A WALKING AND RUNNING BIOMECHANICAL ANALYSIS OF

FEMOROACETABULAR IMPINGEMENT

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ABSTRACT

Context: Femoroacetabular Impingement (FAI) is a bony deformity that leads labral tears, pain, and osteoarthritis. It is currently unknown how hip strength and walking and running biomechanics change over serial time points post-operatively in this population. ***Objective:*** Compare a battery of functional outcome measures pre- and post-operatively in FAI and healthy controls. ***Design:*** Causal comparative; Independent samples t-test for between subjects and matched pairs t-test for within subjects design, as well as correlations of variable relationships. ***Setting:*** Hospital and Research Laboratory. ***Participants:*** 12 unilateral FAI (11F, 1 M; age 30.6 ± 7.6, height 1.7 ± 0.1, weight 73.1 ± 13.1) and 10 controls (7F, 3 M; age 31.7 ± 6.1, height 1.7 ± 0.1, weight 68.4 ± 15.0). ***Intervention:*** Data were collected at an initial or pre-operative session (FAI within two weeks of hip arthroscopy) and again three- and six-months later. ***Main Outcome Measures:*** Clinical and radiographic data were collected at a local hospital, while all other data were collected in the laboratory. Patient related outcomes surveys (PROS) included the Hip Outcome Score and UCLA activity score. Max voluntary isometric hip and knee and strength were collected via hand held dynamometry. Walking and running data were collected via three-dimensional motion capture. ***Results:*** FAI participants PROS were lower than controls, except for the UCLA at six-months (8.7±1.6 vs. 8.8±1.9). The FAI group improved hip and knee flexion and extension strength over time, but remained weaker than controls. In sagittal and frontal plane muscles, the FAI group only had 67% of the strength as the controls. Hip external rotation strength was greater in FAI versus controls at both three- (21.1± 9.6 vs 10.7±4.6, $P<0.05$) and six-months (18.8±8.6 vs. 12.0±5.9). The decreased strength found in the FAI group correlated to their decreased HOS scores ($r>0.4$). Walking velocity in the FAI group was 17%, 12%, and 10% slower than controls at the pre-operative, three-month and six months sessions, respectively. Hip motion was decreased in the sagittal and during walking, but not during running. The pelvis and hip frontal plane motions were reduced during both walking and running. The transverse plane motion during walking and running favored external rotation at the hip and lower leg, whereas the controls preferred an IR position. ***Conclusions:*** There are many differences between groups pre-operatively that persisted at both three- and six-months post-operatively. The FAI patients in this study had worse PROS, were weaker in most of their hip musculature, and displayed abnormal walking and running patterns that may be attributed to both pain and weakness. Therefore, full recovery from hip arthroscopy takes protracted periods of time greater than six-months. ***Keywords:*** Femoroacetabular -Impingement, Hip Arthroscopy, Labrum Tear, Hip Strength, Walking and Running Biomechanics. ***Word Count:*****443**

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PART I

Introduction

Femoroacetabular impingement (FAI) results from femoral or acetabular bony deformities and has been recognized as a primary hip pathology that may lead to mechanical damage and premature development of osteoarthritis/osteoarthrosis (OA)[18, 96, 159, 167, 169]. The femur prematurely contacts the acetabulum during motion, leading to lesions of the labrum and cartilage, which contribute to early degenerative joint changes[96]. Osseous impact of the proximal femur and acetabular rim occurs at terminal hip flexion (100-110°) and more so when the hip is internally rotated (15-20°) in a flexed position[19, 23]. Additionally, painful internal rotation from a flexed and adducted position (FADIR test) is the trademark clinical finding of labral tears[186]. Radiographic imaging aids clinical findings and is used for the determination of FAI subtypes.

Pincer impingement results from excessively deep or retroverted acetabulum that increase coverage of the femoral head and neck, predisposing them to abnormal impact forces of the labrum and countercoup lesions in the posterior joint[96]. Radiographically pincer impingement is identified by lateral center edge angles (LCEA) > 40[9], negative angles of inclination (AI)[9], and signs of retroversion (cross over sign (COS)[134], posterior wall sign (PWS)[234], and ischial spine sign (ISS)[139]). Cam impingement results from a non-spherical femoral head, recognized radiographically by alpha angles > 50°[211]. The cam bump leads to abrading of the superior acetabulum in an anterior to posterior direction, damaging the adjacent labrum and cartilage.

Symptomatic FAI leads to abnormal hip strength[48], walking[127, 148, 237], squatting[158], and stair climbing[237] pre-operatively compared to controls. Walking motion is reduced in the sagittal, frontal, and transverse planes[127, 148, 237] and kinetic force distribution is altered[127, 148]. Additionally, abnormal hip flexion reversals may be present[238]. The result is a protective gait that may result from the attenuated motions[35], or secondary to weakness developed pre-operatively from disuse[48]. However, no studies have examined both gait and strength in a single study to compare these findings. Additionally, post-operative results of walking studies present contentious results[35, 237, 238]. Walking may return to normal a year after the minimally invasive arthroscopic procedure[237], whereas several key variables may worsen following the more invasive surgical hip dislocation[35]. Currently, it is unknown how walking gait changes over time post-operatively, since no studies examined more than one time point, or earlier than ten months after surgery[35, 237, 238].

A better understanding of the current post-surgical biomechanics and strength at specific time points in these patients may lead to new treatment insights. Many of the patients are not only trying to return to walking and other activities of daily living, but also sport and actions that are more demanding. However, to our knowledge no studies have examined running biomechanics in samples with these hip conditions. Therefore, the purpose of this study was to examine hip strength and the walking and running biomechanics in pre-and post-operative FAI patients compared to controls. We hypothesized that FAI patients will demonstrate decreased strength and abnormal walking and running gait compared to healthy matched controls pre- and post-operatively.

Research Hypotheses

1. We hypothesized that the Hip Outcome Score and UCLA activity scores would improve over time in FAI patients, but not reach the level of controls by six months post-operatively. There are considerable post-operative restrictions on impactful movements, therefore we did not expect a level equivalent to the controls at the study's conclusion.
2. We hypothesized that the HOS and UCLA would be positively and significantly correlated to strength measures at each session. Strength is required for adequate sport and activities of daily living (ADL) satisfaction and we anticipated strength and patient related outcome surveys to linearly increase at each post-operative session.
3. We hypothesized that strength would be decreased in the involved limb and compared to controls pre-operatively. By three months, we expect there to be no differences in the FAI group bilaterally or versus controls, since most will have had a regiment of physical therapy.
4. We hypothesized that walking and running gait would be significantly different for the FAI group pre-operatively and that there would be fewer differences post-operatively compared to controls. We believe that gait will normalize once physically therapy is completed and full strength is regained.

Methods

Research Design

This is a case control study designed to investigate of walking and running biomechanical differences of unilaterally involved FAI patients compared with controls. Prospective FAI patients and controls were asked to participate in three data collection sessions. Independent variables were limb (involved/uninvolved) and group (experimental/control). The control group included participants of similar age and anthropometric features. Dependent variables included pre- and post-operative anthropometrics, radiographic measures, patient related outcome surveys (PRO), hip strength, and biomechanical kinematics and kinetics.

Participants

Participants consisted of 22 volunteers, with the experimental group (n = 12) recruited from two orthopedic practices of board certified surgeons. All FAI patients underwent initial conservative treatment, which included at least one of the following: activity modification, physical therapy, non-steroidal anti-inflammatories, or intra-articular injections. The FAI group inclusion criteria included examination by either one of three orthopedic surgeons (RD, WB, & SC), failure of conservative measures, visible cam deformity (alpha angle > 50°) and/or pincer impingement (LCEA > 35°, AI < 10°, and/or positive signs of retroversion: COS, ISS, and PWS). The exclusion criteria were bilateral symptoms, endocrine system dysfunction, avascular necrosis or chondrolysis, radiographic appearance of osteoarthrosis, and any other medical

condition that may adversely affect walking and running gait. The control group (n=10) were recruited from the local community and denied a history of significant lower extremity injury and surgeries. Prior to the study, all participants signed informed consent or assent forms approved by the Human Studies Program and Institutional Review Board (Appendix A).

Data Collection and Storage

Participants reported to the Universities Human Performance Laboratory for all data collections, which consisted of the following: anthropometrics/demographics, patient related outcome surveys (PROS) (UCLA Activity Score, Hip Outcomes Score), gait analysis, and hip/knee strength testing (hip flexion, extension, abduction, adduction, internal/external rotation; knee flexion and extension). A single board certified athletic trainer (BF) and key personnel collected data pre-operatively (no more than two weeks prior to surgery) and three- and six-months post-operatively. Running data were not collected at the three-month post-operative session due to physician restrictions (Table 1).

Table 1. Time of data collection sessions

			Post-Operative	
Groups	Trials	Pre-Operative	Three-Month	Six-Month
FAI &	Walking	X	X	x
Controls	Running	X		x

Radiographic and clinical data were de-identified and numerically coded. All data, as well as the key to the participant code, were stored on the password protected desktop computer in the office of the attending surgeons that is secured by electronic medical records computer system (EPIC and Synapse).

Patient Related Outcomes Surveys

Responses to the UCLA Activity Score (UCLA) and Hip Outcome Score (HOS)[187] were recorded during all data collection sessions. The UCLA (Appendix B) is a validated single question and 10 item survey, that is recommended for monitoring physical activity levels[268]. The HOS (Appendix B) is a valid[187] and reliable[188] 31 question survey designed to assess outcomes after hip arthroscopy. Once calculated, the HOS produces activities of daily living (HOS_{ADL}) and sport (HOS_S) subscales. Within the HOS, there are three single answer questions pertaining to self-reported current level of function. One is a single question that asks how they would rate their current function (normal, nearly normal, abnormal, or severely abnormal). Then the ADL and sport current level of function questions ($CLOF_{ADL}$, $CLOF_S$) are a self-reported single values from 0-100. These subjective surveys determine activity level, pain, function, and quality of life measurements.

Hip and Knee Strength Assessment

Eight (six hip, two knee) positions of isometric maximum voluntary contractions (MVC) were collected on an adjustable Triton® treatment table (DJO Global, Vista, California, USA) using the MicroFet2 Hand Held Dynamometer (HHD) (Hoggan Health Industries Inc., West Jordan, UT). The HHD was placed between the examiner's hand and the marked test site, at an 80% distance from each hip or knee segment to standardize lever arms amongst individuals of varying heights. A goniometer helped to position tested muscle groups. Participants were instructed to exert pressure maximally and to sustain pressure for three seconds. Participants were also instructed to begin resistance upon the command "Go" and to rest upon the command "Stop". Following a submaximal familiarization trial, maximal trials were recorded

first on the uninvolved side, then the involved side to allow for a minimum of 90 seconds rest between trials. Differences greater than 10% between two trials, led to a third trial. Any trials with attempts to rotate the trunk, use adjacent muscles, or deviate motion were discarded and re-assessed. All muscle testing were performed in gravity dependent positions[116], except for hip abduction and adduction, which were collected supine to avoid increased risk of hip joint pain. Data were collected sequentially in the following order: prone (hip extension and knee flexion), supine (hip abduction and adduction), and seated (hip internal and external rotation, hip flexion, and knee extension). Pain level during each of the eight positions was assessed using a 0-10 scale.

3D Gait Analysis

Anthropometric data included height via a wall-mounted stadiometer (model 67032, Seca Telescopic Stadiometer, Country Technology, Inc., Gays Mills, WI, USA); weight via a calibrated scale (Detecto Inc., Webb City, MO, USA); standing leg length; distance between anterior superior iliac spines (ASIS); and ankle and knee joint widths via anthropometer calipers (DKSH, Zurich, Switzerland). Leg lengths and inter ASIS distance were measured via standard tape measure.

Biomechanic data collection utilized a 27 retro-reflective marker set, with placements at specific anatomic landmarks (Appendix C). Static calibration trials were recorded to create individualized models, which include calculation of body segments, joint centers, and neutral positions. Participants then walked 10 meters down a runway, shod for all trials at a self-selected velocity, so as not to alter normal walking gait. Next, shod running trials were recorded

in a similar manner as the walking trials, with set velocity of 4.0 meters per second (± 10%). Speedtrap II infrared sensors (Brower Timing Systems, Draper, Utah) were placed four meters apart, in the middle of the runway to measure velocities. A successful trial is defined as: completion of the pass through the data collection field at a consistent velocity, walking with the head up, and landing with one foot completely on the force plate with no obvious change in stride or targeting the force plate.[65, 286, 287]

A three-dimensional (3D) motion capture system (Vicon, Inc., Centennial, Colorado, USA), including 20 motion capture cameras (seven MX 3+ and six MX 13) and software (Nexus version 1.7.1) were used to capture, smooth, and reduce kinematic gait data. Force plates (Advanced Mechanical Technology Incorporated, Boston, Massachusetts, USA) were embedded flush with the floor surface to collect kinetic data during gait trials. Marker trajectories were collected at 240Hz and smoothed using a Woltring filter (mean square error cutoff of 10), and then time synchronized with kinetic data collected at 960Hz. Gaps were filled with a cubic spline polynomial routine within the Nexus software and an inverse dynamics approach was used to obtain weight-normalized hip, knee and ankle moments. Ground reaction forces and moments were filtered using a fourth-order low-pass Butterworth filter (10 Hz cut-off frequency) to reduce risk of artificial moment impact peaks[152, 153]. Three successful trials for each leg were captured at each session, the gait variables of interest were averaged for subsequent analyses.

Determination of abnormal reversals (change in slope of the extension curve)[238] began with visual inspection of the flexion-extension graph. The prevalence of FAI and control

participants and number of trials with reversals was recorded. The location (% of stance where reversal began), duration (% of stance the reversal lasted), and average change in sagittal plane position of the hip, knee, and ankle, for all reversals were also calculated and averaged for each individual.

Statistical Analysis

Descriptive statistics including means and standard deviations (SD) were generated for participant demographics and dependent variables. Group differences between the FAI and controls (involved FAI vs. right limb in controls) were analyzed via one-tailed independent samples t-tests. The within limb changes over time, and compared with the uninvolved limb, were assessed via one-tailed matched pairs t-tests. All group differences were analyzed via Levene's homogeneity of variance test for the assumption that the variances between groups were equal at $P < 0.05$[172]. Effect size was determined with Cohen's *d*, with 0.20, 0.50, and 0.80 representing small, moderate, and large differences, respectively[196]. For the independent samples t-test, Cohen's d is calculated by subtracting group differences and dividing by the pooled standard deviations (SD). For the matched pair's *d* calculation, there is a correction for the pooled SD by factoring in the correlation coefficient (*r*) between measures (Formula 1). Pearson's product moment correlation coefficients were used to determine the linear relationship between strength and PRO surveys. All statistical analyses were completed using SPSS v19 (IBM SPSS Statistics, IBM Corporation, Armonk New York, USA) with an alpha level set at $P \leq 0.05$.

Formula 1. Cohen's *d* for matched pair's t-test

$$\frac{group\ mean\ 1 - group\ mean\ 2}{(SDr1 + SDr2)/2}\ ,\ \text{where SDr} = (\text{SD1 or 2}) * \sqrt{2 * (1 - r)}$$

Operational Definitions

Activities of daily living (ADL)

Angle of Inclination (AI)

Anterior superior iliac spines (ASIS)

Current level of function (CLOF)

Cross over sign (COS)

Dorsiflexion (DF)

External Rotation (ER)

Femoroacetabular Impingement (FAI)

Flexion abduction external rotation test (FABER)

Flexion adduction internal rotation test (FADIR)

Ground reaction force (GRF)

Hand Held Dynamometer (HHD)

Hip Outcome Score (HOS)

Internal Rotation (IR)

Ischial spine sign (ISS)

Lateral center edge angle (LCEA)

Osteoarthritis (OA)

Patient related Outcome (PRO)

Plantarflexion (PF)

Posterior wall sign (PWS)

Range of motion (ROM)

Signs and symptoms (S/S)

Standard deviation (SD)

Results

Participants

Table 2. Participant Age and Anthropometrics

		Pre-operative			Three-Months			Six-Months Post		
	Group	N	Mean	SD	N	Mean	SD	N	Mean	SD
Height (m)	Con	10	1.7	0.1	7	1.7	1.1	4	1.7	0.1
	FAI	12	1.7	0.1	9	1.7	0.1	6	1.7	0.1
Weight (kg)	Con	10	68.4	15.0	7	66.2	10.7	4	65.4	5.6
	FAI	12	73.1	13.1	9	73.7	16.2	6	78.7	19.4
BMI (kg/m^2)*	Con	10	22.4	2.5	7	22.0	0.7	4	21.7	0.6
	FAI	12	24.7	3.5	9	24.8	4.1	6	26.1	4.9
Age	Con	10	31.7	6.1						
	FAI	12	30.9	7.6						

m=meters, kg=kilograms, BMI=body mass index, N=number, SD=standard deviation, *= FAI significantly higher BMI at Pre-Operative (P=0.05)and Three- (P=0.05) and Six-Months Post-Opreative (P=0.04).

Participant anthropometrics and age are found in table 2. A total of twenty-two participants, 10 controls (3 M, 7 F) and 12 FAI (1 M, 11 F), were available for the pre-operative session. Post-operatively, two FAI participants were unable to attend follow up sessions. One participant was excluded from the study after being diagnosed with a herniated disc of the lumbar spine. Another did not respond to attempts for post-operative scheduling and did not return for any follow-up appointments to her surgeon. No sessions were missed and participants will continue to be advanced prospectively.

There were no significant differences between groups for height, weight, or age at any time (table 2). Body mass index was significantly higher in the FAI group pre-operatively by $2.3 m/kg^2$ (d = 0.16), three-months by $2.8 m/kg^2$ (d = 0.96) and six-months by $4.4 m/kg^2$ (d = 1.60). Though not statistically significant, the FAI group weighed an average of 13.3 kg more than

controls at the six-month session (P = 0.08, d = 1.00), though this may stem from not all the participants having reached the six month session as of the completion of the findings presented.

FAI Clinical and Radiographic Data

All FAI participants pre-operatively displayed unilateral signs and symptoms (s/s) (nine right, three left) and had a mean duration of symptoms of 20.6 months ± 22.4 (range = 3-72 months). Eight had s/s of at least one year, while the remaining four had s/s no greater than six months. All 12 FAI participants had a positive flexion-adduction-internal rotation (FADIR) test, 25% had positive flexion-abduction-external rotation (FABER) tests, and 42% had positive straight leg tests. There were three cam and six pincer isolated deformities, as well as three with mixed pathologies. Therefore, in the 12 FAI participants, there were six cases of cam and nine cases of pincer deformities. There were no significant differences in the comparison between the inter-ASIS distance measured on radiographs versus tape measure (P = 0.32). The mean difference was only 5.6 mm. The cam deformities alpha angles were significantly reduced by 15.4°, as viewed on post-operative frog lateral films (P = 0.02, d = 1.66). There were seven instances of positive COS, three PWS, and five ISS in the nine pincer types. Both measurements of Pincer FAI improved, with the LCEA significantly decreased by 3.4° (P = 0.03, d = 0.78) and the AI increased by 0.9° (P = 0.32). One participant was missing post-operative radiographic assessment and was removed from this particular analysis.

Table 3. FAI Participant Clinical Findings

ID	Involved Limb	Deformity	S/S (months)	Inter-ASIS (mm) RG	Inter-ASIS (mm) TM		Clinical Exam FADIR	Clinical Exam FABER	Clinical Exam SLR
1	R	Mixed	3	305.7	300.0		1	0	1
3	R	Pincer	3	348.2	302.0		1	0	0
5	L	Cam	24	268.4	261.0		1	1	0
7	L	Mixed	13	304.0	290.0		1	1	0
8	R	Pincer	6	295.5	267.0		1	1	1
9	R	Pincer	12	371.0	309.0		1	0	0
10	L	Cam	72	339.5	345.0		1	0	1
11	R	Cam	24	312.0	296.0		1	0	0
12	R	Mixed	6	236.2	323.0		1	0	1
13	R	Pincer	12	278.4	320.0		1	0	0
14	R	Pincer	60	285.0	290.0		1	0	1
15	R	Pincer	12	297.2	271.0		1	0	0
Mean			20.6	303.4	297.8	#	12	3	5
Min			3.0	236.2	261.0				
Max			72.0	371.0	345.0				
SD			22.4	36.6	24.6				

L=Left, R=Right, S/S= Duration of Signs and Symptoms, Inter-ASIS= distance between Anterior Superior Iliac Spines, RG=Radiograph, TM=Tape Measure, FADIR=Flexion Adduction Internal Rotation Test, FABER=Flexion Abduction External Rotation Test, SLR= Straight Leg Raise Test, #= number of positive cases

Table 4. Cam FAI Participants Alpha Angles on Frog Lateral Radiographs

ID	Pre-Operative°	Post-Operative°	Δ
1	87	48	39
5	50	41	9
7	50	34	16
10	56	45	11
11	42	43	-1
12	58	40	18
Mean	57.2	41.8	15.3
Maximum	87	48	39
Minimum	42	34	-1
SD	15.7	4.8	13.4

Δ= Delta (Post- minus Pre-operative)

Table 5. Pincer FAI Participants Radiographic Data

	Pre-Operative		Post-Operative			
ID	LCEA°	AI°	LCEA°	AI°	LCEA° Δ	AI° Δ
1	29	9	24	7	5	2
3	35	-7	N/A	N/A	N/A	N/A
7	41	-1	31	1	10	2
8	31	3	33	2	2	1
9	33	3	25	11	8	8
12	33	10	31	8	2	2
13	48	-3	43	-3	5	0
14	36	3	37	1	1	2
15	33	4	33	6	0	2
Mean	35.4	2.3	32.1	4.1	4.1	2.4
Max	48	10	43	11	10	8
Min	29	-7	24	-3	0	0
SD	5.8	5.4	6.1	4.6	3.5	2.4

LCEA=Lateral Center Edge Angle, AI= Acetabular Inclination Angle, Δ= Delta (change from Pre- to Post-Operative. N/A= Patient did not have follow up radiographs

Patient Related Outcomes Surveys

All controls reported feeling "normal" at each session when asked to rate their current level of function. Pre-operatively the FAI group reported three as "nearly normal", six "abnormal", and three "severely abnormal" (table 6). The FAI group had significantly lower HOS_{ADL}, HOS_S, $CLOF_{ADL}$, and $CLOF_S$ at all three session versus controls (table 7). The pre-operatively session contained the largest effect sizes between groups for the HOS_{ADL} (d = 3.57), HOS_S (d = 3.86), $CLOF_{ADL}$ (d = 3.18), $CLOF_S$ (d = 4.24), and UCLA (d = 1.48).

There was a trend toward decreasing differences in the HOS_{ADL}, $CLOF_{ADL}$, and $CLOF_S$ surveys (d = 1.92-2.78) between groups at three-months. This was due to improvements in the HOS_{ADL} (12.9 points ± 15.2; P = 0.02 d = 1.80), $CLOF_{ADL}$ (15.0 points ± 19.2; P = 0.03 d = 1.65),

$CLOF_S$ (20.6 points ± 33.3; $P = 0.05$, $d = 1.30$) surveys in the FAI group. There was no change in the FAI HOS_S ($P = 0.32$) and UCLA ($P = 0.44$) scores three months post-operatively. One FAI participant reported a decline in function, moving from "nearly normal" to "abnormal", two remained unchanged, and four improved from "abnormal" to "nearly normal".

At six months, the FAI group was just as active as the controls, as indicated by similar UCLA scores ($P = 0.5$, $d = 0.05$). From the three to six-month sessions, the FAI group significantly improved HOS_{ADL} by 8.5 points (81.6 ± 14.2 to 90.2 ± 7.6; P = 0.05, $d = 1.85$), HOS_S by 30.1 points (55.1 ± 30.0 to 85.2 ± 21.1; $P = 0.04$, $d = 1.96$), and UCLA by 2.2 points (6.5 ± 1.2 to 8.7 ± 1.6; $P = 0.3$, $d = 2.32$). The $CLOF_{ADL}$ and $CLOF_S$ improved by an average of 11.7 and 23.3 points, however these were not statistically different between the three- and six-month sessions ($P = 0.13$ and $P = 0.06$, respectively). Three reported feeling normal, one remained nearly normal, one improved to feeling abnormal, and one declined to feeling abnormal.

Table 6. Self-Reported Normalcy

Participant	Pre-Operative	Three-Month	Six-Months
1	nearly normal	nearly normal	normal
3	abnormal	N/A	N/A
5	severely abnormal	severely abnormal	abnormal
7	nearly normal	abnormal	normal
8	abnormal	nearly normal	nearly normal
9	severely abnormal	normal	normal
10	abnormal	nearly normal	abnormal
11	nearly normal	nearly normal	N/A
12	abnormal	N/A	N/A
13	abnormal	nearly normal	N/A
14	abnormal	nearly normal	N/A
15	severely abnormal	N/A	N/A

Table 7. The Hip Outcome Score and UCLA Activity Scores in FAI and Control Groups

	Pre-Operative				
Group	HOS_{ADL}*	HOS_S*	$CLOF_{ADL}$*	$CLOF_S$*	UCLA*
FAI	63.6 ± 14.1	41.7 ± 21.3	55.8 ± 19.5	35.4 ± 24.0	6.2 ± 2.0
Controls	99.7 ± 1.0	100 ± 0	100 ± 0	100 ± 0	8.9 ± 1.9

	Three-Months Post-Operative				
Group	HOS_{ADL}*†	HOS_S*	$CLOF_{ADL}$*†	$CLOF_S$*†	UCLA*
FAI	80.0 ± 12.4	52.8 ± 25.8	71.7 ± 20.5	55.6 ± 27.6	6.2 ± 1.2
Controls	100 ± 0	100 ± 0	100 ± 0	100 ± 0	9.3 ± 1.1

	Six-Months Post-Operative				
Group	HOS_{ADL}*	HOS_S*	$CLOF_{ADL}$*	$CLOF_S$*	UCLA
FAI	90.2 ± 7.6	85.2 ± 21.1	85.0 ± 12.2	80.0 ± 17.9	8.7 ± 1.6
Controls	100 ± 0	100 ± 0	100 ± 0	100 ± 0	8.8 ± 1.9

*=Significantly Different Between Groups at *P<0.5*

†= FAI Group Significantly Different than FAI Pre-Operative Session at *P<.05*

‡= FAI Group Significantly Different than FAI 3 Month Session at *P<.05*

Hand Held Dynamometry

Pain

The only reported instances of pain with HHD came from the involved limb of the FAI group. Half of the FAI participants (6/12) experienced pain in at least one position during HHD at the pre-operative session and reported a mean pain of 4.8/10. Furthermore, 33% experienced pain with knee extension, 25% with hip flexion, extension, adduction, and IR, 16.7% with knee flexion and hip ER, and 8.3% with hip abduction. In the six with painful HHD, they averaged 3.5 painful positions, and one experienced pain in all eight positions.

Post-operatively, only three of nine participants (33%) experienced pain in only four positions at the three months session. All three had painful hip flexion (33%), two hip IR (22.2%), and one each of hip adduction and knee extension (11.1%). The averaged pain reported decreased to 1.9/10. By six-months, only one FAI participant had pain with HHD,

reporting 3/10 pain with hip flexion, hip IR, and knee extension. This person had not reported pain at either the pre-operative or the three-month sessions.

Strength Differences between Groups

The strength difference results between groups across all session are found in table 8. The FAI group was significantly weaker in seven of the eight positions pre-operatively ($P < 0.01$, effect size= 1.35-1.71). Only hip ER was similar between groups (controls 13.5 lbs. ± 8.1, FAI 9.0 ± 7.3), however this was approaching significance ($P = 0.09$). Overall, the FAI group only had 57% of the strength of controls across the eight positions (range = 52 - 66%), which equated to an average of 16.9 lbs. ± 6.8 lbs. of force (range = 4.5 - 28.3 lbs. of force).

The FAI group was weaker in six of the eight positions at the three-month session ($P < 0.05$, d =1.16 -1.49). Hip adduction was decreased in the FAI group, but this was trending toward significant difference and had a moderate effect size of $d = 0.49$ (23.5 lbs. ± 10.5 vs. 32.0 lbs. ± 9.7; $P = 0.06$). The strength of the FAI increased to 66% of the level of controls across those seven measures (range = 61% - 74%), as they increased their strength by an average of 14.5 lbs. ± 5.6 (range 8.5 - 24.4 lbs.). These three-month findings exclude hip external rotation, as the FAI group was stronger than controls (21.1 lbs. ± 9.6 vs. 13.5 lbs. ± 8.1; $P < 0.01$, $d = 0.85$) due to an FAI increase of 12.1 lbs. coupled with a 2.8 decrease from controls.

Six-months postoperatively, the FAI group was significantly weaker than controls in six of the eight positions, except for hip IR ($P = 0.31$) and ER ($P = 0.20$). In these six positions, the FAI individuals only had 67.7% of the strength of controls and were weaker by an average of

11.3 lbs. ± 10.4. When including hip IR and ER, their total strength improved to 83.9% of the overall strength of controls.

FAI Strength Changes Over Time

The FAI strength changes over time in both limbs can be found in table 9. Three-months after surgery the FAI improved their knee flexion (5.9 lbs., d = 0.93), hip IR (2.7 lbs., d = 1.55) and hip ER (11.1 lbs., d = 0.76) strength in their involved limb. Hip flexion (7.0 lbs., d = 0.52) and adduction (4.6 lbs., d = 0.48) moderately improved. At six months, hip flexion (d = 0.89) and knee extension (d = 1.93) significantly improved from the three-month session by 4.5 and 9.7 lbs., respectively. In the six participants who had reached the six-month session, only half of strength measures significantly improved and these were in the sagittal plane (flexion and extension at the hip and knee). None of the frontal and transverse motions significantly changed from the pre-operative values. Only hip flexion and external rotation pre-operatively were significantly lower compared to the uninvolved limb.

Table 8. Hand Held Dynamometry Between Groups (lbs. of force)

			Pre-Operative					Post-Operative									
								Three-Months					Six-Months				
						Group Difference					Group Difference					Group Difference	
Joint	Position	Group	N	Mean	SD	Lbs.	%	N	Mean	SD	Lbs.	%	N	Mean	SD	Lbs.	%
Hip	EXT	CON	10	46.3	9.0	-19.8*	57	7	44.1	10.9	-15.3†	65	4	48.4	11.7	-17.6*	64
		FAI	12	26.5	14.7			9	28.8	15.0			6	30.8	3.5		
	FLEX	CON	10	60.1	19.6	-28.3*	52	7	63.1	24.1	-24.4*	61	4	67.9	10.2	-24.5*	64
		FAI	12	31.8	14.0			9	38.8	10.7			6	43.3	11.4		
	ABD	CON	10	39.7	10.8	-15.6*	60	7	39.9	14.2	-12.0†	70	4	45.0	11.9	-14.8†	67
		FAI	12	24.1	8.5			9	28.0	9.6			6	30.2	6.3		
	ADD	CON	10	36.1	14.6	-17.2*	52	7	32.0	9.7	-8.5†	74	4	37.5	6.4	-10.7†	72
		FAI	12	18.9	9.2			9	23.5	10.5			6	26.8	8.2		
	IR	CON	10	25.6	5.5	-11.9*	53	7	26.8	7.0	-10.4*	61	4	14.9	4.2	1.7	111
		FAI	12	13.7	8.4			9	16.4	7.5			6	16.6	5.8		
	ER	CON	10	13.5	8.1	-4.5	66	7	10.7	4.6	10.5†	198	4	12.0	5.9	6.8	156
		FAI	12	9.0	7.3			9	21.1	9.6			6	18.8	8.6		
Knee	EXT	CON	10	48.4	13.9	-19.8*	59	7	53.2	18.0	-19.3†	64	4	60.0	6.4	-16.4*	73
		FAI	12	28.6	14.8			9	33.9	15.4			6	43.7	9.4		
	FLEX	CON	10	41.1	14.6	-18.7*	54	7	40.0	16.0	-11.6†	71	4	42.8	11.1	-15.0*	65
		FAI	12	22.4	12.6			9	28.3	9.7			6	27.8	6.1		
	Mean					-17.0	57				-14.5	66				-11.3	84
	SD					6.8	5				5.6	47				10.4	33

*=Significant Difference P<0.01,†= Significant Difference P<0.05

EXT=extension, FLEX= flexion, ABD=abduction, ADD=adduction, IR=internal rotation, ER=external rotation

Table 9. FAI Strength Over Time and Between Limbs (pounds of force)

			Pre-Operative			Three-Months			Six-Months	
Joint	Position	Limb	N	Mean	SD	N	Mean	SD	N	Mean	SD
Hip	Ext	U	12	30.3	11.1	9	29.6	12.5	6	30.8	6.9
		I	12	26.5	14.7	9	28.8	15.0	6	30.8 †	3.5
	Flex	U	12	37.1 *	11.3	9	39.0	10.1	6	41.0	5.1
		I	12	31.8	14.0	9	38.8	10.7	6	43.3 †‡	11.4
	Abd	U	12	26.7	8.3	9	27.4	10.2	6	34.0 *	5.8
		I	12	24.1	8.5	9	28.0	9.6	6	30.2	6.3
	Add	U	12	19.6	8.2	9	25.9	8.3	6	25.6	8.9
		I	12	18.9	9.2	9	23.5	10.5	6	26.8	8.2
	IR	U	12	12.6	6.4	9	17.5	9.5	6	19.5	7.5
		I	12	13.7	8.4	9	16.4 †	7.5	6	16.6	5.8
	ER	U	12	14.1 *	8.6	9	18.8	9.4	6	17.6	7.6
		I	12	9.0	7.3	9	21.1 †	9.6	6	18.8	8.6
Knee	Ext	U	12	32.0	9.3	9	37.4	12.9	6	41.5	7.2
		I	12	28.6	14.8	9	33.9	15.4	6	43.6 †‡	9.4
	Flex	U	12	24.1	9.6	9	26.9	12.1	6	28.1	6.8
		I	12	22.4	12.6	9	28.3 †	9.7	6	27.8 †	6.1

U=Uninvolved Limb, I=Involved Limb, Ext=extension, Flex=flexion, Abd=abduction, Add=adduction, IR=internal rotation, ER=external rotation

*=Significantly Different between limbs (P<0.05)

†=Significantly Different from Pre-Operative Session (same limb)(P<0.05)

‡= Significantly Different Than Three-Month Session (same limb)

Correlations of HOS and UCLA with HHD

The HOS_{ADL} and HOS_S were significantly and positively correlated ($r > 0.40$) to 83.3% of strength measures pre- and post-operatively (Table 10). Hip ER was inconsistent between data collection sessions, switching from small positive correlations pre-operatively, to moderate to large negative correlations post-operatively. Additionally, the UCLA was also positively correlated ($r > 0.398$) to seven of the eight strength measures pre-operatively and at the three-month post-operative session. However at six-months post-operatively, there were no significantly positive correlations between the UCLA and strength measures.

