

**TRUNK AND LOWER LIMB BIOMECHANICS DURING STAIR
CLIMBING IN PEOPLE WITH SYMPTOMATIC
FEMOROACETABULAR IMPINGEMENT COMPARED TO
ASYMPTOMATIC HEALTHY INDIVIDUALS**

by

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Abstract

Introduction: Femoroacetabular impingement is a pathomechanical hip condition that leads to pain, impaired physical function and, if left untreated, potentially hip osteoarthritis. It has been shown that those with femoroacetabular impingement exhibit altered gait characteristics during level walking and stair climbing, and decreased muscle force production during isometric contractions. However, to-date no studies have looked at muscle activation during dynamic movements such as stair climbing in this patient population. Purpose: The purpose of this study was to compare three-dimensional gait kinematics of the trunk and lower limb joint kinetics, and activation of the hip, knee and ankle musculature during stair climbing in those with femoroacetabular impingement and pain free controls. Methods: Trunk, hip knee and ankle kinematics, as well as hip, knee and ankle kinetics and EMG activity of nine lower limb muscles were collected during stair climbing for 20 people with femoroacetabular impingement and compared to 20 pain-free individuals. Results: Those with femoroacetabular impingement had significantly increased peak trunk forward flexion angles ($p=0.01$) and external hip flexion moments (0.01), and decreased peak external knee flexion moments (0.01) and lateral gastrocnemius activation ($p=0.04$) compared to the control group. Conclusion: Findings from this study indicate that those with FAI may increase their trunk forward flexion to potentially compensate for reduced gastrocnemius activation, to decrease the demand on the quadriceps or as a response to pain. However, a trunk lean may also be a potential cause of FAI due to increased external hip flexion moments. This should all be taken into account by clinicians when rehabilitating those with FAI.

Preface

This thesis contains the work of a research study conducted by Connor Hammond under the supervision of Dr. Michael Hunt with guidance from Dr. Gillian Hatfield and Dr. Jayne Garland. The study design, data analysis, and writing the manuscript were primarily the work of the candidate. Data collection was performed by the candidate with the help of Dr. Gillian Hatfield. A selection of work from this thesis will be submitted for publication in a relevant peer-reviewed journal.

Ethical approval for this research study was provided by the University of British Columbia Clinical Research Ethics Board on November 12, 2014 and by the Vancouver Coastal Health Research Institute on April 15, 2015. The Clinical Research Ethics Board number is H14-02618 for the University of British Columbia and V14-02681 for Vancouver Coastal Health.

Table of Contents

Abstract.....	ii
Preface.....	iii
Table of Contents	iv
List of Tables	vi
List of Figures.....	vii
List of Abbreviations	viii
Acknowledgements	x
Chapter 1: Background.....	1
1.1 What is Femoroacetabular Impingement?.....	1
1.2 Etiology of FAI	1
1.3 Epidemiology of FAI	3
1.4 Diagnosis of FAI.....	6
1.4.1 Physical Examination	6
1.4.2 Radiographic Imaging	6
1.4.3 MRI/MRA	8
1.5 Treatment of FAI.....	9
1.5.1 Non-operative Treatment of FAI.....	9
1.5.2 Surgical Treatment of FAI.....	9
1.6 Lower Extremity Function in those with Symptomatic FAI.....	10
1.6.1 Passive ROM of the Hip.....	10
1.6.2 Squatting.....	12
1.6.3 Level Walking Gait Analysis	15
1.6.4 Stair Climbing Gait Analysis.....	19
1.6.5 Hip Muscle Strength.....	20
1.6.6 Muscle Activation.....	23
1.7 Thesis Rationale, Objectives and Hypotheses.....	25
1.7.1 Thesis Rationale	25
1.7.2 Objectives	27
1.7.3 Hypotheses.....	27

Chapter 2: Introduction	29
Chapter 3: Methods	32
3.1 Study Design	32
3.2 Study Participants.....	32
3.3 Instrumentation.....	33
3.4 Procedures/Protocol	35
3.5 Data Analysis	38
3.5.1 Kinematic Data.....	39
3.5.2 Kinetic Data.....	40
3.5.3 EMG Data.....	40
3.6 Statistical Analysis	42
3.6.1 Sample Size	42
3.6.2 Statistical Analysis	42
Chapter 4: Results.....	45
4.1 Participant Demographics	45
4.2 Outcome Data.....	46
4.2.1 Kinematic Data.....	47
4.2.2 Kinetic Data.....	48
4.2.3 EMG Data.....	54
Chapter 5: Discussion	58
5.1 Interpretation of Findings.....	58
5.2 Clinical Implications	63
5.3 Study Limitations	66
5.4 Future Directions.....	68
Chapter 6: Conclusions	71
References.....	73
Appendix.....	79
Appendix A: Informed Consent Form	79

List of Tables

Table 1. Biomechanical variables measured.....	44
Table 2. Demographic, questionnaire and radiographic data	46
Table 3. Participant outliers	47
Table 4. Hip, knee, ankle and trunk kinematic data.....	49
Table 5. Hip, knee and ankle kinetic data.....	50
Table 6. EMG data.....	55

List of Figures

Figure 1. Types of FAI.....	3
Figure 2. Alpha Angle.....	7
Figure 3. Centre Edge Angle	8
Figure 4. Marker set used for this study	34
Figure 5. Instrumented staircase	35
Figure 6. Isometric MVC exercises	37
Figure 7. Sagittal plane kinematic and kinetic (external joint moments) ensemble averages about the hip, knee and ankle.....	51
Figure 8. Frontal plane hip kinematic and kinetic ensemble averages	52
Figure 9. Sagittal plane trunk kinematic ensemble averages	53
Figure 10. Muscle activation ensemble averages part 1	56
Figure 11. Muscle activation ensemble averages part 2	57

List of Abbreviations

ANCHOR	Academic Network of Conservational Hip Outcomes Research
BF	Biceps Femoris
BMI	Body Mass Index
CT	Computed tomography
EMG	Electromyography
FAI	Femoroacetabular Impingement
GMax	Gluteus Maximus
GMed	Gluteus Medius
HOOS	Hip disability and Osteoarthritis Outcome Score
Hz	Hertz
IHOT-12	International Hip Outcome Tool
LG	Lateral Gastrocnemius
MG	Medial Gastrocnemius
MHHS	Modified Harris Hip Score
MRA	Magnetic Resonance Arthrogram
MRI	Magnetic Resonance Imaging
MVC	Maximum Voluntary Contraction
NCAA	National Collegiate Athletic Association
Nm/kg	Newton Metres per Kilogram
NRS	Numerical Rating Scale
OA	Osteoarthritis
RF	Rectus Femoris

RMS	Root Mean Square
ROM	Range of Motion
ST	Semitendinosus
TA	Tibialis Anterior
TFL	Tensor Fasciae Latae
THA	Total Hip Arthroplasty
VL	Vastus Lateralis
VM	Vastus Medialis
μV	Microvolts

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Chapter 1: Background

1.1 What is Femoroacetabular Impingement?

Femoroacetabular impingement (FAI) is a result of an abnormal contact or impingement between the proximal femur and the acetabulum of the hip joint (Ganz et al., 2003). This impingement often occurs on the anterosuperior aspect of the pelvis during terminal hip range of motion (ROM) and is more common in young and physically active adults (Beck et al., 2005). If left untreated, FAI can result in pain, damage to the acetabular articular cartilage and osteoarthritis (OA) of the hip (Crawford and Villar, 2005; Ganz et al., 2003; Kowalczyk et al., 2015).

FAI presents as groin and back pain, which is exacerbated by excessive hip flexion and internal rotation during common everyday activities such as: prolonged sitting or walking, deep squats, stair climbing, twisting maneuvers and other athletic activities (Crawford and Villar, 2005; Leunig et al., 2005). This pain can cause avoidance of physical activity, leading to an increased sedentary lifestyle and potential biomechanical adaptations such as limping. Therefore it is very important to understand the function of the lower limb, especially during activities which cannot be avoided. Having a full understanding of this pathology and its effect on the lower extremity will better allow clinicians to rehabilitate those with symptomatic FAI.

1.2 Etiology of FAI

Over the past several decades, FAI has become an increasingly recognized cause of hip pain, yet the development of this pathomechanical hip disorder remains unknown. The current hypothesis for the development of FAI is sport related. Repetitive stresses at the proximal femoral physis from athletic endeavors during skeletal growth have been suggested to increase

the risk of developing FAI (Beck et al., 2005; Byrd, 2014). However, physical activity alone has not been shown to cause FAI (Torry et al., 2006). In addition to high levels of physical activity, genetics and several predisposing conditions have been linked to the development of FAI, including: slipped capital femoral epiphysis with posterior tilt of the femoral head, femoral head necrosis with subsequent flattening, and previous fracture of the femoral head (Crawford and Villar, 2005; Pollard et al., 2010).

FAI is believed to be the result of abnormal contact between the proximal femur and the acetabulum that can result in damage to the articular cartilage and if left untreated, progressive secondary hip OA (Ganz et al., 2008). Proper function of the hip joint is essential to preserving the hip joint and preventing OA. In FAI, bony abnormalities to the femoral head decrease the offset between the femur and pelvis, resulting in impingement during tasks requiring excessive hip flexion (Leunig et al., 2005). This is known as cam impingement (Figure 1). Cam impingement is a result of repetitive loading to the femoral head neck junction, causing the femur to compensate by forming an osseous prominence at the site of the impingement. This is known as the pistol-grip deformity (Wenger et al., 2004). During excessive hip flexion and internal rotation, shearing forces imposed by the pistol-grip deformity are projected onto the anterosuperior aspect of the acetabulum. This causes outside-in trauma to the acetabular cartilage and may result in an avulsion of the cartilage from the labrum (Beck et al., 2005). In addition to bony abnormalities to the femur, excessive femoral head coverage by the acetabulum as a result of abutment from the femoral head neck junction leads to degeneration of the labrum, ossification of the acetabular rim and deepening of the acetabulum (Beck et al., 2005). This is known as pincer impingement (Figure 1). Those with FAI can also have mixed impingement, which is a combined cam and pincer impingement (Figure 1). Additionally, the presence of each type of impingement have varying correlations with hip OA. A recent systematic review

observed that the severity of the cam impingement was strongly correlated with the development of radiographic OA, earlier total hip arthroplasty (THA), and more severe cartilage damage observed at the time of surgery (Kowalczyk et al., 2015). However, long-term studies have failed to correlate pincer impingement to hip OA.

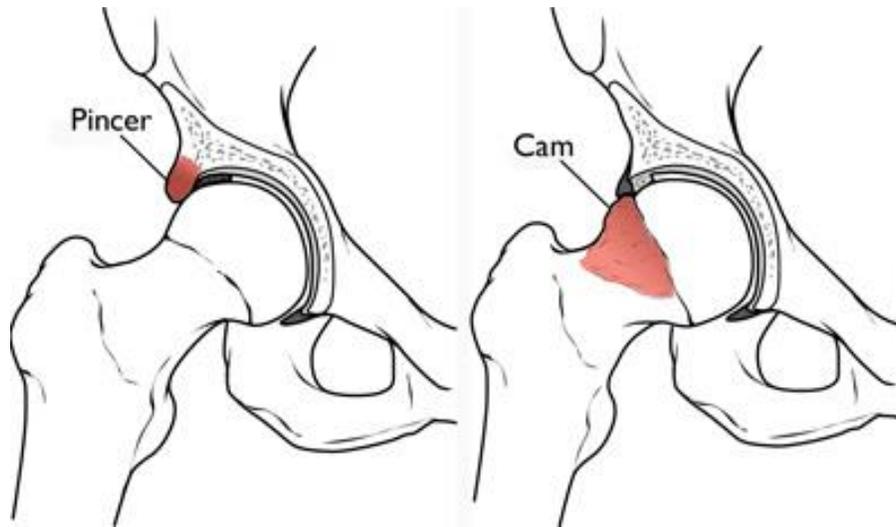


Figure 1. Types of FAI; Two types of FAI, acetabular over coverage or pincer impingement (Left) and larger than average femoral head or cam impingement (Right). **Reproduced with permission from *OrthoInfo*. ©American Academy of Orthopaedic Surgeons. <http://orthoinfo.aaos.org>.**

1.3 Epidemiology of FAI

Recently, the Academic Network of Conservational Hip Outcomes Research (ANCHOR) was created to perform multicentre clinical studies on evaluating diagnostic and treatment options for prearthritic hip diseases. Epidemiological data from the ANCHOR study group on 1076 patients (1130 hips) with symptomatic FAI who underwent surgery, reported that 55% were female and 45% were male (Clohisy et al., 2013). The average patient age was 28.4 years (range 11 - 68 years) with an average body mass index (BMI) of 25.1 (range 15 – 53 kg/m²). The most common length of hip symptoms before surgery ranged from 1 - 3 years. The disease classification from the ANCHOR cohort revealed that of those diagnosed with symptomatic FAI,

48% were cam, combined cam/pincer were in 45% of hips and pincer were in only 8% of hips. The combined cam/pincer impingement was most common in men, whereas the cam only was most common in women. The cohort provided by the ANCHOR study group is uncharacteristic of previous research, as previous reports on FAI have included cohorts that were predominantly males. Clohisy et al. (2013) alludes to the fact that as symptomatic FAI has a high prevalence within athletic individuals, and female participation in the National Collegiate Athletic Association (NCAA) has increased five-fold in the past thirty years. Thus, this increase in females with symptomatic FAI may be due to an increase in sport participation. This association has been established within male athletes but further research into the female subgroup is needed in order to determine a cause and effect relationship (Siebenrock et al., 2011).

Repetitive physical activity may predispose to the development of symptomatic FAI, especially when this physical activity takes place during the developmental years. Therefore, a large amount of the literature has focused on the prevalence of symptomatic FAI within elite athletes. Sports that involve repetitive and supraphysiologic hip ROM may result in remodeling of the hip joint according to the stresses applied to it during phases of development. Sports such as hockey, basketball and soccer have observed a high prevalence of symptomatic FAI within their players (Byrd, 2014). Recently, a systematic review and meta-analysis observed that males who participated in aggressive sporting events (hockey, basketball and jumping sports) during their developing years were significantly (1.9 - 8.0 times) more likely to develop symptomatic FAI compared to male controls (Nepple et al., 2015). While the development of FAI in athletic males is well studied, a lack of research exists as to the development of symptomatic FAI in athletic females. In the one study that evaluated a subgroup of female athletes (N = 25), Johnson et al. (2012) observed that no major differences were observed in the prevalence of the cam deformity between male and female soccer players. However, it has been observed that

symptomatic females have milder cam deformities compared to males (Hetsroni et al., 2013). As sport participation within female adolescents grows in popularity, further research is needed to properly determine the influence of athletic activities on the development of symptomatic FAI within female adolescents.

While FAI has been thought to be a common factor in hip pathology, not all FAI is associated with symptoms. Previous research by Frank et al. (2015) comparing the prevalence of FAI within asymptomatic hips (radiographic evidence of FAI, but no symptoms) observed that 37% of asymptomatic hips showed signs of cam FAI compared to 67% showing signs of pincer FAI. The average age of those in the study was 25.3 ± 1.5 years with 57.2% of them being men and 42.8% women. Additionally, of the 2114 asymptomatic hips analyzed, 33% were athletes. Of those analyzed, 54.8% of the asymptomatic athletic population showed radiographic signs of cam FAI compared to 23.1% of the general population. These values reported by Frank et al. (2015) are much higher than the previously reported prevalence of 14% - 24% (Hack et al., 2010; Reichenbach et al., 2010). This is partially due to the fact that the studies by Hack et al. (2010) and Reichenbach et al. (2010) did not include athletes in their cohorts. Hack et al. (2010) analyzed 200 asymptomatic volunteers who worked at a hospital, whereas Reichenbach et al. (2010) analyzed 1080 asymptomatic male volunteers who were undergoing conscription within the Swiss Army. In addition to asymptomatic individuals having a high prevalence of FAI, labral injury has also been well documented within this population. Previous studies using magnetic resonance imaging (MRI) and magnetic resonance arthrograms (MRA) observed that labral injury was found in 68% of hips (Frank et al., 2015).

1.4 Diagnosis of FAI

Often those with symptomatic FAI get misdiagnosed early on and are treated for a variety of disorders such as back pain, hip pain, groin pain, bursitis, piriformis syndrome, tendonitis of the iliopsoas, groin strain and “growing pains”, before a final diagnosis of FAI is finally reached (Byrd, 2007). This was evident in a previous study by Clohisy et al. (2009) in which a cohort of 51 patients saw an average of 4.2 ± 2.9 health care providers before a diagnosis of FAI was made. A diagnosis of FAI is typically made through a combination of physical examination, diagnostic imaging radiographs and MRI or MRAs. Of course, this only occurs in those with symptomatic FAI (radiographic evidence of FAI and painful hips), as those with asymptomatic FAI (radiographic evidence of FAI but no symptoms) are not often diagnosed because they do not present with pain and/or lack of hip mobility and therefore see no need to visit a clinician.

1.4.1 Physical Examination

Physical examinations by a clinician reveal some restriction in the ROM of the hip. This is tested using an impingement test. During this test, the subject is supine with the hip rotated internally, flexed to 90° and adducted. This induces shearing forces on the acetabular rim and, if accompanied by pain, indicates the presence of an anterior labral lesion, often caused by FAI and other hip pathologies. In a previous study to evaluate the accuracy of the impingement test for FAI, it was observed to have a sensitivity of 56% and the specificity of 100% (Hananouchi et al., 2012). Therefore, the results of the impingement test in conjunction with radiographic evidence are often needed in order to accurately diagnose symptomatic FAI.

