic variables measured during sit-to-stand were the largest angular displacements in the hip and pelvis in each plane within 20% of the total sit cycle before and after seat-contact. The kinetic variables measured during sit-to-stand were the largest moments of force generated at the hip in each plane within the 20% of the total sit cycle after seat-off. This was because kinetic values could only be calculated when the full bodyweight of each participant was on the force plates. The peak positive and negative mechanical powers values were also measured, but with no temporal parameters. The same variables were measured for stand-to-sit in the same manner, except the maximum hip moments of force were recorded in the 20% of sit cycle before seat-contact. Most peak values occurred within close temporal proximity to seat-off or seat-contact, since this was the most demanding aspect of the sit and stand manoeuvres. However there was some temporal variability of peak locations in the frontal and transverse planes, so temporal parameters were specified. Values were limited to the most demanding and relevant time-frame to prevent peak values occurring at the beginning of the cycle being averaged with peak values occurring at the end of the cycle. As with walking, each participant's five standing or sitting trials were averaged together, and then ensemble averaged for the control and cam FAI groups and compared.

Statistical Analysis

One-way between-subjects ANOVAs ($\alpha = 0.05$) were run to ascertain significant differences between the control group and the FAI group for all dependent variables: peak 3-D hip and pelvis walking angles and total ROM in each plane; peak 3-D moments of force and mechanical powers generated at the hip during walking; peak 3-D hip and pelvic sit-to-stand and stand-to-sit kinematics; peak 3-D hip moments of force and mechanical powers

generated at the hip during standing and sitting. One-way between-subject ANOVAs ($\alpha = 0.05$) were also run on walking speed, and step length to ensure they did not interfere with other results. All ANOVAs were calculated using SPSS 15.0 software (SPSS Inc., Chicago, USA).

After finding large variability in frontal and transverse kinematic and kinetic variables for sitting and standing, a series of F-tests ($\alpha = 0.05$) were run to compare the standard deviations between the cam FAI and control group using MedCalc software version 9.5.2.0 (Frank Schoonjans, Mariakerke, Belgium).

RESULTS

Walking

The cam FAI group had a significantly lower peak hip abduction ($p = 0.009$) angle, significantly less total frontal hip ROM ($p = 0.003$), and significantly lower total sagittal hip ROM ($p = 0.047$) during level gait than the matched control. Peak hip abduction occurred at the beginning of swing phase, but the decrease in abduction (or increased adduction) continued throughout the swing phase (Figure B3). The largest difference in sagittal hip ROM occurred in hip extension at the end of stance phase (Figure B4). The cam FAI group also had significantly less pelvic ROM in the frontal plane ($p = 0.004$) than the control group during level gait, with the largest decreases occurring at the beginning of stance phase, and just after toe-off (Figure B5). There were no other significant differences in kinematic variables, nor were there any significant differences in kinetic variables between the FAI and control groups. Finally, there were no significant differences between walking speed and step length between the two groups.

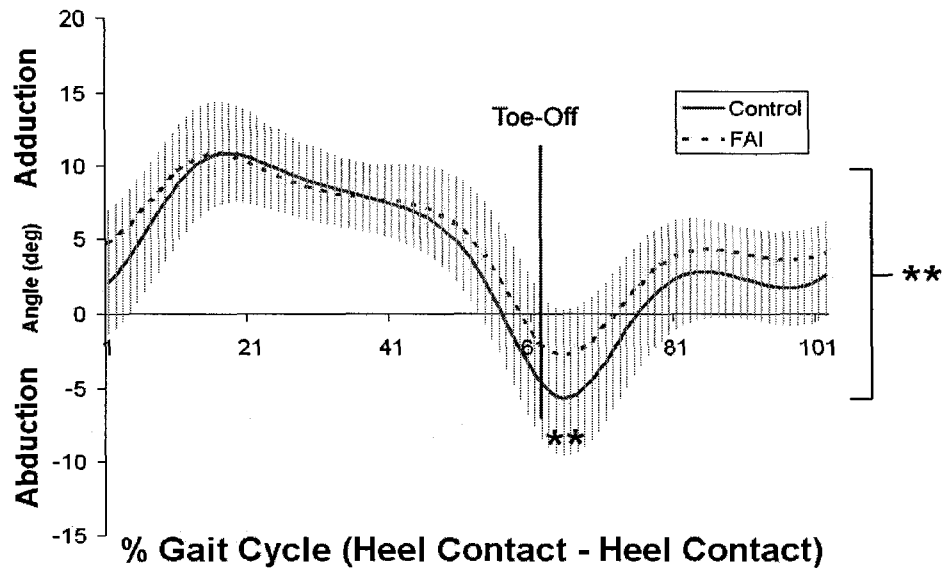


Figure B 3. Mean ($\pm$ standard deviation) of frontal hip angles of the FAI and control groups during level gait. The FAI group had decreased peak hip abduction ($p = 0.009$), and decreased total frontal hip ROM ($p = 0.003$).

'*' = $p < 0.05$

'**' = $p < 0.01$

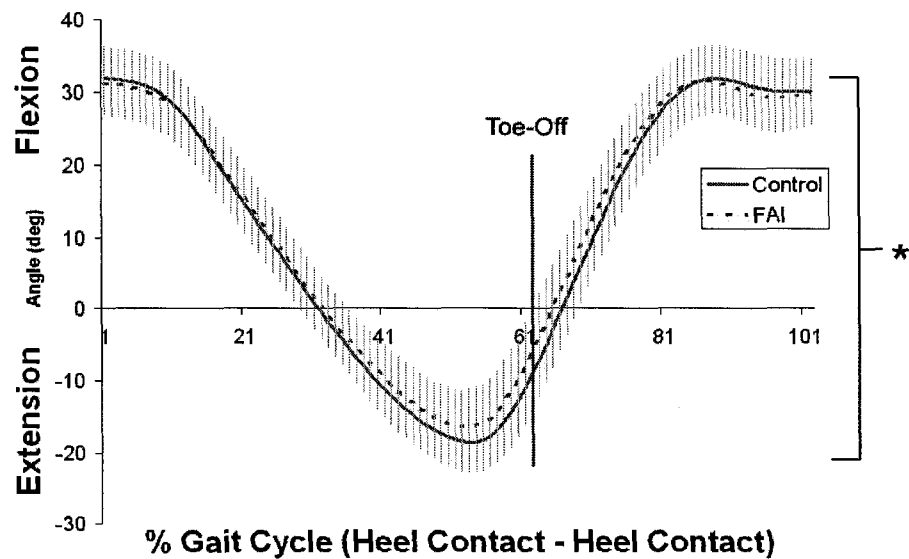


Figure B 4. Mean ($\pm$ standard deviation) of sagittal hip angles of the FAI and control groups during level gait. The FAI group had decreased total sagittal hip ROM ($p = 0.047$).

'*' = $p < 0.05$

'**' = $p < 0.01$

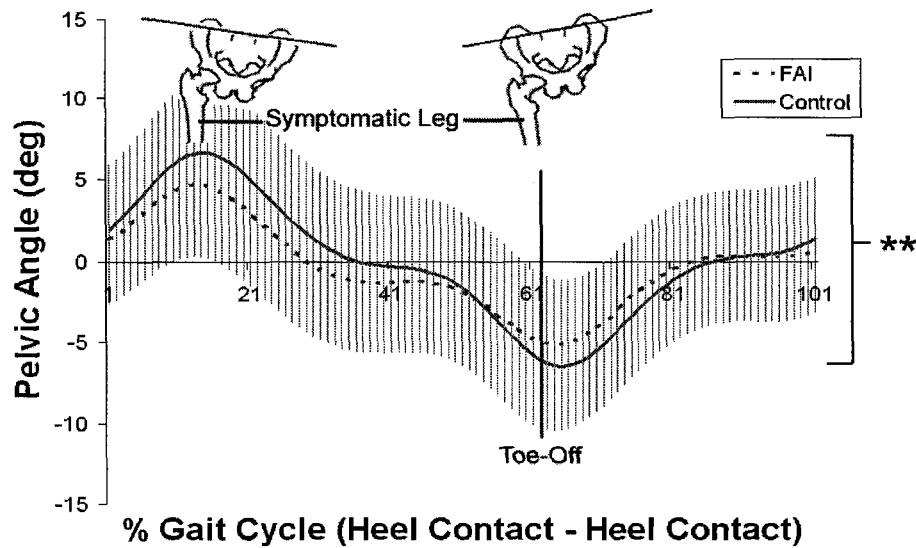


Figure B 5. Mean ($\pm$ standard deviation) of frontal pelvic angles of the FAI and control groups during level gait. The FAI group had decreased total frontal pelvic ROM ($p = 0.004$).

‘*’ = $p < 0.05$

‘**’ = $p < 0.01$

Note: The symptomatic leg notified in the diagrams is the leg landing on the force plate (0-62% gait cycle), and lifting off into swing phase (62-100% gait cycle).

There were no differences between any peak kinematic or kinetic variables in sit-to-stand or stand-to-sit. However, the FAI group had significantly higher variability in peak frontal kinematics for both sit-to-stand ($p < 0.001$) (Figure B6-a), and stand-to-sit ($p = 0.012$) (Figure B7-a) than the matched control. The FAI group also had higher variability in the moment of force generated at the hip during sit-to-stand ($p = 0.020$) (Figure 6-b), and stand-to-sit ($p = 0.004$) (Figure 7-b) than the control group.