Table 10. Correlations between Strength Measures and the HOS and UCLA Surveys

			Pre-Operative			Three-Months			Six-Months		
Joint	Motion		HOS_{ADL}	HOS_{Sports}	UCLA	HOS_{ADL}	HOS_{Sports}	UCLA	HOS_{ADL}	HOS_{Sports}	UCLA
Knee	FLEX	r	.648**	.627**	.581**	.477*	.485*	.565*	.579*	.609*	0.351
		Sig.	0.00	0.00	0.00	0.03	0.03	0.01	0.04	0.03	0.16
		N	22	22	22	16	16	16	10	10	10
	EXT	r	.629**	.589**	.530**	.495*	.496*	.644**	.750**	0.265	-0.232
		Sig.	0.00	0.00	0.01	0.03	0.03	0.00	0.01	0.23	0.26
		N	22	22	22	16	16	16	10	10	10
Hip	FLEX	r	.637**	.620**	.560**	.598**	.629**	.681**	.795**	0.544	0.012
		Sig.	0.00	0.00	0.00	0.01	0.01	0.00	0.00	0.05	0.49
		N	22	22	22	16	16	16	10	10	10
	EXT	r	.743**	.763**	.711**	.610**	.655**	.595**	.607*	0.517	0.087
		Sig.	0.00	0.00	0.00	0.01	0.00	0.01	0.03	0.06	0.41
		N	22	22	22	16	16	16	10	10	10
	ABD	r	.672**	.678**	.660**	.479*	.432*	.551*	0.474	0.306	0.026
		Sig.	0.00	0.00	0.00	0.03	0.05	0.01	0.08	0.20	0.47
		N	22	22	22	16	16	16	10	10	10
	ADD	r	.626**	.614**	.527**	.611**	.553*	.608**	.603*	.722**	0.159
		Sig.	0.00	0.00	0.01	0.01	0.01	0.01	0.03	0.01	0.33
		N	22	22	22	16	16	16	10	10	10
	IR	r	.632**	.617**	.398*	.651**	.702**	.634**	0.453	0.52	0.094
		Sig.	0.00	0.00	0.03	0.00	0.00	0.00	0.10	0.06	0.40
		N	22	22	22	16	16	16	10	10	10
	ER	r	.375*	0.313	0.033	-0.366	-.435*	-.507*	-.663*	-.756**	-0.104
		Sig.	0.04	0.08	0.44	0.08	0.05	0.02	0.02	0.01	0.39
		N	22	22	22	16	16	16	10	10	10

** Correlation is significant at the 0.01 level, *Correlation is significant at the 0.05 level;
r=correlation coefficient, HOS=Hip Outcome Score, ADL=activites of daily living, UCLA=UCLA activity score,FLEX=flexion, EXT=extension, ABD=abduction, ADD=adduction, IR=internal rotation,

Gait Analysis

Walking Velocity

The control group walked significantly faster (meters/second) than the FAI group at both the pre-operative (1.57 m/s ± 0.2 vs. 1.31 m/s ± 0.1; $P < 0.01$, $d = 1.67$) and three-month sessions (1.60 m/s ± 0.2 vs. 1.41 m/s ± 0.1; $P = 0.01$, $d = 1.35$). The FAI group significantly improved their pre-operative walking velocity from 1.29 m/s ± 0.1 to a three-month velocity of 1.41 m/s ± 0.1 m/s ($P < 0.01$). At the six-month session, the controls remained faster, however this was not statistically significant (1.57 m/s ± 0.2 vs 1.42 m/s ± 0.1; P = 0.08, $d = 1.03$).

Walking Gait Differences

There were 14 kinematic and 12 kinetic pre-operative differences between groups (means, SD, and *P* values in table 11). FAI group had increased motion at the ankle and knee, but decreased motion at the hip and trunk. They displayed greater ankle dorsiflexion max angle (d = 0.87), excursion ($d = 1.15$), and mean velocity ($d = 1.00$) and greater knee flexion max angle ($d = 1.64$) and sagittal excursion ($d = 1.05$). At the frontal plane of the knee, the FAI group had greater knee varus excursion ($d = 1.07$) and maximum varus velocity ($d = 1.20$). Conversely, the FAI group walked with decreased hip sagittal excursion ($d = 0.90$), maximum hip extension velocity ($d = 0.77$), and reached maximum extension velocity earlier in stance ($d = 1.05$). At the trunk, the FAI group walked with less frontal plane pelvis excursion ($d = 0.79$) and spine excursion ($d = 0.94$) compared to controls.

All kinetic significant differences were lower in the FAI group compared with controls. Force related differences included maximal ground reaction force (GRF) ($d = 0.15$), loading rate

(*d* = 0.74), maximum breaking force (*d* = 1.17), and maximum propulsive force (*d* = 1.05). The external moment significant findings were ankle plantarflexion (PF) (*d* = 1.07), eversion (*d* = 1.27), and ER (*d* = 0.74), knee extension (*d* = 0.86), adduction (*d* = 1.08), IR (*d* = 0.73), and ER (*d* = 1.14), and hip ER (*d* = 0.81).

There were 14 kinematic and 8 kinetic differences between groups three-months post-operatively (Table 12). The FAI group again had increased ankle DF excursion (*d* = 0.98) and knee flexion excursion (*d* = 1.01), as they did pre-operatively. Maximum hip adduction (*d* = 1.18), maximum hip ER (*d* = 0.91), and maximum IR velocity (*d* = 1.02) were also increased versus controls. Conversely, the FAI group had decreased maximum knee varus (*d* = 1.35), hip sagittal excursion (*d* = 1.02), mean extension velocity (*d* = 1.57), and maximum abduction (*d* = 0.29), pelvis angle (*d* = 1.36), and spine angle (*d* = 1.15).

Kinetically, eight variables were decreased in the FAI group versus controls. Two were ground reaction forces and included maximum GRF (*d* = 1.71) and maximum breaking force (*d* = 1.53). The significantly lower external moments were maximum ankle PF moment (*d* = 1.00) and IR (*d* = 1.20), knee adduction (*d* = 2.88) and IR (*d* = 1.49), and maximum hip IR (*d* = 1.13).

Table 11. Pre-Operative Walking Gait Differences Between Groups

	Kinematics				
		Mean ± SD		t-test	
Joint	Variable	CON (n=10)	FAI (n=12)	t	P Value
Ankle	Max DF°	12.4 ± 3.1	15.1 ± 3.1	2.00	0.030
	DF Excursion°	7.5 ± 4.1	11.6 ± 3.0	2.73	0.007
	Mean DF V (°/s)	15.5 ± 7.5	21.8 ± 5.1	2.34	0.015
Knee	Max Flexion°	30.6 ± 4.6	37.4 ± 8.5	2.26	0.018
	Flexion Excursion°	33.1 ± 4.3	37.9 ± 4.8	2.47	0.012
	Max Flexion V (°/s)	381.1 ± 75.9	334.7 ± 46.5	1.76	0.047
	Varus Excursion °	3.8 ± 2.5	7.5 ± 4.4	2.31	0.016
	Max Varus V (°/s)	97.7 ± 24.6	156.9 ± 74.2	2.41	0.013
Hip	Sagittal Excursion°	49.0 ± 6.5	43.9 ± 4.8	2.10	0.025
	Extension V (°/s)	180.0 ± 27.2	156.4 ± 33.6	2.33	0.015
	Time Max Extension (%)	99 ± 0.3	82 ± 32	1.75	0.048
	Max ER°	8.4 ± 8.5	15.5 ± 10.2	1.77	0.047
Trunk	Pelvis Excursion°	12.6 ± 3.9	10.3 ± 1.9	1.83	0.041
	Spine Excursion°	17.0 ± 5.4	13.5 ± 2.0	2.12	0.024

	Kinetics				
		Mean ± SD		t-test	
	GRF	CON (n=10)	FAI (n=12)	t	P Value
	Max GRF (N/Kg)	11.9 ± 0.82	10.8 ± 0.63	3.68	0.001
	Loading Rate (N/S)	4365.8 ± 2010.0	2937.1 ± 1812.1	1.75	0.048
	Max Breaking (N/Kg)	2.45 ± 0.54	1.89 ± 0.41	2.79	0.006
	Max Propulsion (N/Kg)	2.33 ± 0.42	1.97 ± 0.26	2.49	0.011
		Mean ± SD		t-test	
Joint	Moments (Nm/Kg)	CON (n=10)	FAI (n=12)	t	P Value
Ankle	Max Plantarflexion	0.3 ± 0.01	0.2 ± 0.01	2.50	0.011
	Max Eversion	0.06 ± 0.03	0.02 ± 0.02	2.98	0.004
	Max External Rotation	0.02 ± 0.01	0.04 ± 0.03	1.70	0.053
Knee	Max Extension	0.5 ± 0.1	0.4 ± 0.1	2.01	0.029
	Max Adduction	0.9 ± 0.2	0.7 ± 0.2	2.54	0.010
	Max Internal Rotation*	0.2 ± 0.04	0.2 ± 0.04	1.71	0.052
	Max External Rotation	0.3 ± 0.01	0.2 ± 0.1	2.70	0.007
Hip	Max External Rotation	.02 ± 0.07	0.1 ± .06	1.91	0.036

DF=Dorsiflexion, V=velocity, N=Newtons, KG-kilograms, GRF=ground reaction force

Note: *IR actual values 157.0 N·mm/kg ± 38.8 vs. 186.6 N·mm/kg ± 42.1; P=0.05, *d* =0.73

There were eight kinematic and two kinetic differences between groups at six-months post-operatively (Table 13), with the FAI group having increased ankle and knee kinematic patterns. At the ankle, FAI group no longer utilized dorsiflexion as much as pre-and three-months sessions, and even had increased ankle PF (*d* = 1.50). However, the knee maintained greater max flexion (*d* = 1.54) and flexion excursion (*d* = 1.73) and decreased hip sagittal excursion (*d* = 1.09). In the transverse plane, controls favored hip IR (*d* = 1.78), while the FAI group utilized more hip ER (*d* = 1.45) and walked with a more externally rotated foot progression angle (*d* = 2.37). Kinetically, only max GRF (*d* = 1.33) and maximum breaking force (*d* = 1.09) were different between groups.

Table 12. Three-Months Post-Operative Walking Gait Differences Between Groups

Kinematics

Joint	Variable	Means ± SD CON (n=7)	Means ± SD FAI (n=9)	t-test t	t-test P Value
Ankle	DF Excursion°	7.0 ± 2.0	9.8 ± 3.8	1.783	0.048
	Ankle IR at TO°	12.4 ± 3.8	19.0 ± 8.3	1.906	0.039
Knee	Flexion Excursion°	30.7 ± 7.0	37.2 ± 5.8	2.035	0.031
	Knee Varus at HS°	3.5 ± 1.2	-1.2 ± 3.6	3.333	0.003
	Max Varus°	7.4 ± 1.9	3.2 ± 4.3	2.439	0.015
Hip	Sagittal Excursion°	51.2 ± 7.4	44.7 ± 5.3	2.056	0.030
	Time Max Extension (%)	100.0 ± 0.0	74.6 ± 38.1	1.745	0.052
	Hip Abduction at HS°	3.9 ± 2.4	0.8 ± 4.7	2.377	0.016
	Max Adduction°	5.6 ± 3.6	10.8 ± 5.2	2.239	0.021
	Max Abduction°	10.3 ± 3.1	4.2 ± 4.3	3.174	0.004
	Max External Rotation°	11.9 ± 9.1	21.0 ± 10.8	1.780	0.049
	Max IR V (°/s)	288.8 ± 85.6	461.5 ± 251.7	1.728	0.053
Trunk	Max Pelvis°	7.2 ± 1.7	4.0 ± 3.0	2.513	0.013
	Max Spine °	9.0 ± 2.2	5.2 ± 4.5	2.069	0.029

Kinetics

	GRF (N/Kg)	Means and SD CON (n=7)	Means and SD FAI (n=9)	t-test t	t-test P Value
	Max GRF	12.3 ± 1.0	11.1 ± 0.4	3.224	0.003
	Max Breaking	2.7 ± 0.5	2.1 ± 0.3	3.176	0.004

Joint	Moments (Nm/Kg)	Means and SD CON (n=7)	Means and SD FAI (n=9)	t-test t	t-test P Value
Ankle	Max Plantarflexion	0.3 ± 0.1	0.2 ± 0.1	1.866	0.042
	Max Internal Rotation	0.2 ± 0.1	0.1 ± 0.1	2.398	0.016
Knee	Max Adduction	1.0 ± 0.2	0.7 ± 0.1	3.162	0.004
	Max Internal Rotation	0.2 ± 0.0	0.1 ± 0.1	2.812	0.007
	Max External Rotation	0.03 ± 0.02	0.02 ± 0.01	2.038	0.031
Hip	Max Internal Rotation	0.20 ± 0.03	0.14 ± 0.06	2.408	0.015

TO= toe-off, HS= heel-strike, GRF= ground reaction force, IR= Internal Rotation

Table 13. Six-Months Post-Operative Walking Gait Differences Between Groups

	Kinematics				
		Mean ± SD		t-test	
Joint	Variable	CON (n=4)	FAI (n=6)	t	P Value
Ankle	Max Plantarflexion°	15.2 ± 5.1	22.2 ± 4.2	2.363	0.023
	ER Foot Progression°	1.8 ± 1.9	8.1 ± 3.4	3.371	0.005
Knee	Max Flexion°	32.1 ± 3.1	40.9 ± 8.3	1.994	0.041
	Flexion Excursion°	33.4 ± 1.5	40.6 ± 6.7	2.081	0.036
Hip	Sagittal Excursion°	49.3 ± 6.0	43.6 ± 4.4	1.758	0.059
	Max Abduction°	8.9 ± 2.4	6.1 ± 2.4	1.794	0.050
	Max Internal Rotation°	9.0 ± 3.6	1.7 ± 4.6	2.664	0.015
	Max External Rotation°	11.2 ± 5.0	20.7 ± 8.1	2.063	0.037

	Kinetics				
		Mean ± SD		t-test	
	GRF (N/Kg)	CON (n=4)	FAI (n=6)	t	P Value
	Max GRF	12.2 ± 1.0	11.2 ± 0.5	2.164	0.031
	Max Breaking	2.7 ± 0.6	2.1 ± 0.3	2.068	0.036

ER=external rotation, GRF=ground rection force

FAI Walking Gait Changes of the Involved Limb

There were 22 significant changes, 11 kinematic and 11 kinetic, within the nine involved limbs of FAI participants from the pre-operative to three-month session (Table 14). They decreased their ankle DF excursion (*d* = 1.11), mean DF velocity (*d* = 1.55), and foot progression angle (*d* = 0.78) three-months post-operatively. At the knee, varus angle (*d* = 1.32), varus excursion (*d* = 0.84), and timing of max varus (*d* = 1.78) were also decreased at three-months. Conversely, the FAI involved limb increased max ankle PF (*d* = 1.73) mean knee flexion velocity (*d* = 0.39), and hip sagittal excursion (*d* = 0.80), mean extension velocity (*d* = 0.87) and max adduction velocity (*d* = 0.77) at the three-month session.

The nine FAI participants saw an increase in 11 kinetic variables at three-months. They increased their max GRF (*d* = 0.64), loading rate (*d* = 0.76) max breaking force (*d* = 0.89), and max propulsion (*d* = 1.75) forces. Five external moments were increased, which included ankle DF (*d* = 0.96) and eversion (*d* = 0.70), knee flexion moment (*d* = 1.07), and hip flexion (*d* = 1.81), adduction (*d* = 1.44), and ER (*d* = 0.67).

Table 14. Significant Gait Changes of FAI Involved Limbs During Walking at Three-Months (n=9)

	Kinematics									
		Pre-Operative		Three-Months		Paired Differences			
Joint	Variable	Mean	SD	Mean	SD	Mean	SD	t	P value
Ankle	Max Plantar Flexion	15.8 ±	6.5	19.7 ±	6.8	3.9	2.3	5.103	0.001
	Dorsiflexion Excursion	12.3 ±	3.3	9.1 ±	3.4	-3.2	3.0	3.250	0.006
	Mean DF V (°/s)	22.8 ±	5.4	17.6 ±	6.1	-5.2	6.5	2.417	0.021
	ER Foot Progression°	6.2 ±	4.7	4.0 ±	3.5	-2.2	3.0	2.222	0.029
Knee	Max Flexion V (°/s)	326.0 ±	46.1	355.5 ±	31.5	29.5	34.7	2.549	0.017
	Max Varus	8.3 ±	6.8	3.2 ±	4.3	-5.1	4.4	3.444	0.005
	Time Max Varus	92.8 ±	21.4	70.5 ±	37.4	-22.3	33.4	2.001	0.040
	Varus Excursion	8.5 ±	4.6	4.5 ±	3.6	-4.0	4.8	2.516	0.018
Hip	Sagittal Excursion	43.3 ±	5.3	44.5 ±	5.1	1.2	1.5	2.352	0.024
	Mean Extension V (°/s)	76.3 ±	7.5	82.0 ±	8.6	5.7	6.6	2.592	0.016
	Max Adduction V (°/s)	74.2 ±	10.8	86.6 ±	15.4	12.4	16.5	2.253	0.027

	Kinetics								
		Pre-Operative		Three-Months		Paired Differences			
	GRF (N/Kg)	Mean	SD	Mean	SD	Mean	SD	t	P value
	Max GRF	10.7 ±	0.6	11.2 ±	0.5	0.5	0.8	1.876	0.049
	Loading Rate (N/s)	2974.7 ±	2044.3	4266.9 ±	2127.7	1292.3	1699.3	2.281	0.026
	Max Breaking	1.9 ±	0.5	2.1 ±	0.3	0.2	0.4	2.064	0.037
	Max Propulsion	1.9 ±	0.3	2.3 ±	0.3	0.3	0.2	4.516	0.001

		Pre-Operative		Three-Months		Paired Differences			
Joint	Moments (Nm/Kg)	Mean	SD	Mean	SD	Mean	SD	t	P value
Ankle	Max Dorsiflexion	1.42 ±	0.17	1.51 ±	0.01	0.09	0.01	2.549	0.017
	Max Eversion	0.003 ±	0.002	0.005 ±	0.003	0.002	0.003	2.004	0.040
Knee	Max Flexion- loading	0.02 ±	0.01	0.04 ±	0.02	0.01	0.02	2.464	0.020
Hip	Max Flexion	1.05 ±	0.04	1.22 ±	0.05	0.02	0.02	3.105	0.008
	Time Max Extension (%)	73.1 ±	24.7	60.2 ±	31.5	-12.8	20.9	1.849	0.051
	Max Adduction	1.04 ±	0.01	1.18 ±	0.02	0.14	0.01	3.819	0.003
	Max External Rotation	0.01 ±	0.01	0.01 ±	0.01	0.00	0.01	1.993	0.041

DF= dorsiflexion, V=velocity, GRF= ground reaction force

There were six FAI participants with both three- and six-month data for comparison. There were five variables, one kinematic and four kinetic, that changed. Knee IR position at toe-off decreased (17.3° ± 4.8 to 10.9° ± 3.1; *P* = 0.01, *d* = 1.57), whereas max breaking force (2.1 n/kg ± 0.3 from 2.0 n/kg ± 0.4; *P* = 0.05, *d* = 0.88), max propulsion force (2.3 n/kg ± 0.3 from 2.2 n/kg ± 0.4; *P* = 0.04, *d* = 1.03), knee ER moment (0.003 Nm/kg ± 0.002 from 0.002 Nm/kg ± 0.001; *P* = 0.05, *d* = 1.14), hip abduction moment (0.21 Nm/kg ± 0.01 from 0.16 Nm/kg ± 0.01; *P* = 0.03, *d* = 0.57) increased.

Running Gait Group Differences

Two FAI participants were unable to run pre-operatively secondary to hip joint pain and both were able to run six months post-operatively without pain. The FAI and control groups had 15 (eight kinematic, seven kinetic) pre-operative significant differences (table 15). Only hip transverse plane excursion was different between groups during the swing phase, with the FAI group utilizing more ROM than controls (*d* = 1.21). During stance phase, the FAI group had six decreased variables, with only eversion excursion (*d* = 0.79) occurring at the ankle. At the hip and pelvis, the FAI group displayed decreased frontal plane motions versus controls. Abduction (*d* = 0.79), excursion (*d* = 0.92), and abduction at toe-off (*d* = 0.82) were decreased at the hip, while the pelvis lacked upward tilt (*d* = 0.85) and excursion (*d* = 1.14). Conversely, the FAI group were more flexed at the knee at toe-off (*d* = 1.11) and had larger knee frontal plane motions, which included knee varus excursion (*d* = 0.75), max varus velocity (*d* = 1.20), and mean varus velocity (*d* = 0.92). In the transverse plane the FAI's had greater knee IR position at toe-off (*d* = 1.08) and max hip ER (*d* = 0.77).

Kinetically, the FAI group had decreased max GRF (*d* = 1.57) and max propulsion (*d* = 1.00) forces. Two moments, ankle DF (*d* = 0.81) and knee adduction (*d* = 0.73) were also decreased. The FAI group had three increased moments (one ankle, two hip) compared to controls. They were max knee flexion during loading (*d* = 0.79), hip extension (*d* = 1.50), and hip max IR moment (*d* = 1.14). Additionally, the max hip extension moment occurred earlier in stance (*d* = 0.96).

Table 15. Pre-Operative Running Gait Significant Differences Between Groups

	Kinematics						
		CON (n=10)		FAI (n=10)		t-test	
Joint	Variable	Mean	SD	Mean	SD	t	P Value
Ankle	Eversion Excursion°	32.3	± 6.3	25.9	± 9.8	1.748	0.049
	Plantarflexion ° TO	2.2	± 4.8	10.0	± 9.0	2.413	0.013
Knee	Varus Excursion°	5.1	± 2.6	7.4	± 3.5	1.642	0.059
	Max Varus V (°/s)	210.0	± 54.6	386.4	± 237.3	2.29	0.017
	Mean Varus V(°/s)	63.2	± 33.4	105.8	± 58.4	2.025	0.029
	Knee Varus ° TO	2.9	± 5.6	9.4	± 6.4	2.475	0.012
Hip	Max Abduction °	6.1	± 5.9	2.3	± 3.7	1.763	0.047
	Frontal Plane Excursion°	12.9	± 3.2	9.4	± 4.4	2.061	0.027
	Hip Abduction ° TO	6.0	± 5.9	2.0	± 3.8	1.884	0.038
	Mean Abduction V (°/s)	56.8	± 12.2	43.2	± 18.6	1.948	0.033
	Max External Rotation°	7.8	± 8.8	15.2	± 10.5	1.728	0.050
Pelvis	Max Upward Tilt°	7.8	± 2.2	5.8	± 2.5	1.961	0.033
	Frontal Excursion°	14.3	± 2.9	10.7	± 3.4	2.578	0.009

	Kinetics						
		CON (n=10)		FAI (n=10)		t-test	
	GRF (N/Kg)	Mean	SD	Mean	SD	t	P Value
	Max GRF	26.0	± 2.3	23.0	± 1.5	3.645	0.001
	Max Propulsion	3.7	± 0.4	3.3	± 0.4	2.356	0.015
Joint	Moments (Nm/Kg)	Mean	SD	Mean	SD	t	P Value
Ankle	Max Dorsiflexion	2.91	± 0.03	2.66	± 0.35	1.828	0.042
Knee	Max Flexion - Loading	0.03	± 0.15	0.37	± 0.14	1.812	0.043
	Max Adduction	2.3	± 0.4	2.0	± 0.4	1.665	0.056
Hip	Max Extension	1.6	± 0.6	2.9	± 1.2	3.215	0.003
	Time of Max Extension (%)	70.9	± 22.8	52.0	± 16.5	2.191	0.021
	Max Internal Rotation	0.02	± 0.02	0.10	± 0.01	2.145	0.023

TO= toe-off, V=velocity, GRF= ground reaction force,

The number of significant variables between groups at six-months increased to 25 (17 kinematic, 8 kinetic) from 15 pre-operatively (table 16). During swing phase, the controls had more IR (*d* = 2.08), whereas the FAI group used more ER (*d* = 1.67). The remaining significant variables occurred during stance phase and many were the same findings as compared with the pre-operative differences between groups. There was a pattern of increased frontal plane knee motion in the FAI groups, as well as decreased hip and trunk frontal plane motions. These included increased knee varus position at heel strike (*d* = 0.55), max varus (*d* = 1.25), and varus position at toe-off (*d* = 1.32) and decreased hip abduction (*d* = 2.09), pelvis upward tilt (*d* = 1.80), pelvis excursion (*d* = 1.26), and max spine angle (*d* = 1.20). The FAI group also demonstrated increased foot progression angle (*d* = 3.26), a more flexed knee at heel-strike (*d* = 2.67), max hip ER (*d* = 2.12) and ER at toe-off (*d* = 0.87).

The FAI group had five decreased and four increased kinetic variables versus controls at the six-month session. Max GRF (*d* = 3.57), max DF moment (*d* = 2.00), max ankle IR moment (*d* = 1.38), knee adduction moment (*d* = 1.52), hip ER moment (*d* = 1.44). Increased max knee flexion moment loading (*d* = 1.82), knee stiffness (*d* = 2.2), hip extension moment (*d* = 1.61), hip IR moment (*d* = 2.00).

FAI Running Gait Involved Limb Changes

The FAI group's involved limb decreased ten (seven kinematic, three kinetic), and increased five (three kinematic, two kinetic) variables, at the six-month session compared with pre-operative measures (Table 17). Of the seven kinematic decreases, two were at the ankle and five were at the knee. These include max ankle DF (*d* = 1.56) and DF excursion (*d* = 1.05), as well as knee max flexion (*d* = 1.48), max flexion velocity (*d* = 1.14), mean flexion velocity (*d* =

1.59), and max varus velocity (*d* = 1.98). Decreased kinetic moments were ankle DF (*d* = 5.13), knee adduction (*d* = 0.94), and hip extension (*d* = 1.66). The FAI group's involved limbs increased in knee IR at heel-strike (*d* = 1.10), hip ER at toe-off (*d* = 0.93), and max thorax trunk lateral lean (*d* = 0.94). Kinetically, GRF impulse (*d* = 4.40) and ankle eversion moment (*d* = 1.05).

Table 16. Running Gait Significant Differences Between Groups at Six-Months

	Kinematics							
	Variable	CON (n=4)			FAI (n=6)		t-test	
		Mean		SD	Mean	SD	t	P Value
Ankle	Max Dorsiflexion°	31.3	±	1.2	28.2 ±	2.3	2.392	0.022
	ER Foot Progression°	3.1	±	1.1	11.9 ±	4.3	3.966	0.002
Knee	Flexion at HS°	12.0	±	1.6	18.4 ±	3.2	3.656	0.003
	Max Flexion V (°/s)	605.0	±	108.8	494.5 ±	44.4	2.272	0.027
	Mean Flexion V (°/s)	322.9	±	32.5	256.6 ±	44.9	2.525	0.018
	Varus at HS°	-2.0	±	2.3	4.3 ±	6.0	1.981	0.042
	Max Varus V (°/s)	183.5	±	86.9	265.2 ±	125.2	1.126	0.147
	Varus at TO°	3.7	±	6.6	12.0 ±	6.8	1.898	0.047
Hip	Max Abduction	7.0	±	2.1	2.6 ±	2.1	3.250	0.006
	Max Internal Rotation°	9.2	±	5.6	-1.5 ±	3.5	3.731	0.003
	Max External Rotation°	7.5	±	5.0	22.9 ±	9.5	2.929	0.010
	External Rotation TO°	1.5	±	5.7	12.8 ±	8.4	2.326	0.024
Trunk	Pelvis Frontal Tilt°	8.5	±	1.1	4.8 ±	3.0	2.338	0.024
	Pelvis Frontal Excursion°	13.7	±	1.9	10.3 ±	3.5	1.757	0.059
	Max Frontal Spine°	14.0	±	2.7	9.6 ±	4.6	1.709	0.063

	Kinetics							
		CON (n=10)			FAI (n=10)		t-test	
	GRF (N/Kg)	Mean		SD	Mean	SD	t	P Value
	Max GRF	27.1	±	1.3	22.1 ±	1.5	5.542	0.001
		CON (n=4)			FAI (n=6)		t-test	
	Moments (Nm/Kg)	Mean		SD	Mean	SD	t	P Value
Ankle	Max Dorsiflexion	2.95	±	0.28	2.44 ±	0.23	3.112	0.007
	Max Internal Rotation	0.68	±	0.17	0.48 ±	0.12	2.189	0.030
Knee	Max Flexion- Loading	0.16	±	0.13	0.57 ±	0.32	2.404	0.022
	Knee Stiffness (Nm/°)	2.30	±	0.78	5.05 ±	1.72	2.964	0.009
	Max Adduction	2.27	±	0.16	1.54 ±	0.80	1.769	0.058
Hip	Max Extension	1.38	±	0.54	2.61 ±	0.99	2.258	0.027
	Max Internal Rotation	0.01	±	0.03	0.07 ±	0.03	2.977	0.009
	Max External Rotation	0.52	±	0.08	0.39 ±	0.10	2.038	0.038

TO= toe-off, HS= heel-strike, V=velocity, GRF=ground reaction force

Table 17. Running Gait Stance Changes of FAI Participants at Six-Months

	Kinematics						
		Pre-Operative		Six-Months		Matched Pairs t-test*	
Joint	Variable	Mean	SD	Mean	SD	t	P Value
Ankle	Max DF °	33.0	± 5.3	28.1	± 2.6	2.722	0.027
	DF Excursion °	21.4	± 3.9	14.9	± 5.1	2.311	0.041
Knee	Max Flexion°	44.5	± 4.9	40.8	± 3.3	2.885	0.023
	Max Flexion V (°/s)	598.1	± 89.6	500.2	± 47.1	2.382	0.038
	Mean Flexion V (°/s)	333.3	± 51.0	265.7	± 43.6	3.517	0.013
	Varus° at HS	-1.5	± 3.7	3.3	± 6.1	2.380	0.038
	Max Varus V (°/s)	472.6	± 251.2	260.5	± 139.4	3.132	0.018
	IR ° at HS	9.0	± 9.3	14.6	± 8.4	4.595	0.005
Hip	ER ° at TO	6.2	± 7.9	14.6	± 8.0	2.086	0.053
Thorax	Max Thorax Tilt°	4.0	± 3.4	5.8	± 2.7	2.014	0.057

	Kinetics						
		Pre-Operative		Six-Months		Matched Pairs t-test*	
	GRF	Mean	SD	Mean	SD	t	P Value
	GRF Impulse (N/s)	213.7	± 54.5	225.0	± 60.4	4.238	0.007
		Mean	SD	Mean	SD	t	P Value
Joint	Moments (Nm/Kg)	213.7	± 54.5	225.0	± 60.4	4.238	0.007
Ankle	Max DF	2.6	± 0.2	2.4	± 0.2	4.635	0.005
	Max Eversion	0.07	± 0.09	0.12	± 0.08	2.292	0.042
Knee	Max Adduction	2.3	± 0.5	1.5	± 0.9	3.661	0.011
Hip	Max Extension	3.6	± 1.0	2.6	± 1.1	3.588	0.012

DF=Dorsiflexion, V=Velocity, HS=Heel-strike, IR=Internal Rotation, ER=External Rotation, TO=toe-off, GRF=ground reaction force

Note: *= Five Participants had pre- and six-months data

Abnormal Reversals

The participant and trial prevalence of abnormal hip flexion reversals can be found in table 18. There were no significant difference in the location or duration of reversals between groups in walking (Table 19) or running (Table 20). In addition, there were no significant differences in the hip or ankle positional changes during reversals at any time point for walking

or running. The controls did have greater knee flexion during walking pre-operatively (P = 0.05) and six months post-operatively during running (P = 0.03).