1.4.2 Radiographic Imaging

Radiographic images to diagnosis FAI are taken in two positions. First, an anteroposterior view of the hip may show a flattened head neck junction of the femur or pistol-grip deformity.

Secondly, a lateral view will also show a pistol-grip deformity along with a loss of the femoral neck offset. In addition to viewing a loss of the femoral head neck offset, the lateral view allows the clinician to determine any specific changes to the acetabulum.

The two measurements that clinicians use to diagnose and characterize FAI are the alpha angle and centre edge angle. The alpha angle is determined by drawing a line from the centre of the femoral head down the long axis of the femoral neck, and a line from the centre of the femoral head to the edge of the femoral head (Figure 2). If this angle is greater than 50.5° , it is indicative of cam FAI (Clohisy et al., 2008). The centre edge angle is measured by drawing a vertical line superior of the femoral head and a second line from the femoral head to the lateral edge of the acetabulum (Figure 3). If this angle is greater than 39° , it is indicative of pincer FAI (Notzli et al., 2002).

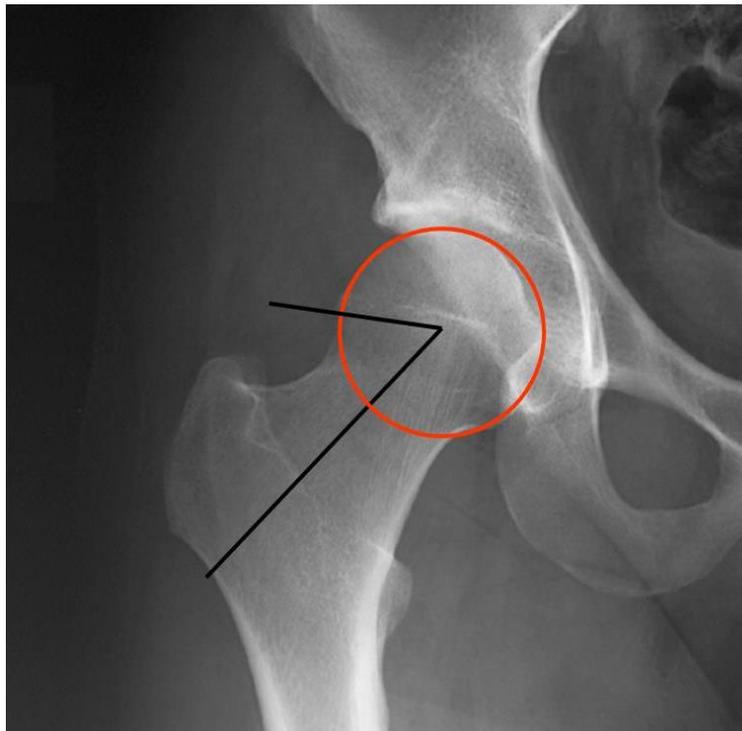


Figure 2. Alpha Angle; Angle formed between a line drawn down the centre of the long axis of the femoral neck and another line to the edge of the femoral head. **Alpha Angle (© 2014 Orthopaedicsone, Michael Taunton and Christian Veillette, by permission)**

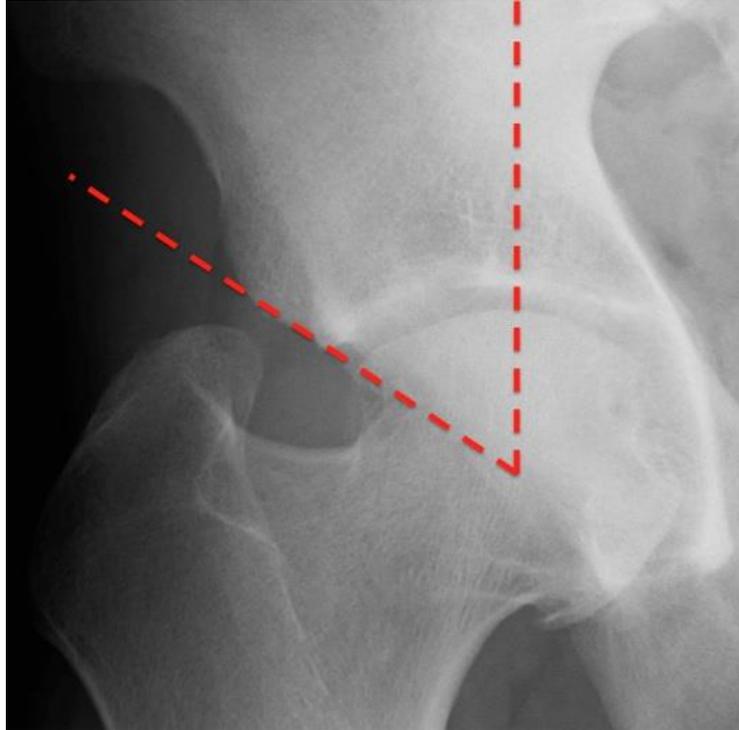


Figure 3. Centre Edge Angle; Angle formed by a vertical line up from the centre of the femoral head and a line connecting the centre of the femoral head with the lateral edge of acetabulum.
Center Edge Angle (© 2014 Orthopaedicsone, Michael Taunton and Christian Veillette, by permission)

1.4.3 MRI/MRA

The use of an MRI is becoming a common tool in the diagnosis of FAI. From a MRI, clinicians are better able to analyze the alpha angle, centre edge angle and determine if any damage has occurred to the labral cartilage. In addition to a standard MRI, the hip joint can be injected with gadolinium. This is called a MRA. Gadolinium makes certain tissues and disease processes more visible, such as labral tears, on the MRI scans. The MRA is now becoming the standard in detecting FAI.

1.5 Treatment of FAI

The primary goal of treatment for symptomatic FAI is to reduce the forces that put the anterior hip and labrum at risk for injury. This can be done non-operatively by physical therapy and pharmaceutically and/or operatively through surgery.

1.5.1 Non-operative Treatment of FAI

After initial diagnosis of FAI, the literature suggests that patients should undergo physical therapy before seeking surgical options (Clohisy et al., 2013; Khan et al., 2015). During physical therapy, those with symptomatic FAI are initially told to avoid compromising positions that involve extreme hip flexion and internal rotation such as cycling and running (Wall et al., 2013). In addition to activity modification, those with symptomatic FAI are typically prescribed exercises aimed at strengthening the gluteus medius (GMed) and maximus muscles. Strengthening these muscles will help improve the posterior glide of the femur, thereby reducing the forces on the anterior aspect of the hip. Additionally, exercise strategies to maximize gluteal activation while reducing iliopsoas and tensor fasciae latae (TFL) activity are encouraged (Kokmeyer et al., 2014). In addition to physical therapy, non-steroidal anti-inflammatory drugs may be prescribed.

1.5.2 Surgical Treatment of FAI

If the conservative treatment of symptomatic FAI fails and the groin and back symptoms are still present, then surgery is the only option. The goal of surgery is to correct the underlying morphological abnormality causing the impingement. This can include a trochanteric osteotomy to remove the cam impingement and/or resection of the excessive acetabular rim to remove the pincer impingement. In addition to correcting the underlying bony abnormalities, a partial resection and repair of the labral cartilage may be required if a tear is present (Crawford and

Villar, 2005). The most common techniques used in the previously mentioned ANCHOR cohort of 1076 patients to correct the aforementioned abnormalities were hip arthroscopy (50.4%) and surgical dislocation (34.4%) (Clohisy et al., 2013).

1.6 Lower Extremity Function in those with Symptomatic FAI

While the underlying anatomical abnormalities associated with symptomatic FAI are widely reported in the literature (Beck et al., 2005; Ganz et al., 2003; Leunig et al., 2009), the effect that symptomatic FAI has on the function of the lower extremity within the literature is scarce. Research has only recently begun to quantify the ROM and biomechanics during functional tasks such as squatting, level walking and stair climbing. In addition to biomechanical analysis, limited research exists as to the effect of symptomatic FAI on strength and activation of the lower extremity musculature. Having a better understanding of the lower extremity ROM, joint loading and muscle activation patterns will better allow clinicians to rehabilitate those with symptomatic FAI.

1.6.1 Passive ROM of the Hip

The majority of research involving ROM within those with symptomatic FAI has been conducted by clinicians, and although they report a loss of ROM, specific values comparing those with FAI to healthy controls are not typically presented within the literature (Jager et al., 2004; Leunig et al., 2005; Siebenrock et al., 2004). However, recent research reporting these values has been conducted to compare this loss of passive ROM in those with symptomatic FAI with healthy controls.

Previous research by Audenaert et al. (2012) used an electromagnetic tracking system to measure passive ROM in those with symptomatic FAI compared to asymptomatic FAI and healthy controls (no radiographic evidence of FAI and no symptoms). The study recruited 18

participants with symptomatic FAI (24 hips), 12 radiographic diagnosed (alpha angle > 55°) asymptomatic FAI participants (24 hips) and 12 healthy control participants (24 hips). It was observed that those with symptomatic FAI had significantly decreased peak hip internal (28.5° vs. 34.1°,) and external rotation (28.9° vs. 38.4°), hip flexion (113.7° vs. 125°) and internal rotation at 90° of hip flexion (16.7° vs. 28°) angles when compared to the healthy controls. In addition to having decreased ROM compared to the healthy controls, those with symptomatic FAI also exhibited decreased peak external rotation and internal rotation ROM at 90 ° of hip flexion when compared to the asymptomatic FAI participants, (28.9° vs. 38° and 16.7° vs. 28.8°, respectively). No significant differences were observed in ROM between the asymptomatic FAI group and the healthy controls. Unfortunately, hip adduction and abduction ROM was not measured and therefore no conclusion can be drawn as to hip mobility in the frontal plane. Therefore, based on the findings of Audenaert et al. (2012), those with symptomatic FAI exhibit decreased passive ROM about the hip when compared to those with asymptomatic FAI and healthy controls. Furthermore, it was observed that those with asymptomatic FAI (radiographic evidence of FAI but no symptoms) exhibit the same hip ROM as those with normal healthy hips.

Similarly, Nussbaumer et al. (2010) used a goniometer to measure passive ROM in those with symptomatic FAI compared to asymptomatic controls that were not radiographed for the presence of FAI. It was observed that those with symptomatic FAI exhibited significantly less peak hip abduction angles when compared to the controls (30.4° vs. 39.3°, respectively). In contrast, although there was a trend towards lower ROM in the symptomatic FAI group, no significant differences were observed with respect to peak hip flexion, adduction, internal and external rotation angles in those with FAI compared to the controls.

Taken together, these studies indicate that that those with symptomatic FAI likely exhibit a decrease in peak internal and external rotation, internal rotation at 90° hip flexion, hip flexion

and hip abduction angles when compared to those with asymptomatic FAI and healthy controls. Having quantifiable values for this loss of ROM in those with symptomatic FAI reported in the literature will better allow clinicians to use passive ROM for diagnostic purposes. Additionally, it was observed that those with asymptomatic FAI exhibit the same ROM as those with healthy hips. This suggests that a loss of passive ROM only occurs when the bony abnormality is combined with symptoms. However, assessing passive ROM is very useful for diagnostic purposes but provides no insight into the ROM during everyday movements. Therefore, looking at hip ROM during dynamic tasks such as deep squatting, walking and stair climbing will provide greater insight into the function of the lower extremity in those with symptomatic FAI.

1.6.2 Squatting

Deep squatting requires a large amount of hip and pelvic motion, encroaching on the normal limits of hip ROM in those with symptomatic FAI. Furthermore, while deep squatting is not in itself a common activity, the excessive hip flexion required to perform the movement is a component in a variety of everyday activities such as sitting in a chair, tying ones shoes and stair climbing. Therefore it is important to understand the biomechanical consequences of symptomatic FAI during the dynamic task of deep squatting.

Using three-dimensional motion analysis technology, Lamontagne et al. (2009) measured peak sagittal plane hip and pelvic kinematics and excursions (difference between the peak flexion and extension) at maximum squat depth in those with symptomatic cam FAI (N = 15) compared to healthy controls (N = 11) with no clinical or radiographic presence of FAI. The maximum depth was standardized by placing a bench behind the participant at a height equal to 1/3 of their tibial length. If the participant was able to squat down, touch the bench with their buttocks, without putting any weight on it while maintaining heel contact, then they were said to

have reached the maximum squat depth. If the participant was not able to touch the bench with their buttocks, the point at which they were closest to the bench was taken as the maximum squat depth. It was observed that only 33% of those with symptomatic FAI were able to reach the required squat depth compared to 91% of controls. Additionally, it was observed that those with symptomatic FAI exhibited a significant decrease in sagittal plane pelvic excursion when compared to the healthy controls (14.7° compared to 24.2°, respectively). When squat depth was included as a covariate, sagittal plane pelvic excursion remained reduced in those with symptomatic FAI compared to the healthy controls. In addition to decreased sagittal plane pelvic excursion, those with symptomatic FAI were observed to have greater peak anterior pelvic tilt at maximum squat depth when compared to the control group. Greater anterior pelvic tilt results in greater acetabular retroversion, leading to increased contact between the acetabular rim and femur. This indicates that at peak squat depth, those with symptomatic FAI predispose the hip to premature contact between the proximal femur and acetabular rim. No significant differences were observed with respect to sagittal plane peak hip angles or excursions in those with symptomatic FAI compared to the healthy controls. Additionally, it was observed that hip kinematics during squatting did not return to normal after corrective surgery had been performed (Lamontagne et al., 2011).

Similarly, Bagwell et al. (2016) used three-dimensional motion analysis to measure pelvic and hip kinematics and joint moments during a maximum squat in those with symptomatic FAI (N = 15) compared to healthy controls (N = 15) with no clinical or radiographic sign of FAI. It was observed that those with symptomatic FAI had diminished squat depth compared to the controls (70% of the participant's leg length compared to 51% of leg length off the ground, respectively). At peak squat depth, those with symptomatic FAI exhibited greater peak anterior pelvic tilt when compared to the healthy controls (23.4° compared to 12.5°, respectively). In

addition to greater peak anterior pelvic tilt, a decrease in the peak internal hip extensor moment was observed in those with symptomatic FAI compared to the healthy controls (0.45 newton metres per kilogram (Nm/kg) compared to 0.56 Nm/kg, respectively). The authors suggest that greater anterior pelvic tilt and a reduction in the hip extensor moment may be due to decreased activation and/or strength of the gluteus maximus (GMax) and/or hamstring muscles. However, electromyography (EMG) and strength data were not collected. Additionally, it was observed that those with symptomatic FAI exhibited significantly decreased peak internal rotation of the hip during a deep squat when compared to the healthy controls (9.5° compared to 15.2°, respectively). However, no differences were observed with respect to peak hip flexion, adduction, abduction, and external rotation angles or peak hip flexor, adductor, abductor and rotator moments during deep squats.

Therefore, previous research looking at the common activity of deep squatting in those with symptomatic FAI suggests that a decrease in squat depth may be caused by premature bone on bone contact between the femur and acetabulum. This was a result of greater anterior pelvic tilt in those with symptomatic FAI compared to healthy controls. Both Lamontagne et al. (2009) and Bagwell et al. (2016) observed greater anterior pelvic tilt at maximum hip flexion, suggesting greater acetabular retroversion in those with symptomatic FAI compared to healthy controls. Additionally, Bagwell et al. (2016) observed a decreased hip extensor moment, indicating hip extensor muscles may play a role in the increased anterior pelvic tilt in those with symptomatic FAI. This suggests that altered activation of the hip extensors may prevent posterior tilt of the pelvis during deep squatting, thereby causing acetabular retroversion and premature bone on bone contact between the acetabulum and femur. This premature bone on bone contact may cause increased bone remodeling at the impingement site, thereby furthering the progression of FAI. This may increase the symptoms and lead to an earlier onset of hip OA.

Therefore, those with symptomatic FAI have been shown to exhibit altered lower extremity function during passive ROM and during the dynamic task of deep squatting. However, while deep squatting is required to perform a variety of movements, it is not in itself a common daily activity. Therefore measuring hip kinematics and kinetic joint moments during more functional tasks such as walking and stair climbing may provide greater insight into the function of the lower extremity in those with symptomatic FAI. Additionally, while decreased hip extension joint moments were observed in those with symptomatic FAI during deep squatting, further research is required on muscle activation during dynamic tasks such as level walking and stair climbing in order to determine whether reduced hip kinematics and kinetics are a result of altered muscle activation.

1.6.3 Level Walking Gait Analysis

Early gait analysis research showed that patients with FAI have a “near normal” gait presentation, but that pain may result in certain compensations (Zebala et al., 2007). However, measures were only reported about the hip and the compensatory mechanisms undertaken about the trunk, knee and ankle were not discussed. Since walking is the most common activity of daily living, it is important to quantify the function of the lower extremity during such activities as level walking and stair climbing in those with symptomatic FAI. Furthermore, understanding the biomechanical alternations undertaken by the trunk, knee and ankle to compensate for the hip are required to quantify the function of the lower extremity.