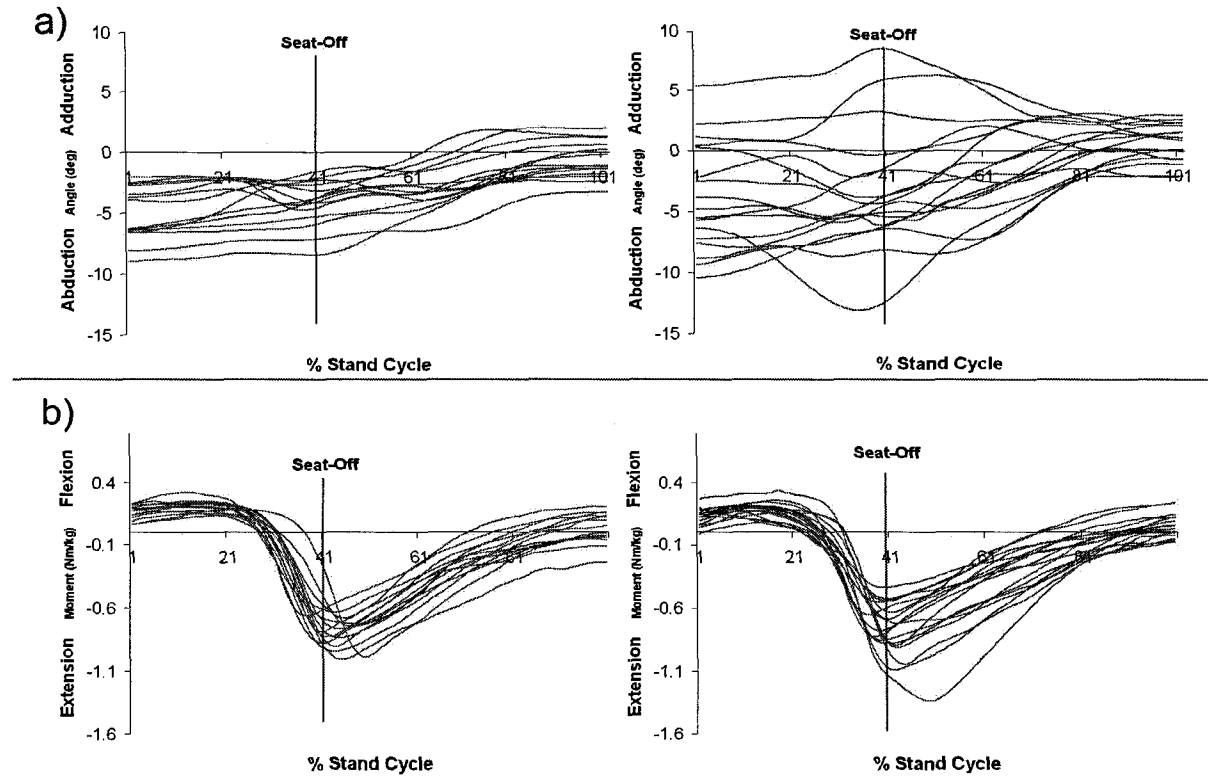


Figure B 6. Variability in stand strategy.

a) Control (left) and FAI (right) frontal angles of the hip during sit-to-stand. FAI individuals had significantly higher peak frontal angle variability ($p < 0.001$) within $\pm 20\%$ seat-off.

b) Control (left) and FAI (right) extension moments of the hip during sit-to-stand. FAI individuals had significantly higher variability in extension moment ($p < 0.020$) within 20% stand cycle after seat-off.

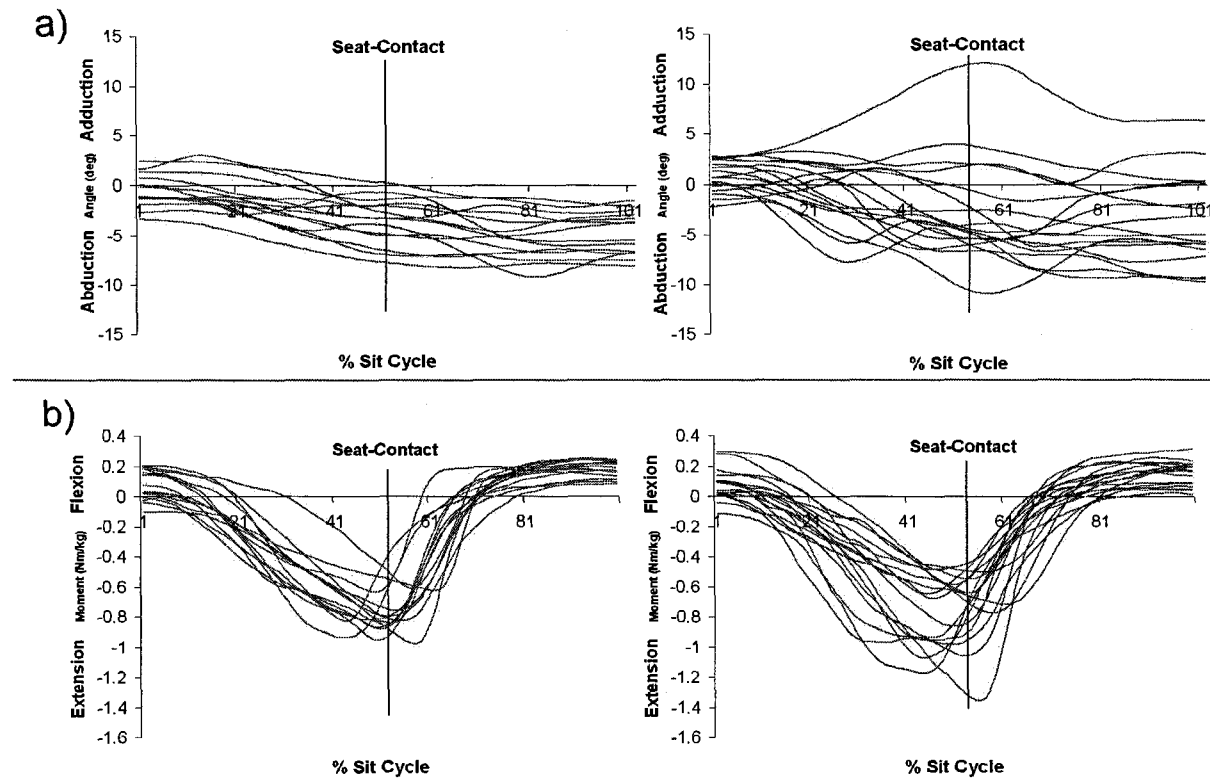


Figure B 7. Variability in sit strategy.

a) Control (left) and FAI (right) frontal angles of the hip during sit-to-stand. FAI individuals had significantly higher peak frontal angle variability ($p < 0.012$) within $\pm 20\%$ seat-contact.

b) Control (left) and FAI (right) extension moments of the hip during sit-to-stand. FAI individuals had significantly higher variability in extension moment ($p < 0.004$) within 20% stand cycle before seat-contact.

DISCUSSION

Walking

Multiple studies have been undertaken to define the differences in the biomechanics of gait on a level surface between healthy populations and pathological populations.^{26, 36, 52, 53} Level gait is the most common and repetitive voluntary movement humans perform²⁷ and is essential to independent living. Furthermore, it is a dynamic and complicated movement requiring the combined coordination of many muscles acting across multiple joints. Because of its key role in independent living, it is important to understand how gait is affected by cam FAI. Moreover, due to the complex nature of this movement, insight into the modality and

pathomechanism of FAI can be gained by noting differences in the affected and adjacent joint(s).

Our original hypotheses concerning the hip kinematics and kinetics were that there would be no significant differences in any variables between the cam FAI and control group. It was expected that due to the relatively low hip mobility required for level gait²³⁻²⁷ compared to the maximal passive ROM in participants with FAI²², that there would be no differences in hip biomechanics during level gait. Our hypotheses were validated for hip kinetics, but not for kinematics. We found no significant differences between the control and cam FAI groups for the mean peak 3-D kinetics generated at the hip during walking. However, contrary to our hypotheses, we found that the cam FAI group had a decrease in mean peak hip abduction, total frontal ROM, and total sagittal ROM during level gait than the control group. We did not form hypotheses on the affect of cam FAI on pelvic kinematics during level gait but found that participants with cam FAI had a significantly attenuated frontal pelvic ROM as compared to the healthy controls.

The symptomatic hip in the FAI group had a decreased peak hip abduction ($p = 0.009$) occurring just after toe-off, but the hip continued in increased adduction throughout swing phase and into the beginning of stance as compared to the control group (Figure3). This decrease in peak hip abduction was the primary contribution to the decreased total frontal ROM of the hip during gait ($p = 0.003$). Attenuated abduction was consistent with the common characterization of gait in patients with SCFE as mentioned by Rab,¹⁷ but there were no differences in external rotation which was also mentioned as characteristic gait in patients with SCFE. It has been well documented that individuals with FAI have limited maximal hip abduction compared to controls.^{9, 54, 55} However, the mean peak passive abduction angles in the literature range from 25-52°,^{9, 22, 33, 54, 55} compared to the mean

abduction angle of 3° in walking from our results. Since the limited abduction angle achieved during gait is so much lower than the recorded peak passive abduction angle in the literature, it is unlikely that it is caused by restricted mobility. It appears that decreased hip abduction during gait is unrelated to restricted mobility, although determining the cause of reduced hip frontal ROM during gait is beyond the scope of this study.