Table 18. Walking and Running Abnormal Reversal Prevalence

		Walking					
		Pre-Operative		Three-Months Post		Six-Months Post	
	Overall Prevalence	Control	FAI	Control	FAI	Control	FAI
	Participants	8/10	9/12	5/7	6/9	3/4	5/6
	% of Trials	48%	48%	26%	62%	50%	56%
Limb	Within AR Group	Control	FAI	Control	FAI	Control	FAI
Involved	Participants	N/A	8/9	N/A	6/6	N/A	5/5
	% of Trials	N/A	71%	N/A	70%	N/A	83%
Uninvolved	Number of Subjects	8/8	7/9	5/5	5/6	3/3	4/5
	% of Trials	60%	54%	57%	90%	67%	83%

		Running			
		Pre-Operative		Six-Months Post	
	Overall Prevalence	Control	FAI	Control	FAI
	Participants	8/10	10/11	3/4	6/9
	% of Trials	72	88	33%	67%
		Pre-Operative		Six-Months Post	
Limb	Within AR Group	Control	FAI	Control	FAI
Involved	Participants	N/A	9/10	NA	5/6
	% of Trials	N/A	90%	N/A	87%
Uninvolved	Participants	8/8	10/10	3/3	5/6
	% of Trials	90%	87%	44%	73%

AR=abnormal reversals

Table 19. Abnormal Walking Reversals Data Between Groups

		Pre-Operative			Three-Months			Six-Months		
Variable	Group	N	Mean	SD	N	Mean	SD	N	Mean	SD
Hip Δ°	CON	7	1.0	0.89	5	0.84	0.79	2	1.64	0.08
	FAI	9	0.7	0.63	8	1.25	0.79	5	1.33	0.85
Knee Δ°	CON	7	9.1	4.49	5	7.68	4.10	2	13.27	3.44
	FAI	9	5.7	3.09	8	8.96	2.50	5	9.70	2.50
Ankle Δ°	CON	7	1.0	3.81	5	2.20	2.19	2	1.77	0.52
	FAI	9	1.7	2.52	8	2.63	2.78	5	3.02	4.07
Location	CON	7	10.0	3.8	5	11.1	11.4	2	10.0	2.8
(% Stance)	FAI	9	8.8	4.6	8	9.6	2.9	5	9.4	3.1
Duration	CON	7	6.0	2.9	5	4.0	2.1	2	7.0	1.4
(% Stance)	FAI	9	5.5	2.5	8	5.5	1.5	5	5.8	1.6

Δ°=difference between min and max angles during reversals

Table 20. Abnormal Reversals Data During Running Between Groups

Variable	Pre-Operative				Six-Months		
	Group	N	Mean	SD	N	Mean	SD
Hip Δ°	CON	8	3.1	2.4	3	1.4	0.8
	FAI	10	3.8	4.0	6	1.5	1.2
Knee Δ°	CON	8	17.3	5.3	3	16.1	2.6
	FAI	10	15.6	5.7	6	11.4	3.2
Ankle Δ°	CON	8	11.2	4.1	3	9.1	5.2
	FAI	10	11.7	3.7	6	10.1	4.4
Location (% Stance)	CON	8	20.4	4.5	3	18.9	3.7
	FAI	10	18.7	2.7	6	18.7	4.7
Duration (% Stance)	CON	8	13.3	3.8	3	12.0	0.0
	FAI	10	11.7	3.6	6	10.5	3.6

Δ°=difference between min and max angles during reversals

Discussion

The findings from this study indicate that FAI participants show many signs of improvement three- and six-months after surgery, but still have many deficits compared to healthy controls. Radiographic improvement was found for both cam and pincer type FAI, which is an important initial step, since some of the highest rates for hip arthroscopy failure are related to residual deformity[29, 60, 239]. The greatest improvements by three months were in the HOS_{ADL} and decreased pain during HHD testing. The only FAI participant that had pain at six-months complained of a recent minor setback, which did not preclude her from completing the test procedures secondary to pain. The PRO surveys and walking gait were mostly parallel between groups by six-months, but there was still improvement needed in their hip and knee strength and running gait at six-months.

The HOS scores were similar to recently published values from larger samples both pre-operatively[75, 103, 130] and three-[75] and six-months[75, 130] post-operatively (Figure 1.) (Note: in all figures controls are blue and FAI are green). The minimal clinically important difference (MCID) has been estimated to be changes of five in the HOS_{ADL} and six in the HOS_S[146]. The HOS_{ADL} in our study more than tripled the MCID at three months (16.4 point improvement) and doubled by six-months (10.2 points improvement). The HOS_S almost doubled the MCID by three-months (11.1 point improvement), but was more than five times larger between three- and six-months (33.4 point improvement). This increase in HOS_S coincided with the large increase in UCLA activity score. Both groups reported an average current activity of 8/10, which is "I regularly participate in active activities such as fast walking, golf, or bowling". The FAI participants were

on post-operative restrictions until six-months, so this may provide some evidence of precaution adherence.

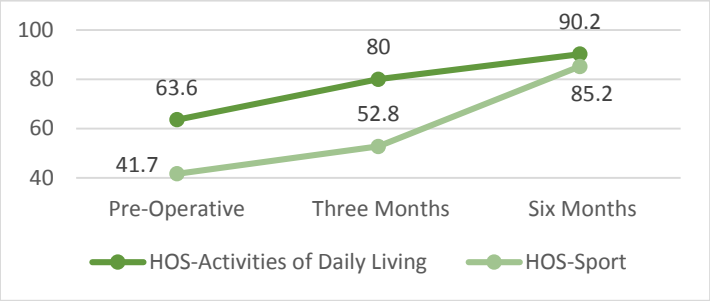


Figure 1. The Hip Outcome Score in FAI participants.

The FAI groups' hip and knee strength also improved at both the three- and six-month post-operative sessions. The hip rotators and knee flexor muscles plateaued in strength by three-months, as none significantly changed between the three- and six-month sessions. The hip flexors, hip extensors, and knee extensors did not improve until the six-month session. The only study to collect post-operative strength measures in FAI patients had a single follow-up time at 2.5 ± 0.2 years post-operatively[47], so direct comparison is difficult. Casartelli et al. found that only the hip flexors failed to return control levels, whereas in our study only hip IR and ER were similar between groups at six-months. It's possible that strength recovery will take extended periods of time for several reasons. The FAI post-operative precautions included no running or impact activities until six-months, which was reflected by lower UCLA scores (Figure 2). Because of these

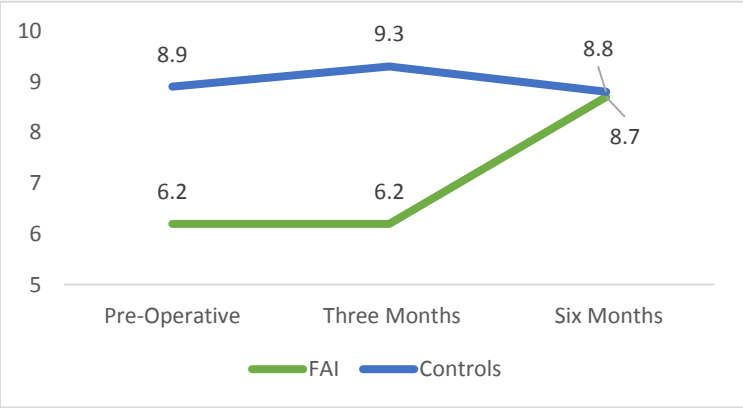


Figure 2. The UCLA Activity Score in FAI and Control Groups.

restrictions, it may have been difficult to improve sport related strength and satisfaction with physical therapy alone, as the quantity and quality of rehabilitation was not measured. Furthermore, those with decreased satisfaction in ADL and sport performance were also likely to be weaker, as indicated by the correlations between HOS scores and strength measures. Since strength is necessary for adequate ADL and sport performance, this may further validate the HOS as a useful clinical measure in hip arthroscopy patients. These strength and PRO deficits may also help explain some of the findings from the gait analyses.

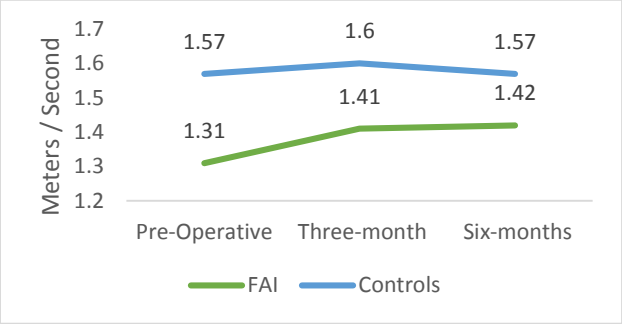


Figure 3. Walking Velocity in FAI and Controls.

There were many similar findings during walking and running gait and others that were unique to each trial type. The FAI group had a decreased walking velocity at all three sessions (Figure 3); making interpretation of the kinetic findings more difficult. Since gait is powered by the hip pulling into flexion and the ankle pushing at toe-off, a decrease in one will increase demand for the other[175]. If these demands are not met, then gait velocity is likely to decrease. This effect did not carry over to running, as velocity was set at 4.0 m/s ± 10%.

Pain avoidance was likely the initial motive for the decreased walking velocity, but even after pain subsided, the FAI walking velocities only slightly improved at the three-month session compared to controls. It is possible the controls were marginally faster walkers than the FAI group, even though both were in a normal range of velocity. It is also possible that this slower walking is a learned compensation during the recovery process, as the FAI group was 10% slower at six-months. The implication of these slower velocities may be specific gait changes.

Pre-operatively there was decreased hip extension velocity and earlier max hip extension angle than controls and sagittal hip excursion (Figure 4) was decreased at all three sessions. This may have limited capability of the hip flexors eccentric action during the second half of stance.

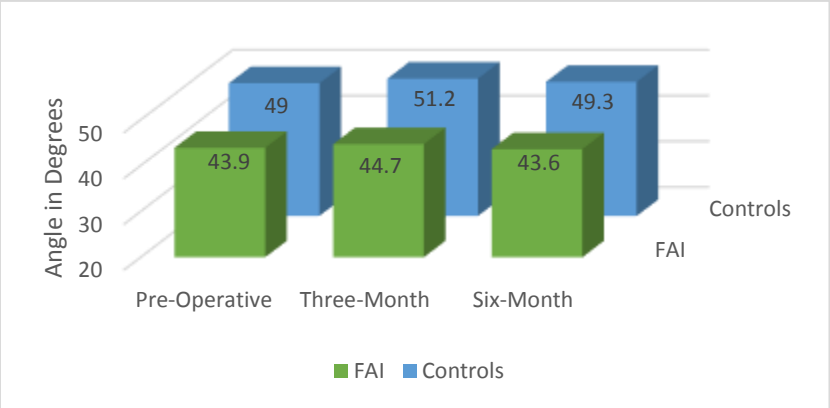


Figure 4. Sagittal Plane Hip Excursion during Walking. Note: all significantly different between groups (P<0.05)

There was also increased DF kinematics (Figure 5) in the FAI group compared to controls during walking, that was likely related to the decreased hip motion. The kinetic product of these hip and ankle findings were the decreased propulsive and breaking forces (Figure 6). Post-operatively, the FAI group significantly increased their max PF angles, possibly due to the reluctance to utilize hip ROM. This may have developed in order to increase walking velocity, that was found between the pre-operative and three-month sessions (Figure 3).

The FAI also utilized more knee flexion excursion at all three sessions during walking versus controls. Quadriceps weakness eccentrically leads to increased knee flexion during weight acceptance, and the knee extensor strength in the FAI group did not improve until the

six-month session. Ankle PF and knee flexion at six-months was greater in the FAI group, propulsive forces were similar between groups at six-months, but hip ROM remained lower.

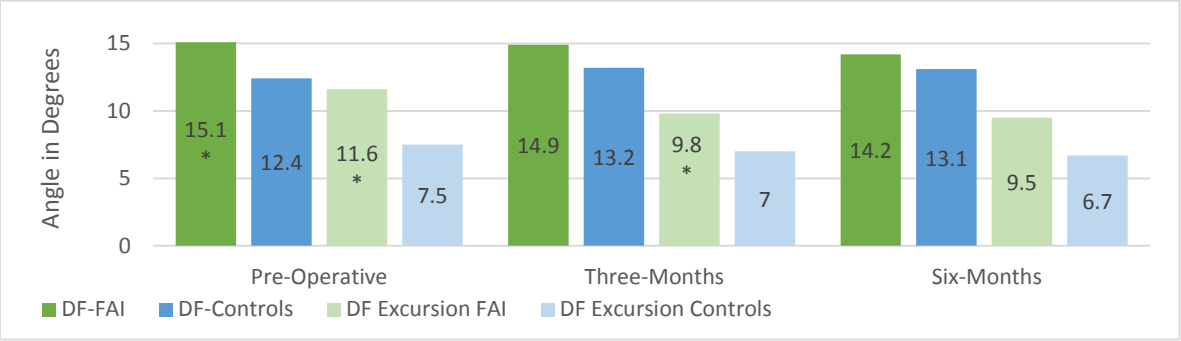


Figure 5 Ankle Dorsiflexion (DF) and Dorsiflexion Excursion.=significant difference between groups (P<0.05)*

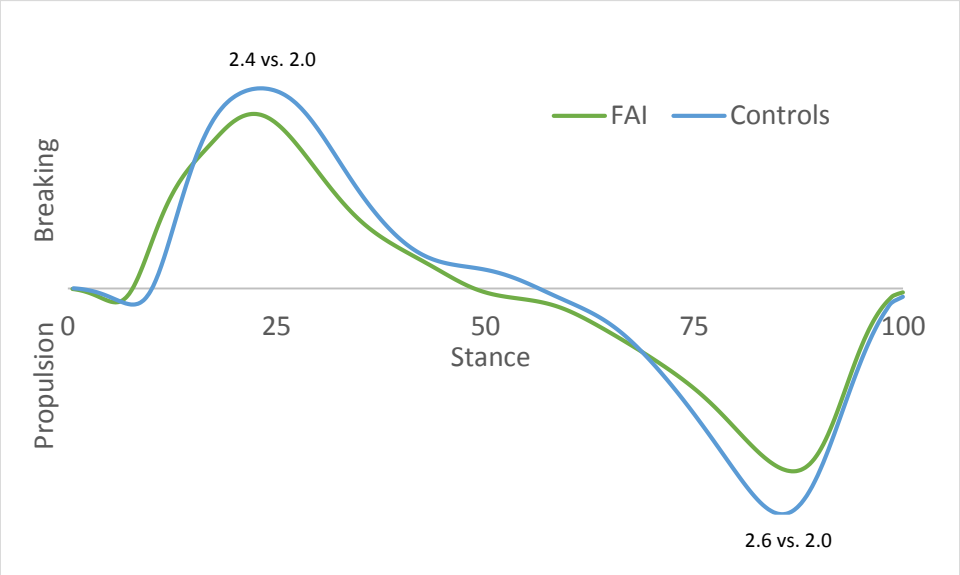


Figure 6. Representative Breaking and Propulsive Forces (N/Kg) in FAI and Control Participants during Walking Pre-Operatively

Sagittal hip motion was relatively equal between groups during running. This was particularly true for hip excursion pre-operatively (FAI: 77.1 ± 11.2, Controls: 76.5; $P = 0.44$) and post-operatively (FAI: 75.6 ± 11.2, Controls: 70.5 ± 7.3; $P = 0.22$). It is possible that when self-selecting walking velocity, they choose to minimize hip usage. When forced into faster

velocities of running, they have no choice but to use full ROM of the hip to accomplish locomotion, since the hip flexors and extensors and knee extensors are responsible for the increased stride frequency by rapidly accelerating the legs in the air faster[76].

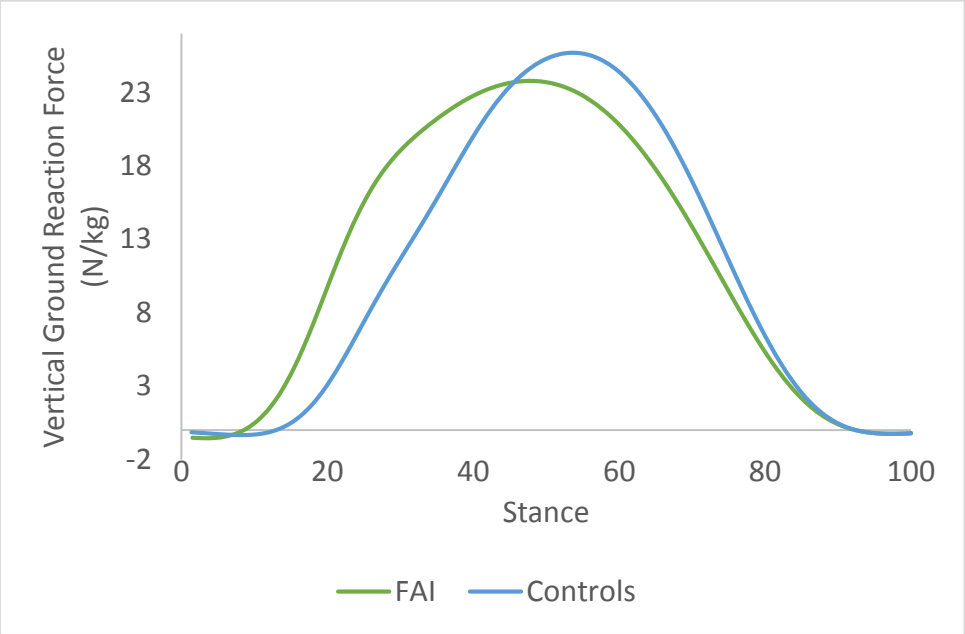


Figure 7. Representative Max Vertical GRF in FAI and Control Participants during Running

Note: Peak values were different between groups (P<0.05) and impulse was similar .

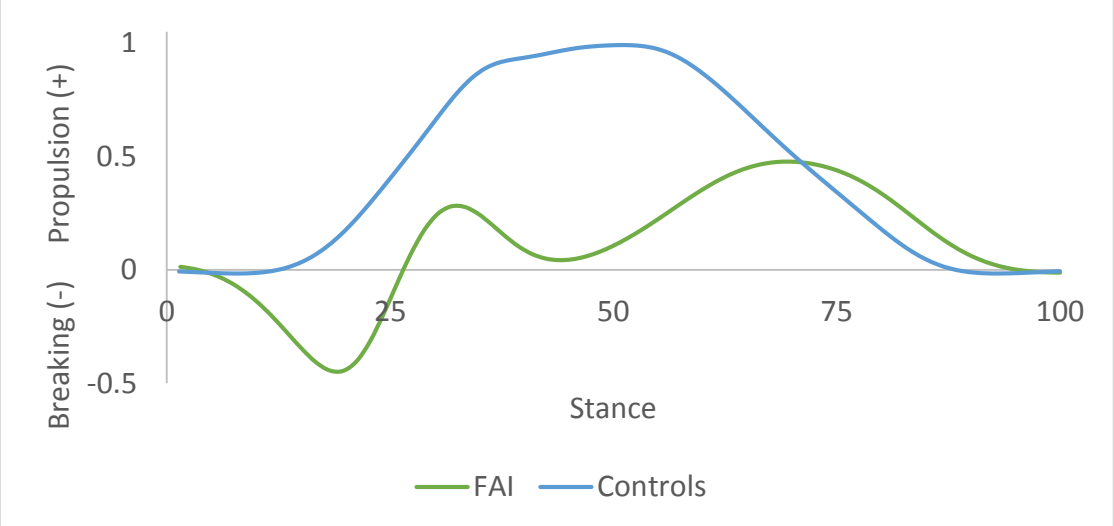


Figure 8. Representative Anterior-Posterior GRF in FAI and Control Participants during Running

Abnormal hip flexion reversals were more prevalent in our sample of both FAI and controls than previously reported[237, 238]. In both Rylander et al. studies, they reported a decrease in the number of participants and trials in which reversals were found at one year after surgery. It is possible that the reversal will also decrease in our sample by one year post-operatively, since the latest follow-up in this study is six-months. However, that would not explain the high prevalence seen in controls as well. Neither of the previously published studies reported their filtering routines for the kinematics. It is conceivable that the hip flexion graphs present in this study were not smoothed in a similar fashion, thereby producing different appearances. Another explanation may be that these brief periods of re-flexing the hip are related a loading mechanism. Though max vertical GRF (Figure 7) had lower peaks in the FAI group, the impulse, or average vertical GRF over the entirety of stance, was similar between groups. The FAI group dispersed forces over a longer period of stance, and there were cases of high breaking forces during the loading response (Figure 8). It is possible that these abnormal reversals occur during, or are at least related to, these periods of breaking during limb acceptance in walking and running.

Decreased frontal plane pelvis angle[127], hip excursion[148], and hip abduction[148] and adduction[127] angles have been previously identified in symptomatic FAI during walking. In the current study, frontal plane upward tilt of the pelvis and hip abduction was attenuated compared to controls during walking and this decrease was amplified during running (Figure 9). The FAI group pre-operatively walked with a decreased pelvis excursion, max pelvis angle, and decreased hip abduction angle at three-months. They also increased their hip adduction peak

angles at the three-month session. During running, the FAI group had decreased pelvis upward tilt and frontal excursion, as well as decreased hip abduction pre- and post-operatively. The FAI group's decreased pelvis angles may be explained by the fact that they had less hip abduction strength than controls (Figure 10), since this muscle group is responsible for maintaining contralateral upward tilt of the pelvis during stance[173]. Since the hip angle is measured relative to the pelvis, a lower pelvis tilt directly decreases the amount of hip abduction[212, 246]. This explains why we see a decreased hip abduction angle both pre- and post-operatively in the FAI group during running. These frontal plane changes were not limited to the hip, and they have also directly affected the knee.

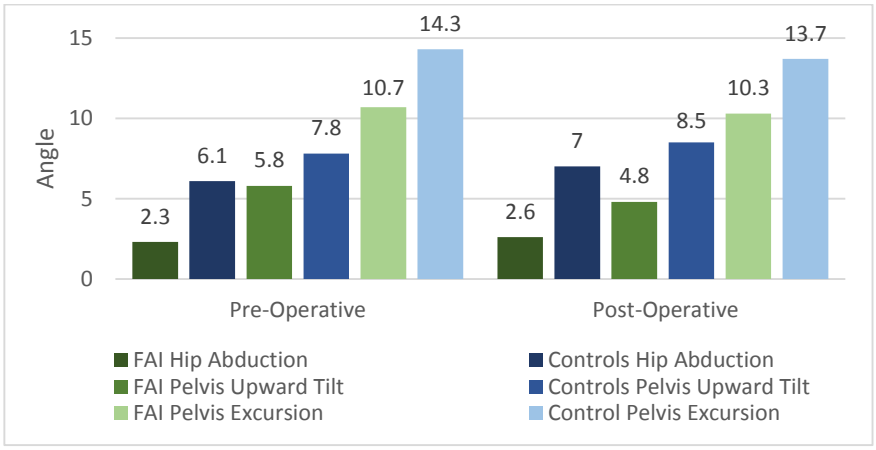


Figure 9. Pelvis and Hip Frontal Plane Angles during Running (all significantly different between groups, P<0.05).

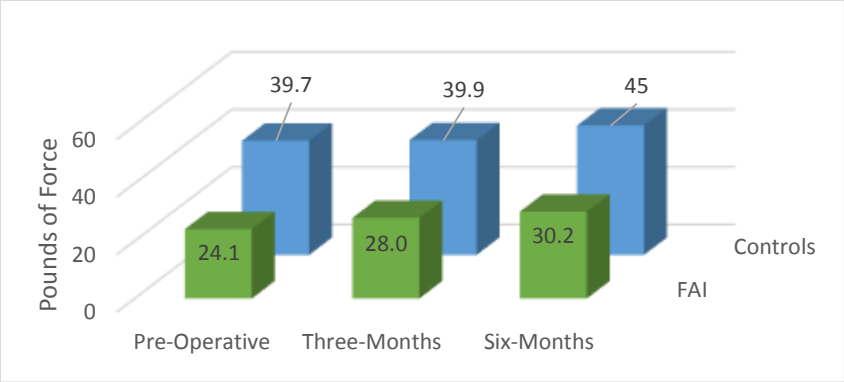


Figure 10. Hip Abduction Strength between Groups at Pre-and Post-Operative Sessions (FAI significantly weaker at each session, P<0.05).

The frontal plane of the knee showed several increased varus kinematic patterns (knee adducted) pre-operatively during walking and running. Varus excursion and velocity returned to control levels during walking by three-months (Figures 11), but remained different post-operatively during running (Figure 12). Varus angle is typically the greatest during weight acceptance in early stance, and then progressively moves into less varus, or a valgus position, in terminal stance. Increased motion in the frontal plane may be related to lack of muscular control of the knee via the FAI's weaker hip abductors, which help in controlling the femur[229]. Excessive frontal plane motion of the knee has been linked to acute and chronic knee pathologies[115, 229, 288], so it will be important to see if these findings remain at the one year session. Though not previously reported in the literature, this group of patient may be at a greater risk of knee pathologies if training regiments and sporting activities increase over time.

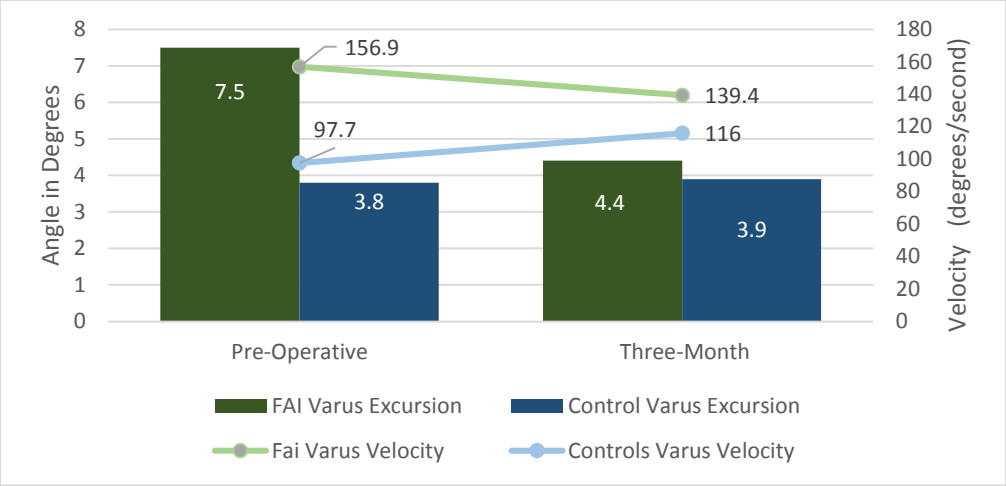


Figure 11. Varus Excursion and Velocity during Walking in FAI and Control Groups (Significantly different between groups pre-operatively, P<0.05).

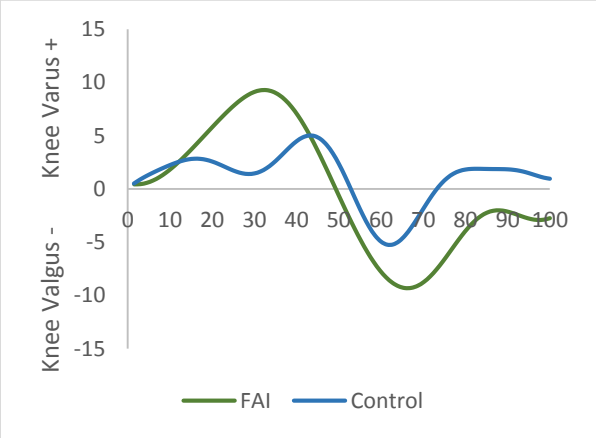


Figure 12. Representative Graph of Frontal Plane Knee Angle during Post-Operative Running.

During walking and running at all sessions, the FAI group preferred to utilize hip ER (Figure 13) and maintained a more externally rotated foot progression angle during running (Figure 14) post-operatively. This should be expected, as hip internal rotation was painful for the FAI group during pre-operative clinical exams and during HHD. All 12 FAI participants had positive FADIR tests for pain, and IR on average was the most painful position during HHD (when painful 6/10). Therefore, pain avoidance compensatory patterns may have resulted secondary to a reluctance to move into hip IR, and continued post-operatively.

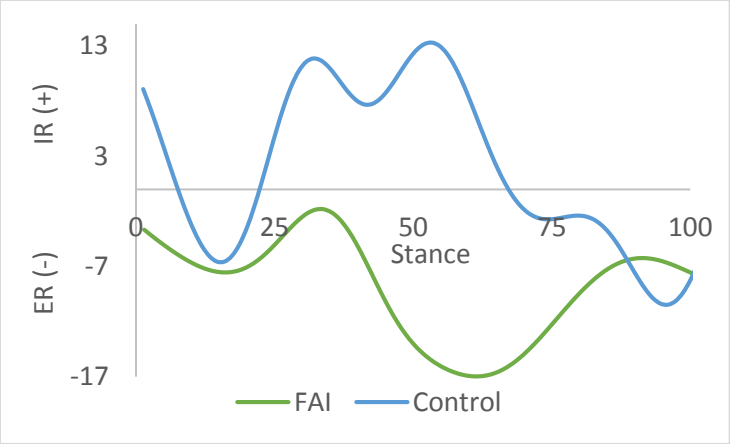


Figure 13. Representative Hip Internal (IR) and External (ER) Rotation in FAI and Control Participants at Six-Months during Running.

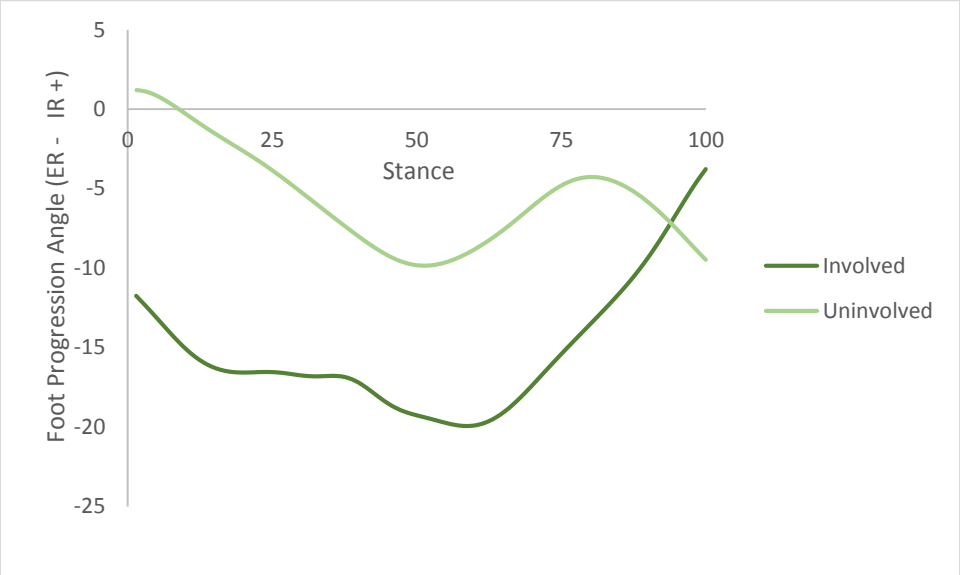


Figure 14. Foot Progression Angle in a Single FAI Patient at Six-Months Post-Operatively.