Using three-dimensional motion analysis, Kennedy et al. (2009), Brisson et al. (2013) and Diamond et al.(2016) measured hip kinematics and joint moments during walking in those with symptomatic FAI compared to asymptomatic healthy controls with no clinical or radiographic sign of FAI. All three studies reported that those with symptomatic FAI exhibited significantly

decreased sagittal plane hip excursion, resulting from reduced peak hip extension during level walking when compared to the healthy controls. Additionally, Brisson et al (2013) and Kennedy et al. (2009) observed that those with symptomatic FAI had reduced frontal plane hip excursion compared to the healthy controls during level walking. Specifically, Kennedy et al. (2009) observed that those with symptomatic FAI exhibited significantly lower peak hip abduction angles when compared to the healthy controls. A reduction in the sagittal plane ROM suggests that soft tissue restriction may be the cause as the hip does not approach the end of the available range, where the bony impingement might occur. This restriction may occur to prevent the ligaments and tendons of the anterior hip from being aggravated by the bony impingement during movements in the sagittal plane. This aggravation can lead to snapping hip syndrome, inflammation and general tightness, and may be a factor in the difference between symptomatic and asymptomatic FAI and why those with symptomatic FAI tend to take a leave from their sport. Additionally, a reduction in the frontal plane excursion and peak abduction angle suggests that a compensatory strategy was utilized in those with symptomatic FAI as the peak abduction angle was well below the terminal ROM. However, despite differences in dynamic ROM, no evidence of impaired hip joint moments was found in all three studies. Furthermore, only Brisson et al. (2013) reported measures about the knee and ankle in order to determine and compensatory mechanisms undertaken about these two joints. It was observed that those with symptomatic FAI exhibited greater peak knee flexion joint moments compared to the healthy controls (Brisson et al., 2013). This indicates that in those with FAI, greater load is applied to the knee potentially as a compensatory mechanism to protect the hip. However, no measures were reported about the trunk.

In addition to the previous studies that screened their healthy control population for radiographic signs of FAI, Hunt et al. (2013) and Rylander et al. (2013) compared hip joint

kinematics and moments in those with symptomatic FAI to asymptomatic controls that were not radiographed for the presence of FAI. Both Hunt et al. (2013) and Rylander et al. (2013) observed that those with symptomatic FAI exhibited altered joint angles in all three of the sagittal, transverse and frontal planes when compared to the asymptomatic controls. With respect to the sagittal plane, both Hunt et al.(2013) and Rylander et al. (2013) observed that those with symptomatic FAI exhibited a reduction in the sagittal plane excursion. This was a result of a reduction in the peak hip extension observed by Hunt et al.(2013), and lesser peak hip flexion observed by Rylander et al. (2013). This provides further evidence to a restriction of the soft tissue of the anterior hip in an attempt prevent aggravating movements that expose this tissue to the bony impingement. Additionally, upon visual inspection of the sagittal plane kinematic profile, Rylander et al. (2013) observed the presence of a “reversal”. A reversal is defined as a second-order change of the slope of the sagittal plane curve and has been observed in those with end stage hip OA (Foucher et al., 2012).

With respect to the transverse plane, Hunt et al. (2013) observed a reduction in peak internal rotation, while Rylander et al.(2013) observed a reduction in the transverse plane excursion in those with FAI compared to the controls during level walking. This reduction in transverse plane ROM suggests that those with FAI may limit movement when approaching positions of bony impingement in order to prevent aggravation to the soft tissue. However, there were inconsistent findings with respect to the frontal plane during level walking; Hunt et al. (2013) observed less peak hip adduction, whereas Rylander et al. (2013) observed lesser peak abduction in those with symptomatic FAI compared to the controls. Additionally, only Hunt et al (2013) reported hip joint moments. It was observed that those with symptomatic FAI exhibited decreased external peak hip flexion and external rotation moments compared to the controls during level walking (Hunt et al., 2013). This indicates that those with FAI may undergo

kinematic changes in order to reduce the joint loading about the hip. Furthermore, both Hunt et al. (2013) and Rylander et al. (2013) failed to report any measures about the trunk, knee or ankle; therefore the compensatory mechanisms in which these two joints may undergo in order to protect the hip are unknown.

Therefore, those with FAI have been shown to elicit altered hip kinematics in the sagittal, frontal and transverse planes during walking. Additionally, those with FAI have been observed to exhibit altered hip joint loading. These alterations to hip kinematics and kinetics in those with FAI may be undertaken in order to prevent aggravation of the soft tissue from the bony impingement. Furthermore, a reduction in hip joint moments may indicate that altered muscle activation may be involved in those with FAI. However, despite the observed kinematic and joint moment differences at the hip between those with and without symptomatic FAI, the lack of generalized biomechanical differences may suggest that level walking may not be sufficiently challenging to properly assess hip functional limitations in the presence of FAI. While the literature is consistent in that those with symptomatic FAI exhibit reduced sagittal and frontal plane excursion, inconsistencies amongst researchers as to whether this occurs during peak flexion or extension and peak adduction or abduction exists. Additionally, only one study observed a reduction in peak joint moments about the hip. Therefore, if level walking may not be challenging enough to accurately quantify hip biomechanics then a more challenging task requiring greater hip requirements may provide a better understanding of the functional implications of FAI.

This notion is further supported by the fact that previous research shows inconsistencies on the effect of gait biomechanics during level walking after corrective surgery; Rylander et al. (2013) observed that hip joint biomechanics returned to normal, whereas Brisson et al. (2013) observed that they did not. This suggests that the underlying cause of altered kinematics in those

with symptomatic FAI may not solely be a result of the bony impingement and that altered muscle activation may be a factor. Additionally, further data on knee and ankle kinematics during functional tasks are needed in order to fully understand the mechanism in which symptomatic FAI affects the function of the lower extremity.

1.6.4 Stair Climbing Gait Analysis

Stair climbing requires greater ROM and muscle activation about the hip compared to level walking (Andriacchi et al., 1980; Nadeau et al., 2003). In the presence of a disability such as FAI, this may pose quite the challenge. Therefore it is important to understand the function of the lower extremity during stair climbing in those with FAI.

A recent study by Rylander et al. (2013) measured hip and pelvic kinematics in those with symptomatic FAI compared to non-radiographed asymptomatic controls during stair climbing. It was observed that those with symptomatic FAI exhibited less peak hip extension (-11.4° vs. -6.6° , where negative values indicate that the hip remained in flexion rather than going into true extension), sagittal plane excursion (54.8° vs. 60.0°) and peak internal rotation (7.1° vs. 12.1°) of the hip compared to the asymptomatic controls during stair climbing (Rylander et al., 2013). Additionally, it was observed that those with symptomatic FAI exhibited greater peak anterior pelvic tilt (20.8° vs. 14.3°) and transverse plane pelvic excursion (13.8° vs. 8.3°) when compared to the controls. Furthermore, as part of this study it was observed that hip and pelvic kinematics during stair climbing did not return to normal one year post-corrective surgery, despite a reported reduction in pain. A reduction in peak hip angles may be a result of soft tissue restriction as the hip did not approach on the limits of ROM (Kennedy et al., 2009). This may be a compensatory measure to limit the stress on the soft tissue of the anterior hip as it is stretched across the bony impingement. Greater stress on the anterior hip soft tissue as a result of rubbing

against the bony impingement may result in inflammation, tightness and pain. This reduction in hip ROM was compensated for by greater pelvic movement, however, an increase in pelvic movement during stair climbing may be contributing to lower back pain, a common occurrence in those with symptomatic FAI (Clohisy et al., 2009). Unfortunately, hip and pelvic joint moments and muscle activation were not reported, and therefore this theory can only be speculated. Furthermore, Rylander et al. (2013) failed to report any measures about the trunk, knee or ankle.

Therefore, under the more strenuous activity of stair climbing, Rylander et al. (2013) observed similar biomechanical differences in people with FAI as observed during level walking. Furthermore, the continued abnormal hip and pelvic motion during stair climbing post-corrective surgery indicates that hip function is not restored in tasks that require greater hip ROM. Unfortunately, only one publication exists on those with symptomatic FAI during stair climbing, and only hip kinematics were reported. Therefore in order to fully understand the function of the lower limb in the presence of FAI, further research reporting on hip, trunk, knee and ankle kinematics and joint moments needs to be explored in order to understand the compensatory mechanisms in which these two joints may undergo in order to protect the hip. Furthermore, as it has been shown that hip and pelvic kinematics do not return to normal after the bony impingement has been corrected, underlying muscle alterations may be a contributing factor to the altered biomechanics. Therefore, looking at muscle strength and activation will give greater insight into the function of the lower limb in those with symptomatic FAI.

1.6.5 Hip Muscle Strength

The objective measures of physical function in those with symptomatic FAI have been increasingly studied. The literature has shown that those with symptomatic FAI exhibit altered

hip kinematics in all three of the sagittal, frontal and transverse planes during a variety of movements, including: squatting, walking and stair climbing. However, it is important to understand the specific muscle weaknesses that may contribute to these altered movements. The majority of the literature focuses on hip muscle strength for diagnostic and rehabilitation purposes, and although many authors anecdotally report hip muscle weakness, a lack of quantification of muscle weakness in FAI has been highlighted in clinical commentaries (Kokmeyer et al., 2014). Indeed, only a small number of studies have been conducted to actually quantify hip muscle weakness in those with symptomatic FAI.

Previous research conducted by Casartelli et al. (2011) measured isometric maximal voluntary contraction (MVC) strength of the hip flexor, extensor, abductor, adductor, internal rotator and external rotator muscles in those with symptomatic FAI compared to non-radiographed asymptomatic controls. All MVC values were normalized to body mass and calculated as a percent difference. It was observed that those with symptomatic FAI exhibited significantly reduced peak hip adduction (28%), flexion (26%), external rotation (18%) and abduction (11%) strength when compared to the controls. However, although the peak hip extensor and internal rotator strength was found to be 18% and 25% weaker than that of the controls, these values were not found to be statistically significant. Similarly, Diamond et al. (2015) compared isometric and isokinetic hip muscle strength in those with symptomatic FAI compared to an asymptomatic control group that had no clinical or radiographic sign of FAI. During isometric hip exercises it was observed that those with FAI exhibited a significant 20% decrease in peak hip abductor strength. This value is almost double that of (Casartelli et al., 2011). As hip abductor muscles are critical in maintaining a neutral pelvis during single leg weight bearing tasks, any weakness in this muscle group may result in excessive adduction leading to impingement of the hip. As those with FAI have been shown to exhibit pain during

excessive hip adduction, a decrease in hip abductor strength may be a contributing factor. Furthermore, although those with symptomatic FAI exhibited substantial peak muscle weakness in internal rotation (24%), extension (23%), and flexion (16%) of the hip, they were not found to be statistically significant. This is contrary to previous research which observed a weakness in hip flexor, adduction and external rotator muscle groups (Casartelli et al., 2011). This may be a result of different populations, in that Diamond et al. (2015) recruited more females and they were significantly older (seven years). In addition to maximum isometric strength, Diamond et al. (2015) also tested isokinetic strength during internal and external rotation between those with symptomatic FAI compared to healthy controls. However, no significant differences were observed.

Therefore, the values observed by both Casartelli et al. (2011) and Diamond et al. (2015) support clinicians' findings in that those with symptomatic FAI exhibit significant hip muscle weakness. Additionally, the hip muscle weakness observed by that of Casartelli et al. (2011) and Diamond et al. (2015) are similar to that observed in those with hip OA, which have been reported to exhibit a 23% and 20% decrease in overall hip strength when compared to health controls (Arokoski et al., 2002; Rasch et al., 2005). As FAI has been thought to be a precursor to hip OA, it is important to understand the function of the lower extremity in those with FAI to restore function and potentially prevent the development of hip OA. Unfortunately, activation patterns of the primary muscles involved in each movement were not measured by Casartelli et al. (2011) and Diamond et al. (2015), instead choosing to focus on muscle strength/weakness.

While there are inconsistencies in the literature as to the magnitude and location of this weakness, there is agreement that those with symptomatic FAI exhibit significant hip abductor weakness when compared to asymptomatic healthy controls (Casartelli et al., 2011; Diamond et al., 2015). Additionally, it was also observed that those with symptomatic FAI exhibit hip flexor,

adductor and external rotator weakness (Casartelli et al., 2011). Having quantifiable values of muscle weakness in those with symptomatic FAI reported in the literature will better allow clinicians to use hip muscle strength for diagnostic and rehabilitative purposes. However, while assessing isometric and isokinetic muscle strength in those with FAI is very important for diagnostic purposes, it provides little insight into how this weakness is associated with the function of the lower extremity. Therefore, looking at muscle activation, especially during dynamic tasks such as stair climbing, will provide a better understanding of the function of the lower extremity in those with FAI.

1.6.6 Muscle Activation

Though those with FAI have been shown to exhibit hip muscle weakness when compared to healthy controls (Casartelli et al., 2011; Diamond et al., 2015), the mechanisms in which this occurs are unknown. Understanding the neuromuscular activity of hip musculature is crucial to determining the overall function of the lower extremity in those with FAI.

Casartelli et al. (2011) measured the root mean square (RMS) magnitudes of rectus femoris (RF) and TFL muscles during maximal isometric hip flexion between those with symptomatic FAI and asymptomatic non-radiographed controls. As previously stated, those with FAI exhibited a 26% weakness in hip flexion strength when compared to the controls (Casartelli et al., 2011). It was also observed that those with symptomatic FAI exhibited significantly lower TFL RMS, measured in microvolts (μV), during hip flexion compared to the controls (401 μV vs. 582 μV). However, no significant difference was observed with respect to RF RMS during hip flexion. This indicates that those with symptomatic FAI exhibit an impaired ability to voluntarily activate the TFL muscle, which may play a part in the previously stated hip abductor weakness. However, the underlying reason for this is unknown. Furthermore, muscle activity

was only measured in two muscles during hip flexion, yet Casartelli et al. (2011) measured hip strength during extension, adduction, abduction and internal and external rotation. Therefore, the activity of other hip muscles involved in the isometric movements that exhibited weakness were not measured.

While assessing isometric muscle activity is important for diagnostic purposes as well as a basic understanding of muscle function, it provides little insight into the function of the lower extremity during dynamic tasks. Therefore, looking at hip muscle activity during dynamic tasks will provide greater insight into the function of the lower extremity in those with FAI. However, no studies to date have reported hip muscle activity during any dynamic task in those with symptomatic FAI. While, muscle activity in those with FAI during stair climbing has yet to be reported in the literature, previous research on those with hip OA has. As FAI has been shown to elicit similar isometric hip muscle force production and reduced hip extension and adduction kinematics during level walking as in those with hip OA, it is plausible that those with FAI may exhibit similar muscle activity as those with hip OA (Rasch et al., 2005; Zeni et al., 2015). GMed activity in people with hip OA has been measured through surface EMG as a percent of their maximum during a step up task in their affected leg and compared to healthy controls (Dwyer et al., 2013; Sims et al., 2002). Results from Dwyer et al. (2013) and Sims et al. (2002) indicate an increase in GMed activity in those with hip OA compared to those without. As the presentation of hip OA and FAI are similar, it is possible that those with symptomatic FAI may exhibit the same GMed activation patterns during stair ascent as those with hip OA. However, no studies to date have reported muscle activation patterns in people with FAI during any dynamic task.

1.7 Thesis Rationale, Objectives and Hypotheses

1.7.1 Thesis Rationale

It is widely accepted that those with FAI present with groin and back pain that is often exacerbated by excessive hip flexion, adduction and internal rotation and can cause avoidance of physical activity (Ganz et al., 2003). Therefore, it is very important to understand the function of the lower extremity during common activities in those with FAI, especially activities which cannot be avoided. This will better allow clinicians to rehabilitate those with symptomatic FAI.

Level walking and stair climbing are two of the most common activities of daily living. Therefore it is very important to understand the function of the lower extremity in those with symptomatic FAI during these activities. Despite the kinematic and kinetic differences observed at the hip between those with and without symptomatic FAI during level walking, conflicting results within the literature suggest that level walking may not be sufficiently challenging to properly assess hip functional limitations in the presence of symptomatic FAI (Brisson et al., 2013; Diamond et al., 2016; Hunt et al., 2013; Kennedy et al., 2009; Rylander et al., 2013). Thus a more demanding task such as stair climbing might be needed to challenge the neuromuscular system in order to accurately quantify the function of the lower extremity. Previous research conducted by Rylander et al. (2013) looking at stair climbing observed that those with symptomatic FAI exhibited similar kinematic alterations compared to level walking. However, no measures of joint moments were reported. Unlike level walking, the greater ROM about the hip required to perform stair climbing may provide more consistent findings about the function of the lower extremity in those with symptomatic FAI. Additionally, it was observed that one year post corrective surgery for symptomatic FAI, hip kinematics returned to normal during level walking but not for stair climbing or deep squatting (Lamontagne et al., 2011; Rylander et al.,

2013). This suggests that hip function may be restored to normal during “simpler” tasks such as level walking, but not for the more demanding task of stair climbing. Therefore measuring hip kinematics and joint loading during the more demanding task of stair climbing may allow for the identification of more consistent gait alterations that will allow researchers to accurately assess the function of the lower extremity in those with FAI.

Additionally, the continued abnormal hip motion post-corrective surgery indicates that hip function is not restored in tasks that require greater hip ROM and biomechanical demands. This suggests that reduced hip ROM may not solely be a result of the actual boney impingement, and that altered muscle activation may play a role in the reduced ROM during tasks requiring greater hip ROM. Unfortunately, no studies to-date have reported muscle activity during any dynamic movement in people with symptomatic FAI. However, previous research on those with hip OA observed an increase in GMed muscle activity during stair climbing when compared to healthy controls (Dwyer et al., 2013; Sims et al., 2002). As those with symptomatic FAI have been shown to elicit similar muscle weakness and gait patterns as those with hip OA (Rasch et al., 2005; Zeni et al., 2015), it can be hypothesized that similar muscle activation patterns during stair climbing when compared to healthy controls. Additionally, there is a lack of reporting of measures about the trunk, knee or ankle joints in an attempt to identify potential compensatory measures.