The FAI groups also had a decrease in sagittal ROM during gait ($p = 0.047$) which resulted primarily from hip extension, with very little difference in hip flexion. Other than greatly exaggerated reports of hip extension from computer simulated ROM studies,^{54, 55} no report of maximal hip extension in cam impingement could be found. Therefore, it can not be determined if the decrease in hip extension during gait is a result of limited maximal hip extension or an unrelated compensatory strategy.

The decrease in pelvic frontal ROM during gait ($p = 0.004$) is also difficult to interpret. Since there is no information on the peak pelvic mobility in participants with FAI, the decrease in frontal pelvic ROM during gait may be a result of restricted maximal pelvic mobility at the sacro-lumbar joint. However, this is just speculation, and the decrease in frontal pelvic ROM during gait could be unrelated to reduced maximal pelvic mobility. Future research should address the effect of cam FAI on peak pelvic mobility.

Sit-to-Stand and Stand-to-Sit

The only hypotheses which were made for sit-to-stand and stand-to-sit were that there would be no statistical differences between the FAI and control groups for peak sagittal angular displacement within the $\pm 20\%$ of seat-off or seat-contact, and that there would be no corresponding sagittal kinetic differences. These hypotheses were supported by our results. For the remaining frontal and transverse peak hip angles, moments of force, and mechanical powers, and for peak 3-D pelvic angles, there were also no significant differences between

the two groups. However, the FAI group had a significantly higher level of variability than the control group for peak frontal hip angles ($p < 0.001$) and peak sagittal moments of force generated at the hip ($p = 0.020$) for sit-to-stand and for stand to sit ($p = 0.012$ and $p = 0.004$ respectively).

Cam FAI had the same affect on both sit-to-stand and stand-to-sit manoeuvres. The sit-to-stand frontal kinematics and sagittal kinetics were basically reciprocals of the stand-to-sit results (Figure B7a,b)). For the sit-to-stand trials the control group tended to have relatively uniform frontal hip kinematics starting with the hips in a mild abduction of approximately 5° , and moving towards a neutral frontal position once fully standing. For stand-to-sit, the control group also had uniform frontal hip kinematics, with the hips starting in mild adduction, and moving to mild abduction. The FAI group during sit-to-stand started with more variability than the control group while seated which peaked as they approached seat-off, and then the variability dropped to being quite similar to the control group once in full stance (Figure B6a). The reverse pattern of this occurred in the stand-to-sit manoeuvre (Figure B7a). The frontal hip angle results from our control group indicate a small frontal hip ROM during 'normal' standing strategy. Since these 'normal' values are far smaller than the recorded maximal passive hip ROM in the FAI group,²² it can be assumed that the increased variability in the stand strategy of the FAI group is not caused by limited ROM. Surprisingly, some FAI participants altered sitting and standing strategy brought the symptomatic hip into adduction while the hip was flexed. This brings the ridge on the femoral head-neck junction towards the acetabular labrum,^{2, 16, 17, 22, 56} which decreases the amount of adduction possible before femoroacetabular abutment. Furthermore, the maximal adduction attained by these few FAI participants occurred at the same time as maximal hip flexion which seems counterintuitive. These results were unexpected, and it is difficult to postulate potential

explanations. However, since both groups were closely matched, the fact that there was increased frontal plane variability in people with an abnormal hip morphology suggests that it is somehow connected to FAI. There was also a large level of variability in the peak normalized extension moment of force generated at the hip in the FAI group compared to the control group during sitting and standing (Figure B6)b) and B7)b)). It is beyond the scope of this study to explain why the FAI group had a higher level of variability in frontal hip kinematics and in hip extension moments during the sit-to-stand manoeuvre, but it is likely that these varying techniques were a result of compensatory strategies adapted based on the cam FAI pathology. These results are very difficult to explain given the current database on FAI, which highlights the need for more research on how cam FAI affects movement.

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The Effect of Cam Femoroacetabular Impingement on Hip and Pelvic Kinematics during Maximum Squat

Running title: 3D Motion Analysis of FAI Patients

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Each author certifies that his or her institution has approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

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Abstract

Femoroacetabular impingement (FAI) causes abnormal contact at the anterosuperior aspect of the acetabulum in activities requiring a large hip range of motion (ROM). While deep squatting is not itself a common daily activity, it is a component of frequent activities, such as tying shoes and proper lifting technique and requires a large hip and pelvic ROM. We therefore addressed the following questions in this study: (1) Does FAI affect the kinematics of the hip and pelvis during a maximal depth squat? And (2) does FAI decrease maximal normalized squat depth? We measured the effect of cam FAI on the three-dimensional kinematics of the hip and pelvis during a maximal depth squat as compared with a healthy control group. Fifteen participants diagnosed with cam FAI and 11 matched control participants performed unloaded squats while three-dimensional motion analysis was collected. Patients with FAI had no differences in hip kinematics during squatting but had decreased sagittal pelvic range of motion compared to the control group ($14.7 \pm 8.4^\circ$ vs $24.2 \pm 6.8^\circ$, respectively). The FAI group also could not squat as low as the control group ($41.5 \pm 12.5\%$ vs $32.3 \pm 6.8\%$ of leg length, respectively), indicating the maximal depth squat may be useful as a diagnostic exercise. Limited pelvic ROM in FAI patients reduces their capacity to perform motions requiring large amplitudes at the hip joint such as maximum squat. Restricted pelvic sagittal ROM could represent a key factor in the pathomechanics of FAI.

Level of Evidence: Level III, therapeutic study. See the Guidelines for Authors for a complete description of levels of evidence.

INTRODUCTION

Femoroacetabular impingement (FAI) is an increasingly recognized cause of hip pain in young active adults^{1, 40} and is believed to be a cause of hip osteoarthritis (OA).^{10, 12, 21, 25, 38, 39} In fact, according to a study by Tanzer et al.³⁸ all 125 hips with idiopathic OA had a pistol-grip deformity, which is indicative of cam FAI. There are two types of FAI, both of which are defined by an anatomic abnormality of the acetabulum and/or the proximal femur that cause abnormal contact at the hip, primarily at the anterosuperior acetabular rim. Pincer impingement is related to overcoverage of the acetabulum, such as with acetabular retroversion and coxa profunda.¹⁸ Cam impingement is caused by decreased concavity of the femoral head-neck junction, most commonly in the anterosuperior region.^{2, 14, 22, 26} Decreased femoral head-neck concavity causes a forceful inclusion of the abnormal femoral head-neck junction into the peripheral acetabulum.¹⁰ The resulting shear forces can produce abrasion of the acetabular articular cartilage combined with a potential tearing of the articular cartilage from the acetabular labrum in the anterosuperior rim of the acetabulum.¹⁰ This damage occurs at the limits of hip range of motion (ROM) during assisted flexion, internal rotation, and adduction.^{15, 24} Many common daily activities, such as prolonged sitting, squatting, stair climbing, and athletic activities requiring a large ROM, produce hip pain in people with FAI.^{4, 10, 21, 24, 40} This pain is thought to occur after the onset of cartilage damage caused by “outside-in” damage of the acetabular labral-chondral junction.²

Cam impingement is most common in relatively young athletes,^{10, 11, 14, 28} who perform activities requiring extreme ROM of the hip that exacerbates the condition. The maximal depth squat is a controlled movement requiring a large ROM of the hip and pelvis, approaching the maximal hip ROM in people with FAI.^{9, 13, 20, 32, 34, 37, 41, 42} This makes it

likely maximal depth squats could be affected by the FAI condition. Furthermore, being a demanding movement, maximal depth squatting acts as an appropriate test for lower limb functionality, which could be useful for diagnosis.^{9, 13} Finally, although deep squatting is not itself a common daily activity, it is a component of frequent activities, such as tying shoes and proper lifting technique, both of which are relevant for the young affected population.

This study addressed the following questions: (1) Does FAI affect the three-dimensional (3D) kinematics of the hip and pelvis during a maximal depth squat? And (2) does FAI decrease maximal normalized squat depth?

MATERIALS AND METHODS

We recruited 32 volunteers for this study: 16 diagnosed with cam FAI and 16 control participants matched by age, gender, and body mass index. We analyzed patients only with cam FAI to prevent there being multiple independent variables. The sample size was determined from a power analysis using pilot data from the first 10 volunteers (five FAI and five control participants) to determine the effect size and with squat depth as the key dependent variable. A total sample size of 22 was calculated using G*POWER 3⁸ with an alpha value set at 0.05 and minimal power set at 80%. The calculated sample size was the minimum required number of participants to achieve desired power based on our pilot data of squat depth, but since we were looking at other variables as well, a larger sample size was chosen to maximize the power of the study. The cam impingement participants were recruited after having a positive impingement test and visible cam morphology on anteroposterior (AP) and Dunn view radiographs.^{7, 21, 27, 35} The alpha angle of Notzli et al.³⁰ was measured on each hip, with a value greater than 50.5° diagnostic of cam-type FAI.