The sample of the FAI participants presented some limitations requiring certain assumptions. Most of the participants were female, which may limit the generalizability of the results. This may be especially true considering that FAI is equally represented between genders[57]. Younger people have greater ability to recover from injury, so the age range (20-45 years) may be suboptimal for generalizing the results to all ages. Lastly, walking velocity and body mass differences may have biased kinetic findings, as the forces and moments are

dependent upon these variables. However, forcing the FAI and controls to alter walking velocity may have unexpected consequences in normal gait patterns as well.

One additional limitation was the testing position for the HHD of hip IR and ER. Cibulka et al. demonstrated that healthy individuals have varying degrees of hip rotation in each limb and therefore, should be tested in their own individualized mid-point of motion[55]. This is to avoid mistakenly testing a shortened or lengthened muscle, as rotational deficits are common in the ipsilateral hip rotators. This could theoretically have an effect on sarcomere length and force production capability of the actin and myosin cross-bridges. Since hip IR/ER strength was tested in 0° of rotation and hip passive range of motion was not measured in our study, it is possible this is a potential source of error in the hip IR/ER HHD. Therefore, results must be viewed acknowledging these limitations, but are not considered to significantly impede this study.

In conclusion, this is the first study to report hip and knee strength and walking and running gait changes pre-operatively and post-operatively at specific time points during recovery from hip arthroscopy. Our primary finding was that FAI patient's recovery occurs in stages. Though never reaching the level of controls, the HOS_{ADL} and HOS_S significantly improved at each session. The FAI group was participating in impactful activity by six months, as the UCLA score was similar between groups. Additionally, the HOS subscales were positively correlated to most hip and knee strength measures, indicating that the stronger a person is, the more likely they will have greater satisfaction in their ADL and sport performance. This finding further validates use of the HOS in hip arthroscopy patients.

The involved limb of FAI participants progressively increased in strength from pre- to post-operatively. However, at six months the hip abductors were still significantly weaker versus the uninvolved side's hip abductors. The FAI group was also significantly weaker than the control group in most of the tested hip and knee strength measures over the course of the study. Therefore, strength training and physical therapy may need to be longer or more intensive in this patient population.

The FAI group's involved limb walking gait parameters changed post-operatively as a result of their increased walking velocity and post-operative adaptations. Increased velocity led to increased ground reaction forces and external moments. Kinematically, most changes were not at the hip joint, but instead they occurred at the knee and ankle. This was most likely due to a reluctance to utilize the hip joint's full range of motion. There were also differences at all three sessions when comparing the FAI group's walking gait to controls. At the study conclusion, these were primarily tri-planar changes in hip, knee, and ankle kinematics, as there were few kinetic differences by six months.

These gait changes were similar and amplified during running, as there were also tri-planar kinematic changes at the hip, knee, and ankle compared to the controls. These were highlighted by increased hip external rotation and frontal plane decreases of the trunk, pelvis, and hip. These variables did not change over time in their involved limbs, which warrants further analyses of rehabilitation and walking and running gait retraining.

Part II
REVIEW OF LITERATURE

Three-Dimensional Biomechanical Analyses of Hip Morphologies

Studies investigating the effects of hip morphology via 3D biomechanics analysis have focused on the squatting motion[157, 158] and level walking gait[35, 127, 148, 238]. The results of these studies indicate that pre-operative walking kinematics[127, 148] and kinetics[127] and stationary squat depth[158] are altered compared to asymptomatic controls, and may remain post-operatively[35, 157]. However, direct comparisons between walking gait studies are complicated by varying methodologies.

Maximal Double Legged Squatting in FAI

Pre-operative cam FAI patients display decreased depth and sagittal plane pelvic motion during maximal squatting, compared with controls[158]. They exhibit 10° less pelvic sagittal plane excursion (14° vs. 24° in controls), 9.2% less squat depth (relative to leg length), and no differences in hip motion. The inability to recline the pelvis may play an important role the development of FAI. The pelvis was anteriorly tilted closer to the femur in cam patients, without a difference of flexion between groups. This may be what leads to the repetitive collisions between the femur and acetabulum, as controls are able to posteriorly tilt the pelvis away from the femur.

Following surgery, 8 of 10 participants were able to posteriorly tilt their pelves at maximal squat depth and all displayed an increased amount of squat depth[157]. The significantly different ROM gains came from the knee and ankle joints, which increased by an average of 11° and 7°, respectively. It's unknown how, or why, there were ROM changes seen at these joints

following surgery. The small sample size (n=10) and two participants with lingering anterior tilt at max squat depth may have led to the non-significant finding for pelvic ROM post operatively. Two limitations of this follow-up study were the wide range of postoperative data collection times (8-32 months) and lack of control group re-tests. Therefore, it is unknown if the range of motion gains were simply from test related learning effects.

Pre-operative Walking of FAI

Decreased kinematic measures at the hip and pelvis during walking have been identified compared to controls in all three planes. Decreased sagittal plane hip excursion[148], abnormal flexion-extension reversals[238] (Fig. 16), and decreased peak extension[127, 148] have been found in FAI patients. The largest differences occur during terminal stance for hip extension, when the hip ligaments and the iliopsoas are maximally stretched and possibly contributing to anterior joint pressure and/or pain. Femoral anteversion was not measured in any of the gait studies as well, so it's unknown what effect that could have on the results.

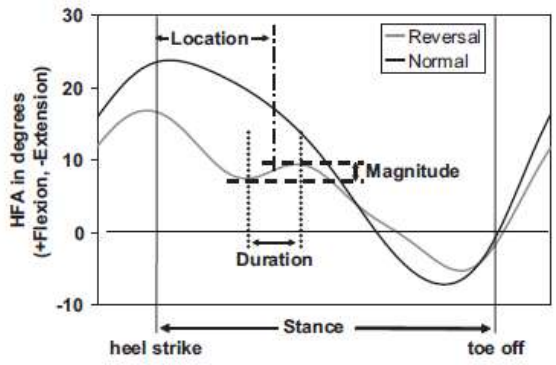


Figure 16. Example of abnormal hip flexion reversal as seen in symptomatic FAI.

Frontal plane pelvis[127] and hip[148] excursion, peak hip abduction[148] and adduction[127] are additionally decreased in symptomatic FAI. Decreased hip abduction was found just after toe off and continued through swing, indicating that this may be muscle related weakness of the

lateral hip muscles. Hip abduction and extension isokinetic torque is correlated to trunk and pelvis positions[89], so strength of the hip cannot be ignored as an important factor in trunk, pelvis, and hip motion during gait. Hip abduction strength at toe off (e.g. in extended hip position) may be aided by action of the tensor fascia lata, or the gluteus maximus during flexed positions. Particularly, Boudreau et al. indicated that the tensor fascia lata controls abduction after gluteus medius initiates it[32]. However, assessment of non-weight bearing hip abduction (as in the swing phase) is typically only assessed in a neutral position, and not flexed/extended[14, 147]. This may further complicate direct relationships. The decrease in frontal plane pelvis excursion was theorized to be caused by limited lumbo-sacral mobility. However neither hip abduction strength nor lumbar mobility were measured, so this relationship remains unclear.

Transverse plane peak internal rotation during stance is also significantly decreased in FAI versus controls[127]. This may be related in part to pelvis positioning and weakness of lateral hip muscles similarly to the frontal plane deviations, or a result of preoperative pain. Hunt et al. indicated that pain was likely a main factor in their findings[127]. It has been hypothesized that FAI patients may adopt compensation strategies, or "protective gait", when walking, as actual impingement of the joint during walking is not likely since terminal ranges are not reached[148]. The decreased joint motions have also been implicated as possible causes for altered external moments seen during walking.

Kinetics alterations have been found in pre-operative FAI patients compared with controls as well. Decreased external moments in early stance flexion and late stance external rotation have been found[127]. These findings indicate that the need for internal hip flexion

moments in early stance and IR moments in late stance are reduced. However in the Hunt et al. study, 40% of the sample had bilateral deformity and no effort was made to control for the significant velocity and cadence differences between FAI and control groups. The effect of these two points casts doubt on the results of Hunt et al., as Kennedy found no kinetic differences after controlling for spatiotemporal differences[148]. Lastly, peak stance values were assessed across these studies, with no effort to isolate specific phases of gait (e.g. kinematics and kinetics at initial contact or toe off).

Post-operative Walking Gait in FAI

Surgical success as measured via 3D gait analysis has only two studies for comparison[35, 238]. The methods differ by sample pathologies, post-operative data collection times, and surgical interventions, creating many issues when trying to compare results. Rylander et al. found a return to normal sagittal plane kinematics and kinetics one year post-operatively in a sample of 11 (6 cam, 2 pincer, and 4 mixed pathology), however did not examine the frontal and transverse planes. Conversely, Brisson et al. found reduced hip frontal and sagittal plane excursion as well as smaller hip abduction and IR moments in (all 10 cam FAI)[35]. There was also decreased power generation at the hip for FAI patients post-operatively. There was 35% and 24% less power versus controls and pre-operative results, respectively. This may be due to the surgical process, muscle atrophy, or part of the methodology flaws for follow up gait analysis. The post-operative follow up gait measurements were not consistent, and may call into question their results (21.1 ± 9.4 months and range= 10–32 months).

Dysplasia Biomechanics

There has only been one study that examined gait in dysplasia[262]. Twenty one patients underwent a Bernese PAO for dysplasia at an average age of 16.1. One year postoperatively all displayed radiographic improvements for the signs of dysplasia by means of LCEA =33°, ACEA=32.5°, and an AI of 10.5°. They found that hip strength and hip abductor impulse during stance could return to normal by one year post-operatively, but hip flexion power could not. They concluded improvement in hip flexion power may be realized with earlier weight bearing, less soft tissue dissection of the hip flexors, or via preoperative strengthening program.

Walking gait in SCFE

Gait related deficiencies following in situ pinning in SCFE have been correlated to increasing severity[254] and compared with controls[283]. Gait velocity, pelvic obliquity (down on affected side), and trunk obliquity were abnormal, so even when walking slower, post SCFE patients still had trunk and pelvis swaying compensations. The involved hip became more extended, more adducted, and more externally rotated throughout the gait cycle and foot progression angle became more external, as found by both studies.[254, 283] The results may be due to femoral retroversion, secondary to the changes at the proximal femur, resulting in external rotation of the entire limb. Westhoff et al. additionally found the involved limb to become more stiff in the sagittal plane at the hip, knee and ankle[283]. This may have led to their findings of decreased concentrically and eccentrically work production at hip, knee, and ankle on the involved side compared to the uninvolved side and controls.

The internal hip extension (r = 0.91), knee flexion (r = 0.88), and ankle dorsiflexion moments (r = 0.97) decreases were correlated to increased severity. In many cases the hip abduction moments were greatly decreased, and became a hip adduction moment in the severe slips.[254] These are believed to result from the deformity of the proximal femur.

In conclusion, femoral and acetabular bony morphologies of the hip may be recognized initially via radiographs, but often require advanced imaging CT and MRI to accurately assess pathology. The combinations of these deformities, and how they lead pathological conditions and gait deviations, are becoming better understood. Future research is warranted to elucidate these complex relationships and how they lead to altered gait and OA.

Normal Human Walking and Running Gait

Gait Cycle Phases and Events

Though walking and running is mere means of moving between two points, its importance to our species cannot be understated. Ability to describe the characteristics of gait becomes a key when clinicians need to address dysfunctional patterns of locomotion. Walking can most simply be described as a series of controlled falls, whereas running has been likened to alternating pogo sticks[214]. Though these views are helpful in an overall sense of understanding, they do not help clinically.

Human bipedal gait is a phenomenon of the most extraordinary complexity[244]. In an attempt to describe this complex movement, gait is broken down into several distinct phases and events that define the entire cycle. The gait cycle is the period from initial contact of one

foot with ground to the next initial contact of the same foot with the ground[214, 219]. It is divided into stance (ST) and swing (SW) phases, which account for 60% and 40% of the walking gait cycle, respectively. Stance phase describes the time in which a foot is contact with the ground, whereas during swing the foot is off the ground. Stance begins with foot initial contact (IC) and can be divided into four subphases: loading response (LR), midstance (MS), terminal stance (TST), and preswing (PSW). Less time is spent in stance as forward velocity increases. Toe off may occur at 35-40% of the cycle during sprinting or running, and elite sprinters may toe off as low as 20%. [214] Swing phase begins with toe-off (TO) and ends at IC. The three subphases of swing are initial (ISW), mid- (MSW) and terminal (TSW) swing.

Walking gait can additionally be defined in terms which limbs are in contact with the ground, or supported[214, 219]. Double support, or when both feet are in contact with the ground, occurs at the first and final 10% of stance. These correspond to the LR and PSW phases. Single support were occur when the contralateral leg is off the ground during swing. Running gait is distinguished by no periods of double support, instead being replaced with “double float”.

Running gait is similarly described in terms of stance and swing as well. However, as velocity is increased above 2.0-2.5 meters per second there are no periods of double support as the each stance phase decreases from 60%, to below 50% of the gait cycle[183, 219, 220]. The swing phase increase to greater than 50% of the cycle and has the added subphase of double float. Running stance may have both an absorption and propulsion subphase, separated by MS. Swing phase is similar to walking, with the added double float at the beginning and end of swing.

Maintaining Gait Efficiency

Gait needs to be as effortless as possible for energy conservation. Perry previously outlined four functions of the LE in accomplishing efficient locomotion[222]. 1) Upright stability is maintained despite ever changing posture. 2) Progression is generated by muscle force and tendon elasticity. 3) Shock from impact with the floor is minimized. 4) Energy needs to be conserved in order to minimize muscular effort, or fatigue. These points are important both in a description of gait and understanding pathologic conditions. They coincide with the theories of propulsion, shock absorption, stance stability, and energy conservation. In other words, the forces absorbed during gait need to be directed in a fashion that assists movement. The tibia, ankle, heel, feet, and toes provides one such mechanism for a smooth advancement.

Sequentially the heel, ankle, forefoot, and toe "rock" during stance[222]. The heel rocker allows for preservation of forward momentum by rolling toward the ground during weight acceptance at IC (presuming a heel strike pattern and not the forefoot or toe strike at IC). Eccentric contraction of the tibialis anterior slows progression of the foot dropping to the surface while simultaneously drawing the tibia forward during the loading response. This energy is also transferred to the eccentrically contracting quadriceps muscles, which is resisting knee flexion, thereby also drawing the tibia forward faster than the femur. At the end of this heel rocker the foot is flat on the ground, tibia is vertical, and the knee is flexed to about 20^{o}. The ankle rocker begins with passive dorsiflexion and eccentric soleus contraction with the foot flat and stationary. The ankle is now a fulcrum point with the forefoot beginning to be in contact with the ground. The tibia continues to advance and becomes a stabilizing base for

future knee extension. The forefoot rocker begins with the heel lifting off the ground and the body's weight in front of the foot's base of support, creating the most force for progression during gait. This force is primarily resisted by strong contraction of the gastroc-soleus complex. Lastly, the medial forefoot and great toe are the last parts of the body to leave the ground during stance during the toe rocker. Now the gastroc-soleus complex is reversing the stored elastic energy and concentrically contracting to thrust the tibia forward.

It's important to note that most of the focus of gait analysis focuses on the lower extremity (LE). This is due to the fact that the LE is the locomotor system, while the HAT are mostly a passive "passenger"[222]. The HAT are considered passive because there is little requirements of it to maintain gait. Malalignment or control of the upper extremity were however have a great effect on the gait patterns of the LE. Comprising 70% of the total body weight, the HAT is where is body's COM is found. Balancing the body during walking relies on keeping the base of support of the lower limbs under this COM, which is typically one third the distance from the hip to the top of the shoulder.[222]

Spatiotemporal Variables

The common spatiotemporal variables also used to describe gait are cadence, stride length, step length, and step width[128, 219]. These measurements describe the displacement between and within the limbs in regard to space over time. Cadence is calculated as an average of steps per unit of time for a given limb and is typically 130 walking (steps/minute) and greater than 215 (steps/minute) in running. Stride length is the distance between the ICs of opposite feet and can be reported as absolute values, or normalized to height to allow comparison

amongst subjects[26]. Step length is the distance between the ICs of the same foot. Additionally, step frequency may be calculated as velocity divided by step length[26]. Step width is the only spatiotemporal measure that describes frontal plane distance. It creates the lateral borders of the body's COM and when sufficient contributes to the maintenance of dynamic stability. These variables simply describe total limb movements. Clinicians need to be able measure spatial movement of joints as well to determine pathologic conditions.

Transitioning from Walking to Running

Increasing forward velocity (*v*) results from taking longer strides or moving the legs quicker. Stride length is inversely related to stride frequency, so to increase velocity you must increase one without a decrease in the other[76]. The main strategy to increase velocity during running (2-7 $m \cdot s^{-1}$) is to increase muscle production during stance to change stride length[76]. This is accomplished by the ankle plantarflexors. Sprinting above 7 $m \cdot s^{-1}$ does not allow the muscle enough time during stance to generate additional power. Therefore, to increase stride frequency is accomplished by rapidly accelerating the legs in the air faster via the hip flexors and extensors and knee extensors[76].

Kinematics

Kinematics describe the three-dimension (3D) angular rotations at all the joints in the body measured in degrees. The three orthogonal dimensions are the sagittal (anterior/posterior pelvic tilt, hip and knee flexion/extension, and dorsiflexion/plantarflexion), frontal (upward/downward pelvic obliquity, hip abduction/adduction), transverse planes (pelvic

and hip medial/lateral rotation). Increasing velocity in direction of progress relies mostly on changes from the sagittal plane. That's why the greatest range of motion comes from the sagittal plane, with much less from the frontal and transverse planes. Total range of motion generally increases in the transition from walking to running and sprinting.[218] Typical kinematic measures for the lower extremity are presented in Table 1 based on previous authors[212, 213, 218, 222].

Table 21. Lower Extremity Walking Gait Kinematic Values

	Joint			
Plane	Pelvis	Hip	Knee	Ankle
Sagittal	10 to 20 (20)	-10 to 30 (40)	5 to 65 (60)	-15 to 20 (35)
Frontal	-5 to 5 (10)	-10 to 10 (20)	-4 to 4 (8)	-5 to 5 (10)
Transverse	-10 to 10 (20)	-10 to 10 (20)	-5 to 5 (10)	0 to 10 (10)†

Mean maximum and minimum joint positions in degrees (total joint excursion).

† denotes foot progression angle

It's important to note that there are two distinctly different methods for describing kinematics. Segmental angles are changes in body position compared to an external reference point (global coordinate systems [GCS]) (e.g. forward pelvic tilt compared to the laboratory). Joint angles are relative changes in position for two adjacent bony segments (local coordinate systems [LCS]) (e.g. change in thigh position compared to the pelvis to give hip angles). For the purpose of describing the 3D kinematics, we were examine the trunk, pelvis, hip, knee and foot in each plane for both walking and running.

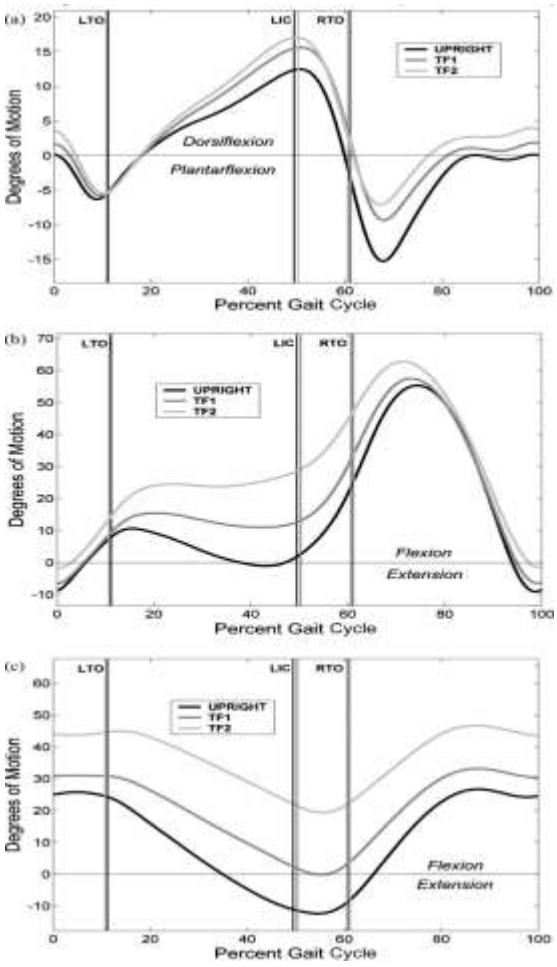
Trunk Kinematics

Movement of the trunk in the three planes has been both a segmental measure (compared to the vertical of the GCS or neutral standing), or a joint angle (compared to the position of an adjacent bone), so it's important to note which a study utilizes[162, 245, 246]. Typically a trunk, lumbo-pelvic, or pelvis angle were be segmental to the GCS, while spine joint angles are thorax relative to the pelvis. You may have two studies that use similar methods, but report vastly different ranges of motion due to the reporting of segmental versus joint angles[246]. Another issue lies with measuring the trunk motion as a single structure, opposed to thoracic, lumbar, and sacral segments. Singular segment analysis does not accurately portray the individual position of the thoracic or lumbar regions. For example, the lumbo-pelvic area can be flexed and rotated, regardless of the thoracic spine, and vice versa. Studies may use the term "trunk" for both instances. Therefore, it is important for readers to understand exactly what movements are being described. Though largely ignored in clinical gait analysis, smoothness and fluidity in trunk motion is necessary for efficient gait [285]. Thus, ignoring trunk positions during gait analysis is imprudent. We gradually, and significantly, increase the amount of sagittal thorax flexion every year during maturation[269]. Around the time of young adulthood we walk and jog with 0-10º of trunk flexion[106, 269]. Position of the trunk is important to lower extremity gait analysis due to its effect on joint angle changes in the lower limbs during gait. Saha et al. examined the effects of segmental forward trunk lean on slow, normal, and fast walking[241]. Forward flexion of the thoracic trunk (or anterior displacement of the trunk COM) leads to sustained periods of knee flexion during stance and greater peak ankle dorsiflexion and hip flexion during normal walking velocities (figure 17).

This has been termed as a “crouched gait” pattern. These changes are believed to be a compensation for maintaining COM over the walking base of support. Perhaps the most interesting consequence is what happens at the hip as forward trunk flexion increases. Maximum hip flexion is increased from 0.9^{o} to 48.8^{o} and hip joint excursion decreases from 41.5^{o} to 30^{o} when moving from 0^{o} to 50^{o} of forward trunk flexion. The loss of excursion is at the expense of hip extension, as the minimum sagittal hip position during a maximally flexed trunk position is around 20^{o} of hip flexion (Figure 17, TF2 line).

Keep in mind the previous examples of segmental sagittal trunk position are being viewed in reference to the GCS. The thorax is flexed forward in space but the actual joint angle is extended (due to the pelvis having more sagittal flexion)[106, 269]. Sagittal spine angles are reported to be about extended 3-5^{o} during walking and about 13^{o} during running[106, 269].

Frontal plane trunk movement is termed lateral trunk flexion, but has also been described as trunk lean, bend, or abduction[162, 222, 285]. During walking the trunk were laterally flex 3-6^{o} toward the stance leg during loading response before returning to neutral[107, 162, 222]. Velocity increases do not change the timing of peak lateral trunk flexion, but excursion and



peak position may increase to as much as 20^{o} when running at 6m·s^{-1}. [246] The spine lateral flexion joint angle were be about 6^{o} during walking and increase to 10^{o} during running[106].

Transverse trunk motion trunk rotation again uses thorax as a segmental GCS angle and spine for denoting joint angles in reference to the pelvis. It has also been termed axial rotation and uses medial/lateral or internal/external rotation descriptors. Transverse thorax angle alternates 2-3^{o} for a total excursion of about 6^{o} during

Figure 17. Average (a) ankle, (b) knee, and (c) hip joint angles during the gait cycle at freely selected normal walking velocities. Left toe-off: LTO, left initial contact: LIC, and right toe-off. TF1: Trunk Flexion 25 degrees, TF2: Trunk Flexion 50 degrees

walking[163, 269]. The transverse spine angle during walking were be higher than its segmental counterpart due to the inverse relationship with the pelvis. The first and last 20% of gait the trunk is medially rotated while the pelvis is laterally rotated and they switch positions during the middle of the cycle with the pelvis medially rotated. This produces transverse spine angles of 5-7^{o} and excursions near 10-14^{o} during walking[162, 163, 269].

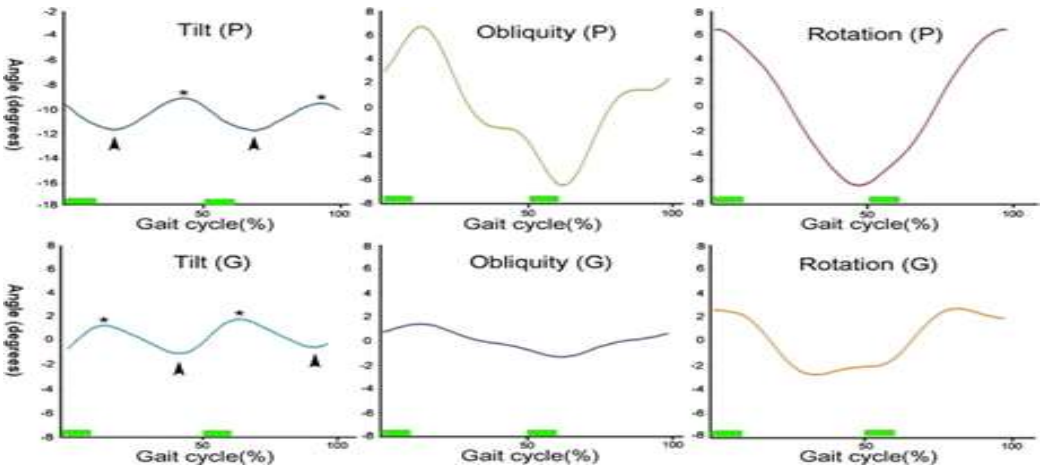


Figure 18. Walking trunk joint angles relative to the pelvis and segmental angles relative to the ground in three planes. *P* = pelvis, G=ground

As velocities increase to running, both thorax and spine transverse angle excursion increases as the upper body medially rotates forward with the arm, while the low back and pelvis laterally rotate backward with the extending leg[246]. The thorax angle has reported to be 10-14º during running velocities of 3.8 m·s^{-1} [73, 106], while the spine angle is 9-12º.

The lumbar trunk movements have been described as joint angles in reference to the pelvis and in reference to the ground, or GCS (Figure 4). During walking the lumber spine typically stays in a flexed position relative to the pelvis, with two peaks during a single gait cycle [245]. The first peak is at about 10% of the cycle and the second during midstance before becoming less flexed during in terminal stance. The lumbar trunk is extended, relative to the pelvis during walking, but appears flexed in relation to the GCS. The total flexion-extension excursion is very low at slow walking velocities (1 m·s^{-1}), around 3º. Typically it is flexed 0-15º over velocities 4 m·s^{-1} during running, with an excursion of 10º at faster velocities [245]. The

timing of peak lumbar trunk flexion is similar between walking and running velocities. The minimal position is around the time of TO and maximal flexion occurs during both early and/or midstance.

Frontal plane lumbar trunk motion laterally flexes about 2-5^{o} and exhibits as much as 10-15^{o} of total excursion during walking[43, 91]. This increases to about 7^{o} during running with an excursion of 10-15^{o} [245]. Many people may walk with more lumbar trunk lateral flexion on a given side in a few, or all, trials. The timing of peak lateral flexion does not vary with increasing velocities. However as mentioned the peak flexion and excursions were.

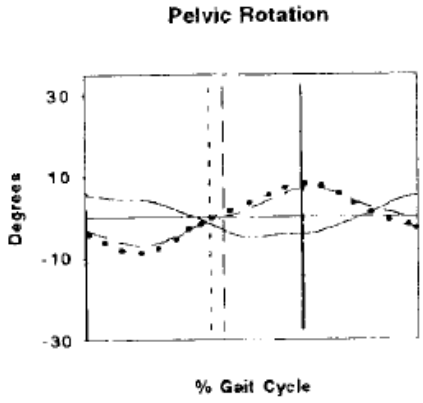
Peak transverse lumbar trunk rotation is 3-6^{o}, typically at ipsilateral foot strike, during walking gait[43, 245]. The transverse excursion were be 6-8^{o} due to people twisting more on one side compared to the other. With running speeds of 4 $m \cdot s^{-1}$ the peak rotation increases a few degrees, as does total excursion. It's important to note that transverse trunk rotation is highly correlated with stride length (r=0.93)[245]. The timing of peak motion were also occur 10-15% sooner. Greater rotation on one side is commonly observed during running as well.

Pelvis Kinematics

Sagittal plane motion of the pelvis is termed anterior and posterior tilt. Standing anterior tilt has been reported to be about 5-10^{o} due to the anatomically lower position of the anterior superior iliac spines (ASIS) compared to the posterior superior iliac spines (PSIS)[174, 222]. During walking the pelvis were anteriorly tilt to about 11-12^{o}, reaching its peak twice during the

gait cycle[212, 222]. The minimal amount of tilt is typically during single leg support. Anterior tilt during running increases to 20° and has a total excursion of 5° [212, 246].

Frontal plane motion of the pelvis is termed pelvic obliquity or lateral pelvic tilt. During walking the pelvis laterally tilts about 4-5° on each side [212]. During running, the pelvis should be tilted upward on the stance leg during weight acceptance and lowering to a neutral position by midstance[246]. This is thought to be important in shock absorption and controlling of a smooth descent. By toe off the pelvis should be tilted downward, with the contralateral limb preparing for IC with an upwardly tilted pelvis. The excursion for this movement is 7-15°[91, 212].



Transverse plane motion of the pelvis, or pelvic rotation, is typically reported in relation to direction of travel (GCS). During stance at walking speeds the pelvis is medially rotated 5-8° during stance to increase step length and laterally rotated 5° during swing [212].

Figure 18. Pelvic rotation kinematics in walking (solid line) and running (dotted lines).

However, this reverses during running as the pelvic rotation is not needed to increase step length, instead becoming a transition point between the rotating trunk and contralateral leg swing (Figure 18). Additionally, the thorax is in-phase (both medially rotated at the same time) with the pelvis at slower walking, and out of phase (one medially rotated with the other laterally rotated) at faster walking and running speeds, or when taking larger steps [176].

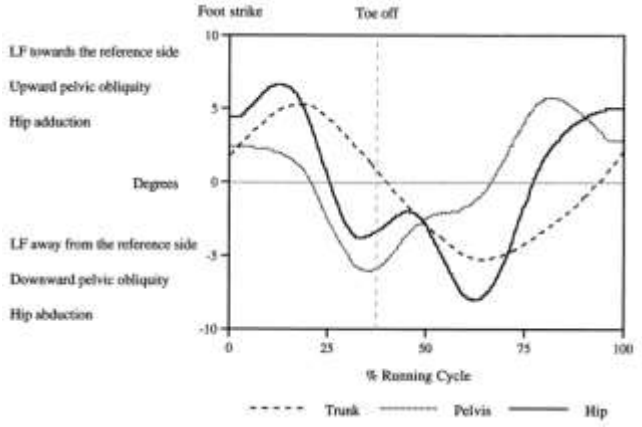
Articulating with the acetabulum of the pelvis is the femur. This is an important joint, as it marks the transition zone from the passive upper extremity to the active locomotor engine of the lower extremity.

Hip Kinematics

The hip is an enarthrodial joint that allows for tremendous range of motion in all three planes. This is advantageous for human movement due the ability to accurately place the foot on various terrain across many different speeds. However, during walking and running most ROM is in the sagittal plane, with much less motion from the frontal and transverse. This is due to the conservation of COM and leads to more efficient movement patterns. The hip has been described as both a segmental measure (thigh to vertical angle) and as a joint angle (femur to pelvis)[222]. This is because movement of either the pelvis or thigh has an effect on the hip angle.

