The Problem of Femoroacetabular Impingement: A Theoretical Framework to Guide Clinical Practice

Christopher Powers, PT, PhD, FAPTA
*University of Southern California*

Alex Weber, MD
*University of Southern California*

Jennifer Bagwell, PT, PhD
*Creighton University*

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Session Objectives

1. Integrate relevant knowledge and understanding of FAI from various disciplines (biomechanics, radiology, orthopaedic surgery, physical therapy).
2. Describe a potential pathomechanical framework for FAI.
3. Describe the role of surgery in the treatment of FAI.
4. Describe the role of physical therapy in the treatment of FAI.

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Session Speakers

Alex Weber MD  
*Assistant Professor*  
*University of Southern California*

Jenny Bagwell, DPT, PhD  
*Assistant Professor*  
*Creighton University*

Chris Powers, PT, PhD  
*Professor*  
*University of Southern California*
**Session Schedule**

3:00-3:10: Introduction & session overview (Powers)

3:10-4:20: Presentation of a pathomechanical model of FAI (Weber, Bagwell, Powers)

4:20-4:35: Treatment implications from a surgeon’s perspective (Weber)

4:35-4:50: Treatment implications from a physical therapist’s perspective (Powers)

4:50-5:00: Questions & Answers

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**Femoroacetabular Impingement (FAI), or hip impingement syndrome**, may affect the hip joint in young and middle-aged adults and occurs when the ball shaped femoral head rubs abnormally or does not permit a normal range of motion in the acetabular socket.

Wikipedia (Accessed 2/22/18)

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**The Problem of Femoroacetabular Impingement**

- Relatively new clinical syndrome
- Thought to be a common cause of hip & groin pain in young, active individuals
- Potential precursor to hip OA
- Etiology still largely unknown
- Treatment approaches vary
- Surgical and conservative treatment outcomes are mixed
What do we know about FAI??

**Literature Associations: Imaging**
- Alpha angle
- Beta angle
- Anterior offset ratio
- Cam deformity
- Pincer deformity
- Pistol grip deformity
- Femoral version
- Acetabular depth
- Acetabular index
- Neck-shaft angle
- Center edge angle
- Cross-over sign
- Acetabular anteversion
- Acetabular inclination
- Acetabular retroversion
- Tonnis angle
- Tonnis grade
- Coxa profunda
- Iliac spine projection
- Pelvis incidence angle

**Literature Associations: Kinematics & Kinetics**
- Excessive anterior pelvic tilt
- Reduced posterior pelvic tilt
- Pelvic drop
- Excessive hip adduction
- Excessive hip internal rotation
- Limited hip internal rotation
- Limited hip external
- Limited hip extension
- Limited hip flexion
- Reduced hip extensor moment
Literature Associations:
Physical Impairments & Clinical Tests

- Hip muscle weakness
- Gluteal inhibition
- Hip capsule tightness
- Limited passive hip internal rotation
- Limited passive hip flexion
- Limited passive hip external rotation
- Positive FABER test
- Positive log roll test
- Positive impingement test

How does one make sense of all this??

Diagram:
Abnormal Bony Morphology
Susceptible Populations and Activities
Chondroaboral Damage
Gluteal Inhibition & Capsular Fibrosis
Bony Abutment (Impingement)
Femoroacetabular Impingement Syndrome (FAIS): Anatomy and Risk Factors

Alexander E. Weber, MD
Assistant Professor of Orthopaedic Surgery
Section of Sports Medicine
Department of Orthopaedic Surgery

FAI Paradigm

What Is FAI?
Warwick International Agreement:

Attempt to **standardize the clinical language** used to describe femoroacetabular impingement

Created the clinical entity known as **femoroacetabular impingement syndrome (FAIS)**

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**Femoroacetabular Impingement Syndrome (FAIS)**

FAIS syndrome is a motion-related clinical disorder of the hip with a triad of symptoms, clinical signs and imaging findings. It represents symptomatic premature contact between the proximal femur and the acetabulum.