Matched control participants were recruited from the Ottawa region using online advertising and through interaction with various community groups. They had no history of serious lower limb injury or surgery and had spherical femoral heads, which were assessed by an AP radiograph.²⁷ Participants from both groups were excluded if hip osteoarthritis was visible on the radiographs or if they had substantial hip joint space narrowing. All FAI patients had only one symptomatic hip. We excluded five of the 16 recruited control participants: three had at least one aspherical femoral head based on an AP radiograph, and the other two could not have the required screening radiographs. One of the patients with FAI was excluded due to the presence of hip osteoarthritis. This left 11 control participants and 15 FAI participants (Table C1). All participants filled out a WOMAC³ questionnaire. Before participating in the study, which was approved by the Ottawa Hospital Research Ethics Board and the University of Ottawa Health Sciences and Science Research Ethics Board, all participants signed an informed written consent.

Table C 1. Participant characteristics ($\pm$ standard deviation).

Condition	Number of Participants	Age (years)	BMI (kg/m ²)	WOMAC	Flexibility
Control	11 (6 males)	34.5 $\pm$ 10.1	23.0 $\pm$ 2.2	99.9 $\pm$ 0.3	29.8 $\pm$ 6.7
Cam FAI	15 (9 males)	35.3 $\pm$ 9.1	23.4 $\pm$ 2.2	75.5 $\pm$ 15.7	21.9 $\pm$ 6.4

The 3D kinematics of squatting was collected using seven Vicon[®] MX-13 cameras (Vicon, Los Angeles, CA) at 200 Hz with retroreflective markers placed on anatomic landmarks according to a modified Helen-Hayes marker set¹⁶ with additional greater trochanter, medial knee, and medial ankle markers to enhance joint center determination accuracy and upper body markers for display purposes only (Figure C1). A height-adjustable bench was used to control squat depth acting as a maximal depth indicator.

For the squat, the participant stood with their feet 10 cm directly in front of the height-adjustable bench. Bench height was lowered to $\frac{1}{3}$ of the participant's tibial height, acting as both a maximal depth indicator and a safety mechanism to prevent falling backwards (Figure C1). Participants were asked to stand with feet shoulder width apart, parallel to each other, and facing anteriorly. Starting from standing, participants were asked to extend both arms anteriorly,¹³ squat down to a maximal depth, and return to a standing position at a self-selected and controlled pace,³¹ maintaining heel contact throughout the movement. Extending both arms anteriorly prevented the temptation to use them for support during the squat and helped with balance during the maneuver. If participants could squat down to the maximal depth, they were told to only touch their buttocks to the bench without transferring any weight and then to ascend back to a standing position. All participants performed five squat trials, and the hip and pelvic kinematics and peak squat depth were calculated for each.

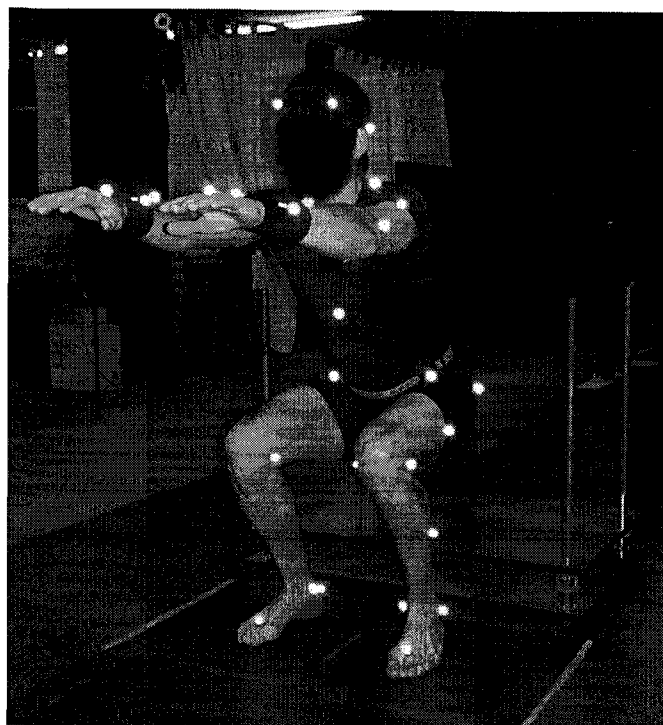


Figure C 1. Instrumented participant performing a maximal depth squat with feet positioned in front of the height-adjustable bench, which is lowered to $\frac{1}{3}$ tibial height.

Lower limb 3D kinematics were calculated according to the methods explained by Kadaba et al.^{16, 17} and Davis et al.⁵ using VICON® Workstation processing software. Hip joint angles were based on local Euler coordinate systems as defined by the aforementioned studies, and pelvic angles were measured relative to a global coordinate system. Joint and segment angles were zeroed based on each participant's neutral position as determined by a standing static trial where feet were parallel and facing anteriorly approximately shoulder width apart.

Squat kinematic variables consisted of the peak hip angle in each plane at maximal squat depth, the peak 3D pelvic angles during descent, ascent, and at peak depth, the total pelvic ROM in each plane, and the maximal attained squat depth. The 3D hip angles were only assessed at maximal squat depth because they tended to only have one peak, which occurred at peak squat depth. The 3D pelvic angular displacements however tended to have a peak during descent, peak squat depth, and again during ascent. Peak squat depth was defined as the lowest point the hip joint centers reached during the squat divided by the participant's total leg height, which was the averaged linear distance from the left and right medial malleoli to their respective anterior superior iliac spine. All five individual squat trials were averaged, and ensemble averaged for each group.

All dependent variables were tested for normality and had skewness and kurtosis values between ± 1.5 , indicating normally distributed data. Differences in maximal hip angular displacement in each plane at peak squat depth and in peak pelvic angles during descent, ascent, and peak squat depth, as well as total pelvic ROM in each plane between the control and cam FAI groups, were determined using one-way between-subjects analysis of variances ($\alpha = 0.05$). When we identified significant differences ($p < 0.05$) in the aforementioned

dependent variables between the two groups we also performed an analysis of covariance run with squat depth included as a covariate. This was to ensure the differences in joint kinematics resulted from the presence or absence of cam FAI, rather than from discrepancies in squat depth. All calculations were processed using SPSS 15.0 software (SPSS Inc, Chicago, IL).

RESULTS

The FAI group had a decreased ($p = 0.005$) sagittal pelvic ROM of $14.7 \pm 8.4^\circ$ (mean $\pm$ standard deviation) compared to $24.2 \pm 6.8^\circ$ in the control group (Figure C2). When squat depth was included as a covariate, mean sagittal pelvic ROM remained reduced ($p < 0.01$) in the FAI group compared to the control group. However, there were no differences in any of the 3D hip angles at maximal squat depth.

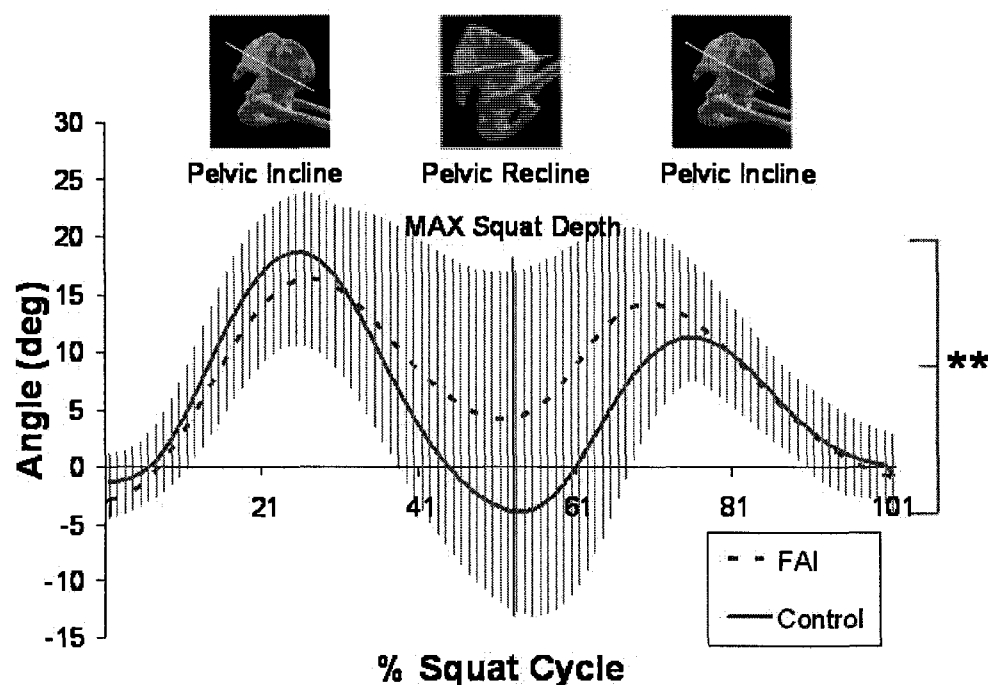


Figure C 2. Mean pelvic pitch of the control group (solid line) and FAI group (dotted line) during maximal depth squat with vertical lines representing standard deviation.

The control group also squatted to a lower ($p = 0.037$) mean maximal squat depth of $32.3 \pm 6.8\%$ of leg length, compared to the $41.5 \pm 12.5\%$ attained by the FAI group. Only five participants from the FAI group (33%) reached the lowest attainable squat depth compared to 10 controls (91%), as indicated by buttocks contact with the height-adjustable bench.