Sagittal plane hip motion is typically very smooth and displaying a sine wave pattern[212]. At IC the hip is flexed about 30^{o} and begins to reverse its position, becoming more extended. By midstance the hip is neutral, then reaches peak extension of 10^{o} near toe off[212]. During running and sprinting peak hip flexion were also occur during terminal swing and increases significantly to 45-60^{o} depending on velocity. Peak hip extension may increase only a few degrees in the transition from walking to running and sprinting, reaching peak values of 20^{o} [212]. The total joint excursion during walking is about 40^{o}, increasing to as much as 70^{o} during sprinting velocities.

The hip displays the largest amount of frontal plane movement of the lower extremity. Generally speaking, during swing it is abducted and reverses to an adducted position during stance. This is highly related to the position of the pelvis in the frontal plane as well[212, 246].



During IC and into midstance, the adduction increases as the limb is loading and absorbing energy.

Figure 19. Frontal plane relationship between the trunk, pelvis, and hip during running.

The trunk is laterally flexed and the pelvis is tilted upward on the ipsilateral side during this period of hip adduction. These three were reverse and the hip were abduct during swing as the pelvis drops and the trunk begins to laterally flex on the contralateral side (Figure 19). The hip were abduct and adduct about to a peak of about 7^{o} during walking and running velocities, with an excursion of $10\text{-}14^{o}$[212, 246]. The frontal plane relationships between the hip and pelvis are also similar during walking and running.

Transverse motion of the hip, or medial and lateral rotation, is the least variable of the hip motions. Typically, it maintains a medially rotated about $5\text{-}10^{o}$ during walking and running. During the absorptive phases of IC and early stance the hip is medially rotated, then returning to a neutral position by toe off. The hip may remain in a neutral position, or may again medially rotate[212, 219, 246].

Knee Kinematics

Sagittal knee flexion-extension has a bimodal flexion peak during the gait cycle. The knee is slightly flexed at IC with the first smaller peak during the LR when the knee flexes 8-15^{o} as part of the shock absorption mechanism[212, 218, 219]. Then the knee reverses, extending from midstance to terminal stance (but remaining in a flexed position and never in true extension), before flexing again to 50^{o} to allow for foot clearance over the ground during swing. This is the second and larger peak knee flexion and occurs during midswing[212, 218]. Sagittal plane knee motion is similar in walking and running, but with an increased excursion as forward velocity increases[212, 218]. This is due to an increased amount of flexion (never true extension, or clinical hyperextension). Knee flexion during swing increases to as much as 70^{o} during running[218] and 100^{o} during sprinting[212]. The most amount of motion at the knee occurs in the sagittal plane, with very little in the frontal and transverse. Its small enough in these two planes that it is not even described in classic biomechanics literature[212, 218, 219].

Frontal plane knee motion is relatively small, abducting and adducting as little as 2-4^{o} in healthy individuals with an excursion of 2-8^{o}[222, 229, 288]. Reaching a peak abduction of 4^{o} at IC the knee then begins to adduct during swing. Though small in healthy individuals, excessive frontal plane motion of the knee has been linked to acute and chronic knee pathologies[115, 229, 288].

Transverse plane knee motion is similarly small with an excursion of 8^{o} and follows the patterns of the pelvis and femur[222]. Generally speaking, when the knee is extended the tibia is laterally rotated and medially rotated in flexed positions. During gait, the tibia is medially

rotated during the loading response of IC and begins to laterally rotate throughout stance as the knee extends. This is part of the screw home mechanism during single leg support. Peak lateral rotation is just a few degrees and occurs at terminal stance. During swing the knee were medially rotate as the knee flexes for foot clearance and in preparation for the next IC. The transverse motion of the tibia (and therefore the knee) is coupled with ankle inversion and eversion (tibia medial rotation and ankle eversion)[25]. During walking the tibia appears to drive foot motions in the form of power flowing proximal to distal. During running velocities this is generally the case, but is more varied as many display distal to proximal power flows [25].

Ankle and Foot Kinematics

The tibia articulates with the superior surface of the talus to form the tibiotalar joint, where ankle dorsiflexion and plantarflexion occurs. This joint in the ankle has the largest ROM, again in the sagittal plane, similarly to the hip and knee. At IC the foot is plantarflexed a few degrees and continues to flex to about 8^{o} by the middle of the loading response[222]. The foot then reverses to dorsiflex throughout stance due to tibial advancement during the heel and ankle rocker, reaching a peak dorsiflexion of 10^{o} in terminal stance. Just prior to toe off the foot were again plantarflex to a peak of about 20^{o} before returning to neutral during swing for foot clearance over the ground. During running and sprinting there is a large increase in both dorsiflexion and plantarflexion, assuming a heel contact pattern. Both can increase to as much as 20-30^{o} with total excursions of 50^{o} in running and 60^{o} in sprinting [212, 219]. The ankle were be dorsiflexed 20-24^{o} during weight acceptance, then reversing to a plantarflexed position during terminal stance.

Distally, the inferior surface of the talus articulates with the superior surface of the calcaneus to form the subtalar joint (calcaneotalar), where ankle (rearfoot) inversion and eversion occur. Ankle motion is usually biplanar due to the 10^{o} frontal and the 20^{o} transverse plane tilts (lateral malleolus is more distal and posterior than medial malleolus), resulting in plantarflexion/inversion and dorsiflexion/eversion coupling[222]. The amount of rearfoot eversion during running is highly dependent on foot strike patterns. Peak rearfoot eversion is 11^{o} during heel striking and decreasing to 9^{o} and 6^{o} with forefoot and toe striking, respectively[228]. Less attention is given to the frontal and transverse motions of the foot and ankle due to their high variability between and within participants [228]. Often the differences within individual trials are as large as the group differences. However, these measures should not be ignored as they are highly correlated to transverse motion of the tibia. Decreased amounts of eversion are found with decreased tibial medial rotation and may play a role in hip and knee pathology.[228]

Transverse motion of the ankle describes position of the foot segment relative the ankle's sagittal plane axis. In other words, it is the angle between the foot vector and the sagittal axis of the shank, projected into the foot's transverse plane. Additionally, authors have used foot progression angle, or foot-placement angle (toed in or toed out) to describe transverse ankle/foot angles relative to the GCS [236]. Foot progression angle is typically laterally rotated 0-15^{o} (toed-out relative to GCS direction of progress) during walking, running, and sprinting[212]. During walking gait the foot is laterally rotated 8-10^{o} during stance before reversing a few degrees just prior to swing, then laterally rotating again to 12^{o} during midswing[219]. The joint excursion were increase from 8^{o} during walking to 14^{o} in running[219].

Only during late stance sprinting were there be true medial rotation, and only for a brief time[212].

The foot is an important site in gait analysis, as it's the first link in the kinetic chain to come into contact with the ground. The ground reaction forces are transmitted distally from the foot up through the rest of the lower limb. The foot typically strikes the ground and follows a sequential pattern of ground contact. First the heel strikes, then the foot is flat with the heel and forefoot during midstance, and finally just the forefoot in contact during terminal stance[222]. Heel only contact with the ground is the first 12-15% of the gait cycle, before the forefoot touches down. For the next 20% of the gait cycle, both the heel and forefoot are in contact with the ground. Most commonly the fifth metatarsal were be the first part of the forefoot to contact the ground (71%), and small percent were touch down with all the metatarsals at once (22%). As the heel rises from the ground only the forefoot is in contact, with this at about 31% of the cycle to the time of toe off. It is common for either the first digit, or all five toes to be the last contact point with the ground.[222] These foot mechanics are important in gait analysis as the kinetic forces change greatly with different types of foot strike patterns.

Kinetics

Kinetics are the study of the forces that create movement[218, 235]. Forces have vector quantities, meaning they have direction and magnitude. Additionally, the point in which the force is applied is a key factor in determining angular motions. This is an important factor when studying human gait as both linear and angular accelerations typically occur simultaneously. When a force is applied over an area, this is called pressure (e.g. foot contact with the ground).

The study of kinetics is based upon Newtonian principles, and are measured in Newton's (N), with 1N the amount of force needed to accelerate a 1kg mass by 1 m/s^2.

Ground reaction forces, joint moments, and joint powers provide insight into how the body accomplishes movement. This requires anthropometric measures of the individual as well as simultaneous acquisition of kinematics and the ground reaction forces, with the latter measured by a surface embedded force plate.

Ground Reaction Forces

Gravity forces bodies in motion to return to earth while walking and running. When the human body comes into contact with the Earth an impact force is delivered and the body absorbs the shock. These multiplanar ground reaction forces are distributed proximally up the kinetic chain from the center of pressure (COP) when the foot comes into contact with the ground. These ground reaction forces (GRF) are can be displayed as a single vector with direction and magnitude, or divided into their respective planes [219, 222]. The three main components of the GRF are the vertical, fore-aft, and medial/lateral forces. Vertical ground reaction forces (vGRF) is the largest and the peak is usually 1.3-1.5 times the person's body mass during the loading response (absorption) and push-off (propulsion) creating a bimodal appearance graphically [218]. The decrease valley seen in midstance results from the body rolling forward over a stationary foot during the ankle rocker and tibial advancement[222]. During running the vGRF is 2-3 times body mass and typically displays a small impact during the loading response and larger peak for the propulsion phase of stance.[218] Vertical ground reaction forces increase linearly with gait velocity up to about 60% of maximum sprinting, then plateaus

despite increasing velocity[145]. Another cause for the differences seen in walking and running vGRF is in the vertical displacement of the center of mass (COM) of the body. The COM is most vertical at midstance in walking and at its lowest vertical position at midstance in running[220].

Fore-aft forces during walking are typically much smaller than vGRF with braking absorption forces the first half of stance, then switching to propulsion pushing forces the second half[218]. At running velocities, the timing is similar, with considerably less braking forces. This decrease, or complete absence, in braking force makes sense during running as it increases forward progression and efficiency. Runners also were decrease the frequency of heel striking, moving to forefoot or toe striking[145]. This is due to longer strides and the decreased need for braking ability. Medial-Lateral are the smallest GRF, at about 10% of body mass. There are medial forces during walking and running in first 40% of stance, with some lateral forces the middle 20% of running stance.

Many factors play a role in the size and shape of these GRF. Body mass, velocity, rate of loading, walking/running style, and mechanical properties of foot and surface interface have been previously identified as some of these factors that contribute to force characteristics [145].

Joint Moments and Power

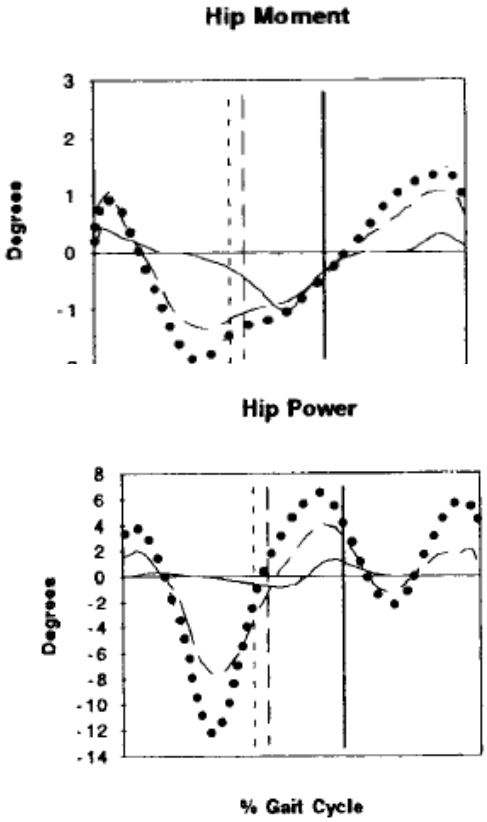
Joint torque forces, or moments, describe off center forces (not through an object's center of mass and rotation) occurring a known distance from an axis of rotation[194, 218]. The product of force (F) and the perpendicular distance from the center of rotation, or moment arm, (*d*) yields the magnitude of force (M=F*d*). These moments are reported in Newton meters

(Nm) and are often scaled to body mass (Nm/kg), or body mass and leg length. These forces can be calculated from kinetic and kinematic (angular motions) data to describe net, or summed, effects[235]. To maintain upright gait and forward progression (i.e. not collapsing) the body generates internal moments created by muscles, tendons, ligaments, and other soft tissues crossing a joint. Moments are calculated via inverse dynamics, which allows for only net forces of hip flexion in this case, and should not be thought of as hip flexor strength. Conversely, external moments describe the forces that gravity and the ground place upon the body. For example at the hip, an external hip flexion moment is created at initial contact as the upper body and thigh are forced closer together. This is countered by an internal hip extension moment as the gluteal muscles contract to resist collapsing forces and maintain upright gait. However, internal moments should not be thought of as muscle strength[212]. Even though the gluteal muscles are contracting, there may be co-contraction, albeit smaller magnitude, of the hip flexors muscles. The resultant action at the joint is dominated by the hip extensors, which is why the net result is an internal hip extension moment.

The product of joint moments and the concurrent angular velocity at a joint is called power[219]. This value indicates the rate and type of work produced for a joint. When the muscles are eccentrically contracting (lengthening), energy is being absorbed and stored. Conversely, when the muscles concentrically contracting (shortening) power is being created to progress gait. Again these power values are net, or summed effects, at a joint as are moments. The study of kinetics during the gait cycle tends to focus on the sagittal plane forces, similarly to kinematics. This is due to the fact that the largest amount of forces are directed in this plane. There is an initial internal hip extension moment peak of 0.7 Nm/kg at IC as the hip is flexed

about 30-40^{o} during walking [219]. This continues through stance as the hip extenders are concentrically contracting and generating a peak of 0.6 W/kg of power. These were reverse once the hip reaches terminal stance and peak hip extension. The anterior capsule and hip flexor muscles are lengthened eccentrically to slow progression of extension and create power via absorption. The hip flexors create an internal hip flexion moment peak of about -0.7 Nm/kg during the swing phase as the leg is progressing forward and generating -0.6 W/kg of concentric power [219]. This reverses a second time in the gait cycle during terminal swing as the leg is preparing for the next IC and the hip is extending from concentric action of the hip extenders once again.

Increased hip extensor and flexor activity are required for the faster velocities of running and sprinting. The graphical patterns of the internal moments for the hip are almost identical to walking, but larger in quantity [212](Fig. 20). The first internal extensor moment is larger than 1.0 Nm/kg during stance with a corresponding increased power generation of the concentrically contracting hip extensors. Then the most noticeable sagittal plane changes occur as the internal hip flexor moment significantly increases to -1.5 Nm/kg during late stance through mid-swing. The impulse of the moment appears to more than double (Fig. 20).

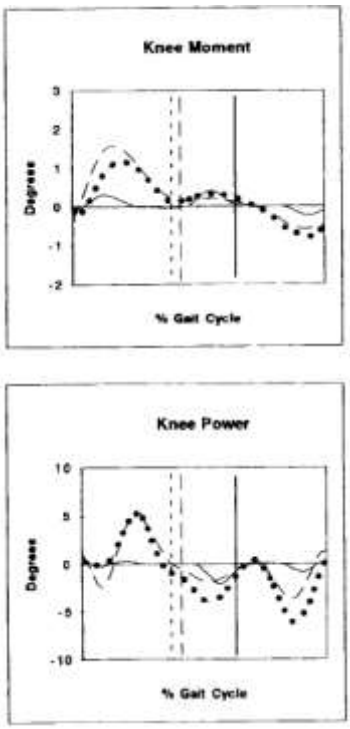


The hip flexors are eccentrically contracting during late stance to absorb power prior to toe off, which then becomes a concentric generation of power through mid-swing to advance the limb. The end of the cycle features an internal hip extension moment with power absorption as the hip extensors contract to slow the rate of hip flexion, then changes to a concentric power creation in lower the leg in preparation for the next IC.

Figure 20. Internal Hip Moments (above) and Joint Power (below) during Walking (solid line), Running (long dash), Sprinting (dots).

The knee is flexed at IC and there is an internal knee extension moment peak of 0.53 Nm/kg, and tapers off throughout the loading response during walking[219]. This corresponds with the quadriceps eccentrically contracting and -1.05 W/kg of power absorption seen during the first 15% of the gait cycle. The knee then extends briefly and 0.59 W/kg of power is generated concentrically by the quadriceps as the leg is straightened through midstance[107]. There is a net knee flexor moment during terminal stance that is due to eccentric contraction of the gastroc-soleus complex and power absorption. These aid in creating bending and clearance for the foot during swing. The majority of swing there is little kinetic activity in the knee until

terminal swing. The knee flexors (hamstrings) are eccentrically contracting to slow the rate of knee extension and absorbing power, before concentrically contracting to bring the leg back down in preparation for the next IC.[219]



Knee sagittal plane kinetics during running are graphically similar, but with larger peaks [212] (Figure 21). There is a large peak internal knee extension moment at IC of 1.4 Nm·kg, which again tapers off the remaining of stance[219]. The first half is dominated by -5.36 W/kg of absorption power due to the eccentric quadriceps activity as the knee slightly flexes[219].

Figure 21. Knee moments (above) and powers (below) for walking (solid line), running (dashed line) and sprinting (dotted line).

This is a fivefold increase in power absorption compared with walking. This reverses during midstance as the quadriceps then concentrically contract to extend the knee and generate power. The peak knee flexor moment is during double float and terminal swing as the hamstrings are eccentrically contracting to limit knee extension, as seen by the large amount of power absorption[212]. The amount of loading and power absorption of the hamstrings is significantly enlarged with increasing velocity[53]. This power is then reversed to a generation force as the hamstrings are concentrically contracting before IC[212, 219].

Ankle sagittal plane kinetics at IC are highly dependent on foot strike patterns. Heel strikers were have an initial internal dorsiflexion moment and power absorption as the ankle dorsiflexors eccentrically slow the decent of the foot towards the ground and to avoid foot slap[219]. Those who land with a flat foot were not have this finding. Regardless of foot strike patterns, there is an internal plantarflexion moment coupled with eccentric power absorption of -0.79 W/kg at the gastroc-soleus as the shank advances over the stationary foot. The plantarflexion moment grows and peaks at 1.26 Nm/kg as 3.5 W/kg of power is generated by the concentric contraction of the gastroc-soleus complex during terminal stance and push off[219].

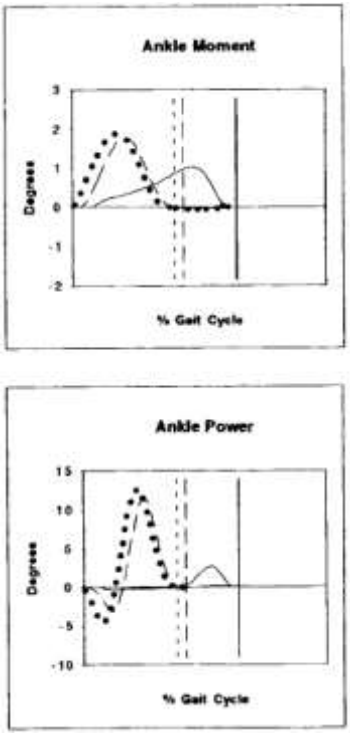


Figure 22. Ankle moments (above) and powers (below) for walking (solid line), running (dashed line) and sprinting (dotted line).

Foot strike patterns similarly affect running ankle kinetics compared with walking, as heel strikers display an initial internal dorsiflexion moment. The loading response of both contact patterns show large internal plantarflexion moments the majority of stance, peaking around 1.7 to 2.0 Nm/kg[212, 219] (Figure 9). Stance power is bimodal with an initial period of -5.13 W/kg of absorption from eccentric gastroc-soleus activity. The plantarflexors then concentrically contract during propulsion to generate 9 to 13 W/kg of power.[219, 285]

Frontal plane kinetics are lower in magnitude than the sagittal plane[218], but are equally important as they been found in many pathologic conditions at the hip, knee, and ankle.[88, 213,

[222, 229, 288] Normal kinetics have a high degree of inter-subject variability as a result of this low magnitude[218, 219]. During walking and running velocities the hip is adducted at IC and the gluteus medius (and other hip abductors) eccentrically slow the rate of contralateral pelvis drop via small net power absorption[218]. This reverses at midstance as the hip slightly abducts to elevate the pelvis for swing. The peak internal hip abduction moment is 0.46 and 1.4 Nm/kg for walking and running, respectively[209, 218]. The peaks occur right at the transition from eccentric to concentric contraction of the hip abductors to aid in propulsion and foot clearance (via pelvis elevation). The primary finding from power graphs demonstrate mid- to terminal stance generation peaks of 0.24 and 0.77 W/kg for walking and running, respectively. The knee has similar frontal power production as the hip but with less moment forces.

The frontal plane forces at the knee are typically reported in external moments. The ground reaction force usually falls medial to the knee which were dictate the typical patterns of force distribution[220]. During stance the hip is adducted, creating peak external knee adduction moments (KAM) of 0.12 to 0.28 Nm/kg during walking[177, 218]. The magnitude of the KAM is related to the vertical and mediolateral GRFs as well as the muscle action of the gluteus maximus & medius, vasti muscles of the thigh, and the gastroc-soleus complex[220]. Increased activity of the hip abductors may contribute to increased peak KAM[113, 220], as they contribute to the vertical and mediolateral GRFs, however this is remains controversial. Henriksen et al. found that experimentally decreased hip abduction activity and decreased internal hip abduction moments have not led to increasing KAM[113]. However, the muscles known to counteract KAM and create internal knee abduction moments are primarily the vasti during the first half of stance and the gastroc-soleus during the second half of stance[220] (Figure 22).

During running, the amount of KAM may be dependent upon the amount knee adduction (varus) excursion[288]. Those who have larger knee adduction angles were increase peak KAM 32%, from 0.78 to 1.14 Nm/kg. This is believed to be a result of weak hip abduction muscles, but again this finding remains controversial[220, 288].

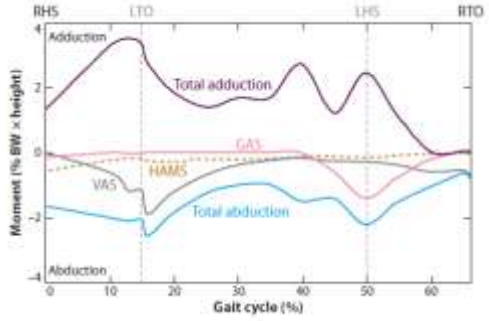


Figure 22. Frontal plane stance knee moments and muscle contributions (GAS=gastroc-soleus, HAMS=hamstrings, VAS=vasti)

Frontal plane ankle kinetics have a high degree of inter-subject variability[80, 180], so discussion of this plane is more challenging. Eng et al. found that heel striking at IC were create an internal eversion ankle moment, which peaks at 20% of the gait cycle in young adults[80]. During midstance this were reverse to an internal inversion moment, before switching back to an internal eversion moment during terminal stance[80] (Fig. 23). However, MacWilliams et al. found that adolescents only have internal eversion moments during stance and lack the reversals found by Eng[80, 180]. The power is similarly high in variability, with net generation during early stance and absorption in terminal stance[80] (Fig. 23).

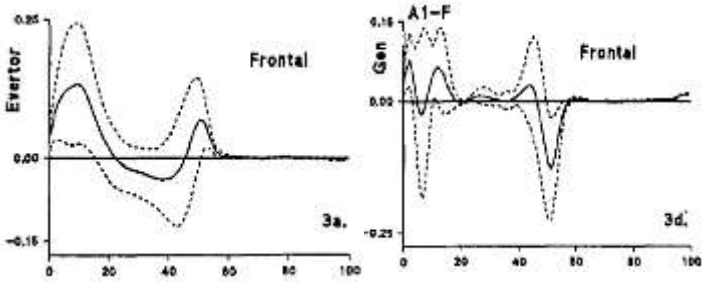


Figure 23. Frontal plane internal ankle moments (left) and power (right) over one gait cycle.

The transverse plane during walking displays the smallest internal moments and power generation of the lower extremity[80]. During the loading response there is an external rotation

moment at both the hip and knee to resist external forces driving hip and knee internal rotation that accompany knee flexion[80]. There is net power absorption seen in early stance as the eccentrically acting external rotators of the hip and passive ligaments of the knee are slowing the forward rotation of the pelvis and femur. The forces in late stance reverse to an internal rotation moment at both the hip and knee during knee extension. The ankle differs in that there are only external rotation moments in the transverse plane and the power absorption/generation is highly variable.[80]

The hip maintains an internally rotated position during running stance and produces only 9% of the joint's total internal moments[84]. The external rotator moment peaks at 0.3 Nm/kg, compared to the much larger sagittal and frontal plane forces of 1.5 and 1.3 Nm/kg, respectively. As the hip resists the internal rotation forces, eccentric contractions create absorption power of about -0.2 W/kg.[84] The knee and ankle moments and powers are miniscule in the transverse plane[84, 195]. During the loading response the tibia internally rotates to create ankle external rotation and there is power absorption at both joints. Only 6.4% of the total absorption power in the transverse plane comes from the ankle and 7.4% at the knee.[195]

Measurement of Hip Strength

Strength Deficits in Femoroacetabular Impingement

Casartelli et al. were the first to describe hip strength in a group with hip impingement. Pre-operative FAI patients have hip flexion, abduction, adduction, and external rotation strength deficits of 11-28% when compared to controls[48]. Additionally, the tensor fascia late

has significantly lower electromyography (EMG) activity in FAI participants. Strength was assessed via the biodex iskoketic dynamometer (hip flexion/extension) and HHD (hip abduction, adduction, IR, and ER) for isometric maximal voluntary contractions. Authors hypothesize that the strength deficits may stem from pain/fear of pain, atrophy (duration of symptoms not reported), or reduced activation. The same researchers separately studied muscle fatigue in pre-operative FAI patients[46]. Isometric and isokinetic hip flexor strength deficits of 16-21% were again found compared with controls. However, during the fatigue protocols, no differences were found between FAI and control groups. The authors concluded that FAI patient's pain during dynamic activities may not be related to hip flexor fatigue difference compared to healthy populations.

In Casartelli et. al's follow-up study, post-operative strength improved in the hip muscles, except for the flexors, when tested 2.5 ± 0.2 years after arthroscopy[47]. In fact, the hip extensors and internal and external rotators were post-operatively stronger than controls. It should be noted that the controls were only tested once and not over repeated measures. Four of the eight FAI patients were not completely satisfied with the outcome of their hip arthroscopy, but the study did not identify hip weakness as a causative factor in these poorer outcomes.

Handheld and Isokinetic Dynamometry Assessments

Isometric maximal voluntary contraction (MVC) strength assessment of the hip muscles can be assessed via HHD[7, 10, 15, 28, 46-48, 55, 56, 72, 101, 105, 121, 122, 138, 151, 166, 247, 257] and isokinetic testing[47, 48, 72, 101, 122, 138, 257]. Though the isokinetic dynamometer is considered the gold

standard for strength assessment, HHD is a valid and reliable alternative when considering the application ease, cost, time, and portability[257]. Direct comparison of these studies is difficult due to the wide-ranging methodologies, particularly in the case of the HHD literature.

Factors that limit study comparison include testing procedures and statistical analyses. Procedural differences include type of force application, length of contraction, positioning of the hip, and distance of the HHD from the joint center. Typical contraction lengths range from 3-7 seconds, depending on the type of application force. The two types of HHD forces applied are either a "make test" in which the patient applies force against an examiner or fixed HHD, or the "break test" in which the examiner pushes against the patient's resistance force. Perhaps the most important factor is patient positioning, which greatly determines force production.

Hip flexion has been assessed from a range of 0-90°[121, 122, 138], the abductor/adductor muscles in varying side lying positions[7, 10, 48, 55] and short or long levers[151], and the internal/external rotators in seated or supine and neutral or individualized midpoints. Bloom et al. provided evidence that the rotators should be tested in seated position[28], while Chibulka et al. added that they should be tested in each person's own mid-point range of motion. This is to avoid mistakenly testing a shortened or lengthened muscle, as rotational deficits are common in the ipsilateral hip rotators[55].

Additionally, the data analysis may include number of trials collected, various scaling routines, and averaging of trials for analysis. Typically, two to three trials are collected, scaled via body mass, then averaged together[46-48, 55, 56, 72, 105, 121, 122, 138]. These may also be reported as torques if also scaled by distance of the lever arm.

Anatomy of the Hip

Acetabulum

The hip is an enarthrodial joint that is comprised of the articulation between the acetabulum of the pelvis and the head of the proximal femur. The acetabulum ("vinegar cup" in Latin) is the junction of the three bones of the pelvis, the ilium, ischium, and pubis[51]. At birth these are separated by hyaline cartilage prior to fusing[189]. The ossification center of the bones is the y-shaped triradiate cartilage, which closes during adolescence[137, 232]. The fossa of the acetabulum is approximately 5 cm deep and lies within the smoothed lunate surface[189]. The circumferential ridge of the acetabulum is round in 60% of people and helps cover the femoral head[190]. The inferior acetabular notch, spanned by the transverse acetabular ligament, is the only area without a prominent ridge.[129, 137] Females have smaller articular surfaces adjacent to the acetabular notch, which has been attributed to a substantially greater notch width[150]. The inferior notch spans 7-5 o'clock position.

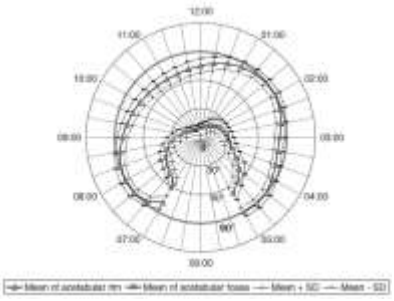


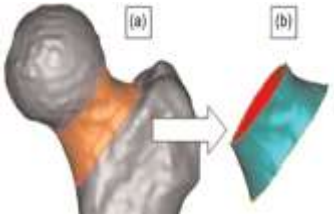
Figure 24. Average rim (triangles) and fossa orientations in 104 asymptomatic acetabuli.

The acetabular opening is normally oriented anteriorly, laterally, and inferiorly, complementing the superior medial orientation of the femoral head to aid stable alignment[137]. Imaging studies of asymptomatic populations indicate a normal acetabulum has 20° anteversion[37, 221], 40-55° inclination[37, 90, 150] and 35° of superior lateral coverage[37, 90]. Anteversion of the acetabulum is initially assessed qualitatively via radiographs features[139, 140, 142, 234], but is best quantified via computed tomography (CT)[67, 69, 99, 221, 281]. Previous studies have indicated that males typically

have 3-5° less anteversion than females[114, 150, 190, 197, 267], and no differences exist between Caucasians and African Americans[267]. However, there is lack of information relative to other races related anteversion differences. It's possible that there are genetic variations in amongst races that lead to differing hip morphologies, as evident by Asians displaying less superior lateral acetabular coverage[78, 263]. The normal hip joint should have complementary relationship between the acetabulum and femur leading to about 75% total coverage of the femoral head[68, 69]. Abnormal coverage, either excessively deep or shallow, is associated with hip pathologies[68, 69, 266] and often lead to altered proximal femur anatomy as well[260].

Femur

The human femur features a proximally round head sitting upon a hyperboloid (rectangle with two concave parallel lines) shaped neck (Fig. 25)[191]. This transitions to the bowed shaft with its greater and lesser trochanters, ending distally with the medial and lateral condyles[189]. The head and neck are characterized by several measures that describe the roundness of the head and the amounts of angulation between the head, neck, shaft and condyles. The head is about 55 and 48 mm in diameter for males and females, respectively[274]. The sphericity of the head in relation to the neck is determined by the alpha angle, which is normally 40-50°[211, 273]. This is the angle between a line connecting neck axis and a line through the center of the head to the point where sphericity is lost. The neck is typically anteverted about 10-20° from a reference line between the femoral condyles[114, 272, 273] (Fig. 25) and connects to the shaft at an angle of 125° (angle of inclination)[273].



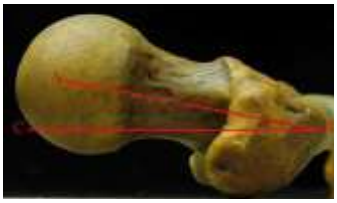


Figure 25. The proximal femur (a) and a hyperboloid shaped neck (b), then an anteverted neck (right).