Lever of agreement: mean score 9.8 (95% CI: 9.0 to 10.6).

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**Clinical Definition of FAIS**

FAIS Triad:
1. Symptoms – mechanical
2. Signs – decreased ROM
3. Radiographic findings of FAI
Who is at risk for FAIS?

- More clearly defined for *cam-type* than pincer-type FAIS
- Contributions:
  - Genetic predisposition
  - Gender
  - Physical activities
  - Pelvic kinematics
Susceptible Populations

- Intrinsic Factors
  - Genetics
  - Gender
  - Hip Kinematics

- Extrinsic Factors
  - High Intensity Athletics

Secondary Conditions
- Legg-Calves-Perthes Disease (LCPD)
- Slipped Capital Femoral Epiphysis (SCFE)
- Post-Traumatic

Susceptible Populations

- Intrinsic Factors
  - Genetics
  - Gender
  - Hip Kinematics

- Extrinsic Factors
  - High Intensity Athletics

Secondary Conditions
- Legg-Calves-Perthes Disease (LCPD)
- Slipped Capital Femoral Epiphysis (SCFE)
- Post-Traumatic
How do the bony changes develop?

- **Genetics**
  - Siblings of patients treated surgically for FAIS had elevated risk of cam deformities (Pollard, JBJS, 2010)

- **Gender**
  - Male predisposition for cam-type
  - Female predisposition for pincer-type (Nakahara, J Ortho Research, 2011)

- **High-intensity athletics**
  - Repetitive flexion-internal rotation on the femoral physis (Philippon, AJSM, 2012)

- **Hip kinematics**

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High-intensity athletics

- Abnormal anterior-superior epiphysial forces in athletes compared to controls (Siebenrock, CORR, 2004; Siebenrock, CORR, 2013)

- Juxtaphyseal microtrauma or physeal shear at time of growth plate closure (Keizer, JOS, 2014)

- Microtrauma as a possible SCFE variant (Albers, CORR, 2010)

- No harm in arthroscopic treatment with open physes (Cvetanovich, Weber, JS, 2016)

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Who develops symptomatic FAIS?

- Risk factors
  - Abnormal body mechanics
  - Genetically susceptible individuals
  - High-intensity athletics

- Cyclic progression of FAIS
  - Chondral degeneration
  - Cartilage injury
  - Structural changes
  - Symptomatic FAIS involvement

- Risk factors:
  - Abnormal body mechanics
  - Genetic predisposition
  - High-intensity athletics

- Cyclic progression of FAIS:
  - Chondral degeneration
  - Cartilage injury
  - Structural changes
  - Symptomatic FAIS involvement
Who develops symptomatic FAIS?

Remember, not all bony abnormalities result in symptomatic FAIS

• Prevalence of radiographic signs of FAIS in asymptomatic populations
  • 54.8% in athletes vs. 23.1% in general population (Frank, Arthroscopy, 2015)
  • Radiographic FAIS in NFL combine athletes
    • 71% of NFL combine athletes had one radiographic finding consistent with FAIS, but only 31.4% symptomatic (Larson, Arthroscopy, 2013)
    • 93.4% of NFL combine athletes with pelvic radiographs had FAIS findings without current hip symptoms (Frank, Arthroscopy, 2015)

Who develops symptomatic FAIS?

• Largely unanswered question
  • Activity-level related?
  • Resultant damage to articular soft tissue structures (chondrolabral damage)?