DISCUSSION

This study evaluated squat motion since it is an important component of daily activities and a valuable indicator of lower limb mobility.^{9, 13} As mentioned previously, the maximal depth squat is a relatively demanding activity that requires a high level of hip mobility,^{9, 13} and our results suggest it also requires a large sagittal ROM of the pelvis.

There are some limitations inherent to joint kinematic studies resulting from generic calculations, marker misplacements,⁶ joint centre determination and skin or clothing artefacts.^{23, 33} However, precautions were used to minimize the potential errors caused by these limitations. All marker placements were performed by the same investigator (MK) which decreased inter-participant variability. The primary effect of misplaced markers is an incorrect joint offset, which was corrected by zeroing all joint angles based on a neutral static trial. Finally, participants wore skin-tight clothing to minimize trunk and pelvic clothing artifacts, and the slow controlled nature of the squatting movement minimized skin artifacts.²³

A further limitation was posed by only analyzing the squat maneuver. This may reduce the extent to which our results may be generalized. However, this activity was chosen because it is an important component of various common daily activities, and because of its potential in helping to diagnose FAI. Squatting requires a large sagittal hip and pelvic ROM that most

control participants, but few FAI patients can fully achieve making it a useful diagnostic exercise. Finally, any limitations in hip or pelvic mobility that prevent the accomplishment of maximal squat depth could be indicative of a general restriction in joint mobility.

Surprisingly, we observed no differences in any of the hip kinematic variables between the impingement group and the control group. However, we observed more than one squat strategy in both groups. The majority of both groups used one modality, but five impingement participants and two control participants used different techniques. The main differences occurred in the frontal plane. The dominant strategy consisted of hip abduction throughout the squat maneuver, whereas the less dominant strategy consisted primarily of hip adduction throughout the maximal depth squat. Grouping these varying squat strategies increased variability and may explain the lack of differences between the two groups.

Perhaps the most important finding was the difference in the total sagittal pelvic ROM during the maximal depth squat between the control and FAI groups. Not only was the pelvic ROM lower in the FAI group, it was also independent of squat depth. As previously mentioned, all kinematic data were zeroed based on a standing trial, so the values are relative and give no insight on neutral pelvic orientation. The control group had much more sagittal pelvic ROM than the FAI group, with an increased incline during descent and ascent and increased recline at peak squat depth. The largest difference occurred in recline at peak depth (Figure C2). The more the pelvis is reclined, the less acetabular retroversion occurs, which is associated with anterior FAI.^{29, 34-36} In fact, it has been suggested pelvic retroversion may be caused by pelvic incline.³⁴ This indicates pelvic recline orients the acetabulum in such a way as to minimize contact between the acetabular rim and the anterosuperior femoral head-neck junction by decreasing anterior femoral head coverage.¹⁹ Sagittal pelvic mobility is integral in flexing the

torso to maintain balance and in allowing the large hip flexion angles essential for deep squats. Since the FAI group had decreased pelvic pitch in a maximal capacity maneuver requiring high levels of sagittal pelvic ROM, it is reasonable to assume cam FAI causes a general decrease in sagittal pelvic ROM. Reduced sagittal pitch may predispose the hip to premature contact between the proximal femur and the acetabular rim, especially in movements requiring high levels of hip flexion.

There was also a difference in maximal squat depth, with the control group squatting deeper than the FAI group. However, as mentioned previously, there were no differences in the kinematics of the hip between the two groups. Since pelvic orientation does not directly contribute to squat depth, it was not responsible for the discrepancy in squat depth. The decreased squat depth in the impingement group was likely attained with a small combined contribution of the hip, knee, and ankle. Explaining the exact mechanism for the discrepancy in squat depth is outside the scope of this study, but decreased maximal squat depth in the cam FAI population could make maximal squat depth a useful exercise for diagnosing hip impingement.

Reduced sagittal pelvic ROM in patients diagnosed with cam FAI provides new insight regarding the multifactorial nature of FAI. It is well documented that cam FAI is a result of decreased concavity of the femoral head-neck junction,^{2, 14, 22, 26} which causes jamming of the proximal femur into the acetabulum.^{15, 24} There is further evidence that cam impingement is caused by decreased femoral anteversion,¹⁵ which reduces femoral neck clearance from the acetabular rim during hip flexion and internal rotation.^{15, 24} However, the idea that pelvic mobility may also be partially responsible for the abnormal contact between the anterosuperior acetabulum and the proximal femur is novel. Decreased sagittal pelvic

mobility is an important manifestation of cam FAI, which highlights the multifactorial nature of this condition. Furthermore, it may represent a key feature in the pathomechanics of FAI and is an area of ongoing research.

One unresolved issue in FAI is the explanation of what causes the anatomic abnormality of cam FAI to be symptomatic in some but asymptomatic in others with the same hip morphology. Approximately 8% to 12% of hips with the anatomic variation indicative of cam FAI are asymptomatic.^{11, 28, 41} The prevalence of asymptomatic FAI is further supported by the fact that three of our healthy controls were excluded due to the presence of aspherical femoral heads consistent with cam-type impingement but had no hip pain or physical limitations. Since decreased pelvic mobility may be a contributing factor of symptomatic cam FAI but is not assessed in current diagnosis, it could provide pathologic insight into the cause of symptomatic cam FAI. However, since this study provides no insight on the sagittal pelvic ROM of asymptomatic patients with decreased femoral head-neck concavity, no conclusions can be made. Future research should compare the pelvic mobility of symptomatic and asymptomatic patients with FAI anatomic variations.

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General Discussion

There were a series of limitations which reduced the extent to which the results could be generalized to the cam FAI and control populations. These limitations are addressed, and the precautions implemented are covered in the three included articles.

The results of the effect of cam FAI on maximal dynamic ROM, walking, sit-to-stand and stand-to-sit and squatting are presented and discussed in the included articles called: *The Effect of Cam Femoroacetabular Impingement on Hip Kinematics during Maximal Dynamic Range of Motion*; *Hip Biomechanics of Cam Femoroacetabular Impingement During Daily Activities*; *The Effect of Cam FAI on Hip and Pelvic Kinematics during Maximum Squat*, respectively. Therefore only a brief summary of the key results and their implications are presented here.

Dynamic Range of Motion

It is well documented that cam FAI causes a decrease in passive ROM (Eijer et al., 2001; Ito et al., 2004; Philippon et al., 2007; Reynolds et al., 1999; Wyss et al., 2007; Zebala et al., 2007), and produces bony limitations within the normal range of motion (Kubiak-Langer et al., 2007; Tannast et al., 2007). Furthermore, it is not uncommon for patients to notice limited hip motion even before the onset of pain, (Wyss et al., 2007) indicating a loss of functional mobility. Although it should be noted that all the aforementioned studies assessed the maximal passive hip ROM with externally applied forces in a clinical setting. The precise level of self-generated dynamic hip ROM in participants with cam FAI has not been previously tested. Since the repetitive microtrauma in activities of daily living which exacerbate cam FAI (Ito et al., 2004; Leunig et al., 2005) and lead to hip OA (Ganz et al.,

2003; Leunig et al., 2003; Tanzer & Noiseux, 2004) occurs within the functional range of hip mobility, it is important to measure the extent to which cam FAI limits dynamic self-generated hip ROM. This will allow for a deeper understanding of cam FAI, and is necessary for predicting which movements will exacerbate the condition in developing conservative treatments.

The symptomatic leg of the cam FAI group having a significantly lower peak hip abduction and internal rotation with the hip flexed to 90° than the matched control group which supported our hypotheses, and coincided with the results from passive hip ROM in the literature (Ito et al., 2004; Kubiak-Langer et al., 2007; Notzli et al., 2002; Philippon et al., 2007; Tannast et al., 2007; Wyss et al., 2007). These mobility restrictions in the FAI group likely occur from abutment between the anterosuperior acetabular rim, and the abnormal femoral head-neck junction.

It was also postulated that the FAI group would have a lower peak hip flexion than the control group as indicated by passive hip ROM in the literature (Ito et al., 2004; Kubiak-Langer et al., 2007; Tannast et al., 2007; Wyss et al., 2007). However, this was not supported by our results. This discrepancy between passive assisted and dynamic self-generated hip mobility is likely due to the inability to achieve true peak hip flexion without external assistance (Blandine Calais-Germain, 1993). Thus, it is likely that self-generated hip movement cannot produce enough flexion to elicit bony limitation in FAI participants without additional externally applied forces.

It was hypothesized that there would be no differences in external hip rotation or total transverse hip ROM with the hip flexed at 90° based on conflicting results in the passive ROM literature. However, the studies which predicted no difference in hip flexed external rotation were based on computer simulations which neglected all soft tissues, which greatly

exaggerated their results, and discredited these values for live hip mobility (Kubiak-Langer et al., 2007; Tannast et al., 2007). Thus there was only support in the passive hip ROM literature for attenuated hip external rotation in the FAI group (Ito et al., 2004). The support of both reduced internal and external flexed hip rotation by passive hip ROM studies, also indirectly supports our findings of a decreased total hip transverse ROM (Ito et al., 2004; Kubiak-Langer et al., 2007; Notzli et al., 2002; Tannast et al., 2007; Wyss et al., 2007).

Another unexpected finding was that despite the lack of significant difference in peak hip flexion between the two groups, the cam FAI group had attenuated total sagittal hip ROM. This finding was primarily a result of decreased hip extension, which was not significant, but was a strong trend ($p = 0.054$). Decreased hip extension in the FAI group was not found in any passive hip ROM literature.