Therefore, while those with FAI report pain and loss of physical activity, this will be the first study to quantify joint kinematics and moments about hip, trunk, knee and ankle during stair climbing. Furthermore, this will be the first study to assess muscle activation patterns in those with FAI during a dynamic task. The underlying cause of symptomatic FAI is not fully understood, however, having a better understanding of the kinematics, moments, and muscle

activation about the three lower limb joints will better allow clinicians to rehabilitate those with symptomatic FAI.

1.7.2 Objectives

The objective of this study was to perform a complete biomechanical analysis of this patient population examining kinematic, joint moments and muscle activation of all three lower limb joints on the study limb during the dynamic task of stair climbing, specifically:

- 1) To compare ankle, knee, hip and trunk kinematics during stair in those with and without symptomatic FAI.
- 2) To compare ankle, knee and hip moments during stair climbing in those with and without symptomatic FAI.
- 3) To compare lower limb muscle activation patterns during stair climbing between those with and without symptomatic FAI.

1.7.3 Hypotheses

- 1) During stair climbing, those with symptomatic FAI will exhibit altered kinematics in the sagittal plane on the study limb when compared to those without symptomatic FAI.
 - Decreased hip joint peak angles and excursion.
 - Increased ankle, knee and trunk peak angles and excursion.
- 2) During stair climbing those with symptomatic FAI will exhibit altered joint moments in the sagittal plane on the study limb when compared to those without symptomatic FAI.
 - Decreased hip joint loading (as quantified by peak external joint moments).
 - Increased ankle and knee loading.
- 3) During stair climbing those with symptomatic FAI will exhibit altered muscle activity on the study limb when compared to those without symptomatic FAI.

- Greater integrated and peak GMed electrical activity as a percentage of their maximum.
- Greater integrated and peak muscle activation about the knee and ankle joint as a potential compensatory mechanism for the hip.

Chapter 2: Introduction

FAI is a pathomechanical process, believed to be the result of abnormal contact between the proximal femur and the acetabulum, that has been linked to labral tears and early development of hip OA in young and active adults (Ganz et al., 2003). Typically, those with symptomatic FAI present with groin and back pain that is often exacerbated during movements requiring excessive hip flexion, adduction and internal rotation in common everyday activities such as: prolonged sitting or walking, deep squats, stair climbing and other athletic activities (Crawford and Villar, 2005; Leunig et al., 2005). This pain can cause avoidance of physical activity. Therefore, it is important to understand the biomechanical consequences of symptomatic FAI during common activities, especially those which cannot be avoided.

Previous research reporting hip kinematics during level walking in those with symptomatic FAI reported small differences in all three planes of movement (Brisson et al., 2013; Diamond et al., 2016; Hunt et al., 2013; Kennedy et al., 2009; Rylander et al., 2013). In addition, only one study has reported altered joint moments about the hip; observing that those with FAI exhibit reduced peak external hip flexion and external rotation moments (Hunt et al., 2013). The overall similarity in walking biomechanics between those with and without symptomatic FAI suggests that level walking may not be a challenging enough task to accurately differentiate the biomechanics of the hip in the presence of symptomatic FAI.

In contrast, stair climbing is an important daily activity, especially for younger, more active people and has been shown to require greater ROM and joint loading of the hip compared to level walking in healthy individuals (Nadeau et al., 2003). Therefore, this task may provide greater sensitivity in detecting a clinical difference in people with FAI than compared to level walking. However, only one study has been published reporting hip biomechanics during stair

climbing in people with FAI. Rylander et al. (2013) observed less peak extension and internal rotation in those with symptomatic FAI compared to healthy participants during stair climbing. Additionally, it was observed that one year post corrective surgery, hip joint kinematics returned to “normal” during level walking, but not for stair climbing. This suggests that hip function is returned to normal during “simpler” tasks such as level walking, but not in tasks requiring greater hip demands such as stair climbing. This continued abnormal hip motion during stair climbing post-corrective surgery suggests that altered hip biomechanics may not solely be a result of the actual boney impingement, and that altered neuromuscular patterns may play a role in the reduced motion during tasks requiring greater hip ROM. However, no studies to-date have reported muscle activity during any dynamic movement in people with FAI, including stair climbing.

Previous research on isometric muscle force production has observed that those with FAI exhibit a significant decrease in hip flexion, adduction, abduction and external rotation muscle force and reduced TFL activity during hip flexion when compared to healthy controls (Casartelli et al., 2011; Diamond et al., 2015). This indicates that those with FAI exhibit muscle weakness and dysfunction that may impact movement biomechanics. Unfortunately, no research on muscle activation has been conducted during stair climbing, let alone any dynamic task, in those with FAI. However, previous research on those with hip OA observed greater GMed muscle activity when compared to healthy controls during a step up task (Dwyer et al., 2013; Sims et al., 2002). As FAI has been shown to elicit similar hip kinematics and strength values as in those with hip OA as reported by Rasch et al. (2005) and Zeni et al. (2015), this may suggest that those with FAI may exhibit the similar muscle activation differences during stair climbing as those with hip OA. To best inform clinical management of FAI, further research is required to elucidate the effects of FAI on challenging tasks such as stair climbing.

Therefore, the purpose of the present study was to assess kinematic, kinetic and muscle activation patterns of the hip and lower limb during stair climbing in those with symptomatic FAI compared to an asymptomatic healthy population. It was hypothesized that those with symptomatic FAI would exhibit reduced hip joint motion and larger external hip joint moments in the sagittal plane. It was also hypothesized that those with FAI would exhibit increased activation of the GMed muscle. Furthermore, it was hypothesized that those with FAI would exhibit significant alterations in trunk, knee and ankle kinematics, knee and ankle kinetics and muscle activation as a compensatory mechanism for the observed changes at the hip.

Chapter 3: Methods

3.1 Study Design

This was a cross-sectional, comparative study examining kinematic, kinetic and electromyographic outcomes of the hip, knee and ankle and trunk kinematics during stair climbing in individuals with symptomatic FAI compared to age- and sex-matched healthy individuals. The study was approved by the University of British Columbia Clinical Research Ethics Board.

3.2 Study Participants

Twenty individuals scheduled to undergo arthroscopic debridement surgery for confirmed symptomatic FAI (diagnosed through imaging) were identified by the same orthopaedic surgeon (M. Gilbert) at his orthopaedic injuries clinic at UBC Hospital. Twenty age- and sex-matched (age \pm 3 years) healthy individuals were recruited from a convenience sample within the university community to act as controls. These participants were categorized as “healthy” based on a self-reported absence of hip and back pain.

Inclusion Criteria for those with symptomatic FAI and healthy controls

- i)* Between the ages of 16-40 years
- ii)* Diagnosed with FAI by orthopedic surgeon and on wait list for surgery; OR are healthy with no history of lower leg injury (controls)

Exclusion Criteria for those with FAI and healthy controls

- i) A history of lower body injuries or conditions that impair the measurement of walking or hip strength (other than FAI for those in the FAI group)
- ii) A history of neurological injury that affects walking
- iii) Diagnosed hip osteoarthritis, defined as tonnis grade greater than 1
- iv) Planned or previous lower limb joint replacement

3.3 Instrumentation

Lower extremity kinematic, kinetic and electromyographic data were collected from participants as they performed stair climbing trials. Selection of study limb and non-study limb was done using the following criteria: in the case of both unilateral and bilateral FAI participants the study-limb was defined as the limb undergoing surgery, while selection of the study limb for healthy participants was randomly selected.

Three-dimensional kinematics were collected at 100 Hertz (Hz) using a ten-camera motion capture system (Motion Analysis Corporation, Santa Rosa, CA) with forty-two passive reflective markers affixed to the skin over anatomical landmarks following a modified Helen Hayes marker set similar to that used by Rylander et al. (2013). Markers were placed bilaterally on the distal aspects of the second metatarsal, posterior aspect of the calcanei, medial and lateral malleoli, medial and lateral femoral epicondyles, anterior shank and thigh, the anterior superior iliac spines, and the L5-S1 junction. Semi-rigid plastic plates with four tracking markers each were secured to the lateral shanks and thighs. Markers were also fixed bilaterally to the acromion process, olecranon processes, radial styloid and the right scapula (Figure 4). Ground reaction force data were collected from two floor mounted force platforms (AMTI, Watertown, MA)

sampling at 2000Hz. A set of stairs were embedded into the force plates, consisting of three steps that were each 29cm deep by 60cm wide by 18cm high (Figure 5).

Muscle activity of the lower limb was measured using nine wireless bipolar surface EMG (Delsys, Natick, MA) electrodes at a sampling rate of 2000Hz. The nine surface EMG electrodes were placed in line with the muscle fibers on the following muscles: GMed, RF, vastus medialis (VM), vastus lateralis (VL), tibialis anterior (TA), semitendinosus (ST), biceps femoris (BF) and the medial (MG) and lateral (LG) gastrocnemius (Figure 4). Placement of the electrodes was consistent with international guidelines (www.seniam.org) and were validated by muscle palpitation during isometric contractions. Prior to electrode placement, the skin was shaved and cleaned using a 70% alcohol swab.

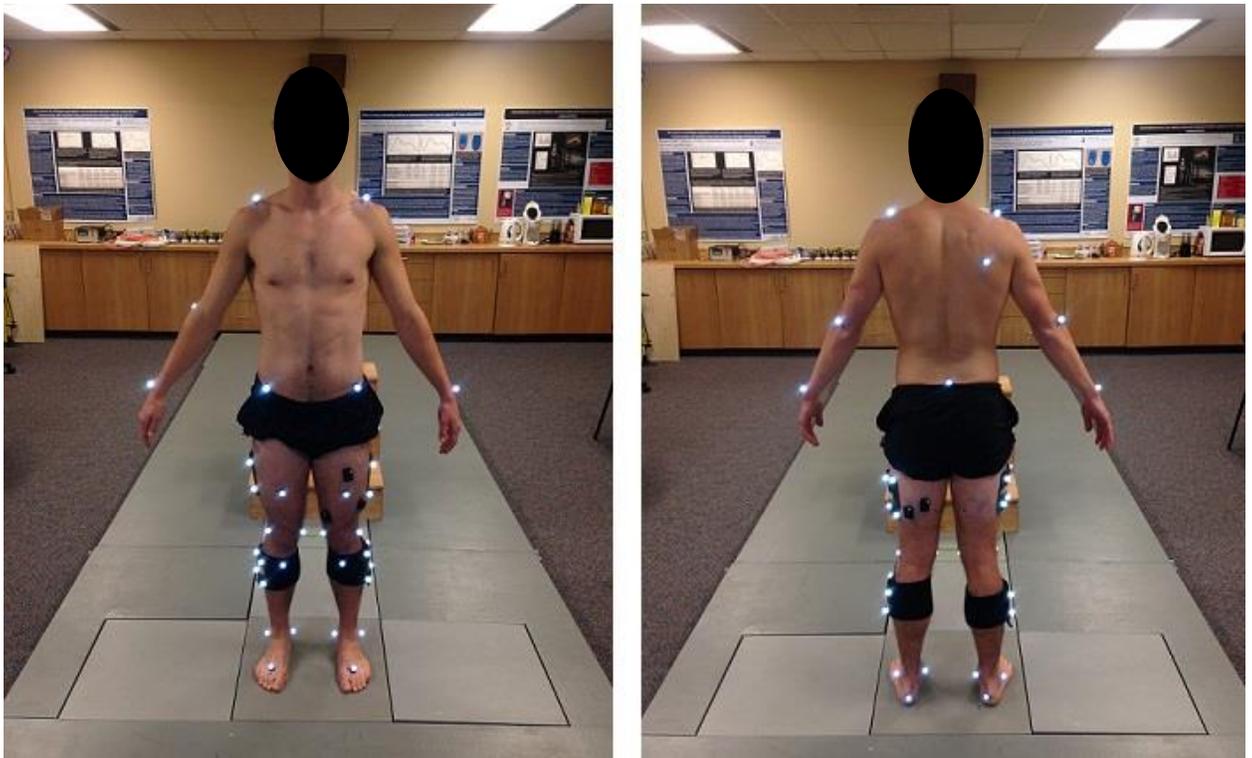


Figure 4. Marker set used for this study; 42 reflective markers following the modified Helen Hayes marker set and EMG electrodes.



Figure 5. Instrumented staircase; Photo of the instrumented staircase.

3.4 Procedures/Protocol

All participants completed and signed an informed consent form approved by the university's committee for research ethics. Participants then completed the Modified Harris Hip Score (MHHS), International Hip Outcome Tool (IHOT-12) and Hip disability and Osteoarthritis Outcome Score (HOOS) questionnaires to assess self-reported hip pain and function during everyday activities. Additionally, self-reported hip pain and difficulty during stair ascent and overall average pain in the past week were self-assessed by an 11 point numerical rating scale (NRS) (terminal descriptors of 0 = "no pain", and 10 = "worst pain possible"). These self-report questionnaires are valid and reliable instruments that have been used in many studies for people with hip disabilities (Nilsson et al., 2003). Identified "healthy control" participants who reported any hip pain during the past week on the NRS were deemed to not meet inclusion criteria for that particular group, and were immediately excluded from further testing.

Participants were then outfitted with nine wireless bipolar surface EMG electrodes on the study limb as detailed above (section 3.3). Once the electrodes were placed on the muscles, participants were asked to perform six isometric MVC exercises; 1) hip abduction in prone, 2) knee flexion in prone with knee at 90° of flexion, 3) knee extension while sitting with hip and knee at 90° of flexion, 4) ankle dorsiflexion while sitting with hip and knee at 90° of flexion and ankle touching the floor, 5) ankle plantar flexion while standing on one leg and 6) hip flexion while supine with hip flexed to 50° (Figure 6). Resistance was provided by the researcher. MVC exercises were used to elicit maximal voluntary electromyographic amplitude of the participants' muscles. Each MVC was performed three times, with the first time being a practice/warm up and the subsequent two at maximum effort. Participants were asked to perform the tasks with maximum effort to ensure that the captured MVC electrical activity was accurate. A countdown of "ready", "set", "go" was given, followed by the participants exhibiting maximal effort during each movement for three seconds. Following a three second countdown the researcher told the participants to "relax". A thirty second rest period was given between attempts to ensure the muscle was fully recovered for the next trial. After all MVCs were completed, a resting trial was performed in which the subject laid down on a treatment bed fully relaxed for eight seconds while resting muscle bias data were collected. This allowed the researcher to capture the underlying muscle activity when no movement was performed.

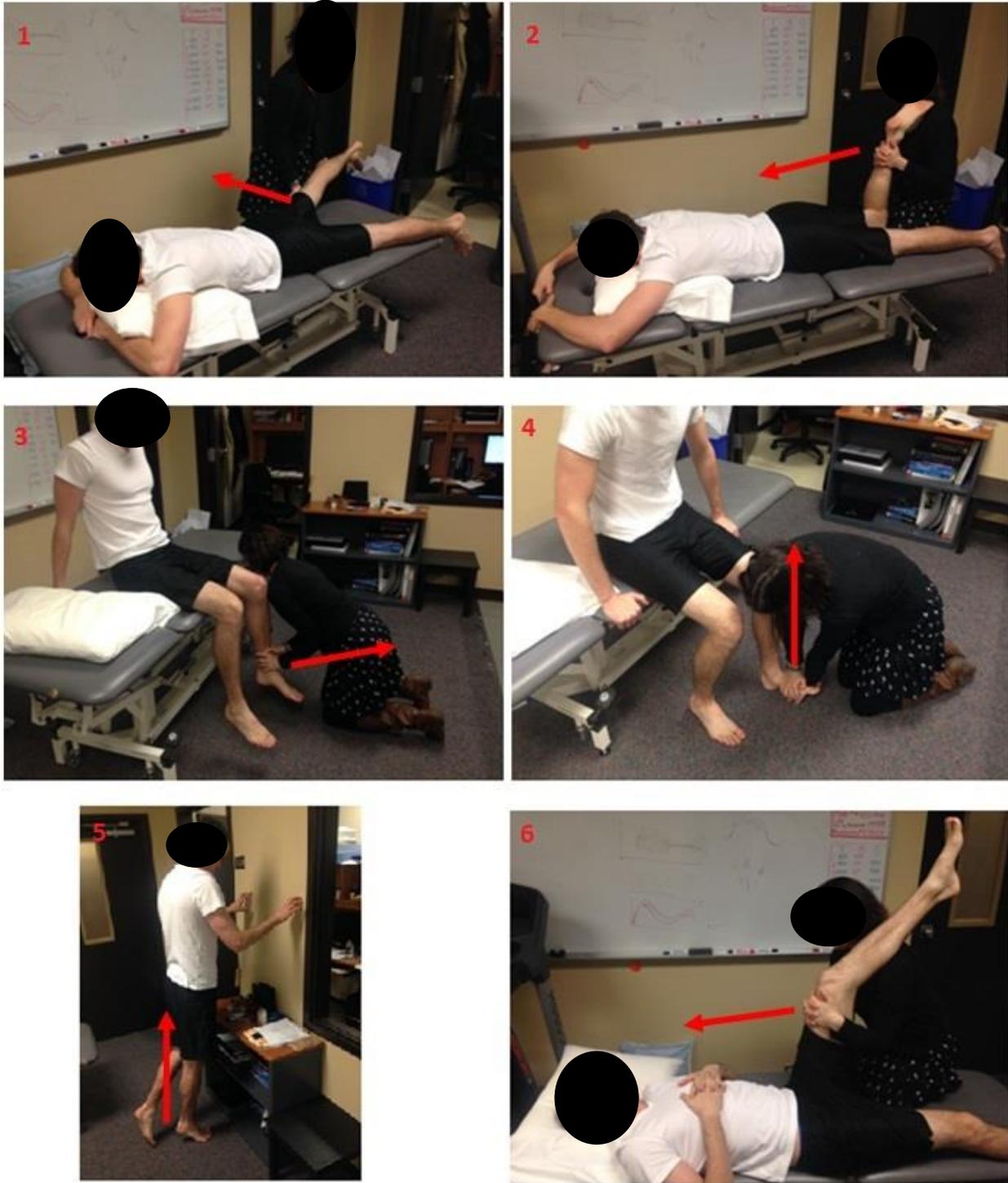


Figure 6. Isometric MVC exercises; The six MVC exercises: 1) hip abduction, 2) knee flexion, 3) knee extension, 4) ankle dorsiflexion, 5) ankle plantar flexion and 6) hip flexion.