Cartilage

Articular cartilage is a type of hyaline cartilage is found at the end of opposing bones and in a synovial joint[189]. Healthy articular cartilage is optimized to reduce friction and distribute weight evenly throughout the joint[193]. In the hip these layers of cartilage are thin ranging from 0.32 mm to 2.83 mm on the femoral head and from 0.95 mm to 3.13 mm on the acetabulum[206, 289]. Mainly composed of water (60-80%), type II collagen (15-20% of weight), and proteoglycans (PGs) (3-10% of weight) [276, 289]. The protein cores of PGs are lined by attachments to glycosaminoglycans (GAG). These two, PG and GAG, help attract sodium which then draw water into the tissue to generate the swelling pressure of cartilage[184]. The breakdown of these structures coupled with an increase in inflammatory biomarkers has been linked to reduced contact stress coping and may lead to osteoarthritis[22, 108, 193, 230, 256].

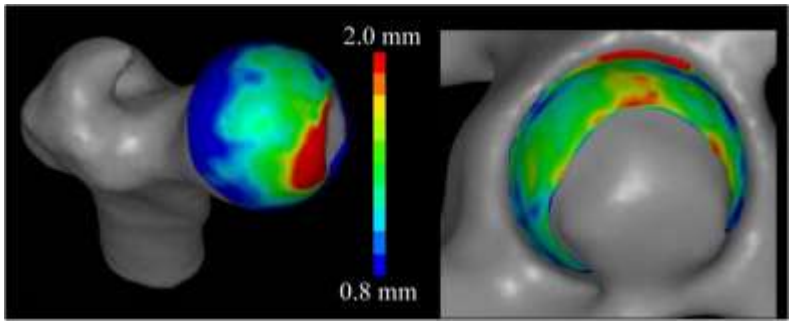
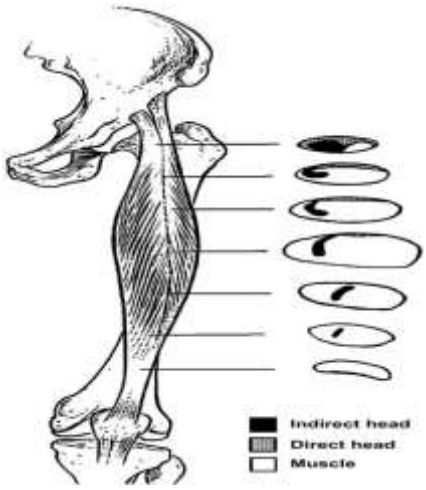


Figure 26. Typical cartilage thickness in the femur (left) and acetabulum (right) with red indicating the densest concentrations.

Soft tissue structures

Muscles Acting on the Hip

The muscles of the hip are separated into the anterior, posterior, and medial compartments[202]. The anterior compartment contains the flexors of the thigh and extensors of the knee. The iliacus originates from the iliac fossa and as far anteriorly as the anterior superior and inferior iliac spines (ASIS and AIIS)[66] and the psoas muscles from the lumbar vertebrae[202], however it's common for both to have variant slips[135]. They form a common tendon and insert on the femur at the lesser trochanter and pectinieal lines. Near the level of anterior acetabular labrum and lying directly anterior, iliopsoas has circumference of 64mm and is 55% muscle belly and 45% tendon[6]. Due to their close proximity, the iliopsoas has been implicated as a possible contributor to labrum pathology[6, 27, 74]. The iliopsoas group are the main thigh flexors but are also aided by the pectineus, sartorius, and rectus femoris.



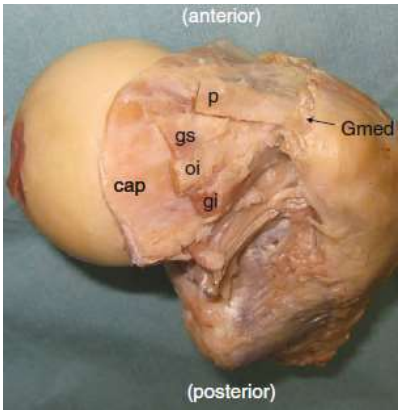
The rectus femoris is an important muscle as it is biarticulate and spans both the hip and knee joints[202]. Proximally there are two heads of origin, the direct head at the AIIS and the indirect head from the superior acetabular ridge[109, 125]. The two conjoin to form a tendon, with the direct head contributing to the superficial portion and the indirect forming a deep intramuscular part[31, 144](Figure 27).

Figure 27. Origin and tendon composition of the two heads of the rectus femoris.

Deep to the superior portion of the rectus lies the iliocapsularis muscle, which has only recently been recognized[12, 280]. It originates from the AIIS and inserts distally near the lesser trochanter. It appears as though this muscle is a key active stabilizer in people with dysplastic hips as it hypertrophies compared with deeper socket hip that rely on bony stability[12].

The medial compartment is comprised of the adductor longus, brevis, and magnus, gracilis, and obturator externus[202]. They typically originate from the pubis, with the exception of the adductor magnus which also has a "hamstring" part from the ischial tuberosity. They insert along the linea aspera and femur with the exception of the gracilis which crosses the knee to insert at the pes anserine and the obturator externus at the trochanteric fossa of the proximal femur[189, 202]. Due to its orientation the obturator externus is believed to be a steadying force of the head in the acetabulum.

The posterior compartment features the gluteal muscles (maximus, medius, minimus), tensor fascia lata (TFL), the short hip rotators (obturator internus and superior and inferior gemelli), piriformis, and quadratus femoris[189, 202]. The gluteus maximus is a powerful hip extensor that originates on the ilium, sacrum, and coccyx and inserts at the iliotibial band (ITB) and gluteal tuberosity. The gluteus medius, minimus, and TLF originate on the lateral ilium and insert on the greater trochanter and ITB, working together to abduct the hip. The piriformis, quadratus femoris, and short external rotators were also abduct the hip when the thigh is flexed. When extended, they were externally rotate the hip. The short external rotators are small muscles that form a conjoined tendon (Fig. 28) that is key in maintaining dynamic hip stability[131]. Insertion variability of the tendons are high, possibly placing them at risk of being



damaged during capsular release during the direct anterior[131] and posterior approaches[284] for total hip arthroplasties.

Figure 28. The short external rotator muscles insertions. p = piriformis; gs = gemellus superior; oi = obturator internus; gi = gemellus inferior; Gmed = gluteus medius; cap = capsule.

Capsule and Ligaments

The articular joint capsule consists of strong and dense fibers arranged in a cylindrical shape connecting the margins of the acetabulum to the proximal femur[278]. The capsule ranges from 2-6 mm in thickness and is 18-33 mm long[282]. The thickest fibers are located in the anterior superior portion at the 1-2 o'clock positions[282], possibly being related with intra-articular adhesions[17]. The capsule is additionally supported by four distinct ligamentous structures.

The iliofemoral (ligament of Bigelow), ischiofemoral, and pubofemoral ligaments (Fig. 29) represent thickenings of the joint capsule that reinforce and stabilize the hip joint[123, 189, 202, 278]. They are responsible for femoral head stability in the acetabulum by resisting translation and limiting extremes of motion, which is aided by their spiraling orientations. The fourth

structure is the deeper zona orbicularis, which surrounds the deep portion of the femoral neck supporting like a sling[278].

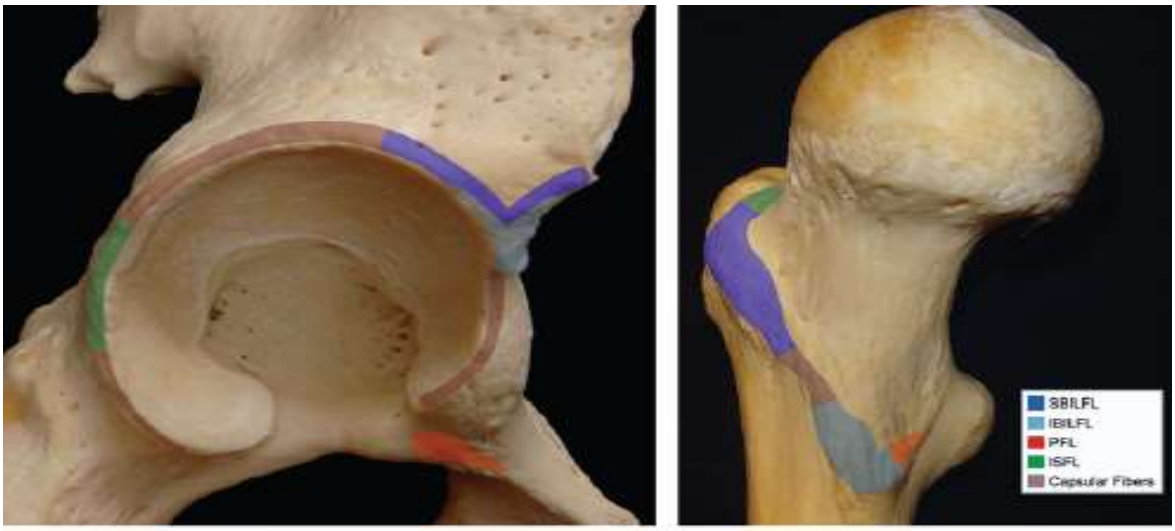


Figure 29. Proximal (left) and distal (right) attachments of the capsular ligaments.

Aiding in femoroacetabular stability are the ligamentum teres (LT) and transverse acetabular ligament (TAL). The LT's two distinct bands, the ischial and pubic fascicles, insert into the femoral head's central fovea[50] from their origin on the transverse acetabular ligament. It is most taut in the combined flexion, adduction, and external rotation position when the hip is in an open pack position. Micro-instability and subluxations of the hip are believed to a direct result of isolated LT tears in the shallow hip socket[50]. However, the exact role of the LT in hip stability is not clearly understood, as it is used as a labral graft without incidences of instability[252]. The transverse acetabular ligament which spans the inferior acetabular notch to "complete" the circular shape blends into the acetabular labrum to provide extra coverage of the femoral head[51, 77, 123].

Labrum

The labrum is a fibrocartilagenous rim that attaches to the acetabular margins to deepen and extend femoral head coverage, thereby enhancing stability and congruity[86]. It functions to seal in joint fluids, increase joint fluid pressure, and enhance joint lubrication[85-87], which has the benefits of providing nutrients, distributing forces symmetrically, and reducing wear on the adjacent cartilage. Compromise of labrum function, secondary to disruption of the acetabular seal or tears, decreases the force required to overcome the sealing effect, leads to decreased joint stability, and increases in cartilage friction possibly predisposing them for osteoarthritis[64, 255]. Additionally, dysplastic acetabuli rely more heavily on the labrum to dissipate joint forces across the joint[112] and therefore, labrum repair is preferred over debridement in the case of labrum pathology due to its ability to restore some of the fluid seal effect[42].

The labrum is also thought to contribute to pain and inflammation of the hip. It is populated with unique highly active fibrochondrocyte-like cells that express, release proinflammatory enzymes and cytokines, and react to a pro-inflammatory stimuli[71]. Pain-associated free nerve ending expression, via nocio-receptors, are located predominantly at its base and decrease toward the periphery[110]. It is in these ways disturbed tissue function relates to clinical pathology.

Neurovascular Anatomy

The blood supply to the hip is a complex system with many variations in distribution patterns and anastomoses that link together[54, 132, 189, 202]. The bony acetabulum is primarily supplied by the superior and inferior gluteals and the obturator arteries[132].

Blood supply to the proximal femur is primarily achieved via the medial and lateral femoral circumflex arteries (MFC and LFC)[54] and is additionally supported by an anastomosis with the inferior gluteal artery near the obturator externus tendon[102]. The MFC and LFC arteries leave the femoral triangle, arising from either the femoral or deep profunda arteries, and form an extra-capsular ring around the femoral neck[54]. These give rise to the cervical ascending arteries (retinacular arteries) that travel up the neck to the head to form an intra-capsular anastomosis ring[54] (Fig. 30).

Figure 30. Blood supply to the femoral head via the cervical ascending arteries anastomoses.

The capsule is thought to receive its innervation from at least seven different nerves, which include the obturator, femoral, sciatic, superior/inferior gluteals, nerve to quadratus femoris, and accessory obturator nerve[141].

Morphological variations of either the femur or pelvis can have serious implications on human movement and the long term health of the joint[1, 3, 18, 44, 96, 159, 167, 169, 253]. The acetabulum often presents as too shallow or overly tight, whereas the femoral head may lose sphericity. Excessive torsion of bone may also confound these abnormal features as well. How and why many of these morphologies develop is still under debate. Progression to

symptomatic hips is multifactorial and many developmental theories exist. A clear understanding of these acetabular and femoral conditions is paramount to guiding diagnosis and treatment.

Theories of Developmental Etiology

Development of the hip is multifaceted and complex. Unfortunately there is a lack of longitudinal studies examining the causes of hip morphology. However there are possible, embryologic[49], genetic[13, 120, 277], mechanical loading[120], and evolutionary[118-120] factors that lead to hip morphologies.

The hip joint of the human fetus is held in a position of at least 90° of flexion, initially influencing the relationship between the acetabulum and the femoral head[49]. Part of the femoral head may be contact with the anterior portion of the acetabulum in this fetal position. Local mechanical stimuli provides positional information, guiding genetic patterning of the musculoskeletal anatomy[120]. Static loading is needed for bone modeling, whereas motion is mainly involved in joint development[120]. There may be a mutually dependent counterbalance needed between the acetabulum and femur during development that is key for avoiding risk factors for pathology[37, 260] . Once the hip shapes are irregular, it were lead to predictable wear patterns that predispose for early osteoarthrosis (OA)[165, 198, 199, 253, 261]. These early findings do not elucidated what leads to morphologies and hip pathology, but led to attempts to define genetic and loading history factors.

Genetics and loading of the hip may initially affect how the shapes of the hip develops and the type of cartilage a person has. The additional amount of loading over the course of

one's life may then play into how pathology and OA develop (Fig. 31)[120] . Gene expression is varied across different ethnicities. Asians have been shown to have fewer hip morphology variants that lead to disease compared with Caucasian Americans and Europeans[78, 117]. Additionally, high intensity sports (i.e. increased load history) during adolescence, just after physeal closure, is associated with a higher rate of developing hip morphology[2, 45, 249]. It is important to remember that simply having hip morphology, weak cartilage type, and high load histories were not always lead to pathology, but increases relative risk.

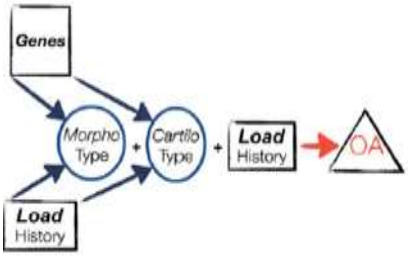


Figure 31. Theory of how genes and loading history lead to hip pathology.

Human Bipedalism and Hip Morphology

It's been said that evolution has no plan or goal, it just happens[119]. Humans are unique in that we are the only mammals that exclusively use bipedal gait as our primary form of motion. Man's evolutionary journey features many remarkable modifications, two of the most important are bipedal gait and encephalization, or an increased brain to body size[119]. The human skeleton has undergone great changes to accommodate for these two key aspects of human evolution[119, 178, 179].

There are several key points in the development of human bipedalism with important differences at the lumbosacral spine and pelvis. Humans have a far more lordotic lumbar spine compared to chimpanzees and other mammals[119, 178]. The human lumbar vertebrae exhibit wider lamina and space in the zygapophyses joints leading to increased lumbar lordosis and changing sagittal plane dynamics.[178] Utilizing full hip and pelvis range of motion increases

leverage and ability to create negative energy eccentrically, making gait more efficient for humans during extended periods of locomotion.[178] However, it allows humans to fully extend the hip during gait, which is a position shown to place a great deal of strain on the anterior lateral labrum and possibly placing it at greater risk of tears[240].

The human pelvis also became shorter, wider, and more curved compared to the long and flat pelvis seen in monkey, ape, and chimp pelves[119] (Fig. 32). This led to exceedingly mobile lower spines than the great apes (short backed primates). The wings of the ilium now curve forward changing the pull of the gluteus medius, gluteus minimus, and tensor fascia lata from extensor to abductors. Lack of abductors explains why chimpanzee-centric gait has a trendelenburg trunk sway[136]. This also changes the amount of compressive stress on the superior neck of the femur, leading to changes in ossification patterns amongst different species. The femoral neck of a stance phase limb is loaded with 80% of the total body weight as a cantilevered beam and supports the upper body and swinging limb.[119, 178]

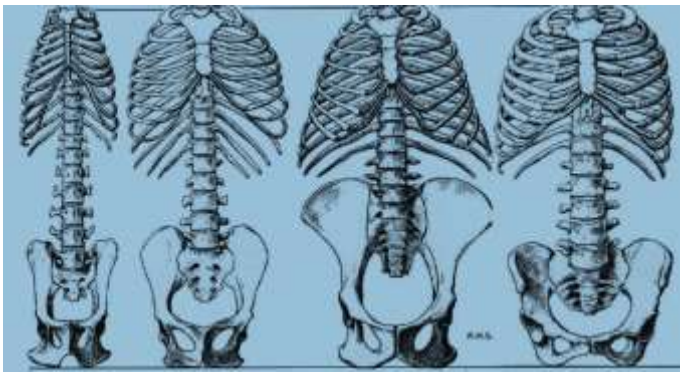


Figure 32. Spine and pelvis of a monkey, ape, chimpanzee and human.

Increasing of the pelvic cavity and enlargement of the birth canal is more accommodating for increasing head and brain sizes. It is currently unknown if the change in human brain size led to pelvic and spine changes, or vice versa. Regardless of which is causative, the resultant wider pelvis spaces the hips further apart changing the requirements

for bipedal gait. Childbirth adaptations illustrate how the abductor moment arm lengthened and increased femoral neck length in order to provide increased stability during gait.[119] Additionally, human females have a delayed secondary ossification center in the pubic symphysis that does not fuse until after the third decade, near the end of childbearing stage. This allows for continued forward growth of the pubic rami to allow increased pelvic outlet space and rotational births[119].

Upright walking has also repositioned alignment to a vertical pelvis and extension of the hip. Quadrupeds were maintain a covered femoral head by the acetabulum whereas bipedal gait creates an uncovering of the anterior joint (Fig. 33). This places the human hip at greater amount of acetabular rim loading, as adequate femoral head and neck offset is required for anterior joint clearance[3, 4, 45, 96, 158]. These adaptations may necessitate a greater amount of anteversion in both the human femur and acetabulum for normal joint motions.

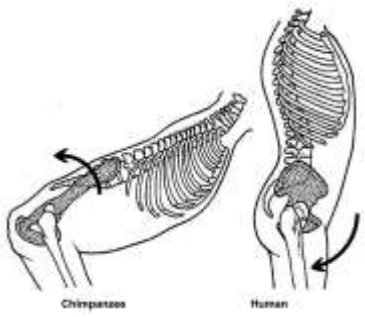


Figure 33. Extension of the lumbar spine, pelvis, and femur leads to anterior joint uncovering in humans.

The evolutionary changes to the orientation of the hip bones accommodate for upright bipedal gait. Chimps and apes have laterally facing acetabuli whereas humans are anteriorly facing (Fig 34). This anteverted position reduces the prominence of the ischial spines in the pelvic cavity and is advantageous for human childbirth[119, 221]. Retroversion, or decreased anteversion, of the pelvis has been shown to include the entire segment containing both the

acetabulum and the ischial spines[139, 140, 221]. Women have been shown to have more anteversion than males at all levels of the acetabulum using three dimensional computed tomography,[114, 221] which may lend support to this argument. Femoral morphology is vastly more variable across species as anteversion is beneficial to resist bending forces in both quadruped and biped walking[118, 119]. Retroversion of the femur is much more common in chimps and apes than humans (Fig. 34).[119] Additionally, decreased femoral anteversion, or absolute retroversion especially when combined with a retroverted acetabulum, is associated with the OA related degeneration of cartilage and bone in humans[272].

Figure 34. Femoral retroversion allows an orangutan to scratch its back with their toes.

Human Acetabular Deformities

Acetabular morphologies lead to unequal stress distribution that can predispose for early joint degeneration. Acetabular abnormalities are described via focal features, or on a spectrum of femoral head coverage that lead to distinct pathologies. The overly tight hip may suffer from pincer deformity or acetabular protrusio whereas the under-covered and shallow hip has acetabular dysplasia[170].

Though having the appearance of semi lunar or a hemi-sphere, the acetabulum has considerable inter-person variation[190]. The anterior acetabular ridge has been identified to have four configurations[190]. Sixty percent of pelves have a curved appearance, 25% are angular, 10% irregular, and 5% are straight. Half of people have bilaterally similarities and there appears to no gender effect on type of configuration or bilateral differences. The posterior

ridge almost always has a semicircular curve. The variations seen in the anterior ridge can affect the amount of acetabular version by an average of 6°.[190] In the tight hip, decreased acetabular anteversion may have serious clinical implications.

Pincer Deformities

Architectural abnormalities of the acetabulum that lead to over-coverage of the femoral head may predispose the femoral neck for direct impaction on the acetabular rim complex during hip motion[265]. These may stem from a complex combination of focal or global morphologies that lead to pincer type femoro-acetabular impingement (P-FAI)(Fig. 35).[170] These morphologies lead to crushing injuries of the acetabular labrum and the articular cartilage at the margin, as well as leveraging type lesions in the posterior inferior joint[96, 265]. The deformities and predictable patterns of damage are thought to be a precursors for early development of osteoarthritis and arthrosis[96].

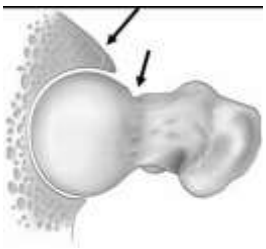


Figure 35. Anterior over-coverage leading to pincer impingement at the head neck junction and leading to labrum crushing injury.

Clinical presentation of P-FAI is typically groin, hip, and trochanteric pain that is exacerbated with increased activity[96, 168, 170]. Pain can also be noted with cutting and pivoting sporting activities, rotations in activities of daily living, and getting in and out of a chair[160]. Positive exam findings are particularly sensitive in the presence of labrum tears and cartilage breakdown. Range of motion may be limited and painful, as are provocative maneuvers[160]. The flexion-adduction-internal rotation (FADDIR) tests the anterior superior acetabular

rim/labrum[104] and flexion-abduction-external rotation (FABER) test were assesses the posterior-inferior rim/labrum[160]. Pincer deformity is focally caused by a retroverted acetabulum.

The normal human acetabulum is anteverted 20° at the horizontal midline[190, 221, 272]. Moving cranially there is a trend toward decreasing anteversion with an average of 15° in the superior level[221]. Retroversion has been defined as the tendency for the acetabulum to open posteriorly such that the anterior rim is more lateral than normal[221]. Absolute retroversion, or relative decrease in anteversion, is a common finding in many hip pathologies compared with asymptomatic populations[82]. This type of pincer deformity can stem from an excessive anterior wall, a deficient posterior wall, or a rotation of the entire pelvic segment.[100, 134, 139, 234]

Coxa profunda and acetabuli protrusio are features of P-FAI that have been historically associated with an excessively deep acetabulum leading to over-coverage[30] (Fig. 36). However, profunda's role in contributing to P-FAI is still not clearly defined, or understood. Isolated coxa profunda may not to lead to pathological states, however this needs further elucidation[8].

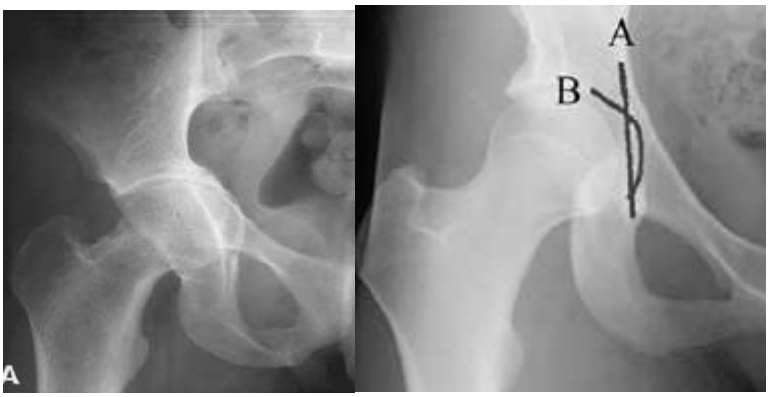


Figure 36. Examples of Acetabuli Protrusio (left) and Coxa Profunda (right; A=ilioischial line, B= acetabular fossa).

Hips with protrusio have similar reactive changes circumferentially along the posterior-inferior rim and head-neck junction as seen in retroversion P-FAI, so there is more clinical evidence compared to profunda[18, 171]. Protrusio deformities on the acetabular side are found alongside femoral deformities and secondary arthrosis of the joint space[171] (Fig. 37). The acetabular roof often has a downward sloped inclination, excessive superior lateral coverage, and covers a significant amount more of the femoral head compared with normal populations (82% vs. 71%)[69, 171]. The femoral head appears more medial, has lower center of rotation (relative to the greater trochanter) and decreased neck shaft angles[171]. The etiology of these deformities is currently unknown, but surgical intervention is indicated to slow the rate of arthrosis progression.

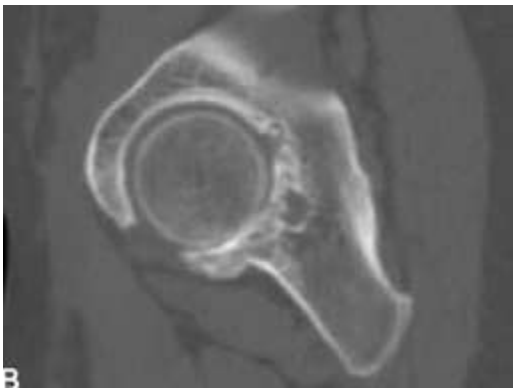


Figure 37. Excessive coverage and posterior joint degeneration as seen in patient with Acetabuli Protrusio.

Acetabular Dysplasia

Dysplastic hips are on the opposite end of the spectrum compared with pincer deformities, as they feature deficient coverage of the femoral head[259]. It is characterized by a decreased weight bearing zone which leads to shearing forces directed to the rim, labrum, and cartilage resulting in early OA[164, 210, 259](Fig. 38). Neonatal screenings help identify infants at risk for dysplasia (congenital/developmental dislocations) using physical examination and typically

resolve with use of bracing[34]. Often the signs and symptoms are dormant until adolescence or young adulthood, even in absence of childhood disease[164].

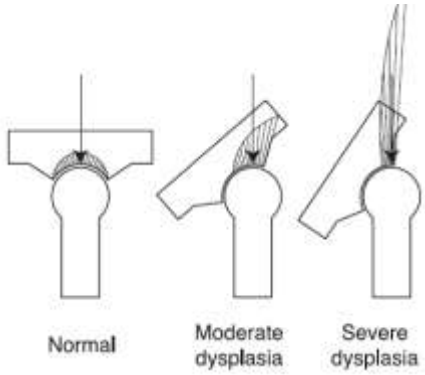


Figure 38. Schematic representation of force distribution in normal and dysplastic hips.

Young adults with symptomatic dysplasia present with moderate to severe insidious pain, with an associated limp, and/or trendelenburg gait, and positive FADDIR test. Activity exacerbates pain while rest were alleviate.[217] In addition to the clinical signs and symptoms, dysplasia is identified by advanced imaging. Typically, the acetabulum has an upslope and deficient coverage anterior and laterally (Fig. 39).

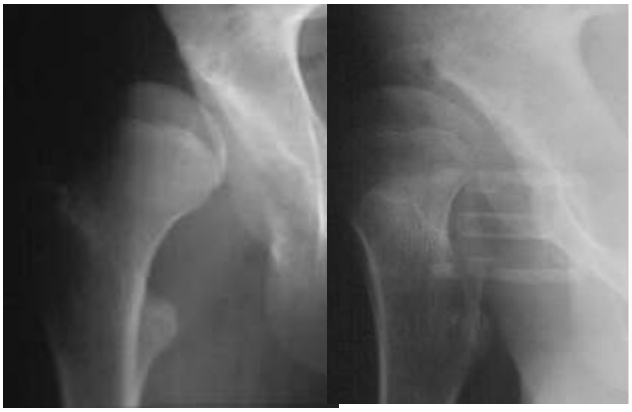


Figure 39. 16 year old with severe dysplasia and under-coverage laterally (AP-left) and anteriorly (false profile-right) on radiographs.

Femoral Deformities

Femoral deformities of the proximal femur typically effect the sphericity or offset between the head and neck. These deformities create an incongruent joint articulation with the acetabulum, leading to mechanical impact, damage to the labrum and cartilage, and early onset

of OA.[96, 182, 199, 201, 253, 264] Childhood diseases like Legg-Calve-Perthes disease (LCPD) and slipped capital femoral epiphysis (SCFE) lead to obvious deformities than can be monitored over the course of ones' life, however development of cam type FAI deformities are still far from being clearly understood[250]. They are typically classified according to radiographic and advanced imaging measures of the proximal femur.

Residual Deformity from Childhood Diseases

Residual deformities often remain following childhood disease and can lead to significant pain and discomfort into adult life. Misshaped femoral heads, physeal growth disturbances, and acetabular remodeling are common following the osteonecrosis of LCPD and lead to complex deformities[149] . The head takes on a flattened mushroomed appearance that leads to irregular motion and intra- and extra-articular impingement[264].

The common impingement areas are anteriorly between the head and anterior-superior acetabulum, laterally with the greater trochanter and ilium, and posteriorly with the greater/lesser trochanters and ilium[264]. Clinically, these patients present with pain patterns similar to other hip conditions[227], including pain exacerbated by activity[149, 264]. Motion is limited in all planes compared to normal populations and is *not* useful in determining the location of impingement because the joint congruency is so irregular[264].

Though a separate entity than LCPD, SCFE similarly results in loss of sphericity and head-neck offset leading to impingement patterns, labrum tears, and early OA[201, 253]. Shearing forces through a weak physis during adolescence leads to posterior migration of the head, creating a prominent anterior metaphysis. Regardless of slip severity, this prominence of the metaphysis

strikes the labrum and cartilage during hip range of motion, particularly flexion and internal rotation[201]. The model of post-SCFE rim impaction was an initial key to identifying cam type impingement in FAI[95].

Cam FAI

Cam deformities typically results from a non-spherical femoral head and decreased offset with the neck, which may lead to a jamming effect into the acetabulum with hip flexion[96]. This can be either a focal phenomenon (prominence or flattening of the head-neck junction) or a globally dismorphic head that results in a relatively larger neck[250]. Males are almost four times more likely to present with cam deformities, and there may be genetic and racial implications for relative risk[250], but further research into these areas is warranted.

Incongruent hip joints as a result of cam deformity are at a greater risk of impingement. This is true of LCPD and SCFE because of their obvious deformity, but also the more subtle cam type FAI deformity. Most often symptomatic FAI patients are physically active young adults[2, 45, 96, 97, 143, 207, 249]. The etiology of the isolated cam deformity is under debate, but one theory is that cam morphology is an adaptation of bipedal running[118]. A recent study showed that adolescent basketball players displayed physeal alterations compared to a relatively inactive age-matched control group[249]. The more active group had 12-15% more physeal extension toward the neck in anterior superior quadrant, possibly leading to cam type deformity. Additionally, two other studies have found that increase sporting activities may lead to cam deformity compared to controls[2, 45]. Agricola et al. found that elite youth soccer athletes as young as 12 years of age have cam deformities, and there is a higher prevalence compared with

controls[2]. These soccer athletes formally trained an average of 8 hours per week, however activity levels for controls were not reported[2]. Furthermore, Carsen et al. found when activity levels are high, adolescents are at a higher risk for cam morphology[45]. Those with cam morphology were more active during weekdays (6.56 ± 1.00 vs. 4.43 ± 0.32 hours per week) and particularly on Saturdays (7.06 ± 1.59 vs. 2.94 ± 0.51). Therefore, the large volume of vigorous sporting activity may create proximal femur changes at the head-neck junction.

The hip extension in bipedal gait diminishes the mechanical advantage femoral anteversion can have on shear forces on the capital physis[120]. The physis is nearly horizontal in neonates (less than one month old), but tilts approximately 30° more vertical in adolescence. Both mechanisms render the capital physis more vulnerable to shear forces.[120] These findings indicate a possible etiology of cam deformity. However, it's important to keep in mind the presence of cam does not necessarily lead to clinical and symptomatic FAI.

The initial clinical presentation of cam FAI is similar to other hip pathologies. There is pain and loss of range of motion, especially in flexion and internal rotation as the femur and acetabulum come into contact[11, 23, 96, 154, 290]. Provocative tests, such as the FADDIR, as almost always positive[104, 156, 185, 231, 271]. Once FAI is clinically suspected, advanced imaging can be useful to detect acetabular and proximal femur deformity and determine the amount of damage to the labrum and cartilage.