Reduced hip flexion, flexed internal rotation, and abduction in the cam FAI group all coincide well with the proposed pathomechanism of the condition. All these movements bring the abnormal anterosuperior femoral head-neck bulge indicative of cam FAI into close proximity with the anterosuperior ridge of the acetabulum (B Calais-Germain, 1993; Ganz et al., 2003; Ito et al., 2004; Kubiak-Langer et al., 2007; Philippon et al., 2007; Tannast et al., 2007), resulting in a jamming of the bony prominence into the acetabular rim (Ito et al., 2001; Leunig et al., 2005). However, hip flexed external rotation and extension rotate the anterosuperior femoral head-neck junction away from the acetabular rim (Blandine Calais-Germain, 1993; Rab, 1999). One important anatomical function of the iliofemoral ligament is to restrict hip extension (Bucholz & Wheelless, 1982; B Calais-Germain, 1993), and according to a study assessing the location of herniation pits on the femoral neck in hip surgery patients the iliofemoral ligament and the iliopsoas muscle tendon both restrict external rotation as well (Bucholz & Wheelless, 1982; Blandine Calais-Germain, 1993;

Daenen et al., 1997). These soft tissues are the primary restraints of external rotation and extension of the hip rather than bony contact. Thus decreased dynamic flexed external rotation and sagittal ROM of the hip in participants with cam FAI compared to healthy controls is likely a result of abnormal soft tissue restriction. The effect of soft tissue on hip mobility in cam FAI should be investigated in future research.

Walking

Level gait is the most common and repetitive voluntary movement humans perform (Winter, 1983) and is essential to independent living, making it important to determine if FAI affects its performance. However, the affect of FAI on the biomechanics of level gait has not been quantitatively measured before. Walking is a complicated manoeuvre which involves the combined coordination of multiple muscles acting across numerous joints. Thus, assessment of how cam FAI affects the kinematics and kinetics of the hip and pelvis during walking can provide insight into the pathomechanism of this condition.

It was hypothesized that there would be no differences in level gait between the two groups for all biomechanical hip variables tested. These hypotheses were supported for all kinetic variables, but there were differences in peak hip abduction, total frontal ROM, and total sagittal ROM during level gait. No hypotheses were generated for pelvic kinematics during gait, but it was found that the FAI group had attenuated frontal pelvic ROM compared to the control group.

Participants diagnosed with cam FAI had a decreased peak hip abduction occurring just after toe-off compared to the control group, and continued in increased adduction throughout the swing phase. This was consistent with common gait characteristics of patients with slipped capital femoral epiphysis (Rab, 1999) which results in a similar

abnormal morphology, and is a proposed aetiology of cam FAI (Goodman et al., 1997; Kassarian et al., 2007; Siebenrock et al., 2004). The attenuated hip abduction during gait was the main contributor to the decreased frontal ROM in the FAI group compared to the control group. Cam FAI also caused an attenuated sagittal hip ROM during gait relative to the control values which was primarily a result of reduced hip extension. The frontal kinematic hip values were well within the previously reported maximal passive hip mobility values of FAI participants (Philippon et al., 2007), and were also well within the dynamic frontal ROM values from this study, indicating that the attenuation of hip abduction during level walking was not a result of bony limitations. It is difficult to interpret the cause of reduced abduction during the swing phase of gait in participants with FAI, and further research is needed to come to any conclusive answer. However, the peak hip extension angles produced during walking in the FAI group came very close to the peak dynamic hip extension values from our study. Thus the attenuated sagittal hip ROM in participants with cam FAI during walking (which was primarily a result of reduced hip extension) is likely due to the same abnormal soft tissue restriction which limited maximal dynamic ROM in the sagittal plane.

The decreased frontal pelvic ROM during gait in the cam FAI group as compared to the control group is also difficult to interpret. Since peak pelvic mobility in FAI participants is not reported in the literature, this limited frontal pelvic ROM could be a result of restricted pelvic mobility, however this is beyond the scope of this study. Future research should look into the causes of these altered gait kinematics in participants with cam FAI.

Sit-to-Stand and Stand-to-Sit

We postulated that there would be no differences in sagittal hip kinematics or kinetics, but made no further postulations for frontal and transverse hip kinematics or kinetics, or 3-D pelvic angles for the sit-to-stand and stand-to-sit manoeuvres between the FAI and control groups. Our results supported the hypothesis that there would be no differences in sagittal hip kinematics or kinetics. Furthermore, there were no differences in mean peak values for any hip or pelvic kinematic variables between the two groups.

However, after seeing the large level of variability in hip kinematics and kinetics during sitting and standing in the FAI group, compared to the relatively uniform trends in the control group, F-tests were run to compare the standard deviations of the two groups. The FAI group had significantly higher variability in frontal kinematics and sagittal kinetics for both sitting and standing manoeuvres compared to the control group. This high level of variability in the FAI group suggests a variety of compensatory strategies in performing these activities either to avoid pain, restricted mobility or other factors.

Squat

The maximal depth squat is an important component of daily activities and a valuable indicator of lower limb proficiency (Flanagan et al., 2003; Isear et al., 1997). It is a demanding activity which requires a high level of hip (Flanagan et al., 2003; Isear et al., 1997) and pelvic mobility. Furthermore, it is a maximal capacity manoeuvre, and very few FAI participants could achieve the maximal attainable squat depth. This makes it likely that any limitations in hip or pelvic mobility which restricted squat depth are a result of a general reduction of maximal hip or pelvic ROM.

In contrast to our hypotheses, there were no differences in sagittal hip kinematics or kinetics between the FAI and control groups for the maximal depth squat. In fact, there were no differences in any 3-D hip kinematic or kinetic variables between the two groups. This was likely due to the fact that there were two different squat strategies used in both the control and FAI groups. The majority of both groups used one modality, but five impingement participants and two control participants used different techniques. The main differences occurred in the frontal plane. The dominant strategy consisted of hip abduction with a corresponding adduction moment throughout the squat maneuver, whereas the less dominant strategy consisted primarily of hip adduction with a corresponding abduction moment throughout the maximal-depth squat. Grouping these varying squat strategies greatly increased variability, and may explain the lack of statistical differences between the two groups. Furthermore, stratifying the control and FAI groups into the two different squat strategies was beyond the scope of this study, as it would reduce the sample size too much—especially in the non-dominant strategy. This should be assessed in future research.

In accordance with our hypotheses, the control group did squat to a lower maximal squat depth than the FAI group. The decreased squat depth in the impingement group was likely attained with a small combined contribution of the hip, knee, and ankle. Decreased maximal squat depth in the cam FAI population could make maximal squat depth a useful exercise for diagnosing hip impingement.

A very interesting finding from the squat protocol was the decrease in sagittal pelvic ROM in the FAI group as compared to the control group independent of squat depth. The control group had an increased pelvic incline during descent and ascent, and increased pelvic recline at peak squat depth. The largest difference occurred in pelvic recline at peak depth

which results in acetabular retroversion and is associated with anterior FAI (Myers et al., 1999; Reynolds et al., 1999; Siebenrock, Kalbermatten et al., 2003; Siebenrock, Schoeniger et al., 2003). Pelvic recline orients the acetabulum in such a way to minimize contact between the acetabular rim and the anterosuperior femoral head-neck junction, by decreasing anterior femoral head coverage (Konishi & Mieno, 1993). FAI participants having reduced sagittal pelvic mobility may predispose the hip joint to premature contact between the proximal femur and the acetabular rim, especially in movements requiring high levels of hip flexion. The potential contribution of sagittal pelvic mobility to the abnormal contact between the proximal femur and the acetabular rim in cam FAI is a novel finding. Furthermore, it could be an important factor in the pathomechanics of FAI.

Summary

Unilateral cam FAI causes significantly decreased dynamic mobility of the symptomatic hip as compared to a healthy matched control group. Participants with FAI had decreased internal and external rotation as well as total transverse ROM of the hip while it was flexed at 90°; decreased hip abduction; and decreased total sagittal hip ROM as compared to the control group.

Cam FAI also affected hip and pelvic kinematics during level gait. The FAI group had decreased peak hip abduction and total frontal hip ROM; decreased sagittal hip ROM; and decreased frontal pelvic ROM during walking as compared to the control group.

Cam impingement also caused higher variability in sitting and standing hip kinematics and kinetics. The FAI group had higher frontal kinematic variability and sagittal kinetic variability in both sitting and standing compared to the control group.

Finally, FAI affected the pelvic kinematics and maximal attainable squat depth in a maximal depth squat. The cam impingement group could not squat as low as the matched control group, and had decreased sagittal pelvic ROM during the maximal depth squat.