Participants were then fitted with forty-two retro-reflective markers as detailed above (section 3.3), and anthropometric data including height, foot width and foot length were recorded. A static trial was recorded to determine joint centres and marker orientations and to calculate body mass. Upon completion of the static trial, the medial femoral epicondyle and medial malleoli reflective markers were removed.

In order to ensure participants performed each stair climbing trial from the same starting position every time, they were asked to ascend the first step repeatedly until they had identified a comfortable starting position. The researcher then marked the location with tape on the floor and informed the participants to use this as a starting position for all subsequent trials (Figure 5). Participants were then allowed to perform practice trials in order to become comfortable with the stair setup. Participants then completed five stair climbing trials, leading with the study limb, barefoot and at a self-selected pace while kinetic, kinematic and electromyography data were simultaneously collected.

3.5 Data Analysis

Kinematic, kinetic and EMG data were normalized to stance phase and averaged across the five stair climbing trials for each participant. Stance phase was defined as foot contact on the first step to toe off of the first step, with a force threshold of 20 Newtons being applied to ensure proper gait events. Ground reaction force profiles were visually inspected for each trial in order to ensure that foot contact and toe off occurred at the correct data point. Stair ascent speed was calculated as the average resultant speed of the body between subsequent foot contacts of the study limb (expressed in m/s).

3.5.1 Kinematic Data

Three-dimensional marker trajectories were filtered using a fourth order lowpass 6Hz Butterworth filter in Cortex 5.3 (Motion Analysis Corporation, Santa Rosa, CA) and exported to Visual 3D (C-Motion Inc, Rockville, MD) for computation of trunk, hip, knee and ankle joint angles according to the joint coordinate system. The foot segment was defined by the medial and lateral malleoli markers and the foot width measured through anthropometrics. The shank segment was defined by two proximal markers (medial and lateral femoral epicondyles) and two distal markers (medial and lateral malleoli). The thigh segment was defined by the medial and lateral femoral condyles, the anterior superior iliac spine, and the hip joint centre (Bell et al., 1989). The trunk segment was defined by the right and left acromion markers and virtual markers on the right and left iliac crests. The virtual iliac crest markers were created using landmarks estimated using the Terry Database (Kepple et al., 1997). The static position of the thigh and shank rigid plates with respect to the segment definition markers were calculated and used to track movements during the stair climbing trials. The origin for the foot coordinate system was located at the midpoint between the two malleoli markers (the anterior-posterior axis was oriented to the midpoint of the measured foot width; the medial-lateral axis was oriented from the medial to lateral malleoli markers; and the vertical axis was orthogonal to the other two axes). The origin for the shank coordinate system was located at the midpoint between the femoral epicondyle markers (vertical axis oriented to the midpoint of the lateral and medial malleoli; anterior-posterior axis orthogonal to the plane formed by the four segment definition markers; and medial-lateral axis orthogonal to the other two axes). The origin for the thigh coordinate system was located at the hip centre (vertical axis oriented to the midpoint of the lateral and medial femoral condyles; anterior-posterior axis orthogonal to the plane formed by the four segment definition markers; and medial-lateral axis orthogonal to the other two axes).

The origin of the trunk coordinate system was located at the midpoint between the virtual iliac crest markers (vertical axis oriented to the midpoint of right and left virtual iliac crest markers, anterior-posterior axis orthogonal to the plane formed by the four segment definition markers, and medial-lateral axis orthogonal to the other two axes). Joint angles were calculated for the distal segment relative to the proximal segment using a Cardan XYZ sequence of rotations with six degrees of freedom (Grood and Suntay, 1983).

Peak joint angles (°) and excursions (maximum minus minimum) in the sagittal and frontal planes were extracted for the hip. Additionally, peak angles and excursions in the sagittal plane for the knee and ankle were calculated. Furthermore, peak trunk forward flexion in the sagittal plane was calculated. Finally, ensemble average curves were plotted for the hip in the sagittal and frontal planes, and the trunk, knee and ankle in the sagittal plane.

3.5.2 Kinetic Data

Ground reaction forces and external joint moments were filtered using a second order 6Hz lowpass Butterworth filter within Visual 3D. An inverse dynamics approach was used to calculate external joint moments about the hip, knee and ankle, and all moments were normalized to body mass (Nm/kg). Peak external joint moments were identified for the hip in the sagittal and frontal planes, and for the knee and ankle in the sagittal plane. Additionally, ensemble average curves of joint moments were plotted for the hip in the sagittal and frontal planes, and the knee and ankle in the sagittal plane.

3.5.3 EMG Data

EMG data were processed using a custom MATLAB program (Mathworks Inc., Natick, MA). First, the average electrical activity for each muscle measured during the resting trial was removed from the EMG signal for each of the stair climbing and MVC trials to ensure all data

measured were a result of muscle activation. Then, all MVC trial and stair climbing EMG data were converted to volts and the known 48 millisecond time delay was removed. Next all data were bandpass filtered using a second order Butterworth filter from 20Hz-500Hz. Then, frequency plots of each muscle were graphed for all MVC and stair climbing trials to determine if noise spikes were present. If noise was present in a muscle during a trial, the trial EMG data for that muscle were discarded. Finally EMG data were full wave rectified and smoothed using a fourth order Butterworth filter at 25Hz.

To calculate peak signal amplitude for each muscle, the maximum electromyographic amplitude produced during all MVC tasks was determined using a 100ms moving average window. This was done by averaging and plotting 100ms subsets of the trial. After the average value from the first 100ms (data points 1-100) subset had been calculated, the subset was then modified by shifting forward one data point and excluding the first data point from the previous subset (data points 2-101), and the average value from this subset was calculated. Following completion of the moving average window, a plot of the calculated subset averages was made and the peak value of this plot was identified for each muscle and MVC task. This peak value was the maximum electrical activity in which the surface EMG was capable of capturing for each muscle. The peak amplitude for each muscle (regardless of task) during the MVC trials was visually checked in order to ensure that the maximum electrical activity was a result of muscle contraction and not due to noise.

Electromyographic amplitude of each muscle during the stair climbing trials was amplitude-normalized to the peak amplitude for the respective muscle from the MVC trials. Integrated EMG of each muscle was then calculated as the area under the curve of the amplitude-normalized signal (%MVC*s). Next, the amplitude-normalized data were time-normalized by expressing the curve as a percentage of stance phase. Finally, the peak activation amplitude for

each muscle during the stair climbing trials was identified (%MVC) and expressed as a percentage of stance. Finally, time- and amplitude-normalized ensemble average curves during stair climbing were plotted for each muscle.

3.6 Statistical Analysis

3.6.1 Sample Size

A minimum sample size of 20 participants was estimated based on previous research by Hunt et al. (2013) comparing level walking in those with FAI and those without ($d=0.90$, $\alpha=0.05$, $\beta=0.8$). To put this into context, the only published study examining stair climbing in people with FAI found an effect size of 0.85 in 17 participants per group with respect to the maximum hip extension angle (Rylander et al., 2013). Therefore, 20 participants per group was targeted to provide adequate statistical power for hypothesis testing.

3.6.2 Statistical Analysis

Descriptive statistics including means, standard deviations and ranges were calculated for both the FAI and control groups on the study-limb only. All data were explored for normality using the Kolmogorov-Smirnov test and for unequal variances using the Levene's test. Outliers were removed if they fell outside 1.5 times the interquartile range above and below the 75th and 25th quartile values for each variable (Tukey, 1977). Specifically, if the mean value across the five trials for a given participant was deemed to be an outlier based on this criterion, the data for that participant was removed for that variable only. Between-group comparisons for all biomechanical outcome variables, summarized in Table 1, were performed using two-tailed independent samples t-tests ($\alpha = 0.05$) for normally distributed data, and the Mann-Whitney U test was used in instances of non-normally distributed data ($\alpha = 0.05$). Independent samples t-

tests were run with both the outliers in and outliers removed. Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS v. 22; IBM Corp., Armonk, NY).

Table 1. Biomechanical variables measured; Kinematic, kinetic and electromyographic variables measured during stair climbing trials

	Hip	Knee	Ankle	Trunk
Kinematics	Peak flexion and extension angle (°) Peak abduction and adduction angle (°) Sagittal plane excursion (°) Frontal plane excursion (°)	Peak flexion and extension angle (°) Sagittal plane excursion (°)	Peak plantar flexion and dorsiflexion angle (°) Sagittal plane excursion (°)	Peak forward flexion angle (°)
Kinetics	Peak flexion and extension moment (Nm/kg) Peak abduction and adduction moment (Nm/kg)	Peak flexion and extension moment (Nm/kg)	Peak Plantar flexion and dorsiflexion moment (Nm/kg)	
Electromyography	Peak (%MVC) and integrated (%MVC*s) GMed EMG Peak (%MVC) and integrated (%MVC*s) RF EMG Peak (%MVC) and integrated (%MVC*s) BF EMG Peak (%MVC) and integrated (%MVC*s) ST EMG	Peak (%MVC) and integrated (%MVC*s) RF EMG Peak (%MVC) and integrated (%MVC*s) VM EMG Peak (%MVC) and integrated (%MVC*s) VL EMG Peak (%MVC) and integrated (%MVC*s) BF EMG Peak (%MVC) and integrated (%MVC*s) ST EMG Peak (%MVC) and integrated (%MVC*s) MG EMG Peak (%MVC) and integrated (%MVC*s) LG EMG	Peak (%MVC) and integrated (%MVC*s) TA EMG Peak (%MVC) and integrated (%MVC*s) MG EMG Peak (%MVC) and integrated (%MVC*s) LG EMG	

Chapter 4: Results

4.1 Participant Demographics

Participant demographic and clinical data is reported in Table 2. The FAI and control groups were similar in age ($p=0.78$), sex ($p=1.0$), height ($p=0.60$) and BMI ($p=0.11$), indicating that our intended matching was successful. Those in the FAI group reported symptom durations for a mean (SD) of 50.9 (38.3) months prior to testing. Those with FAI were moderately impaired based on hip questionnaire scores (Table 2). Those with FAI reported lower scores on the MHHS (70.5 ± 9.9), HOOS (60.1 ± 13.5) and IHOT-12 (3.8 ± 2.1) compared to the controls (100 ± 0.0 , 99.8 ± 0.5 and 9.9 ± 0.03), where a lower score indicates greater impairment. Additionally, those with FAI reported greater scores on the NRS pain scale (4.4 ± 1.9) compared to the controls (0 ± 0.0), where a higher score is indicative of greater impairment. Though those with FAI were scheduled to undergo arthroscopic surgery on their study limb, 10 of the 20 patients (50%) reported bilateral symptoms.

Table 2. Demographic, questionnaire and radiographic data; Mean (SD) data for FAI and control groups.

Variable	FAI Group (n = 20)	Control Group (n=20)
Sex (male:female)	15:5	15:5
Age (years)	27.6 (5.8)	27.1 (5.0)
Height (m)	1.77 (0.08)	1.76 (0.07)
Body mass (kg)	76.6 (12.4)	70.6 (10.9)
BMI (kg/m ²)	24.4 (3.7)	22.8 (3.7)
NRS (0-10)	4.4 (1.9)	0 (0.0)
MHHS (0-100)	70.5 (9.9)	100 (0.0)
HOOS (0-100)	60.1 (13.5)	99.8 (0.5)
IHOT-12 (0-10)	3.8 (2.1)	9.9 (0.03)
Impingement Type		
Cam	3	-
Pincer	1	-
Mixed	13	-
Tonnis Grade		
0	13	-
1	4	-
2	0	-
3	0	-
Alpha Angle (°)	68.8 (9.9)	-
Centre Edge Angle (°)	38.7 (5.8)	-
Tonnis Angle (°)	7.9 (3.2)	-

NRS, numerical rating scale for pain in the last week. A higher value indicates more pain. MHHS, modified harris hip score. A lower value indicates greater impairment. HOOS, hip disability and osteoarthritis outcome score. A lower value indicates greater impairment. IHOT-12, international hip outcome tool. A lower value indicates greater impairment.

4.2 Outcome Data

Those with FAI ascended the stairs at a significantly slower stair ascent speed (0.41 ± 0.07 m/s) compared to the controls (0.46 ± 0.05 m/s; $p = 0.03$). Given that the stair height distance was fixed, this would suggest that individuals with FAI ascended the stairs at a slower speed. Individual outlying data points that were removed are summarized in Table 3.

Table 3. Participant outliers; Summary of individual participant data points that were removed for each outcome measure.

Variable	Group	Participant #
Peak Trunk Forward Flexion Angle	Control	1, 10, 30
Peak Hip Flexion Angle	FAI	29
Hip Sagittal Excursion	Control	24
Peak Hip Abduction Angle	Control	40
Peak Knee Flexion Angle	Control	1, 18, 27
Peak Knee Extension Angle	FAI	23,34,35
	Control	38
Knee Sagittal Excursion	Control	18
Peak Ankle Dorsiflexion Angle	Control	2, 22
Peak Ankle Plantar Flexion Angle	FAI	23,26
Peak Hip Flexion Moment	Control	18
Peak Hip Adduction Moment	FAI	15
Peak Knee Flexion Moment	Control	18
Peak Knee Extension Moment	FAI	26
	Control	3, 18
Peak Dorsiflexion Moment	FAI	26
GM Peak	Control	1, 40
GM Integrated	Control	40
MH Peak	Control	27,40
MH Integrated	Control	27, 36, 40
LG Peak	FAI	11,13
MG Peak	FAI	13
	Control	31
MG Integrated	FAI	13
	Control	3
RF Peak	Control	2
VL Peak	FAI	13
VL Integrated	FAI	13
VM Integrated	FAI	13
TA Integrated	FAI	32

4.2.1 Kinematic Data

Selected peak kinematic data with the outliers removed is summarized in Table 4. Those with FAI exhibited greater peak trunk forward flexion angle ($15.6^{\circ} \pm 4.9^{\circ}$ to $12.0^{\circ} \pm 1.5^{\circ}$, $p = 0.01$) when compared to the controls during stair climbing. No significant kinematic differences

existed at the hip, knee or ankle joints in any plane of movement during stair climbing trials in those with FAI compared to the controls. Group ensemble average curves are present in Figure 7, Figure 8 and Figure 9. Results from the independent samples t-test prior to the removal of the outliers yielded the same statistical results as when the outliers were removed with respect to the kinematic variables.

4.2.2 Kinetic Data

External joint moments with the outliers removed were calculated for the hip, knee and ankle and are summarized in Table 5. Those with FAI demonstrated a greater peak external hip flexion moment (0.97 ± 0.36 Nm/kg) when compared to the controls (0.70 ± 0.19 Nm/kg) during stair climbing ($p=0.01$). Additionally those with FAI exhibited a significantly reduced peak external knee flexion moment (1.24 ± 0.17 Nm/kg) during stair climbing trials when compared to the controls (1.38 ± 0.14 Nm/kg, $p =0.01$). No differences were observed with respect to any other kinetic variables. Group ensemble average curves are shown in Figure 7 and Figure 8.

Prior to the removal of the outliers summarized in Table 3, an independent samples t-test was run. The results demonstrated that all kinetic variables that were significant when the outliers were removed were significant when the outliers remained within the dataset.

Table 4. Hip, knee, ankle and trunk kinematic data; Mean (SD) of lower limb kinematics (degrees) for FAI and control groups, Mean Difference (95% CI) and p-values for group effects as determined by an independent samples t-test.

Joint	Direction	FAI Mean (SD)	Control Mean (SD)	Mean Difference (95% CI)	p-value
Hip	Flexion	60.6 (5.2)	59.8 (4.9)	0.8 (-2.5, 4.1)	0.63
	Extension	-7.1 (8.2)	-6.1 (5.2)	-1.0 (-3.4, 5.5)	0.63
	Sagittal Plane Excursion	52.5 (4.9)	53.1 (3.2)	-0.6 (-3.3, 2.1)	0.67
	Adduction	7.8 (4.1)	10.6 (5.5)	-2.8 (-6.0, 0.3)	0.08
	Abduction	8.5 (4.5)	7.7 (4.1)	0.8 (-3.6, 2.0)	0.55
	Frontal Plane Excursion	16.3 (7.0)	18.8 (7.3)	-2.5 (-7.1, 2.1)	0.28
Knee	Flexion	68.1 (3.4)	68.2 (2.4)	-0.1 (-2.1, 1.9)	0.91
	Extension	-8.5 (3.1)	-8.8 (2.8)	0.2 (-1.8, 2.2)	0.81
	Sagittal Plane Excursion	60.4 (6.7)	60.9 (3.6)	0.5 (-4.0, 3.0)	0.77
Ankle	Dorsiflexion	19.6 (4.2)	20.3 (2.6)	-0.7 (-3.0, 1.7)	0.57
	Plantar Flexion	13.3 (4.0)	12.4 (4.1)	0.9 (-3.6, 1.8)	0.51
	Sagittal Plane Excursion	32.7 (5.2)	33.6 (3.2)	-0.9 (-3.6, 1.9)	0.52
Trunk	Forward Flexion	15.6 (4.9)	12.0 (1.5)	3.6 (1.2, 6)	0.01*

* indicates statistically significant differences $p < 0.05$. A negative mean value indicates that the joint did not truly enter into that direction.