The Acetabular Labrum and Cartilage

The labrum is a fibrocartilagenous rim that attaches to the acetabular margins to deepen and extend femoral head coverage, thereby enhancing stability and congruity[86]. It

functions to seal in joint fluids, increase joint fluid pressure, and enhance joint lubrication[85-87], which has the benefits of providing nutrients, distributing forces symmetrically, and reducing wear on the adjacent cartilage. Compromise of labrum function, secondary to disruption of the acetabular seal or tears, decreases the force required to overcome the sealing effect, leads to decreased joint stability, and increases in cartilage friction possibly predisposing them for osteoarthritis[64, 255]. Therefore, labrum repair is preferred over debridement in the case of labrum pathology due to its ability to restore some of the fluid seal effect[42]. Additionally, dysplastic acetabuli rely most heavily on the labrum to dissipate joint forces across the joint[112], so labrum preservation is key.

Labrum injury is also thought to contribute to pain and inflammation of the hip. It is populated with unique highly active fibrochondrocyte-like cells that express, release proinflammatory enzymes and cytokines, and react to a pro-inflammatory stimuli[71]. Pain-associated free nerve ending expression, via nocio-receptors, are located predominantly at its base and decrease toward the periphery[110]. It is in these ways disturbed tissue function may relate to clinical pathology.

Articular cartilage is a type of hyaline cartilage found at the end of opposing bones and in a synovial joint[189]. Healthy articular cartilage is optimized to reduce friction and distribute weight evenly throughout the joint[193]. In the hip these layers of cartilage are thin, ranging from 0.32 mm to 2.83 mm on the femoral head and from 0.95 mm to 3.13 mm on the acetabulum[206, 289] (Fig. 10). It's mass is mainly composed of water (60-80%), type II collagen (15-20%), and proteoglycans (PGs) (3-10%)[276, 289]. The protein cores of PGs are lined by attachments to glycosaminoglycans (GAG). These two, PG and GAG, help attract sodium, which then draw

water into the tissue to generate the swelling pressure of cartilage[184]. The breakdown of these structures coupled with an increase in inflammatory biomarkers has been linked to reduced contact stress coping ability and may lead to osteoarthritis[22, 108, 193, 230, 256].

Surgical Hip Preservation

Minor hip injuries are amenable to conservative measures, whereas suspected impingement or dysplasia in the presence of labrum and cartilage pathology necessitates surgical interventions. Conservative management should be included in the initial treatment for hip pathologies[92, 243], consisting of patient education on activity and positional modifications, anti-inflammatory medication (oral or intra-articular injection), abductor and core strengthening, and hip motion exercises intended to improve neuromuscular control, posture, and balance[20, 92, 126]. Rehabilitation should avoid painful positions including, but not limited to: straight leg raises, rotation of acetabulum on the femur while loaded, hyperextension, and anterior translation of the femur[126]. However, there is little evidence to support non-operative measures altering the natural history of progressive degenerative changes seen with FAI or dysplasia[126, 133]. Poor outcomes are typical in non-operative patients, particularly in the highly active[126, 133]. As little as 44% of patients are satisfied with non-operative measures and success may be highly dependent on permanent activity modification and/or presence of mild femoral and acetabular deformity[79, 126]. Red flags should be failure of conservative treatment, continued hip and groin pain, and positive provocative tests with physical examination, particularly the FADIR and FABER tests.

Surgical intervention is constantly evolving for the treatment of hip pathologies[160], however the goals are always to repair damaged tissues, restore normal joint congruency and motion, and minimize iatrogenic damage[168]. The underlying hope is that pain is removed, previous activity levels return, and sign of OA slow or stop completely. Hence the umbrella term for this area of specialization, hip preservation. Choice of a particular hip preservation surgery is dependent on many factors including, but not limited to: experiences of the surgeon, age and activity level of the patient, underlying morphologies, and personal preferences of the patient. Most patients should expect successful outcomes, regardless of surgery choice, but there is always risk of failure.

Repair of the torn labrum has the potential to restore labrum functions[42], and has superior short term results in both arthroscopy[161] and open procedures[81], versus debridement (removal). Therefore, preserving as much of the labrum as possible may be crucial to the long term health of the hip joint. Labrum reconstructions using the ligamentum teres[252], iliotibial band (ITB)[70, 224], and gracilis[192], have recently been described for cases in which the labrum is damaged beyond repair. The articular cartilage (or chondral) damage typically encountered in a symptomatic hip may be addressed with microfracture techniques[111, 225]. However, there are no long term studies yet to verify that labrum repair, reconstructions, and microfracture were stop advancement to OA. Repair or removal of damaged tissue and surgical success is most reliant upon correction of the underlying bony morphologies and return to normal mechanics of the joint. The focal bony abnormalities of the femur and acetabulum can be addressed with arthroscopy[20, 21] or open techniques[16]. Osteoplasties (shaving down and reshaping bone) at the head-neck junction and proximal femur can restore a normal offset and often are combined

with acetabular rim resections to reduce anterior over-coverage from focal retroversion. Surgical dislocation of the hip (SDH)[93] allows for full visualization of the joint, but comes with the increased risk of complications due to the complexity of the surgery. These risks are avascular necrosis, neurapraxia, non-union of bones, and heterotrophic ossifications[16]. The dysplastic acetabulum, or severely retroverted and/or deep, is often repaired with a Bernese periacetabular osteotomy (PAO)[94] or reverse PAO[251]. These re-orientate the acetabulum to a normal position and alter the weight bearing zone on the femur. These techniques are aimed at restoring normal hip mechanics and femoroacetabular coverage, as well as providing adequate clearance and stability of the joint during motion.

Surgical success may hinge on patient selections. Currently, those with intermittent activity related pain despite rehabilitation, and no signs of OA, were be most likely to have surgical success. The outcomes of surgery for intra-articular pathologies are positive via subjective and objective measures including: clinical/functional outcomes, radiographic improvements, and quality of life, activity level, and OA surveys.

Treatment for cam and pincer FAI is accomplished by arthroscopy or open procedures. Arthroscopy is often a preferred choice for patients because it is less invasive, has a shorter recovery time, and there are lower risks for complications[16]. Arthroscopy has good results at the 10 year mark[38-40] and SHD is similarly successful at the 5 year mark[204, 258]. Direct comparisons of the two are limited, but the results indicate that either can accomplish surgical goals adequately[24, 36, 293]. The only prospective matched-pair design study of the two indicated that, amongst two groups similar at baseline measures, arthroscopy results in better outcomes than SHD via patient reported surveys[75].

Additionally, evidence seems to indicate that adolescent[83], high school[83, 208], collegiate[41, 208], and elite athletes[41, 205, 208, 223, 226] can return to pre-surgery activity levels with either technique. Professionals may return and recover quicker, but patient survey scores and rates of return to sporting appear similar[181]. The 2-3 year short term results for labrum reconstruction, regardless of graft site, are promising for in both non-athletes[98, 279] and elite athletes[33], especially considering the worse conditions these patients are in compared with those whom the labrum is repairable. A continued follow up of patients is needed to evaluate the long term effectiveness for all these techniques.

Symptomatic hip impingement is common in the years following healed LCPD[215, 216, 264] and SCFE pinned in-situ[124], and surgical intervention primarily addresses the proximal femoral deformity. This can be accomplished via SHD[155, 203], arthroscopy, or intertrochanteric osteotomy (ITO)[155, 203, 242]. The results of SHD for LCPD demonstrate that a majority of patients can have significant improvement in pain and clinical signs and symptoms[215, 248]. Though recommended in the literature, reported patient outcomes for treatment of impingement following healed SCFE are limited to one short term study. Secondary to the heterogeneity of subjects, not many conclusions can be drawn from the results[233]. In LCPD the acetabular side deformities have recently garnered more attention and may warrant consideration for corrective surgeries like PAO[62]. Though not studied in LCPD and SCFE, traditional PAOs have the longest follow-up time times for hip preservation surgeries.

Acetabular dysplasia is typically treated by PAO. The Bernese PAO is the most utilized due to its advantages over other pelvic osteotomies[270] and has good results at the 20 year follow-up[259] since its original inception and publication, in 1983 and 1988 respectively[94]. The

ability to re-orientate the joint in theory should improve static and dynamic stability of the joint and therefore the improve contact and stress distribution as described by finite element analyses[52, 291]. Patient outcomes are also successful following PAO. Pain[58, 59, 61, 63, 275], function[58, 59, 61, 63, 275], and sporting ability[275] improve in the short term, and even in patients older than 40 years of age, with little signs of OA, PAO can be successful[200].

Surgical failure is not typical for hip preservation techniques like arthroscopy[29, 239] and open procedures[239] for FAI, or SHD for LCPD[248]. Failure to reduce pain or advancing signs of OA (substantial cartilage damage, joint space narrowing, and severe pain) may require conversion to total joint replacement[16]. The factors associated with greatest risk for failure of hip preservation are related to demographics, radiographic measures, and surgery related outcomes.

In a multicenter study, 2,386 hip preservation studies were reviewed and identified 359 failures, or about 15% of the total cases[60]. Need for hip preservation revision was related to being female (70% of cases) with an average age of 23. The pathologies of the 359 failures included 35% FAI, 20% dysplasia, 23% SCFE and 12% LCPD. Inadequately corrected structural disease was the primary need for revision, as PAO and arthroscopy for femoral osteochondroplasty were the two most common choices for repeat surgery.[60]

Arthroscopy failure is measured quantitatively via lack of statistical improvement in pain, function or patient satisfaction, and need for revision surgery. Older than 38 to 40 at initial surgery has been identified as a risk for failure in several studies[239]. It appears as though younger patients are much more successful and by the time a person is in their fourth decade

arthroscopy may not have good odds to succeed. This may be related to the amount preoperative cartilage loss and signs of OA. More than 2mm of cartilage loss is significantly related with conversion to arthroplasty. Need for repeat surgery, compared to total hip arthroplasty (THA), is used in younger patients with lower body mass index (BMI) and residual deformity[29]. The average patient of repeated hip preservation is 28 years of age, compared to 50 in THA, with a mean of 25 months between surgeries[29]. Bogunovic et al. reported 4.7% (n=60/1270) of patients needing revision hip preservation, with a breakdown of: 26 FAI, nine dysplasia, two laxity 1 other with no signs of structural deformity. The remaining 22 had residual signs of FAI and dysplasia, 16 and 6 respectively, but underwent elective THA.[29]

Failure of PAO often leads to OA, especially in the presence of continued femoral and acetabular structural deformities[5, 292]. The highest risk of OA is typically seen in someone older than 30 years of age, low pre-operative survey scores, and a non-spherical femoral head. The 10-year survivorship were decrease from 80-100%, down to 70-85% in the presence of femoral head deformity and double the risk for THA[5]. Additionally, resultant femoral head coverage is crucial as retroversion (over-coverage) or extreme anteversion and/or superior lateral under-coverage are significantly related to decreased survivorship.[5] Even when femoral head coverage is returned to normal, there is risk of FAI signs and symptoms, especially in males[292].

In conclusion, hip morphologies are the result of an elaborate combination of influences. The three known sources of hip morphology are traced to the shift for humans to use a bipedal upright gait, a multitude of genetic factors, and loading history of the bones. Often these morphologies are the catalyst for initiating pathological conditions that eventually lead to OA.

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APPENDIX A

WESTERN INSTITUTIONAL REVIEW BOARD APROVAL

INITIAL APPROVAL

FYLER

RENEWALS

INFORMED CONSENT DOCUMENTS

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Certificate of Approval

THE FOLLOWING WERE APPROVED

INVESTIGATOR: Robert Durkin M.D.
1319 Punahou Street
Honolulu, Hawaii 96826

BOARD ACTION DATE: 01/15/2013
PANEL: 5
STUDY APPROVAL EXPIRES: 01/15/2014
STUDY NUM: 1136805
WIRB PRO NUM: 20122141
INVEST NUM: 179843
WO NUM: 1-758235-1
CONTINUING REVIEW: Annually
SITE STATUS REPORTING: Annually

SPONSOR: University of Hawaii, Manoa
PROTOCOL NUM: None
AMD. PRO. NUM:
TITLE:
Analysis of Walking and Running Biomechanics in Femoroacetabular Impingement and Slipped Capital Femoral Epiphysis

APPROVAL INCLUDES:
Investigator
Protocol
Consent Form - Controls [IN0]
Consent Form [IN0]
Partial Waiver of Authorization for Recruitment

WIRB APPROVAL IS GRANTED SUBJECT TO:
The Board requires that all adult subjects and the parents or guardians of minor subjects be able to consent for themselves to be enrolled or enroll a minor in this study. This means that you cannot enroll incapable subjects who require enrollment by consent of a legally authorized representative or minor subjects whose parent or guardian are incapable of providing consent and who require consent by a legally authorized representative.

WIRB HAS APPROVED THE FOLLOWING LOCATIONS TO BE USED IN THE RESEARCH:
Kapiolani Medical Center for Women and Children, 1319 Punahou Street, Honolulu, Hawaii 96826
University of Hawaii, Manoa, PE/A Complex Room 231, 1337 Lower Campus Road, Honolulu, Hawaii 96822

If the PI has an obligation to use another IRB for any site listed above and has not submitted a written statement from the other IRB acknowledging WIRB's review of this research, please contact WIRB's Client Services department.

ALL WIRB APPROVED INVESTIGATORS MUST COMPLY WITH THE FOLLOWING:

1. Conduct the research in accordance with the protocol, applicable laws and regulations, and the principles of research ethics as set forth in the Belmont Report.

2. Although a participant is not obliged to give his or her reasons for withdrawing prematurely from the clinical trial, the investigator should make a reasonable effort to ascertain the reason, while fully respecting the participant's rights.

3. Unless consent has been waived, conduct the informed consent process without coercion or undue influence, and provide the potential subject sufficient opportunity to consider whether or not to participate. (Due to the unique circumstances of

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THE FOLLOWING WERE APPROVED

INVESTIGATOR: Robert Durkin MD
1319 Punahou Street
Honolulu, Hawaii 96826

BOARD ACTION DATE:	02/18/2014
PANEL:	5
STUDY APPROVAL EXPIRES:	01/15/2015
STUDY NUM:	1136805
WIRB PRO NUM:	20122141
INVEST NUM:	179843
WO NUM:	1-823978-1
CONTINUING REVIEW:	Annually
SITE STATUS REPORTING:	Annually

SPONSOR: University of Hawaii, Manoa
PROTOCOL NUM: None
AMD. PRO. NUM:
TITLE:
Analysis of Walking and Running Biomechanics in Femoroacetabular Impingement and Slipped Capital Femoral Epiphysis

APPROVAL INCLUDES:
Consent Form - Controls [IN0]
Consent Form [IN0]
Revised Research Locations (02-10-2014)

WIRB APPROVAL IS GRANTED SUBJECT TO:
Please have all future subjects sign the revised Consent Form(s) specified in this approval.

WIRB HAS APPROVED THE FOLLOWING LOCATIONS TO BE USED IN THE RESEARCH:
Kapiolani Medical Center for Women and Children, 1319 Punahou Street, Honolulu, Hawaii 96826
University of Hawaii, Manoa, PE/A Complex Room 231, 1337 Lower Campus Road, Honolulu, Hawaii 96822
Straub Clinic & Hospital, 888 S. King Street, Honolulu, Hawaii 96813

If the PI has an obligation to use another IRB for any site listed above and has not submitted a written statement from the other IRB acknowledging WIRB's review of this research, please contact WIRB's Client Services department.

ALL WIRB APPROVED INVESTIGATORS MUST COMPLY WITH THE FOLLOWING:

1. Conduct the research in accordance with the protocol, applicable laws and regulations, and the principles of research ethics as set forth in the Belmont Report.

2. Although a participant is not obliged to give his or her reasons for withdrawing prematurely from the clinical trial, the investigator should make a reasonable effort to ascertain the reason, while fully respecting the participant's rights.

Western Institutional Review Board®
1019 39th Avenue SE Suite 120 | Puyallup, WA 98374-2115
Office: (360) 252-2500 | Toll Free: (800) 562-4789
www.wirb.com

Certificate of Approval

THE FOLLOWING WERE APPROVED

INVESTIGATOR: Robert Durkin MD
1319 Punahou Street
Honolulu, Hawaii 96826

BOARD ACTION DATE:	11/11/2014
PANEL:	5
STUDY APPROVAL EXPIRES:	01/15/2015
STUDY NUM:	1136805
WIRB PRO NUM:	20122141
INVEST NUM:	179843
WO NUM:	1-860761-1
CONTINUING REVIEW:	Annually
SITE STATUS REPORTING:	Annually

SPONSOR: University of Hawaii, Manoa
PROTOCOL NUM: None
AMD. PRO. NUM:
TITLE:
Analysis of Walking and Running Biomechanics in Femoroacetabular Impingement and Slipped Capital Femoral Epiphysis

APPROVAL INCLUDES:
Advertisement - Flyer - Having a Hip Arthroscopy This #12544920.0 - As Submitted

WIRB APPROVAL IS GRANTED SUBJECT TO:

WIRB HAS APPROVED THE FOLLOWING LOCATIONS TO BE USED IN THE RESEARCH:
Kapiolani Medical Center for Women and Children, 1319 Punahou Street, Honolulu, Hawaii 96826
University of Hawaii, Manoa, PE/A Complex Room 231, 1337 Lower Campus Road, Honolulu, Hawaii 96822
Straub Clinic & Hospital, 888 S. King Street, Honolulu, Hawaii 96813

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2. Although a participant is not obliged to give his or her reasons for withdrawing prematurely from the clinical trial, the investigator should make a reasonable effort to ascertain the reason, while fully respecting the participant's rights.

RESEARCH SUBJECT INFORMATION AND CONSENT FORM
Controls

TITLE:	Analysis of Walking and Running Biomechanics in Femoroacetabular Impingement and Slipped Capital Femoral Epiphysis
PROTOCOL NO.:	None WIRB® Protocol #20122141
SPONSOR:	University of Hawaii, Manoa
INVESTIGATOR:	Robert Durkin, MD 1319 Punahou Street Honolulu, Hawaii 96826 United States
SITE(S):	Kapiolani Medical Center for Women and Children 1319 Punahou Street Honolulu, Hawaii 96826 United States University of Hawaii, Manoa PE/A Complex Room 231 1337 Lower Campus Road Honolulu, Hawaii 96822 United States Straub Clinic & Hospital 888 S. King Street Honolulu, Hawaii 96813 United States
STUDY-RELATED PHONE NUMBER(S):	Robert Durkin M.D. (808) 983-6000 (24 hour) (808) 945-3766 (office) (808) 942-9837 (fax) Bret Freemyer, MS, ATC (818) 209-7222 (mobile)

In this consent form, "you" always refers to the subject. If you are a parent or guardian, please remember that "you" refers to the study subject.

This consent form may contain words that you do not understand. Please ask the study doctor or the study staff to explain any words or information that you do not clearly understand. You may take home an unsigned copy of this consent form to think about or discuss with family or friends before making your decision.

SUMMARY

You are being asked to be in a research study. The purpose of this consent form is to help you decide if you want to be in the research study. Please read this consent form carefully. To be in a research study you must give your informed consent. "Informed consent" includes:

- Reading this consent form
- Having the study doctor or study staff explain the research study to you
- Asking questions about anything that is not clear, and
- Taking home an unsigned copy of this consent form. This gives you time to think about it and to talk to family or friends before you make your decision.

You should not join this research study until all of your questions are answered. Things to know before deciding to take part in a research study:

- The main goal of a research study is to learn things to help patients in the future.
- The main goal of regular medical care is to help each patient.
- No one can promise that a research study will help you.
- Taking part in a research study is entirely voluntary. No one can make you take part.
- If you decide to take part, you can change your mind later on and withdraw from the research study.
- The decision to join or not join the research study will not cause you to lose any medical benefits. If you decide not to take part in this study, your doctor will continue to treat you.
- Parts of this study may involve standard medical care. Standard care is the treatment normally given for a certain condition or illness.
- After reading the consent form and having a discussion with the research staff, you should know which parts of the study are experimental and which are standard medical care.
- Your medical records may become part of the research record. If that happens, your medical records may be looked at and/or copied by the sponsor of this study and government agencies or other groups associated with the study.

After reading and discussing the information in this consent form you should know:

- Why this research study is being done;
- What will happen during the research;
- Any possible benefits to you;
- The possible risks to you;
- How problems will be treated during the study and after the study is over.

If you take part in this research study, you will be given a copy of this signed and dated consent form.

PURPOSE OF THE STUDY

There will be approximately 180 subjects in this study. The purpose of this research study is to examine how you walk and run for comparison with people who have had treatment for femoroacetabular impingement (FAI) or slipped capital femoral epiphysis (SCFE). This information will be used to help better understand FAI and SCFE.

PROCEDURES

If you decide to take part in this study you will be part of the control group of individuals with normal walking abilities who are similar in age, height, and weight to individuals who have received treatment for FAI and SCFE.

Each data collection session will take approximately 45 minutes. At *each* data collection session you will be asked to:

1. Complete two questionnaires about your hip and your state of mind. These questionnaires include: the UCLA activity score and Hip Outcome Score (HOS)
2. Push as hard as you can into a non-moving strength measuring device in 8 different leg motions:
hip flexion, extension, abduction, adduction, internal rotation, external rotation, knee flexion, and extension. This will be done on both legs.
3. Walk 6 meters (about 20 feet) 6 to 10 times at a self-selected (natural) walking speed.

At some of the data collection sessions you will be asked to run 6 meters (20 feet) 6 to 10 times at a selfselected running speed.

Figure 1. Data Collection Schedule for Control Group

Group		Baseline	3 months	6 months	1 year	2 year
Control	Walking	X	X	X	X	X
	Running	X		X	X	X

RISKS AND DISCOMFORTS

Due to the level of physical activity involved, there is a risk of injury. You may also have some discomfort, muscle cramping or soreness during or after test sessions. Although we have a fall prevention system, there is a chance of falling during the gait trials. There is a very remote chance of cardiac arrest and/or death. These risks are comparable to your activities of daily living.

You cannot participate in this study if you are pregnant because the walking and running biomechanics collected may not accurately represent your normal characteristics. If you are unaware that you are pregnant, participation in this study will result in no more danger to the mother or fetus than normal activities of daily living. However, if you become pregnant or think you might be pregnant during the course of this study, you must inform the researchers, and you will be taken out of the study.

NEW INFORMATION

You will be told about anything new that might change your decision to be in this study. You may be asked to sign a revised consent form if this occurs.

BENEFITS

You will not receive direct/immediate benefits from participating in this study. However, you will obtain information regarding your walking and running gait, functional activity capacity, hip and knee muscular strength, and behavioral characteristics. Results of this study may assist physicians and health care providers to ensure the best clinical outcomes following hip surgery for FAI and SCFE.

PAYMENT FOR PARTICIPATION

You will receive $5 for each data collection session. This money can be applied to your parking and transportation to and from the University of Hawaii Gait Laboratory. You will be paid only for the visits you have completed.

COSTS

You will be responsible for parking and transportation to and from the University of Hawaii, Manoa, Kinesiology and Rehabilitation Science, Human Performance and Gait Laboratory (Sherriff 100). You will be given $5 per data collection session that can be applied toward the parking fee or transportation; however, the money will be given after you arrive at the facility, so it is a reimbursement. The fee for parking at the University of Hawaii, Manoa parking structure is $5 during the week and on weekends. Any other cost associated with parking/transportation over and above the $5 provided will be your responsibility.

You might have unexpected expenses from being in this study. Ask your study doctor to discuss the costs that will or will not be covered by the sponsor. This discussion should include who will pay the costs of treating possible side effects.

ALTERNATIVE TREATMENT

This is not a treatment study. Your alternative is not to participate in this study. Your follow-up care is the same whether or not you are in this study.

USE AND DISCLOSURE OF YOUR HEALTH INFORMATION:

By signing this form you are authorizing the use and disclosure of individually identifiable information. Your information will only be used/disclosed as described in this consent form and as permitted by state and federal laws. If you refuse to give permission, you will not be able to be in this research.

This consent covers all information about you that is used or collected for this study. It includes

- Data about your walking and running

Your authorization to use your identifiable health information will not expire even if you terminate your participation in this study or you are removed from this study by the study doctor. However, you may revoke your authorization to use your identifiable information at any time by submitting a written notification to the principal investigator, Dr. Robert Durkin, 1319 Punahou Street, Suite 630, Honolulu, HI 96826. If you

decide to revoke (withdraw or "take back") your authorization, your identifiable health information collected or created for this study shall not be used or disclosed by the study doctor after the date of receipt of the written revocation except to the extent that the law allows us to continue using your information. The investigators in this study are not required to destroy or retrieve any of your health information that was created used or disclosed for this study prior to receiving your written revocation.
By signing this consent form you authorize the following parties to use and or disclose your identifiable health information collected or created for this study:

- Robert Durkin, MD and his research staff for the purposes of conducting this research study.
- Kapiʻolani Medical Center for Women and Children, Straub Clinic & Hospital, and Hawaiʻi Pacific Health
- The University of Hawaiʻi, at Manoa

The individuals named above may disclose your medical records, this consent form and the information about you created by this study to:

- The sponsor of this study and their designees
- Federal, state and local agencies having oversight over this research, such as the Office for Human Research Protections in the U.S. Department of Health and Human Services, Food and Drug Administration, the National Institutes of Health, etc.
- Hawaii Pacific Health (HPH) Officials, the Western Institutional Review Board, and the HPH Office of Compliance for purposes of overseeing the research study and making sure that your ethical rights are being protected.
- The University of Hawaiʻi, at Manoa

Some of the persons or groups that receive your study information may not be required to comply with federal privacy regulations, and your information may lose its federal privacy protection and your information may be disclosed without your permission.

COMPENSATION IN CASE OF INJURY:

No financial compensation or coverage will be routinely provided by the sponsor or study doctor. If you require treatment for any injury or illness related to procedures required by the study, or if you suffer side effects while in the study, you should contact your study doctor, Robert Durkin, MD at 808-983-6000 (24 hours), who will give you the necessary medical care and advice. The cost of this medical care and advice will be billed to you or your medical insurance in the usual manner.

By signing this consent form, you will not give up any legal rights.

VOLUNTARY PARTICIPATION AND WITHDRAWAL

Your participation in this study is voluntary. You may decide not to participate or you may leave the study at any time. Your decision will not result in any penalty or loss of benefits to which you are entitled.

Your participation in this study may be stopped at any time by the study doctor or the sponsor without your consent for any of the following reasons:

- it is in your best interest;
- you do not consent to continue in the study after being told of changes in the research that may affect you;
- or for any other reason.

If you leave the study before the planned final visit, you may be asked by the study doctor to have some of the end of study procedures done.

SOURCE OF FUNDING FOR THE STUDY

This research study is being funded by the University of Hawaii, Manoa.

QUESTIONS

Contact Dr. Robert Durkin at (808) 983-6000 (24 hours) for any of the following reasons:

- if you have any questions about this study or your part in it
- if you feel you have had a research-related injury or
- if you have questions, concerns or complaints about the research

If you have questions about your rights as a research subject or if you have questions, concerns or complaints about the research, you may contact:

Western Institutional Review Board® (WIRB®)
1019 39th Avenue SE Suite 120
Puyallup, Washington 98374-2115
Telephone: 1-800-562-4789 or 360-252-2500
E-mail: Help@wirb.com

WIRB is a group of people who perform independent review of research.

WIRB will not be able to answer some study-specific questions, such as questions about appointment times. However, you may contact WIRB if the research staff cannot be reached or if you wish to talk to someone other than the research staff.

Do not sign this consent form unless you have had a chance to ask questions and have gotten satisfactory answers. If you agree to be in this study, you will receive a signed and dated copy of this consent form for your records.

CONSENT

I have read this consent form. All my questions about the study and my part in it have been answered. I freely consent to be in this research study.

I authorize the use and disclosure of my health information to the parties listed in the authorization section of this consent for the purposes described above.

By signing this consent form, I have not given up any of my legal rights.

_____
Subject Name (printed)

Consent and Assent Instructions:
Consent: Subjects 18 years and older must sign on the subject line below

For subjects under 18, consent is provided by the parent or guardian

Assent:
Verbal assent is required for subjects ages 10 through 17 years using the Assent section below.

_____
Subject Name (printed)

CONSENT SIGNATURE:

_____ _____
Signature of Subject (18 years and older) Date

_____ _____
Signature of
Parent or Guardian (when applicable) Date

_____ _____
Signature of Person Conducting Informed Consent Discussion Date

ASSENT SECTION For Subjects Ages 10 - 17:

Statement of person conducting assent discussion:

1. I have explained all aspects of the research to the subject to the best of his or her ability to understand.
2. I have answered all the questions of the subject relating to this research.
3. The subject agrees to be in the research.
4. I believe the subject's decision to enroll is voluntary.
5. The study doctor and study staff agree to respect the subject's physical or emotional dissent at any time during this research when that dissent pertains to anything being done solely for the purpose of this research.

_____ _____

Signature of Person Conducting Assent Discussion — Date

Statement of Parent or Guardian:

My child appears to understand the research to the best of his or her ability and has agreed to participate.

_____ _____

Signature of Parent or Guardian — Date

RESEARCH SUBJECT INFORMATION AND CONSENT FORM

TITLE:	Analysis of Walking and Running Biomechanics in Femoroacetabular Impingement and Slipped Capital Femoral Epiphysis
PROTOCOL NO.:	None WIRB® Protocol #20122141
SPONSOR:	University of Hawaii, Manoa
INVESTIGATOR:	Robert Durkin, MD 1319 Punahou Street Honolulu, Hawaii 96826 United States
SITE(S):	Kapiolani Medical Center for Women and Children 1319 Punahou Street Honolulu, Hawaii 96826 United States University of Hawaii, Manoa PE/A Complex Room 231 1337 Lower Campus Road Honolulu, Hawaii 96822 United States Straub Clinic & Hospital 888 S. King Street Honolulu, Hawaii 96813 United States
STUDY-RELATED PHONE NUMBER(S):	Robert Durkin M.D. (808) 983-6000 (24 hours) (808) 945-3766 (office) (808) 942-9837 (fax) Bret Freemyer, MS, ATC (818) 209-7222 (mobile)

In this consent form, "you" always refers to the subject. If you are a parent or guardian, please remember that "you" refers to the study subject.

This consent form may contain words that you do not understand. Please ask the study doctor or the study staff to explain any words or information that you do not clearly understand. You may take home an unsigned copy of this consent form to think about or discuss with family or friends before making your decision.

SUMMARY

You are being asked to be in a research study. The purpose of this consent form is to help you decide if you want to be in the research study. Please read this consent form carefully. To be in a research study you must give your informed consent. "Informed consent" includes:

- Reading this consent form
- Having the study doctor or study staff explain the research study to you
- Asking questions about anything that is not clear, and
- Taking home an unsigned copy of this consent form. This gives you time to think about it and to talk to family or friends before you make your decision.

You should not join this research study until all of your questions are answered. Things to know before deciding to take part in a research study:

- The main goal of a research study is to learn things to help patients in the future.
- The main goal of regular medical care is to help each patient.
- No one can promise that a research study will help you.
- Taking part in a research study is entirely voluntary. No one can make you take part.
- If you decide to take part, you can change your mind later on and withdraw from the research study.
- The decision to join or not join the research study will not cause you to lose any medical benefits. If you decide not to take part in this study, your doctor will continue to treat you.
- Parts of this study may involve standard medical care. Standard care is the treatment normally given for a certain condition or illness.
- After reading the consent form and having a discussion with the research staff, you should know which parts of the study are experimental and which are standard medical care.
- Your medical records may become part of the research record. If that happens, your medical records may be looked at and/or copied by the sponsor of this study and government agencies or other groups associated with the study.

After reading and discussing the information in this consent form you should know:

- Why this research study is being done;
- What will happen during the research;
- Any possible benefits to you;
- The possible risks to you;
- How problems will be treated during the study and after the study is over.

If you take part in this research study, you will be given a copy of this signed and dated consent form.

PURPOSE OF THE STUDY

There will be approximately 180 subjects in this study. The purpose of this research study is to examine how you walk and run after your treatment for femoroacetabular impingement (FAI) or slipped capital femoral epiphysis (SCFE).

PROCEDURES

If you decide to take part in this study you fall into *one* of these three groups:

A) If you had FAI or SCFE surgery more than a year ago, you will be asked to complete a single data collection session.