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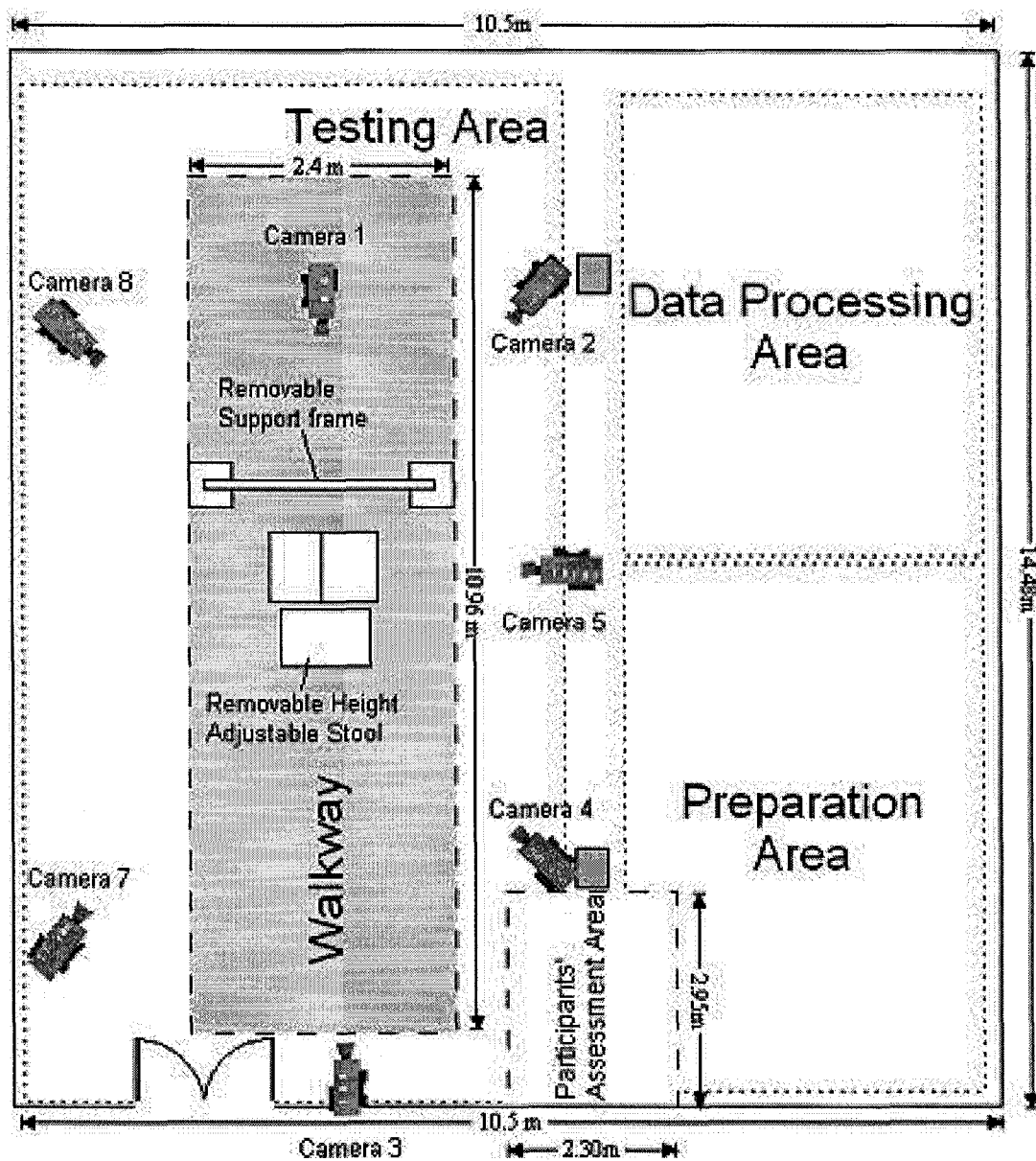
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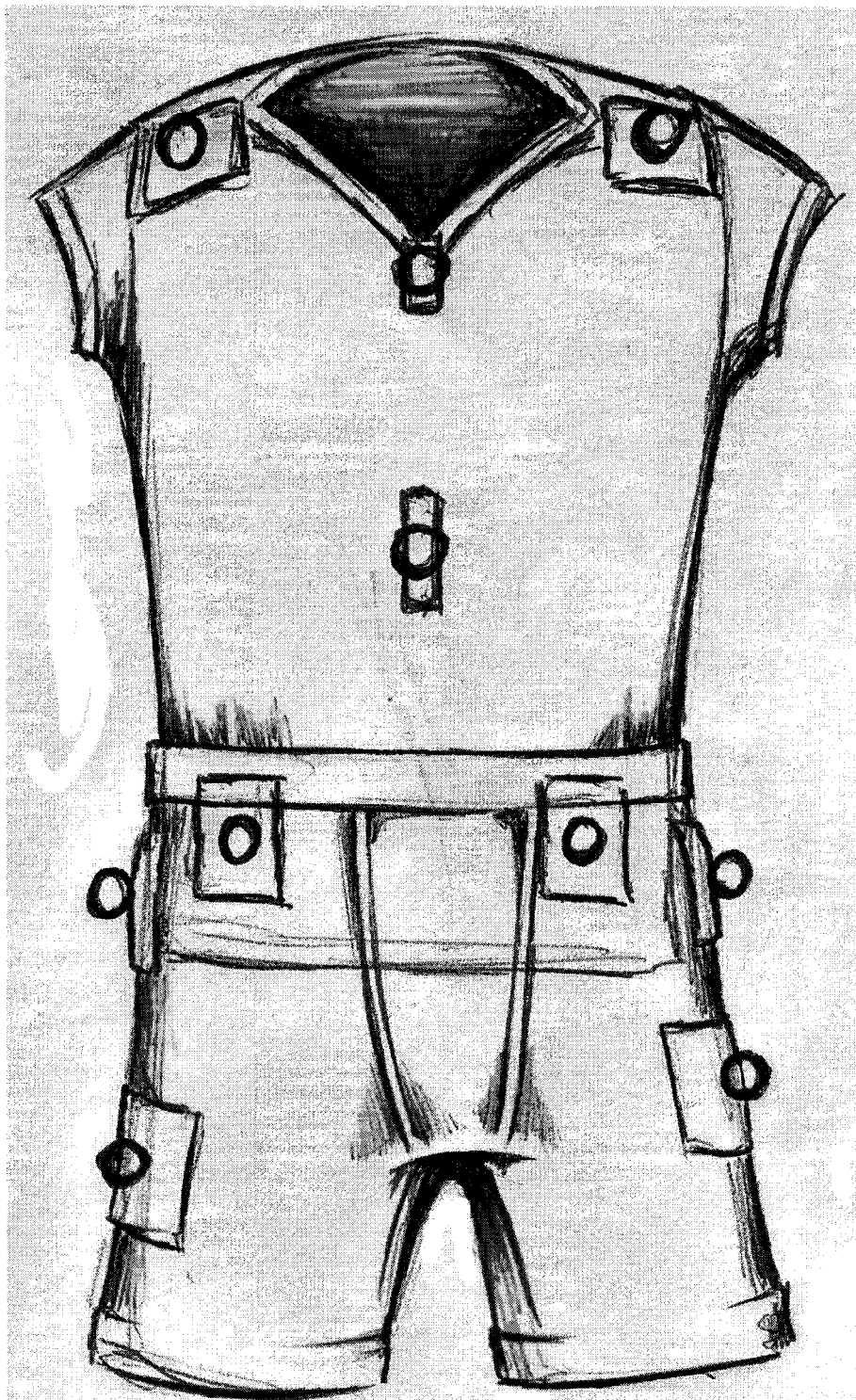
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Lab Set-up