• Physical examination signs and radiographic findings are only 2/3 of the Triad
  • Must have Symptoms
  • Reminder for surgeons more than therapists!
The Role of Kinematics

Asymptomatic
- 14-55% cam morphology

Motions Contributing to Impingement
- Hip Flexion/Pelvis Anterior Tilt
- Hip Adduction
- Hip Internal Rotation

References:
3. Laborie Radiology 2011
4. Frank Arthroscopy 2015
FAI Gait Kinematics

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kennedy (2009)</td>
<td>↓ frontal hip ROM and frontal pelvis ROM</td>
</tr>
<tr>
<td>Brisson (2013)</td>
<td>↓ frontal hip ROM</td>
</tr>
<tr>
<td>Hunt (2013)</td>
<td>↓ speed, hip abduction and IR</td>
</tr>
<tr>
<td>Rylander (2013)</td>
<td>↓ sagittal hip ROM and hip IR</td>
</tr>
<tr>
<td>Kumar (2014)</td>
<td>None</td>
</tr>
<tr>
<td>Hestroni (2015)</td>
<td>↓ pelvic IR and hip abduction at heel strike</td>
</tr>
<tr>
<td>Diamond (2016)</td>
<td>↑ sagittal pelvis ROM</td>
</tr>
<tr>
<td>Diamond (2018)</td>
<td>↓ sagittal hip ROM</td>
</tr>
</tbody>
</table>

Are Kinematics during Gait a Risk Factor?

Sagittal Pelvis Kinematics

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Task</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lamontagne (2009)</td>
<td>Squat</td>
<td>↓ sagittal pelvis ROM</td>
</tr>
<tr>
<td>Rylander (2013)</td>
<td>Step Ascent</td>
<td>↑ pelvic anterior tilt</td>
</tr>
<tr>
<td>Kumar (2014)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Нg (2015)</td>
<td>Drop Jump</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Bagwell (2016)</td>
<td>Squat</td>
<td>More anterior pelvic at peak hip flexion</td>
</tr>
<tr>
<td>Diamond (2017)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Hammond (2017)</td>
<td>Step Ascent</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Diamond (2018)</td>
<td>Step Ascent</td>
<td>Not Significant</td>
</tr>
</tbody>
</table>
Sagittal Pelvis Kinematics

Bagwell ClinBiomech 2016

CAM = 23.4 ± 11.2
CON = 12.5 ± 17.1
p = 0.032*

Ng ClinOrthopRelatRes 2015

Lamontagne ClinOrthopRelatRes 2009

Sagittal Pelvis Kinematics


TABLE 3

<table>
<thead>
<tr>
<th>Symptomatic Hip Condition Group</th>
<th>Symptomatic Hip Healthy Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip flexion to 90°</td>
<td>Hip flexion to 90°</td>
</tr>
<tr>
<td>-1.4±4.9</td>
<td>-4.6±4.3</td>
</tr>
<tr>
<td>Hip flexion to 90°</td>
<td>Hip flexion to 90°</td>
</tr>
<tr>
<td>-7.6±4.4</td>
<td>-13.4±10.6</td>
</tr>
<tr>
<td>Hip flexion to 90°</td>
<td>Hip flexion to 90°</td>
</tr>
<tr>
<td>0.2±8.9</td>
<td>10.2±10.9</td>
</tr>
<tr>
<td>Hip flexion to 90°</td>
<td>Hip flexion to 90°</td>
</tr>
<tr>
<td>-0.1±13.2</td>
<td>-16.3±6.9</td>
</tr>
<tr>
<td>Hip flexion to 90°</td>
<td>Hip flexion to 90°</td>
</tr>
<tr>
<td>0.1±10.2</td>
<td>10.2±10.9</td>
</tr>
</tbody>
</table>

*Significant differences compared to other symptomatic hip conditions group and healthy group.
Clinical Importance of Sagittal Pelvis Kinematics

- 10° anterior pelvis tilt
  - Increased acetabular retroversion
  - With 90° hip flexion, hip IR ↓ 5.9°

- 10° posterior pelvis tilt
  - With 90° hip flexion, hip IR ↑ 5.1°

Sagittal Pelvis Kinematics Discussion

- Possible explanations
  - Lack muscular ability to posterior tilt
  - Lumbopelvic mobility limitation
  - Limited posterior tilt could be a mechanism contributing to increased impingement