B) If you will be having FAI surgery in the near future you will be asked to complete five data collection sessions over the next two years at the following times: one - two weeks before surgery, three months, six months and 1 year and two years after surgery (see figure 1).

C) If you recently had surgery for SCFE (in the last month) you will be asked to complete four data collection sessions over the next two years at the following times: three months, six months, one year and two years after surgery (see figure 1).

Each data collection session will take approximately 45 minutes. At *each* data collection session you will be asked to:

1. Complete two questionnaires about your hip and your state of mind. These questionnaires include: the UCLA activity score and Hip Outcome Score (HOS)

2. Push as hard as you can into a non-moving strength measuring device in 8 different leg motions: hip flexion, extension, abduction, adduction, internal rotation, external rotation, knee flexion, and extension. This will be done on both legs.
3. Walk 6 meters (about 20 feet) 6 to 10 times at a self-selected (natural) walking speed.

At some of the data collection sessions you will be asked to run 6 meters (20 feet) 6 to 10 times at a selfselected running speed.

1. Group A will be asked to run at their only data collection session.
2. Group B (FAI in the future) will begin running at 6 months after surgery.
3. Group C (recent SCFE) will begin running at one year after surgery.

Figure 1. Data Collection Schedule for Groups B and C

Group		Pre-surgery	3 months	6 months	1 year	2 year
B-(FAI)	Walking	X	X	X	X	X
	Running	X		X	X	X
C-(SCFE)	Walking		X	X	X	X
	Running				X	X

Information for all of the groups will also be collected from your medical records and stored on the secured database at Kapi'olani Medical Center for Women and Children. The following items will be reviewed and entered into a data collection spreadsheet:

1. History of hip surgery and other leg surgeries
2. Age, height, weight, and body mass index at the date of hip surgery
3. Pre and post-operative diagnosis
4. History of clinical data (study doctor's physical examination findings)
5. Radiographic (x-ray) measurements of your hip
6. Surgery component characteristics
7. Surgical complications

RISKS AND DISCOMFORTS

Due to the level of physical activity involved, there is a risk of injury. You may have pain in your involved hip during testing. You may also have some discomfort, muscle cramping or soreness during or after test sessions. Although we have a fall prevention system, there is a chance of falling during the gait trials. There is a very remote chance of cardiac arrest and/or death. These risks are comparable to your routine rehabilitation and activities of daily living, and will not affect your recovery from the surgery.

You cannot participate in this study if you are pregnant because the walking and running biomechanics collected may not accurately represent your normal characteristics. If you are unaware that you are pregnant, participation in this study will result in no more danger to the mother or fetus than normal activities of daily living. However, if you become pregnant or think you might be pregnant during the course of this study, you must inform the researchers, and you will be taken out of the study.

NEW INFORMATION

You will be told about anything new that might change your decision to be in this study. You may be asked to sign a revised consent form if this occurs.

BENEFITS

You will not receive direct/immediate benefits from participating in this study. However, you will obtain information regarding your walking and running gait, functional activity capacity, hip and knee muscular strength, and behavioral characteristics. Results of this study may assist physicians and health care providers to ensure the best clinical outcomes following hip surgery for FAI and SCFE.

PAYMENT FOR PARTICIPATION

You will receive $5 for each data collection session. This money can be applied to your parking and transportation to and from the University of Hawaii Gait Laboratory. You will be paid only for the visits you have completed.

COSTS

You will be responsible for parking and transportation to and from the University of Hawaii, Manoa, Kinesiology and Rehabilitation Science, Human Performance and Gait Laboratory (Sherriff 100). You will be given $5 per data collection session that can be applied toward the parking fee or transportation; however, the money will be given after you arrive at the facility, so it is a reimbursement. The fee for parking at the University of Hawaii, Manoa parking structure is $5 during the week and on weekends. Any other cost associated with parking/transportation over and above the $5 provided will be your responsibility.

You might have unexpected expenses from being in this study. Ask your study doctor to discuss the costs that will or will not be covered by the sponsor. This discussion should include who will pay the costs of treating possible side effects.

ALTERNATIVE TREATMENT

This is not a treatment study. Your alternative is not to participate in this study. Your follow-up care is the same whether or not you are in this study.

USE AND DISCLOSURE OF YOUR HEALTH INFORMATION:

By signing this form you are authorizing the use and disclosure of individually identifiable information. Your information will only be used/disclosed as described in this consent form and as permitted by state and federal laws. If you refuse to give permission, you will not be able to be in this research.

This consent covers all information about you that is used or collected for this study. It includes

- Data from your medical record as listed in the procedures section
- Data about your walking and running

Your authorization to use your identifiable health information will not expire even if you terminate your participation in this study or you are removed from this study by the study doctor. However, you may revoke your authorization to use your identifiable information at any time by submitting a written notification to the principal investigator, Dr. Robert Durkin, 1319 Punahou Street, Suite 630, Honolulu, HI 96826. If you decide to revoke (withdraw or "take back") your authorization, your identifiable health information collected or created for this study shall not be used or disclosed by the study doctor after the date of receipt of the written revocation except to the extent that the law allows us to continue using your information. The investigators in this study are not required to destroy or retrieve any of your health information that was created used or disclosed for this study prior to receiving your written revocation.

By signing this consent form you authorize the following parties to use and or disclose your identifiable health information collected or created for this study:

- Robert Durkin, MD and his research staff for the purposes of conducting this research study.
- Kapiʻolani Medical Center for Women and Children, Straub Clinic & Hospital, and Hawaiʻi Pacific Health
- The University of Hawaiʻi, at Manoa

Your medical records may contain information about AIDS or HIV infection, venereal disease, treatment for alcohol and/or drug abuse, or mental health or psychiatric services. By signing this consent form, you authorize access to this information if it is in the records used by members of the research team.

The individuals named above may disclose your medical records, this consent form and the information about you created by this study to:

- The sponsor of this study and their designees
- Federal, state and local agencies having oversight over this research, such as the Office for Human Research Protections in the U.S. Department of Health and Human Services, Food and Drug Administration, the National Institutes of Health, etc.
- Hawaii Pacific Health (HPH) Officials, the Western Institutional Review Board, and the HPH Office of Compliance for purposes of overseeing the research study and making sure that your ethical rights are being protected.
- The University of Hawaiʻi, at Manoa

Some of the persons or groups that receive your study information may not be required to comply with federal privacy regulations, and your information may lose its federal privacy protection and your information may be disclosed without your permission.

COMPENSATION IN CASE OF INJURY:

No financial compensation or coverage will be routinely provided by the sponsor or study doctor. If you require treatment for any injury or illness related to procedures required by the study, or if you suffer side effects while in the study, you should contact your study doctor, Robert Durkin, MD at 808-983-6000 (24 hours), who will give you the necessary medical care and advice. The cost of this medical care and advice will be billed to you or your medical insurance in the usual manner.

By signing this consent form, you will not give up any legal rights.

VOLUNTARY PARTICIPATION AND WITHDRAWAL

Your participation in this study is voluntary. You may decide not to participate or you may leave the study at any time. Your decision will not result in any penalty or loss of benefits to which you are entitled.

Your participation in this study may be stopped at any time by the study doctor or the sponsor without your consent for any of the following reasons:

- it is in your best interest;
- you do not consent to continue in the study after being told of changes in the research that may affect you;
- or for any other reason.

If you leave the study before the planned final visit, you may be asked by the study doctor to have some of the end of study procedures done.

SOURCE OF FUNDING FOR THE STUDY

This research study is being funded by the University of Hawaii, Manoa.

QUESTIONS

Contact Dr. Robert Durkin at (808) 983-6000 (24 hours) for any of the following reasons:

- if you have any questions about this study or your part in it
- if you feel you have had a research-related injury or
- if you have questions, concerns or complaints about the research

If you have questions about your rights as a research subject or if you have questions, concerns or complaints about the research, you may contact:

Western Institutional Review Board® (WIRB®)
1019 39th Avenue SE Suite 120
Puyallup, Washington 98374-2115
Telephone: 1-800-562-4789 or 360-252-2500
E-mail: Help@wirb.com

WIRB is a group of people who perform independent review of research.

WIRB will not be able to answer some study-specific questions, such as questions about appointment times. However, you may contact WIRB if the research staff cannot be reached or if you wish to talk to someone other than the research staff.

Do not sign this consent form unless you have had a chance to ask questions and have gotten satisfactory answers. If you agree to be in this study, you will receive a signed and dated copy of this consent form for your records.

CONSENT

I have read this consent form. All my questions about the study and my part in it have been answered. I freely consent to be in this research study.

I authorize the use and disclosure of my health information to the parties listed in the authorization section of this consent for the purposes described above.

By signing this consent form, I have not given up any of my legal rights.

_____
Subject Name (printed)

Consent and Assent Instructions:
Consent: Subjects 18 years and older must sign on the subject line below
For subjects under 18, consent is provided by the parent or guardian
Assent: Verbal assent is required for subjects ages 10 through 17 years using the Assent section below.

_____

Subject Name (printed)

CONSENT SIGNATURE:

_____ _____

Signature of Subject (18 years and older) Date

_____ _____

Signature of Date
Parent or Guardian (when applicable)

_____ _____

Signature of Person Conducting Informed Consent Discussion Date

ASSENT SECTION For Subjects Ages 10 - 17:

Statement of person conducting assent discussion:

1. I have explained all aspects of the research to the subject to the best of his or her ability to understand.
2. I have answered all the questions of the subject relating to this research.
3. The subject agrees to be in the research.
4. I believe the subject's decision to enroll is voluntary.
5. The study doctor and study staff agree to respect the subject's physical or emotional dissent at any time during this research when that dissent pertains to anything being done solely for the purpose of this research.

_____ _____

Signature of Person Conducting Date
Assent Discussion

Statement of Parent or Guardian:

My child appears to understand the research to the best of his or her ability and has agreed to participate.

_____ _____

Signature of Parent or Guardian Date

APPENDIX B

PATIENT RELATED OUTCOME SURVEYS

THE HIP OUTCOME SCORE

&

THE UNIVERISTY OF CALIFORNIA, LOS ANGELES ACTIVITY SCORE

Hip Outcome Score

Please answer every question with ***one response*** that most closely describes your condition within the *past week*.
If the activity in question is limited by something other than your hip, mark not applicable (N/A).

ADL Subscale

	No Difficulty at All	Slight Difficulty	Moderate Difficulty	Extreme Difficulty	Unable to Do	N/A
Standing for 15 min						
Getting into and out of an average car						
Putting on socks and shoes						
Walking up steep hills						
Walking down steep hills						
Going up 1 flight of stairs						
Going down 1 flight of stairs						
Stepping up and down curbs						
Deep squatting						
Getting into and out of a bathtub						
Sitting for 15 min						
Walking initially						
Walking for approximately 10 min						
Walking for 15 min or more						
Because of your hip, how much difficulty do you have with the following:						
Twisting/pivoting on involved leg						
Rolling over in bed						
Light to moderate work (standing. walking)						
Heavy work (pushing/pulling. climbing. carrying)						
Recreational activities						

How would you rate your current level of function during your usual ADL from 0 to 100, with 100 being your level of function before your hip problemand 0 being the inability to perform any of your usual daily activities?

_____%

Sports Subscale

Because of your hip, how much difficulty do you have with the following:	No Difficulty at All	Slight Difficulty	Moderate Difficulty	Extreme Difficulty	Unable to Do	N/A
Running 1 mile						
Jumping						
Swinging objects like a golf club						
Landing						
Starting and stopping quickly						
Cutting/lateral movements						
Low-impact activities like fast walking						
Ability to perform activity with your normal technique						
Ability to participate in your desired sport as long as you would like						

How would you rate your current level of function during your sports-related activities from 0 to 100. with 100 being your level of function before your hip problem and 0 being the inability to perform any of your usual daily activities?

_____%

How would you rate your current level of function?

□ Normal □ Nearly Normal □ Abnormal □ Severely Abnormal

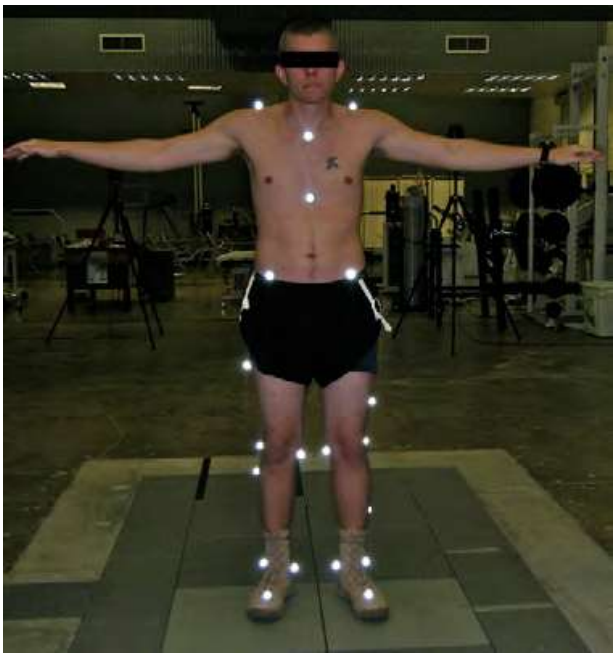
UCLA Activity Score	
Please circle the number which best describes your activity level over the last 6 months. Circle only one response.	
Regularly is once a week or more. Sometimes is once a month or less.	
10	I regularly participate in **impact activities** such as jogging, tennis, skiing, acrobatics, ballet, heavy labor, or backpacking
9	I sometimes participate in **impact activities** such as jogging, tennis, skiing, acrobatics, ballet, heavy labor, or backpacking
8	I regularly participate in **active activities** such as fast walking, golf, or bowling
7	I sometimes participate in **active activities** such as fast walking, golf, or bowling
6	I regularly participate in **moderate activities** such as moderate walking or heavy
5	I sometimes participate in **moderate activities** such as moderate walking or heavy
4	I regularly participate in **mild activities** such as slow walking, limited housework
3	I sometimes participate in **mild activities** such as slow walking, limited
2	I am mostly inactive and restricted to minimal activities of daily living
1	I am wholly inactive dependent on others, cannot leave residence

APPENDIX C

ANATOMICAL LANDMARKS AND PLACEMENT OF

LOWER LEG AND THORAX RETRORELECTIVE MARKER SET

FOR GAIT ANALYSIS



Markers were placed bilaterally at the calcaneus, lateral and medial malleoli, and second metarsal-phalangeal joints at the foot. Moving proximally, they were placed at the lateral shank (left lower, right higher) and medial and lateral epicondyles of the knee (10mm above joint line) and the mid-thigh (left lower, right higher). The pelvis markers were placed at both anterior and posterior superior iliac spines. The thorax was comprised of two at acromioclavicular joint, C7, T10, superior notch of the sternum, and finally the xiphoid process.

APPENDIX D
RAW DATA

Raw Data - Anthropometrics and Age.

Subject ID#	Group	Height (mm)	Weight (kg)	BMI	Height (mm)-3	Weight (kg)-3	BMI-3	Height (mm)-6	Weight (kg)-6	BMI-6	Height (mm)-12	Weight (kg)-12	BMI-12	Age	Gender (F=0, M=1)	Operated Leg (L=0, R=1)	Dominant Leg (L=0, R=1)	Inter ASIS (mm)
CF-011-0	0	1664	57.9	20.9	1654	57.9	21.2							41	0	*	1	290
CF-012-0	0	1918	91.1	24.8	1950	86.2	22.7							27	1	*	1	271
CF-013-0	0	1701	60.9	21.0	1693	64	22.3	1707	62.6	21.48361154				29	0	*	1	243
CF-014-0	0	1616	54.2	20.8	1615	55.9	21.4							28	0	*	1	297
CF-016-0	0	1754	64.4	20.9	1730	66.8	22.3	1743	65.8	21.65863684				34	0	*	1	248
CF-018-0	0	1681	60.4	21.4	1671	59.1	21.2	1683	60	21.18278306				38	0	*	1	260
CF-019-0	0	1786	73.5	23.0	1800	73.8	22.8	1800	73	22.53086642				34	1	*	1	325
CF-020-0	0	1724	60.7	20.4										30	0	1	1	301
CF-021-0	0	1645	62.1	22.9										20	0	*	1	234
CF-022-0	0	1873	99.2	28.3										36	1	1	1	285
F-001-0	1	1815	92.1	28.0	1813	92.5	28.1	1813	96.6	29.4	1818	88.1	26.7	38	1	1	1	300
F-003-0	1	1790	76.4	23.8										29	0	1	1	302
F-005-0	1	1693	62.3	21.7	1684.5	61.4	21.6	1692	61.8	21.6	1683	62.1	21.9	44	0	0	1	261
F-007-0	1	1680	80.7	28.6	1676	81.8	29.1	1699	82.1	28.4	1680	77.7	27.5	20	0	0	1	290
F-008-0	1	1701	51.7	17.9	1702	51.4	17.7	1695	52.2	18.2				34	0	1	1	267
F-009-0	1	1853	97.6	28.4	1857	102.5	29.7	1849	102.3	29.9				32	0	1	1	309
F-010-0	1	1647	73.8	27.2	1639	74.6	27.8	1634	77.3	29.0				34	0	0	1	345
F-011-0	1	1830	68.7	20.5	1810	72.1	22.0							22	0	1	1	296
F-012-0	1	1670	78.9	28.3										39	0	1	1	323
F-013-0	1	1685	67.5	23.8	1678	65.3	23.2							31	0	1	1	320
F-014-0	1	1600	59.4	23.2	1611	62.1	23.9							20	0	1	1	290
F-015-0	1	1637	68.1	25.4										28	0	1	1	271

Raw Data of Clinical and Radiographic (cam, pincer subtypes)

subject id	age at surgery		involved side r=1		FAI (cam=1,pi	duration of s/s	FADIR test	FABER 1=postive	SLR	Inter ASIS (mm)	AP standing?
1	37.8		1		3	3	1	0	1	305.70	1
3	29.8		1		2	3	1	0	0	348.20	1
5	44.7		2		1	24	1	1	0	268.35	1
7	20.6		2		3	13	1	1	0	304.03	1
8	34.9		1		2	6	1	1	1	295.54	1
9	32.7		1		2	12	1	0	0	371.00	0
10	34.5		2		1	72	1	0	1	339.53	1
11	22.2		1		1	24	1	0	0	312.00	1
12	39.2		1		3	6	1	0	1	236.20	0
13	31.0		1		2	12	1	0	0	278.40	0
14	20.8		1		2	60	1	0	1	285.00	0
15	28.9		1		2	12	1	0	0	297.20	0
	31.4	R	9	cam	3	20.6				303.4	
	20.6	L	3	pincer	6	22.44775					
	44.7			combo	3	3					
	7.530593										
						72					
						12					

subject id	ap AA inv-0	ap AA uni-0	ap AI-inv-0	frog AA IN-0	Frog AA UNI-0	ap AA inv-1	ap AA uni-1	frog AA IN-1	Frog AA UNI-1
f-005	34	33	5	50	49	35	31	41	47
f-010	77	77	5	56	54	31	32	45	42
F-011	46	50	12	42	43	49	44	43	*
f-001	78	82	9	87	77	77	82	48	55
f-007	42	80	-1	50	59	36	75	34	61
F-012			10	58		37		40	

subject id	FAI (cam=1,pincer=2, combo=3,	COS (1=involved, 0= no, 2=uninvolved,	pws	iss	ap lcea involved-0	ap AI-inv-0	lcea involved-1	ap AI-inv-1
f-003	2	1	1	1	35	-7		
f-008	2	0	0	2	31	3	33	2
f-009	2	3	0	3	33	3	25	11
F-013	2	0	0	0	48	-3	43	-3
F-014	2	0	0	1	36	3	37	1
F-015	2	3	3	3	33	4	33	6
f-001	3	1	1	0	29	9	24	7
f-007	3	3	0	3	41	-1	31	1
F-012	3	1	0	0	33	10	31	8
		7	3	5				

Raw Data for Hand Held Dynamometry

Subject ID#	Operated Leg (L=0, R=1)	Dominant Leg (L=0, R=1)	Group	HE-U	KF-U	HAB-U	HAD-U	HF-U	HIR_U	HER_U	KE_U	HE_I	KF_I	HAB_I	HAD_I	HF_I	HIR_I	HER_I	KE_I
CF-011-0	N/A	1	0	38.0	17.4	31.0	16.7	40.0	12.0	19.8	32.5	37.5	19.9	29.5	17.3	40.0	22.5	4.6	33.0
CF-012-0	N/A	1	0	59.5	62.5	56.5	64.5	89.0	31.0	36.0	68.0	64.0	62.5	60.0	50.0	87.5	29.0	35.7	65.5
CF-013-0	N/A	1	0	45.0	45.0	38.0	29.0	50.0	16.5	26.5	49.0	49.0	46.0	37.5	30.0	54.5	29.0	10.6	53.0
CF-014-0	N/A	1	0	40.5	34.0	37.0	28.0	46.0	14.6	18.8	37.0	48.0	34.5	32.5	33.0	44.5	20.3	9.9	34.0
CF-016-0	N/A	1	0	42.0	36.0	48.0	42.0	51.5	19.4	31.0	55.5	46.5	39.5	36.5	44.0	55.5	34.0	13.8	48.0
CF-018-0	N/A	1	0	41.0	37.0	34.0	14.7	50.3	14.9	23.7	40.5	40.0	37.0	34.0	15.8	62.0	26.0	16.0	42.0
CF-019-0	N/A	1	0	49.7	56.0	40.5	50.0	78.0	26.0	28.0	62.0	57.0	53.5	46.0	54.0	82.5	33.5	24.0	65.5
CF-020-0	N/A	1	0	45.0	30.0	33.0	26.0	43.0	24.0	24.0	57.5	44.5	34.0	33.0	30.0	43.0	23.0	9.1	49.0
Cf-021-0	N/A	1	0	33.0	24.0	30.0	21.0	37.5	12.1	18.9	29.0	34.0	23.0	31.0	29.5	41.5	19.7	7.1	29.0
CF-022-0	N/A	1	0	40.5	47.0	45.0	51.0	90.0	27.5	27.2	54.0	42.0	61.0	56.5	57.0	89.5	19.3	9.7	65.0
F-001-0	1	1	1	40.0	36.0	35.0	32.0	43.0	24.0	23.0	50.0	35.0	31.0	30.0	33.0	46.0	24.0	24.0	50.0
F-003-0	1	1	1	28.0	25.0	18.1	14.5	29.0	15.8	22.0	25.0	21.0	22.0	17.4	12.8	23.8	18.6	11.2	23.0
F-005-0	0	1	1	24.0	28.5	27.0	30.5	38.5	16.4	7.1	31.0	19.2	24.0	28.0	19.7	29.0	5.9	10.7	30.3
F-007-0	0	1	1	17.0	23.5	18.0	15.5	30.0	24.0	9.2	27.0	18.8	24.0	25.5	17.0	34.0	6.4	20.0	30.0
F-008-0	1	1	1	32.0	15.2	24.5	11.7	29.0	7.6	8.8	25.0	27.0	14.0	24.5	15.0	23.0	11.6	8.2	17.6
F-009-0	1	1	1	31.0	28.5	31.5	24.0	50.0	9.6	28.7	38.0	34.0	32.0	32.0	33.5	49.5	24.2	11.0	39.5
F-010-0	0	1	1	10.6	5.6	13.5	7.9	11.1	13.2	8.0	17.0	7.6	1.3	11.3	5.4	10.3	1.4	10.2	9.3
F-011-0	1	1	1	54.5	42.0	38.0	31.0	49.5	12.1	26.0	46.2	63.5	46.0	34.0	31.0	43.5	24.0	5.9	53.0
F-012-0	1	1	1	28.0	25.5	34.0	21.0	50.0	8.0	3.9	30.5	28.0	30.5	30.0	17.2	46.0	11.4	0.8	20.0
F-013-0	1	1	1	27.0	17.6	33.0	17.0	39.0	7.0	6.8	27.0	11.2	5.1	11.5	8.1	6.4	3.4	0.0	4.5
F-014-0	1	1	1	35.5	24.5	30.5	17.8	40.0	6.9	15.7	32.5	35.0	26.5	31.5	19.6	37.5	13.1	3.1	33.5
F-015-0	1	1	1	35.5	16.7	16.7	11.8	36.0	6.2	10.0	34.5	17.7	12.0	13.3	14.6	32.5	20.8	2.6	33.0
Subject ID#				**HE-U3**	**KF-U3**	**HAB-U3**	**HAD-U3**	**HF-U3**	**HIR_U3**	**HER_U3**	**KE_U3**	**HE_I3**	**KF_I3**	**HAB_I3**	**HAD_I3**	**HF_I3**	**HIR_I3**	**HER_I3**	**KE_I3**
CF-011-0				19.4	21.5	33.0	16.7	44.0	5.3	20.4	37.5	30.5	12.7	29.5	17.2	45.5	17.0	4.5	31.0
CF-012-0				54.0	46.5	54.0	44.5	106.0	30.0	30.6	73.0	60.0	61.0	71.0	39.0	104.5	34.0	15.2	80.0
CF-013-0				46.5	36.5	36.0	33.0	52.2	16.1	27.5	58.5	43.0	42.5	32.0	35.5	51.0	32.0	13.8	56.5
CF-014-0				37.5	32.5	36.0	29.0	46.0	11.0	25.6	43.0	42.5	33.0	37.0	31.0	47.0	29.0	6.3	44.5
CF-016-0				44.5	30.5	39.0	33.0	48.0	17.6	27.0	46.0	40.0	38.5	35.0	36.0	47.0	33.0	12.4	48.0
CF-018-0				33.5	33.5	33.5	15.5	48.5	8.2	22.9	41.0	35.5	35.5	33.5	21.0	56.5	24.0	7.0	39.0
CF-019-0				52	51.5	48.5	50.5	73	27	32.7	68	57.5	56.5	41.5	44	90.5	18.8	15.5	73.5
CF-020-0																			
Cf-021-0																			
CF-022-0																			
F-001-0				32.0	41.0	28.5	33.0	39.0	28.0	28.0	34.0	32.0	38.5	28.0	31.5	46.0	25.0	24.0	34.2
F-003-0																			
F-005-0				12.9	32.0	29.5	27.5	28.7	21.8	11.2	32.2	10.4	30.5	29.5	28.5	24.0	11.4	27.2	31.8
F-007-0				19.0	13.4	13.2	10.3	31.0	26.4	7.6	13.6	18.1	20.8	14.5	11.2	29.0	12.4	19.8	27.0
F-008-0				20.0	14.0	20.0	24.0	26.0	4.6	13.6	27.0	13.4	18.0	15.8	18.8	23.0	12.5	10.0	31.0
F-009-0				33.0	37.5	43.0	33.5	43.5	13.5	29.0	43.0	33.0	34.5	34.0	37.5	50.0	25.5	15.2	45.0
F-010-0				31.5	17.1	19.8	34.5	39.0	23.5	6.6	41.0	30.0	21.5	37.5	15.4	45.5	9.6	30.2	40.5
F-011-0				56.5	43.5	41.0	31.5	58.0	25.5	27.4	60.0	61.0	45.5	38.5	38.0	49.5	25.0	17.1	56.0
F-012-0																			
F-013-0				34.0	16.4	19.5	18.6	48.5	4.7	17.8	40.5	35.5	18.0	18.0	17.9	39.5	6.3	38.0	0.0
F-014-0				27.5	27.2	32.5	20.0	37.0	9.2	27.7	45.0	26.0	27.5	36.0	12.7	42.5	20.2	8.8	40.0
F-015-0																			
Subject ID#				**HE-U6**	**KF-U6**	**HAB-U6**	**HAD-U6**	**HF-U6**	**HIR_U6**	**HER_U6**	**KE_U6**	**HE_I6**	**KF_I6**	**HAB_I6**	**HAD_I6**	**HF_I6**	**HIR_I6**	**HER_I6**	**KE_I6**
CF-011-0																			
CF-012-0																			
CF-013-0				43.5	44.5	40	31	59.5	14.5	27.5	51	40	44.5	37	38	60	12.9	9.3	61
CF-014-0																			
CF-016-0				46	37.5	44.5	34	60	11.6	27.7	62.5	49.5	39	44.5	35	70	16.3	14.9	62
CF-018-0				29.5	36.5	34	16.7	50	14.4	19	50.5	39.5	30.5	36.5	31	60	10.3	5.3	51
CF-019-0				64	52.5	46.5	50	85	35	32.4	65	64.5	57	62	46	81.5	19.9	18.6	66
CF-020-0																			
Cf-021-0																			
CF-022-0																			
F-001-0				31.0	32.0	38.0	31.0	43.0	23.6	24.0	48.0	32.0	28.0	35.5	25.0	57.0	25.0	20.2	48.0
F-003-0																			
F-005-0				35.0	34.0	35.0	33.5	42.5	28.2	9.4	33.0	31.0	32.0	35.0	31.0	39.5	13.2	24.0	30.4
F-007-0				33.0	26.0	24.0	15.1	34.5	19.6	11.4	34.0	28.0	26.5	19.4	17.2	30.0	10.4	20.6	38.0
F-008-0				38.0	26.0	32.0	18.2	36.0	13.0	23.7	39.0	36.0	27.0	26.0	31.0	35.5	20.4	8.2	39.0
F-009-0				30.0	34.0	41.0	36.0	48.5	8.4	25.5	49.5	31.5	35.5	31.5	38.5	57.5	19.1	9.1	55.5
F-010-0				17.9	16.4	34	20	41.5	24	11.3	45.5	26	17.6	34	18.3	40.5	11.2	30.4	51
F-011-0																			
F-012-0																			
F-013-0																			
F-014-0																			
F-015-0																			

Raw Data of the HOS and UCLA Scores

ID number	group	How would you rate your current level of function?	HOS-ADL	HOS-Sports	Current LOF	Sport CLOF	UCLA	function?3	HOS-ADL3	HOS-Sports3	Current LOF3	Sport CLOF3	UCLA3	function?6	HOS-ADL6	HOS-Sports6	Current LOF6	Sport CLOF6	UCLA6
Cf-011-0	0	normal	100.0	100.0	100	100	10	normal	100.0	100.0	100	100	10						
CF-012-0	0	normal	100.0	100.0	100	100	10	normal	100.0	100.0	100	100	10						
CF-013-0	0	normal	100.0	100.0	100	100	10	normal	100	100	100	100	10	normal	100	100	100	100	10
CF-014-0	0	normal	100.0	100.0	100	100	9	normal	100.0	100.0	100	100	9						
CF-016-0	0	normal	100.0	100.0	100	100	5	normal	100.0	100.0	100	100	7	normal	100	100	100	100	6
CF-018-0	0	normal	100.0	100.0	100	100	6	normal	100.0	100.0	100	100	9	normal	100	100	100	100	9
CF-019-0	0	normal	100.0	100.0	100	100	10	normal	100.0	100.0	100	100	10	normal	100	100	100	100	10
CF-020-0	0	normal	100.0	100.0	100	100	9												
CF-021-0	0	normal	100.0	100.0	100	100	10												
CF-022-0	0	normal	97.1	100.0	100	100	10												
F-001-0	1	nearly normal	66.2	52.8	70	30	5	nearly	97.1	83.3	100	85	8	normal	100.0	100.0	90	90	10
f-003-0	1	abnormal	55.9	25.0	45	30	4												
F-005-0	1	severely abnormal	69.1	16.7	30	10	6	severely	69.1	13.9	45	15	6	abnormal	80.9	88.9	90	90	10
F-007-00	1	nearly normal	67.6	47.2	65	25	4	abnormal	61.8	52.8	55	30	5	normal	86.8	75.0	70	70	10
F-008-0	1	abnormal	66.2	61.1	60	80	7	nearly	79.4	33.3	60	60	6	nearly	92.6	100.0	90	80	8
F-009-0	1	severely abnormal	73.5	44.4	75	25	6	normal	95.6	94.4	100	100	8	normal	97.1	100.0	100	100	8
F-010-0	1	abnormal	72.1	41.7	40	25	6	nearly	86.8	52.8	80	50	6	abnormal	83.8	47.2	70	50	6
F-011-0	1	nearly normal	94.1	83.3	90	80	10	nearly	83.8	63.9	75	60	7						
F-012-0	1	abnormal	61.8	36.1	75	60	8												
F-013-0	1	abnormal	48.5	47.2	50	20	4	nearly	79.4	52.8	80	70	5						
F-014-0	1	abnormal	45.6	44.4	30	20	9	nearly	66.2	27.8	50	30	5						
F-015-0	1	severely abnormal	42.6	0.0	40	20	5												