Table 5. Hip, knee and ankle kinetic data; Mean (SD) of lower limb kinetics (Nm/kg) for FAI and control groups, Mean Difference (95% CI) and p-values for group effects as determined by an independent samples t-test.

Joint	Direction	FAI Mean (SD)	Control Mean (SD)	Mean Difference (95% CI)	p-value
Hip	Flexion	0.97 (0.36)	0.70 (0.19)	0.27 (0.08, 0.46)	0.01*
	Extension	0.15 (0.07)	0.14 (0.09)	0.02 (-0.07, 0.03)	0.49
	Adduction	0.79 (0.12)	0.85 (0.12)	-0.06 (-0.14, 0.02)	0.13
	Abduction	-	-	-	-
Knee	Flexion	1.24 (0.17)	1.38 (0.14)	-0.14 (-0.24, -0.04)	0.01*
	Extension	0.41 (0.21)	0.35 (0.11)	-0.06 (-0.17, 0.05)	0.29
Ankle	Dorsiflexion	1.27 (0.21)	1.37 (0.19)	-0.11, (-0.24, 0.03)	0.11
	Plantar Flexion	-	-	-	-

* indicates statistically significant differences p <0.05.

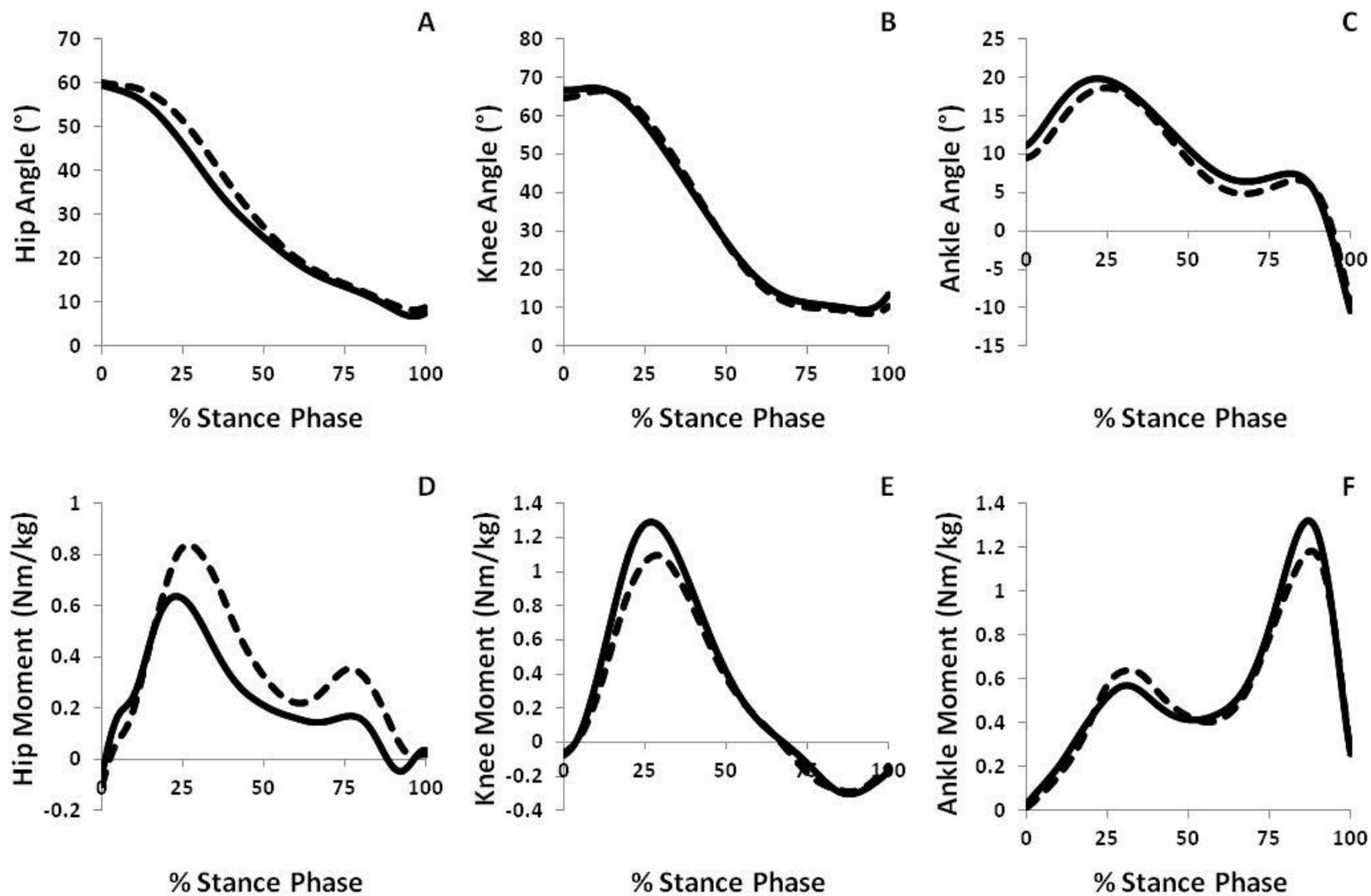


Figure 7. Sagittal plane kinematic and kinetic (external joint moments) ensemble averages about the hip, knee and ankle; A, B, D and E, positive values indicate flexion and negative values indicate extension. C and F, positive values indicate dorsiflexion and negative values indicate plantar flexion. The solid line represents the control group and the dashed line represents the FAI group.

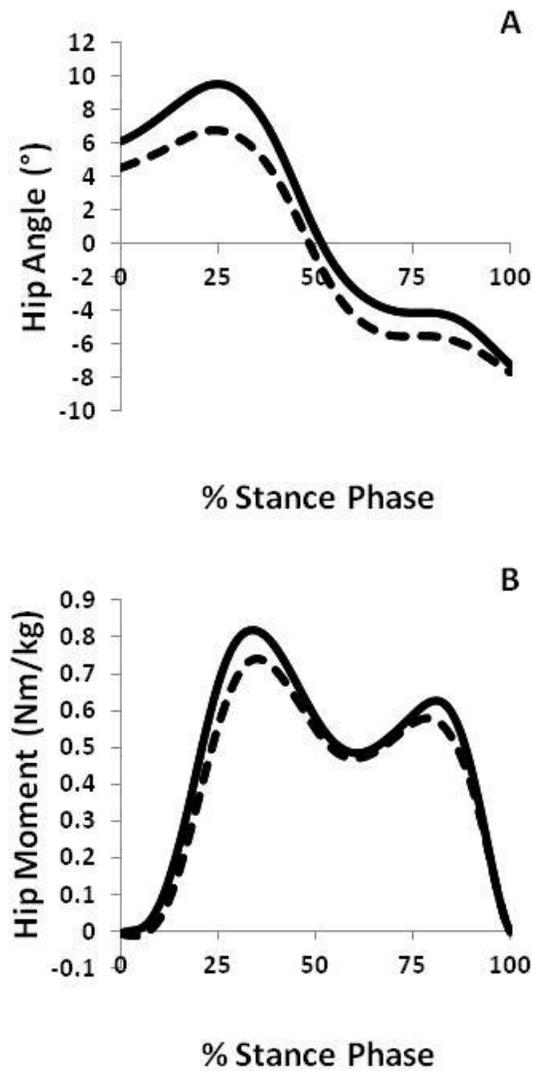


Figure 8. Frontal plane hip kinematic and kinetic ensemble averages; A and B, a positive value indicates adduction and a negative value indicated abduction. The solid line represents the control group and the dashed line represents the FAI group.

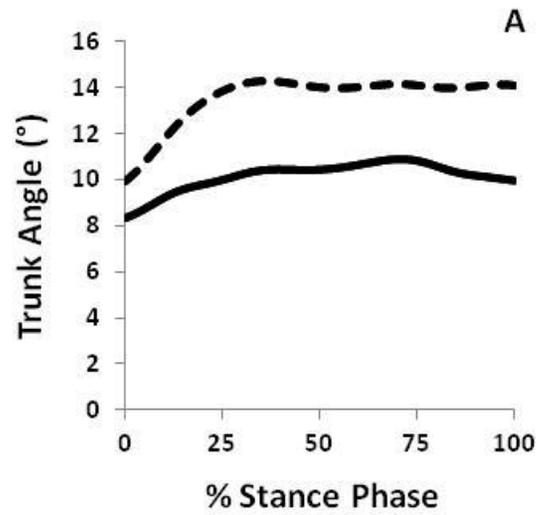


Figure 9. Sagittal plane trunk kinematic ensemble averages; A, a positive value indicates forward flexion of the trunk. The solid line represents the control group and the dashed line represents the FAI group.

4.2.3 EMG Data

Peak and integrated EMG were calculated for each of the nine muscles and are summarized in Table 6. A significant difference existed with respect to peak LG activation, in that those with FAI exhibited significantly lower LG activation (43.5 ± 12.1 %MVC) compared to the controls (52.9 ± 14.1 %MVC, $p=0.04$). Group ensemble average curves during stair climbing are present in Figure 10 and Figure 11. Prior to the removal of the outliers, an independent samples t-test indicated that all EMG variables were not statistically significant. Therefore, a difference in the LG activation became statistically significant once outlying data from two FAI participants were removed (FAI group = 48.7 ± 19.5 %MVC; control group = 52.9 ± 14.1 %MVC; mean difference in LG was, -4.1, 95% CI: -15.1, 6.9; $p=0.45$ with no outliers removed prior to analysis).

Table 6. EMG data; Mean (SD) of peak (%MVC) and integrated (%MVC*s) lower limb EMG for FAI and control groups, Mean Difference (95% CI) and p-values for group effects as determined by an independent samples t-test.

Muscle	Variable	FAI Mean (SD)	Control Mean (SD)	Mean Difference (95% CI)	p-value
Gluteus Medius	Peak	30.5 (13.3)	30.8 (11.7)	-0.3 (-8.7, 8.2)	0.95
	Integrated	10.0 (4.2)	9.8 (4.2)	0.2 (-2.5, 3.0)	0.86
Biceps Femoris	Peak	19.4 (9.4)	24.2 (11.6)	-4.7 (-11.5, 2.0)	0.17
	Integrated	5.5 (3.4)	5.8 (2.5)	-0.3 (-2.2, 1.7)	0.78
Semitendinosus	Peak	22.6 (11.7)	22.6 (6.3)	0.0 (-6.3, 6.3)	0.99
	Integrated	5.1 (3.4)	3.9 (0.7)	1.2 (-0.4, 2.9)	0.13
Medial Gastrocnemius	Peak	53.1 (12.2)	56.4 (12.0)	-3.3 (-11.3, 4.7)	0.41
	Integrated	10.3 (2.1)	9.9 (2.3)	0.4 (-1.1, 1.8)	0.61
Lateral Gastrocnemius	Peak	43.5 (12.1)	52.9 (14.1)	-9.4 (-18.2, -0.5)	0.04*
	Integrated	9.4 (4.1)	8.2 (3.3)	1.0 (-1.4, 3.5)	0.40
Rectus Femoris	Peak	17.1 (9.7)	15.6 (9.1)	1.5 (-4.7, 7.6)	0.63
	Integrated	4.2 (2.6)	3.6 (1.9)	0.6 (-0.9, 2.0)	0.42
Vastus Medialis	Peak	68.8 (22.3)	63.4 (22.2)	5.4 (-8.8, 19.7)	0.45
	Integrated	13.2 (3.2)	11.8 (4.2)	1.3 (-1.1, 3.8)	0.26
Vastus Lateralis	Peak	54.5 (20.4)	53.9 (17.4)	0.5(-11.8, 12.8)	0.93
	Integrated	12.4 (4.3)	11.4 (3.6)	1.0 (-1.6, 3.5)	0.43
Tibialis Anterior	Peak	20.4 (10.0)	24.9 (10.65)	-4.5 (-11.2, 2.2)	0.18
	Integrated	4.0 (1.9)	4.6 (2.3)	-0.6 (-2.0, 0.7)	0.35

* indicates statistically significant differences $p < 0.05$.

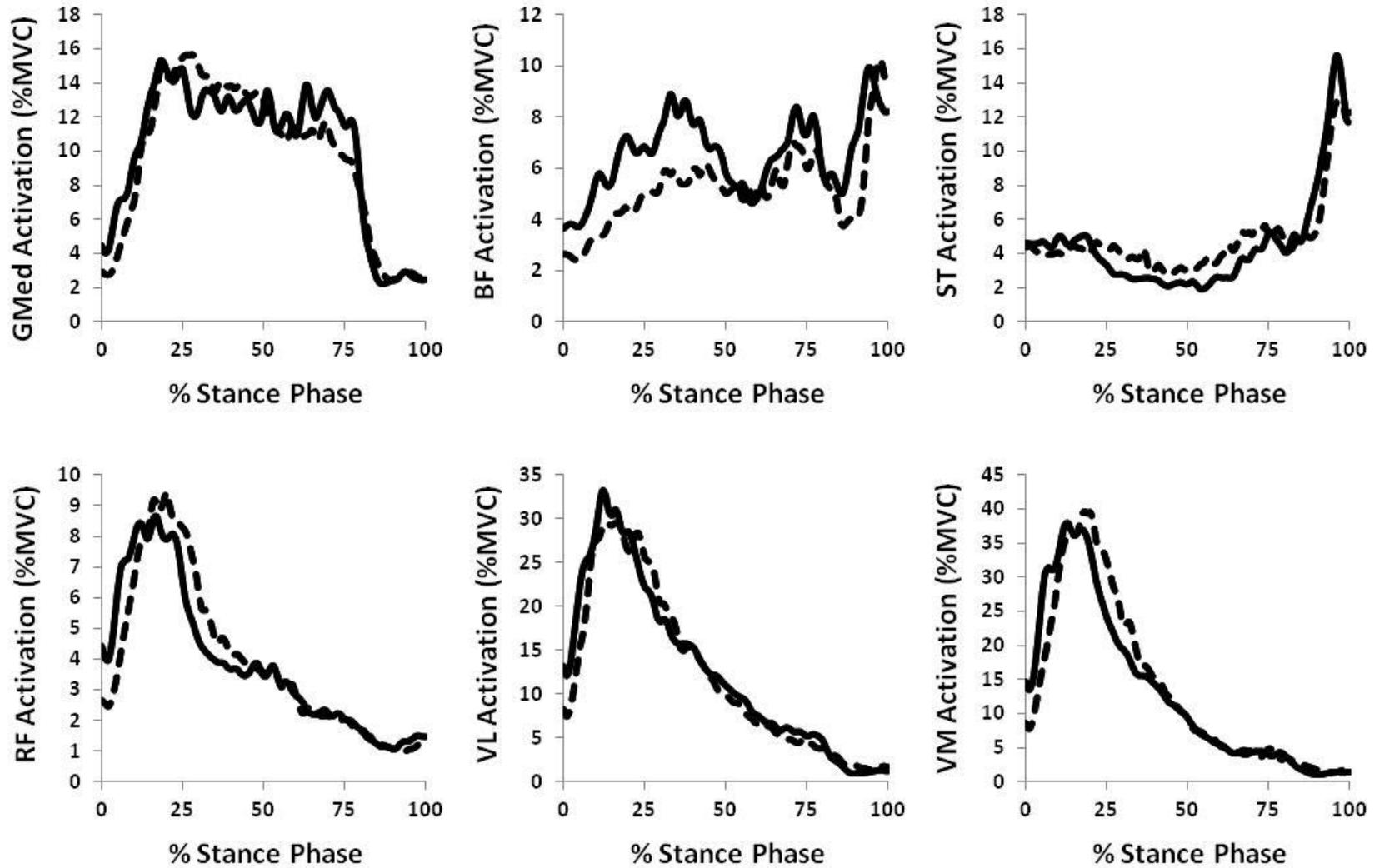


Figure 10. Muscle activation ensemble averages part 1; A solid line represents the control group and a dashed line represents the FAI group.