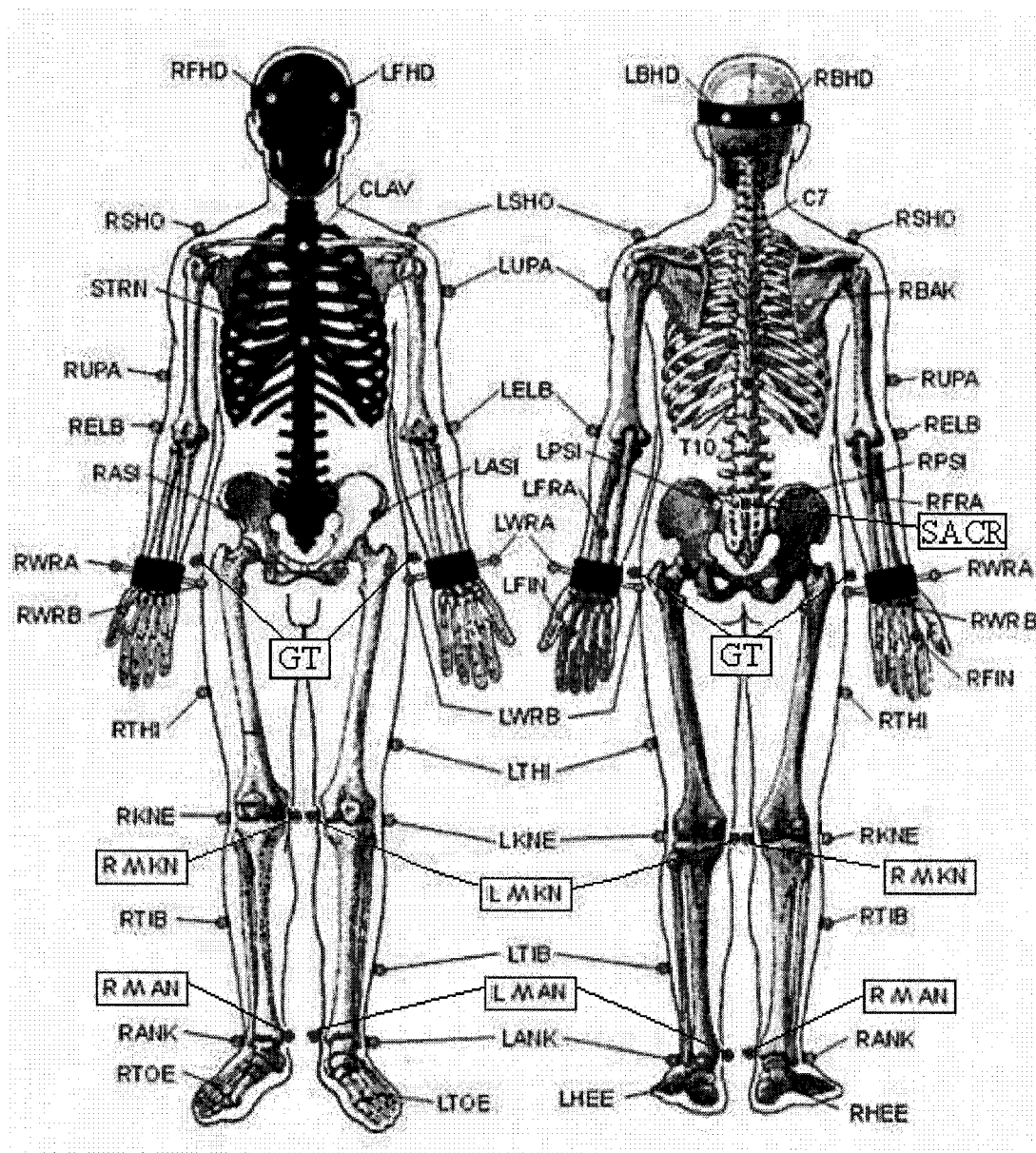
Appendix B

Marker-Suit



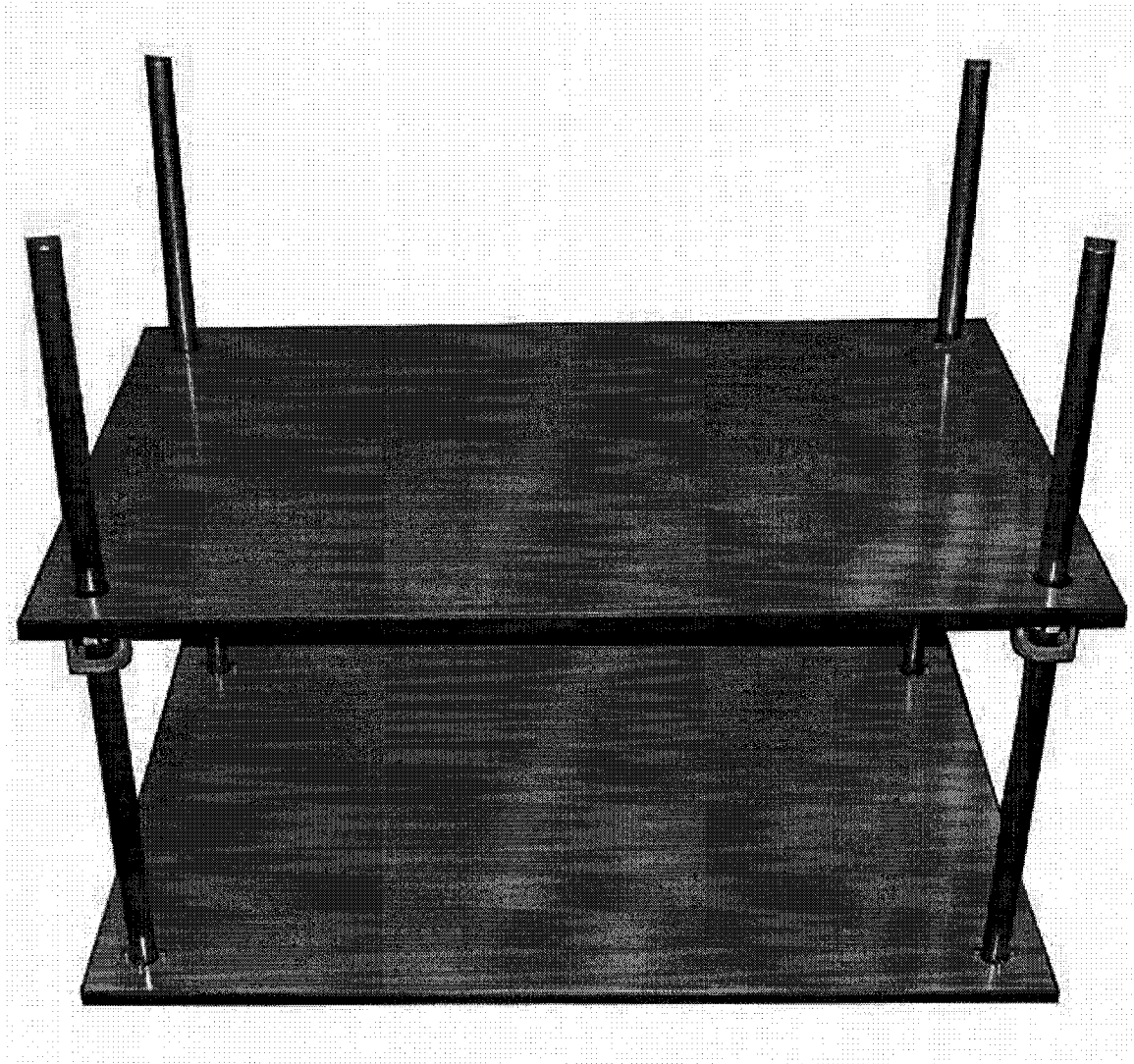
Appendix C

Modified Plug-in Gait Marker Set



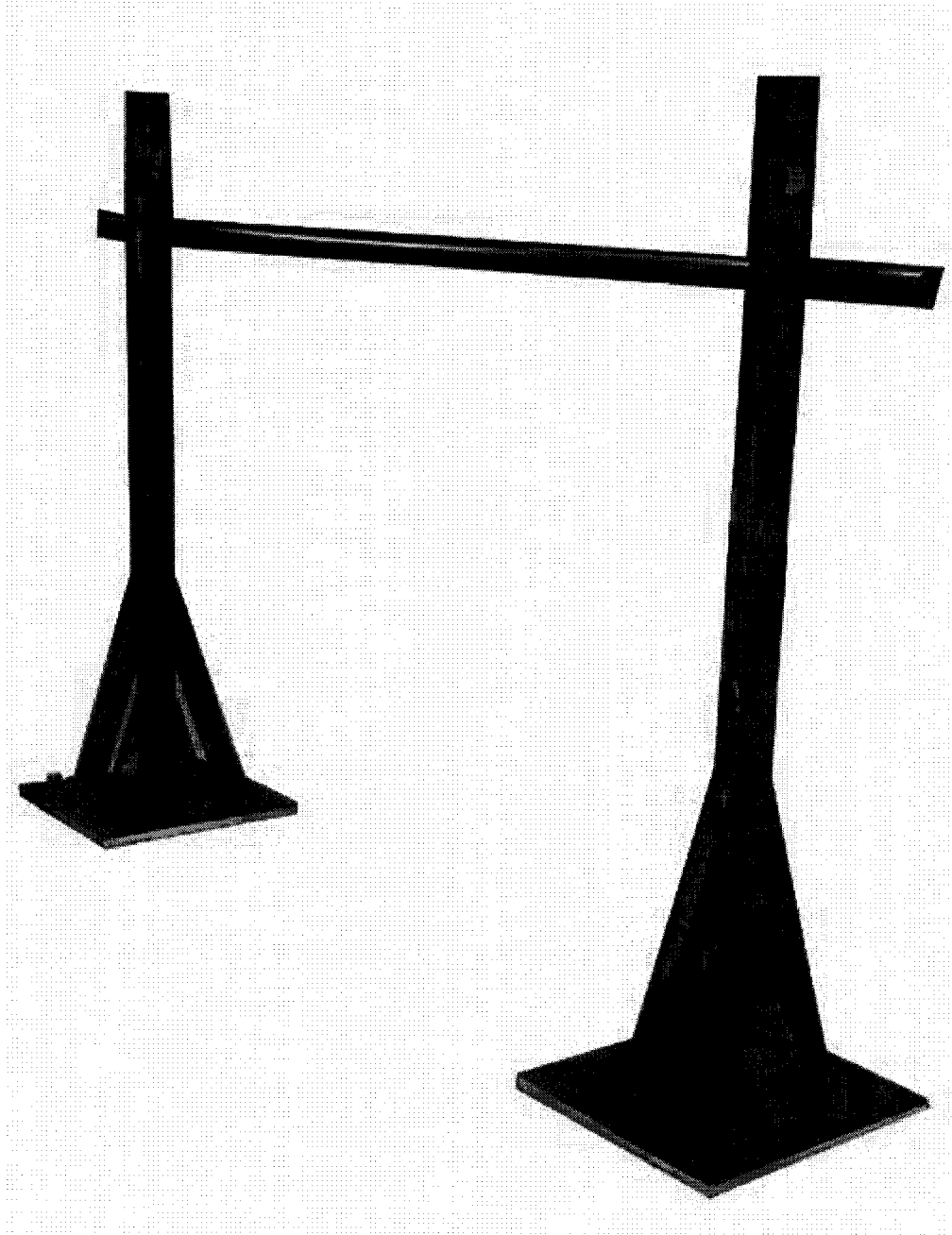
Appendix D

Height-Adjustable Stool



Appendix E

Range of Motion Support Frame



Appendix F

Concept Map