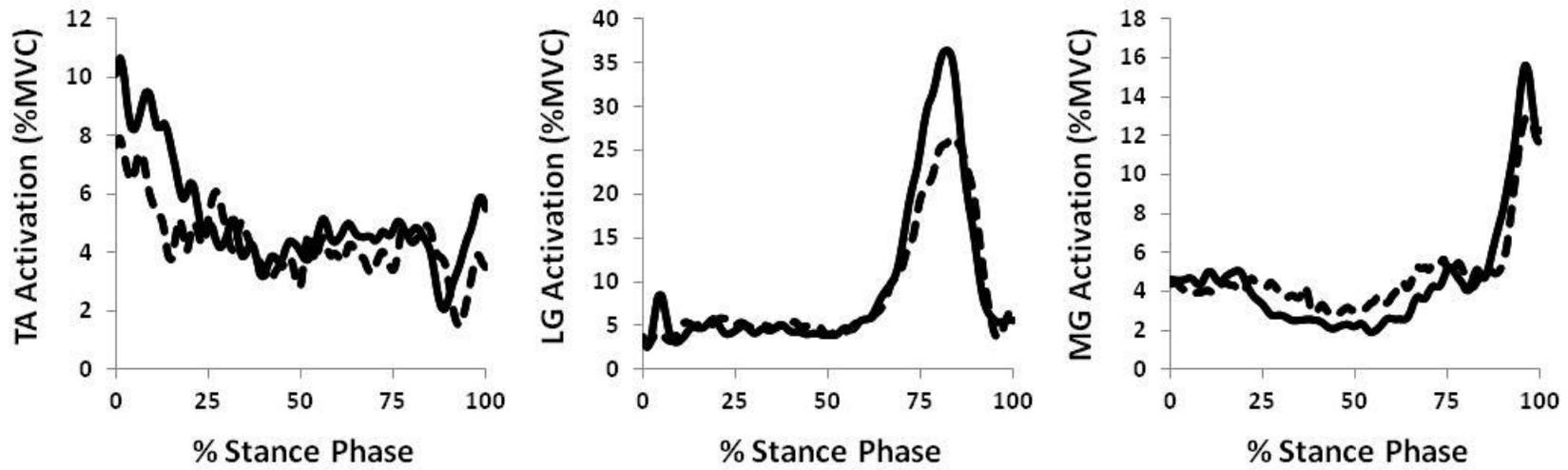


Figure 11. Muscle activation ensemble averages part 2; A solid line represents the control group and a dashed line represents the FAI group

Chapter 5: Discussion

The purpose of the present study was to examine three-dimensional gait kinematics of the trunk, hip, knee and ankle in addition to hip, knee and ankle joint kinetics and muscle activation during stair climbing in those with FAI compared to healthy controls. This will allow clinicians to better understand the biomechanical performance characteristics during stair climbing in those with FAI and help with rehabilitation of this population. The present study showed that those with FAI exhibited multiple gait differences during stair climbing compared to the healthy controls. The results of testing the first hypothesis were partially supported, in that those with FAI exhibited increased forward trunk flexion, however, this did not occur in conjunction with other changes in peak hip joint angles or excursions as these values were similar to that of the control group. The results of testing the second hypothesis were opposite of that hypothesized, in that those with FAI increased the joint loading about the hip and decreased the joint loading about the knee during stair climbing when compared to the controls. This was shown by greater peak external hip flexion moments and lesser peak external knee flexion moments. Finally, the results of testing the third hypothesis of increased GMed activation and increased activation of other lower limb muscles were not supported. Instead, those with FAI only exhibited decreased peak LG muscle activation compared to the control group.

5.1 Interpretation of Findings

To our knowledge, this was the first study to assess muscle activation during a dynamic movement task in those with FAI. Additionally, this was the first study to assess the kinematics of the trunk, knee and ankle and the kinetics of the hip, knee and ankle in order to understand the potential compensatory strategies undertaken by these joints in those with FAI during stair climbing. Our results showed that, for the most part, those with FAI ascended stairs relatively

similar to controls although some biomechanical differences did exist. It was observed that those with FAI exhibited greater peak trunk forward flexion angles and peak external hip flexion moments and lesser peak external knee flexion moments and plantar flexion (LG) muscle activation during stair climbing compared to the controls.

Our findings that the majority of our outcomes were similar between the groups is consistent with previous studies examining gait (Brisson et al., 2013; Diamond et al., 2016; Hunt et al., 2013; Kennedy et al., 2009) and stair climbing (Rylander et al., 2013) differences in people with FAI. One potential explanation for this is that, while more difficult than over ground walking, stair climbing may still not be challenging enough to elicit significant biomechanical differences. Further, unlike walking, stair ascent does not enable multiple strategies for ambulation given that stair height was fixed and therefore not different. As a result, while stair ascent speed was amenable to change, many biomechanical parameters such as step length were predetermined based on the task requirement, and therefore most kinematic requirements were the same. Finally, maximum hip flexion during stair ascent was approximately 60 degrees, which is well below the maximum available range of motion at the hip. As a result, it is unlikely that an anatomical impingement was present which would have caused more differences.

The between-group differences that we observed (namely differences in stair ascent speed, forward trunk flexion, and LG activation) can potentially be explained multiple ways, both from a biomechanical and clinical perspective. As no kinematic differences were observed about the hip, knee or ankle, the first potential explanation is that those with FAI increased the trunk forward flexion angle to compensate for decreased LG activation. The plantar flexor muscles increase their activation toward the end of stance and play an important role during stair climbing as they are required to propel the body up and forward (Andriacchi et al., 1980). In order to compensate for decreased LG activation, those with FAI increased their trunk forward

flexion angle. A greater trunk forward flexion angle moves the centre of mass more anterior, allowing the participant to propel themselves forward by increasing their forward momentum (Bouffard et al., 2011). Unfortunately, to our knowledge a lack of research exists comparing the medial and lateral gastrocnemius activity during stair climbing. However, as previous research on similarly aged, healthy subjects during stair climbing observed a similar MG activation level (41.7 %MVC) as that of the present study, it is likely that the LG activity in the present study is correct (Luder et al., 2015). Therefore, those with FAI may have compensated for decreased plantar flexion activation towards the end of stance, as a result of decreased LG muscle activation. They did so by increasing their trunk forward flexion angle to shift the centre of mass more anterior, increasing their forward momentum. While it is unknown why those with FAI decreased their LG muscle activation, a second possible compensatory strategy suggests that a greater trunk forward flexion angle may be undertaken to decrease the demand on the quadriceps.

Reducing the activity of the quadriceps to prevent the soft tissue, mainly the RF and iliopsoas muscles, on the anterior aspect of the hip from becoming aggravated may be an additional explanation for the increased trunk forward flexion observed in this study. This has been suggested to occur as a result of the soft tissue becoming aggravated by the bony impingement during movements in the sagittal plane that do not encroach on the limits of the hip ROM, such as level walking and stair climbing, (Hunt et al., 2013; Kennedy et al., 2009; Rylander et al., 2013). While not measured in this study, those with FAI have been observed to exhibit significantly less hip flexion force production (Casartelli et al., 2011), and, with the quadriceps being main contributors to stair ascent (McFadyen and Winter, 1988), those with FAI may have increased their trunk forward flexion angle in order to ascend the stairs. Increasing the trunk forward flexion angle moved the line of action of the ground reaction force away from the

hip and closer to the knee, creating a mechanical advantage (Bouffard et al., 2011). This would have caused the sagittal plane moment arm to increase for the hip and decrease for the knee, resulting in a greater peak external hip flexion moment and a reduced peak external knee flexion moment. A greater peak external hip flexion moment would decrease the demand of the quadriceps and increase the demand of the hip extensors. Unfortunately, GMax EMG was not measured in the present study. A reduction in the peak external knee flexion moment has been shown to reduce the quadriceps demand in those with knee OA as well as anterior cruciate ligament deficiency (Asay et al., 2009; Berchuck et al., 1990). It is possible that those with FAI undertook the same compensatory strategy in order to reduce the demand on the RF muscle in an attempt to prevent it from becoming aggravated by the bony impingement. However, despite the possible compensatory strategies undertaken by those with FAI to potentially reduce the activation of the quadriceps, the peak EMG activity of the RF in those with FAI was the same as the control group. This may be due to a slower stair ascent speed in the FAI group compared to the controls. The observed stair ascent speeds of the controls in the present study are similar to that of previous research, which observed that healthy participants ascended stairs at 0.49 m/s (Protopapadaki et al., 2007).

Despite a slower stair ascent speed in the FAI group, the relative muscle activity was similar to that of the controls. EMG activity of the quadriceps is positively correlated with gait velocity during stair climbing (Lewis et al., 2015). As those with FAI have been observed to exhibit weakened hip flexor force production and reduced stair ascent speed, it is possible that those with FAI in the present study managed to compensate for muscle weakness with reduced stair ascent velocity, and thus used the same effort as the control group relative to their maximum. While this has not been observed in those with FAI, it been observed in those with total knee arthroplasty (Bjerke et al., 2014).

A third potential explanation for our findings was that slower stair ascent speed and increased trunk forward flexion was a compensatory response to pain. Reduced gait speed has also been observed to decrease the peak external hip flexion moment during stair climbing (Lewis et al., 2015). Therefore not only may those with FAI adopt slower gait speeds during stair ascent to decrease the muscle activation of RF, but they may also use this strategy to decrease the demands on the hip and the associated pain response. An exploratory subgroup analysis from our FAI cohort looking at HOOS questionnaire data focusing on stair climbing (“What amount of hip pain have you experienced the last week while going up and down stairs” and “during stair ascent, please indicate the degree of difficulty, by which we mean your ability to move around and to look after yourself, you have experienced in the last week due to your hip”) indicated that those who reported some pain (18/20, N=8 mild pain, N=10 moderate or severe pain) or some loss of function (15/20, N=6 mild, N=9 moderate or severe) during stair climbing performed the task differently than those who reported no pain (2/20) or no loss of function (5/20).

Although not statistically significant (likely due to lack of statistical power), differences were observed when comparing stair ascent speed in the two people who reported no pain (0.50 ± 0.04 m/s) compared to the eight people reporting mild pain (0.41 ± 0.05 m/s) and the ten people reporting moderate or severe pain (0.40 ± 0.09 m/s). Similar findings were found when examining peak trunk forward flexion angle ($13.4 \pm 4.1^\circ$, no pain; $15.4 \pm 6.6^\circ$, mild pain; $16.2 \pm 3.7^\circ$ moderate or severe pain). When comparing those who reported no loss of function to those who reported mild and moderate or severe loss of function during stair climbing, differences were observed for ascent speed (0.44 ± 0.06 m/s no dysfunction; 0.44 ± 0.08 m/s mild dysfunction; 0.38 ± 0.07 m/s moderate or severe dysfunction) as well as peak forward trunk flexion angle ($14.1 \pm 7.3^\circ$ no dysfunction; $15.9 \pm 4.7^\circ$ mild dysfunction; $16.3 \pm 3.9^\circ$ moderate or severe dysfunction).

The results from the limited subgroup analysis based on questionnaire data suggest that those with FAI who self-reported greater pain or loss of function ascended the stairs at a slower speed and with greater trunk forward flexion. Similar findings have been exhibited in those with knee OA during level walking, where it was observed that as self-reported pain increased, gait speed decreased and lateral trunk lean increased (Hunt et al., 2010). This may suggest that the biomechanical differences observed in those with FAI during stair climbing are a response to pain as greater differences (i.e. “compensations”) were observed as self-reported pain increased.

A final potential explanation for our findings is that increased trunk forward flexion may be a precursor to the development of FAI. Greater trunk forward flexion moves the line of the ground reaction force away from the hip, thereby increasing the sagittal moment arm which results in a greater external hip flexion moment. A greater external hip flexion moment must be counteracted by internal moments in the opposite direction. This can increase the joint contact forces leading to cartilage breakdown. FAI is often associated with labral tears. Therefore, the development of FAI may be a result of greater forces imposed about the hip as a result of greater trunk forward flexion. Of course, this explanation is speculative and cannot be confirmed with the current cross-sectional study design. Future longitudinal prospective datasets are required to answer this question as well as that of the potential compensatory mechanism related to increased functional and symptomatic disease severity.

5.2 Clinical Implications

The results from the present study suggest that those with FAI exhibit slight biomechanical variations when ascending stairs than pain free, healthy controls. Most of our findings suggest that those with FAI exhibit the same biomechanics about the hip, knee and ankle during stair climbing compared to the controls. However, our results did show that those

with FAI increased their trunk forward flexion angle and external hip flexion moment, and decreased their external knee flexion moment and LG activation. This could be done to possibly compensate for either decreased LG muscle activation, to decrease the demand on the quadriceps, or to minimize pain.

However, increasing the trunk forward flexion angle may be detrimental to those with FAI. Those with FAI had similar knee and hip flexion angles compared to the controls. This flexion of the lumbar spine could potentially increase the loading of lumbar spine resulting in low back pain (Shum et al., 2005). Low back pain is commonly associated with FAI. Therefore, a greater trunk forward flexion angle during stair climbing may be a contributing factor, and certainly not advocated for people with FAI who may be at risk, or already present with, low back pain if possible. As mentioned above, it is possible that a greater trunk forward flexion angle resulted in an increased peak external hip flexion moment that may be associated with greater joint contact forces leading to greater articular cartilage breakdown and development of OA in those with FAI (Ganz et al., 2003).

Additionally, decreased peak LG activation may suggest altered muscle activation in those with FAI during stair climbing. The plantar flexors are important in propelling individuals up stairs as they encroach on roughly 50 % of the MVC amplitude in the controls during stair climbing. As those with FAI exhibited decreased activation, this may hinder their ability to properly ascend stairs. This decrease in activation could be a result of the inability to properly recruit the muscle. This would decrease the ability of those with FAI to ascend stairs properly. Furthermore, decreased peak LG activation may suggest that those with FAI require rehabilitation of this muscle. This may reduce pain and increase the function of those with FAI. Therefore those with FAI may be less likely to seek more invasive treatment options such as surgery. These considerations should be taken into account by clinicians when treating those

with FAI as the gait adaptations undertaken during stair climbing may not only be detrimental to the hip, as evident by greater peak external hip flexion moments, but to the low back as well. However, it is unknown whether people with FAI exhibit plantar flexor weakness, decreased muscle activation, or both. Given the potential link between plantar flexor activity and stair climbing observed in this study, assessment of all lower limb strength should be conducted as part of a thorough objective assessment in this patient population.

It is important to understand the implications of the potential compensatory strategies in those with FAI as it may provide clinicians with possible rehabilitation options. Training the LGs could provide the necessary plantar flexion to propel those with FAI up the stairs. Therefore those with FAI would not have to rely on a greater trunk forward flexion angle to provide the necessary momentum during stair ascent. Additionally, training the quadriceps during stair climbing, specifically the VM and VL, may provide those with FAI the necessary muscle activation to propel them up and forward. Reducing the trunk forward flexion angle during stair ascent could possibly decrease the loading of the low back, which is often a painful area in those with FAI. Additionally, reducing the trunk forward flexion angle would decrease the external hip flexion moment. Reducing the loading about the hip during stair climbing may help prevent the breakdown of cartilage and development of OA.

The potential compensation in which those with FAI increased their peak trunk forward flexion angle may differ based on the self-reported pain or function during stair climbing. Reducing pain and increasing the function during stair climbing may help for rehabilitation of this population. If those with FAI do not experience pain or loss of function for certain tasks, they may be less likely to seek more invasive treatment options such as surgery. Based off the limited questionnaire data, it appeared that those with FAI who self-reported no pain or loss of function during stair climbing exhibited decreased LG activation and trunk forward flexion

compared to those who reported some pain or loss of function. Therefore, those who report no pain or loss of function may increase the trunk forward flexion angle to potentially compensate for decreased LG activation, as those who reported some pain or loss of function exhibited LG activation comparable to the controls. Additionally, it appeared that those with FAI who report some pain or loss of function during stair climbing exhibited decreased peak external knee flexion moments compared to those who reported no pain or loss of function, exhibiting similar peak external knee flexion moments to that of the controls. Therefore those who reported some pain or loss of function may increase their trunk forward flexion angle to potentially reduce the demand on the quadriceps. This is an important factor for clinicians to consider when rehabilitating those with FAI. However, further research is needed to determine if these compensatory strategies are correlated to self-reported pain or loss of function.

5.3 Study Limitations

One limitation is that the stairs are of set height and depth. This imposes gait restrictions on participants and may be the reason for which no kinematic differences were observed about the hip, knee and ankle. This was in contrast with previous research that observed reduced peak hip extension and internal rotation angles in those with FAI compared to controls during stair ascent (Rylander et al., 2013). While the participants were matched for BMI, their height was not reported in the study by Rylander et al. (2013). As previous research by Livingston et al. (1991) observed that shorter participants required greater knee flexion angles to ascend the stairs, it is possible that matching for participant height in the present study nullified any potential kinematic differences about the hip, knee and ankle that were observed in previous studies. Second, our control group was recruited based on the absence of hip and back pain, but not radiographic findings. It is known that approximately 37% of asymptomatic individuals exhibit

radiographic evidence of FAI (Frank et al., 2015). However, previous work by Audenaert et al. (2012) observed that those with asymptomatic FAI exhibited the same passive hip ROM as healthy controls. As hip ROM during stair climbing does not encroach on the limits of ROM, and asymptomatic controls have been observed to elicit the same passive hip ROM as healthy controls, it can be speculated that the asymptomatic control group used in the present study would produce the same kinematics as a healthy control group. However, the asymptomatic control group in the present study was recruited based on the absence of hip and back pain, suggesting that the presence of pain may be an important factor in the biomechanical differences observed between groups.

Another limitation to the present study is that we did not control for gait speed to ensure that natural, self-selected movement biomechanics were captured. Previous research has shown that increased gait speeds during stair climbing led to increased muscle peak activation of the RF, VL, VM, BF, ST, LG and MG and increased peak external hip flexion moments (Lewis et al., 2015). As the FAI group ascended the stairs at a slower ascent speed compared to the control group, it is possible that the FAI group compensated for weakened muscles with decreased ascent speed. This would allow those with FAI to ascend the stairs with the same muscle effort as the controls, masking potential differences in EMG amplitude.

Placement of the EMG electrodes may also have altered the EMG amplitude of a specific muscle. If EMG electrodes are not placed on the centre of the muscle belly, the signal amplitude could have been influenced by tendons, ligaments and crosstalk from other muscles. However, EMG preparation and placement were done according to SENIAM.org guidelines which has been validated within the literature (Hermens et al., 2000). The EMG amplitude may also have been influenced by the MVC exercises. The resistance provided during the MVC for both the medial and lateral gastrocnemius was provided by the weight of the subject and not by the

researcher. The weight of the subject may not have been enough resistance to elicit a proper isometric contraction resulting in the electrical amplitude produced by the muscle being lower than the actual maximum. However, the MG peak activation in the present study was similar to previous research during stair ascent using the same gastrocnemius MVC procedure (Luder et al., 2015). Additionally, GMax EMG was not measured. Therefore, although a greater peak external hip flexion moment was observed in those with FAI, we can only speculate that altered muscle activation of GMax would occur. This is highly likely as GMax is the primary hip extensor muscle and the other two hip extensor muscles (BF and ST), which act primarily as knee flexors and secondarily as hip extensors, did not differ from the controls in their activation levels. Therefore it is likely that differences in GMax muscle activation would have been observed if the present study measured the EMG amplitude of GMax.

Finally, due to the nature of cross-sectional study designs, we were unable to determine a cause and effect relationship of the biomechanical differences in those with FAI compared to the controls. We can only speculate that those with FAI may increase their trunk forward flexion as a compensatory response for decreased LG activation, to reduce the demand on the quadriceps or in response to pain. However, it may be that greater trunk forward flexion leads to the development of FAI. The nature of cross-sectional research design prevents us from being able to determine the exact nature of why a greater trunk forward flexion in those with FAI occurs.

5.4 Future Directions

Although the results suggest that those with FAI may employ compensatory strategies during stair climbing, further research is required to fully understand the mechanisms for why and how this occurs. As most of the biomechanical variables during stair climbing in those with FAI were similar to the controls, it suggests that stair climbing may not be a difficult enough task

to elicit a significant compensatory response in those with FAI. Future research should be conducted on a task that does not impose constraints on human movement, such as cutting motions or lunging. Additionally, future research should be conducted on tasks that have been shown to require greater trunk forward flexion.

Future research should control for gait speed. This will allow for better comparison of muscle activation and joint moments between groups. Additionally, it would be beneficial to include measurements of GMax activation during stair climbing as a greater peak external hip flexion moment was observed in those with FAI.

Further research on muscle force production about the ankle and knee should be conducted in those with FAI. Determining whether those with FAI exhibit muscle weakness about the knee and ankle joints, combined with muscle activation measures during stair climbing, will allow for better understanding of the possible compensatory strategies undertaken in those with FAI. Importantly, understanding muscle weakness throughout the lower limb is necessary to provide optimal rehabilitation of these individuals, with or without surgical intervention. Future research should also focus on the subgroups of FAI based on the self-reported pain and loss of function. It was observed that those with FAI who reported some pain or some loss of function performed the stair climbing task differently than those who reported no pain or loss of function. Recruiting more FAI subjects based on the amount of pain or loss of function they self-report will allow researchers to better understand the different strategies in which those with FAI ascend stairs, and provide insight into functional progression of FAI.

Most importantly, this was the first study to measure trunk kinematics in those with FAI, observing that those with FAI increase the trunk forward flexion angle as a potential compensatory mechanism. Therefore, trunk kinematics, kinetics, and muscle activation of the

erector spinae should be included in future research studies to fully understand the compensations, as well as the implications in which these compensations affect those with FAI.

Lastly, future research should focus more on longitudinal study designs. This will allow researchers to better determine causation. Specifically, this will enable researchers to determine if greater trunk flexion in those with FAI is a result of possible compensatory strategies for other factors, such as reduced LG activation, quadriceps avoidance an increased pain, or if greater trunk forward flexion is a contributing factor to the development of FAI.

Chapter 6: Conclusions

Stair climbing is a common everyday activity that requires greater ROM about the hip, knee and ankle and greater muscle activation compared to level walking. It can be quite challenging with the presence of a disability such as FAI. Therefore it is important to understand the gait strategies in which those with FAI ascend stairs in order for clinicians to better rehabilitate this population. To our knowledge, this was the first study to measure muscle activity during a dynamic movement task in people with FAI. Additionally, this was the first study to measure the kinematics of the trunk, knee and ankle and kinetics of the hip, knee and ankle during stair climbing in this patient population.

The results indicate that those with FAI exhibit greater peak trunk forward flexion angles, increased external hip flexion moments, and decreased peak external knee flexion moments and LG activation compared to the control group. This was thought to occur due to three potential compensatory strategies. The first possible strategy consists of increasing the trunk forward flexion angle to compensate for decreased LG activation. The LG acts as a plantar flexor, driving the body forward during stair ascent. Decreased plantar flexor activation may be compensated for by increased trunk forward flexion, which moves the centre of mass more anterior, increasing the forward momentum and facilitating the stair ascent. The second possible strategy is that those with FAI increase their trunk forward flexion angle to reduce the demand on the quadriceps, specifically the RF muscle. This is achieved by decreasing the external knee flexion moment. This may occur to prevent the soft tissue on the anterior aspect of the hip from becoming aggravated due to the bony impingement. However, no difference in RF EMG was detected despite a decrease in the external knee flexion moment. One possible explanation for this is that those with FAI ascended the stairs at a reduced speed compared to the controls, and combined

with previously reported hip flexion muscle weakness, a slower ascent speed may have been a compensatory mechanism to use the same quadriceps effort as the controls relative to the max. The third possible compensatory strategy is that those with FAI decreased their stair ascent speed and increased their trunk forward flexion in response to pain. It was observed that those with FAI who reported some pain (mild or moderate or severe) or loss of function (mild or moderate or severe) during stair ascent exhibited decreased gait speed and increased trunk forward flexion compared to those with FAI who reported no pain or loss of function.

An increased trunk forward flexion angle does have its implications. Increased flexion of the lumbar spine can cause low back pain, a common problem in those with FAI. Additionally, greater trunk forward flexion angle results in a greater peak external hip flexion moments. This can increase the joint forces about the hip and potentially increase cartilage breakdown and development of hip OA in those with FAI. Therefore, trunk forward flexion may not be a compensatory strategy in those with FAI, but a possible cause of it. However, this is merely speculative and cannot be determined in a cross-sectional study design.

The results from the present study suggest potential gait compensations during stair ascent in those with FAI. However, the reason for why this occurs was not in the scope of this study and further research is required. Regardless, these potential gait compensatory strategies should be taken into account by clinicians when rehabilitating those with FAI. If found to be effective and safe, gait modification focusing on increasing trunk forward flexion could potentially decrease the difficulty of the task by reducing pain and increasing the function in those with FAI. Therefore those with FAI may not feel the need to seek more invasive treatment options such as surgery.

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Appendix

Appendix A: Informed Consent Form

THE UNIVERSITY OF BRITISH COLUMBIA



Department of Physical Therapy

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212, Friedman Building

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Participant Information and Consent Form

Kinetic and kinematic analysis comparing stair ambulation in healthy participants and those with symptomatic femoroacetabular impingement

Principal Investigator: Michael A. Hunt, PhD, PT
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(604) 822-XXXX

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For non-emergency contact numbers:

Motion Analysis Biofeedback Laboratory (604) 822-XXXX

1. Invitation

You are being invited to take part in this research study because you i) were diagnosed with femoroacetabular impingement (hip impingement) or ii) are healthy with no history of lower body injuries, and are between the ages of 16-50. You must have no significant lower body musculoskeletal injuries or neurological conditions that would impair walking or hip strength, no evidence of arthritis of the hip, knee or ankle, no planned or future lower limb joint replacement, and no avascular necrosis (death of bone due to lack of blood supply) of the hip. Finally, you must be able to understand written and spoken English or have the ability to have a family member present to translate.

2. Your participation is voluntary

Your participation is voluntary. You have the right to refuse to participate in this study. If you decide to participate, you may still choose to withdraw from the study at any time without any negative consequences to the medical care, education, or other services to which you are entitled or are presently receiving.

You should be aware that there is a difference for both you and your doctor (if applicable) between being a patient and being a research participant. As a patient, all medical procedures and treatments are carried out for your benefit only according to standard accepted practice. As a research participant, you and your doctor also must take into account the requirements for the research study. These may include procedures and treatments that are not part of standard practice or are not yet proven. This consent form describes the diagnostic and treatment procedures that are being carried out for research purposes. Please review the consent document carefully when deciding whether or not you wish to be part of the research and sign this consent only if you accept being a research participant.

If you wish to participate in this study, you will be asked to sign this form. Please take time to read the following information carefully and to discuss it with your family, friends, and doctor before you decide.

3. Background

Hip impingement is a condition where the contact between the femur (thigh bone) and the pelvis (hip) is poor, resulting in excess friction, causing pain and dysfunction. Repetitive activities that require greater hip range of motion such as stair climbing may increase forces acting on the joint resulting in damage and irritation. As a consequence of this recurring irritation, over time this can lead to early onset of hip osteoarthritis (inflammation of joint due to lack of cartilage in joint) in young and active adults.

During the initial stages of hip impingement simple tasks such as walking or sitting may not be enough to elicit a clinically symptomatic response (pain and discomfort), therefore a more challenging task such as stair ascent may be required. Individuals in this present study will undertake stair climbing tasks to assess the changes in locomotion and muscle activation between health participants and those who with hip impingement.

4. What is the purpose of the study?

Impingement of the hip prevents full movements at that joint and this restriction causes pain. With prolonged pain and altered walking patterns, there is a possibility of developing muscle weakness, which may result in further hip damage. We aim to examine the changes in locomotion and muscle activation during stair ambulation between healthy individuals and people with hip impingement.

You are one of forty individuals being invited to participate in this study that will assess the mechanisms in which hip impingement affects stair ambulation when compared to healthy controls. This study will help us better understand the mechanisms in which hip impingement affects stair ambulation compared to those with healthy hip joints. By identifying discrepancies between the two, we may better be able to diagnose and treat affected hips thereby reducing pain and physical dysfunction in those with “hip impingement”.

5. Who can participate in this study?

You may be able to participate in this study if you are:

- Between the ages of 16-50
- Diagnosed with femoroacetabular impingement or are healthy with no history of lower leg injury
- Be able to participate in moderate activity

6. Who should not participate in this study?

You will not be eligible to participate in this study if you have:

- A history of lower body injuries or conditions that impair the measurement of walking or hip strength
- A history of neurological injury that affects walking
- Have osteoarthritis in the hip, knee or ankle
- A history of avascular necrosis of the hip
- Planned or previous lower limb joint replacement.

7. What does the study involve?

Once Dr. Gilbert has approved you to be a valid femoroacetabular impingement candidate for the present study or you are deemed a healthy control participant, the procedures you can expect will include the following if you agree to participate:

Testing will be performed at Dr. Hunt's Motion Analysis and Biofeedback Laboratory at UBC and will measure walking patterns during stair ascent and descent over one 60 minute testing session. This study will require you to walk up and down a set of stairs five times each while digital cameras and electrodes measure your walking and muscle activation patterns.

During a testing session in the lab, the following measurements will be collected:

Upon completing a questionnaire to assess hip pain and functionality, you will be asked to walk barefoot up and down stairs while wearing shorts and a special t-shirt with 2 holes cut out. Reflective skin markers will be attached to your skin at various sites such as the ankle, knee and hip (additional markers will be placed under your chin and on your back within the holes cut into the t-shirt). We will also measure muscle activity of your lower limb muscles using electromyography. Electromyography is the measurement of the electrical activity in your body that causes your muscles to work. This requires the attachment of electrodes to your skin around your hip joint (similar to how an electrocardiogram measures the electrical activity of your heart). These electrodes only measure the muscle activity and do not provide any electrical stimulation to you.

You will be filmed with special cameras that track the movement of the reflective markers as you walk up and down the stairs. The movement of the markers enables a recreation of a moving "stick figure" on the computer screen that mimics your movements. The image in no way provides any information about your identity, and all files stored on the compute will be recognized only based on your unique coded identifier (ie. not your name).

From the stair climbing tests, we will be able to analyze the movements at your trunk, hip, knee and ankle, as well as the forces acting across each joint. You will complete approximately 10 trials of stair ascent and descent at your own pace.

You must report to the investigator any pain or discomfort during any of the testing procedures. As noted above, you will be required to wear shorts and a t-shirt during all testing.

8. What are the possible harms and discomforts?

You may experience mild skin irritation from the reflective skin markers. Hip impingement participants may also experience mild discomfort during stair ambulation. If this is bothersome, you should inform the tester immediately. In the unlikely event of a medical emergency during the assessment, the research personnel will call 911.

9. What are the potential benefits of participating?

You will not receive any direct benefits from participation in this study. However, the findings from this study may contribute to the development of better diagnosis and treatment options for people with hip impingement.

10. What happens if I decide to withdraw my consent to participate?

You may withdraw from this study at any time without giving reasons. If you choose to enter the study and then decide to withdraw at a later time, you have the right to request the withdrawal of your information collected during the study. This request will be respected to the extent possible. Please note however that there may be exceptions where the data will not be able to be withdrawn for example where the data is no longer identifiable (meaning it cannot be linked in any way back to your identity) or where the data has been merged with other data. If you would like to request the withdrawal of your data [and/or samples], please let your study doctor know. If your participation in this study includes enrolling in any optional studies, or long term follow-up, you will be asked whether you wish to withdraw from these as well.

11. Can I be asked to leave the study?

If you are not able to follow the requirements of the study or for any other reason, the study investigator may withdraw you from the study and will arrange for your care to continue. If you are asked to leave the study, the reasons for this will be explained to you and you will have the opportunity to ask questions about this decision.

12. How will my taking part in this study be kept confidential?

Your confidentiality will be respected. However, research records and health or other source records identifying you may be inspected in the presence of the Investigator and UBC Clinical Research Ethics Board for the purpose of monitoring the research. No information or records that disclose your identity will be published without your consent, nor will any information or records that disclose your identity be removed or released without your consent unless required by law.

You will be assigned a unique study number as a participant in this study. This number will not include any personal information that could identify you (e.g., it will not include your Personal Health Number, SIN, or your initials, etc.). Only this number will be used on any research-related information collected about you during the course of this study, so that your identity will be kept confidential. Information that contains your identity will remain only with the Principal Investigator and/or designate. The list that matches your name to the unique study number that is used on your research-related information will not be removed or released without your consent unless required by law.

Your rights to privacy are legally protected by federal and provincial laws that require safeguards to insure that your privacy is respected. You also have the legal right of access to the information about you that has been provided to the sponsor and, if need be, an opportunity to correct any errors in this information. Further details about these laws are available on request to your study doctor. Your personal identifier/s will also be provided if requested by the sponsor or responsible regulatory agency.

13. What happens if something goes wrong?

By signing this form, you do not give up any of your legal rights and you do not release the study doctor, participating institutions, or anyone else from their legal and professional duties. If you become ill or physically injured as a result of participation in this study, medical treatment will be provided at no additional cost to you. The costs of your medical treatment will be paid by your provincial medical plan.

In case of a serious medical event, please report to an emergency room and inform them that you are participating in a clinical study and that the following person can then be contacted for further information: Dr. Michael Hunt at telephone number: (604) 827-XXXX

14. What will the study cost me?

All research-related medical care and treatment and any related tests that you will receive during your participation in this study will be provided at no cost to you. You will not be

reimbursed for your involvement in this study. However, the costs of parking or taking transit to attend study sessions, if necessary, will be reimbursed. Receipts for parking and transit must be kept and given to the research assistant.

15. Who do I contact if I have questions about the study during my participation?

If you have any questions or desire further information about this study before or during participation, or if you experience any adverse effects, you can contact Connor Hammond at (604) 822-XXXX. You can also speak to the doctor who is the principal investigator, (Dr. Michael Hunt PhD) at (604) 827-XXXX

16. Who do I contact if I have any questions or concerns about my rights as a participant?

If you have any concerns or complaints about your rights as a research participant and/or your experiences while participating in this study, contact the Research Participant Complaint Line in the University of British Columbia Office of Research Ethics by e-mail at RSIL@ors.ubc.ca or by phone at 604-822-XXXX (Toll Free: 1-877-822-XXXX)

17. After the study is finished

Possibility of future research

There may be future opportunities for you to participate in ongoing research. If you are interested in being contacted, please check the appropriate box below. If contacted, you will be asked to read a new letter of information and sign a new consent form.

Please keep my name and contact information so that I may be contacted to learn about future research opportunities or have access to my data in the future.

Copy of Study Results

I would like a copy of the study results. Yes No

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CONSENT FORM

Kinetic and kinematic analysis comparing stair ambulation in healthy participants and those with symptomatic femoroacetabular impingement

Participant Consent

My signature on this consent form means:

- I have read and understood the information in this consent form.
- I have had enough time to think about the information provided.
- I have been able to ask for advice if needed.
- I have been able to ask questions and have had satisfactory responses to my questions.
- I understand that all of the information collected will be kept confidential and that the results will only be used for scientific purposes.
- I understand that my participation in this study is voluntary.
- I understand that I am completely free at any time to refuse to participate or to withdraw from this study at any time, and that this will not change the quality of care that I receive.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I understand that there is no guarantee that this study will provide any benefits to me.

I will receive a signed copy of this consent form for my own records.

I consent to participate in this study.

Signature of Participant Printed Name Date

Signature of Person Obtaining Consent	Printed Name	Date
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If this consent process has been done in a language other than that on this written form, with the assistance of an interpreter/translator, indicate:

Language: _____

Was the participant assisted during the consent process in one of ways listed below?

Yes No

If yes, please check the relevant box and complete the signature space below:

The consent form was read to the participant, and the person signing below attests that the study was accurately explained to, and apparently understood by, the participant (please check if participant is unable to read).

The person signing below acted as an interpreter/translator for the participant, during the consent process (please check if an interpreter/translator assisted during the consent process).

Signature of Person Assisting in Consent Discussion	Printed Name	Date
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