Frontal Plane Kinematics

<table>
<thead>
<tr>
<th>Author (Year)</th>
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<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lamontagne (2009)</td>
<td>Squat</td>
<td>net significant</td>
</tr>
<tr>
<td>Rylander (2013)</td>
<td>Step Ascent</td>
<td>no significant</td>
</tr>
<tr>
<td>Kumar (2014)</td>
<td>Squat</td>
<td>peak hip adduction</td>
</tr>
<tr>
<td>Bagwell (2016)</td>
<td>Drop jump</td>
<td>net significant</td>
</tr>
<tr>
<td>Diamond (2017)</td>
<td>Squat</td>
<td>no significant</td>
</tr>
<tr>
<td>Hammond (2017)</td>
<td>Step Ascent</td>
<td>no significant</td>
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<tr>
<td>Diamond (2018)</td>
<td>Step Ascent</td>
<td>no significant</td>
</tr>
</tbody>
</table>

1. Kumar PMR 2013
2. Biomech 
3. Ortho 
4. Sports 
5. Clinic 
6. Biomech 
7. Ortho 
8. Sports 
9. Clinic
Frontal Plane Kinematics

- Possible explanations
  - Compensation: unload involved hip and ↓ hip flexion²
  - Abductor muscle weakness resulting in altered habitual position¹⁻³
  - Increased hip adduction could be a mechanism contributing to increased impingement

Frontal Plane Kinematics diagram

Transverse Plane Kinematics

<table>
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<tr>
<td>Lamontagne (2009)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Rylander (2013)</td>
<td>Step Ascent</td>
<td>3. peak hip</td>
</tr>
<tr>
<td>Kumar (2014)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Ng (2015)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Bagwell (2016)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Diamond (2017)</td>
<td>Squat</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Hammond (2017)</td>
<td>Step Ascent</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Diamond (2018)</td>
<td>Step Ascent</td>
<td>Not Significant</td>
</tr>
</tbody>
</table>

Transverse Plane Kinematics

CAM = 9.4 ± 7.8
CON = 15.2 ± 9.5
p = 0.041*
Transverse Plane Kinematics Discussion

• Possible explanations
  • Avoidance behavior
  • Impingement

Transverse Plane Kinematics Discussion

Control=112.6 flex, 10.5 abd, 14.8 IR        Cam=106.3 flex, 10.5 abd, 8.9 IR

Transverse Plane Kinematics Discussion

• Possible explanations
  • Avoidance behavior
  • Impingement
  • Hip external rotator weakness or limited external rotation ROM could still contribute to impingement
Kinematics Big Picture

- Kinematic profiles are varied across studies
- Some consistent patterns
- Difficult to ascertain cause-effect relationships
- Evaluate potential kinematic risk factors on an individual basis

Motions which Contribute to Impingement
- Pelvis Anterior Tilt/Decreased Posterior Tilt
- Hip Adduction
- Hip Internal Rotation

Risk Factors
- Abnormal Bony Morphology
- Susceptible Populations and Activities
- Aberrant Hip/Pelvis Kinematics
- Bony Abutment (Impingement)
- Gluteal Inhibition
- Capsular Fibrosis
- Chondrolabral Damage
- Inflammation

Cyclical Progression of FAI
2. Chondrolabral Damage
2. Chondrolabral Damage

- Labral Intrasubstance Tear
- Chondrolabral Wave/Separation
- Chondral Flap/Delamination

3. Inflammation

- Area of active research for our group
- Circulating systemic inflammatory markers are increased with FAI
  - Cartilage oligomeric matrix protein (COMP) and C-reactive protein (CRP) elevated in athletes with FAI compared to healthy controls
  - 24% COMP and 276% CRP increase
  - COMP also elevated in hip osteoarthritis
  - FAI possible “precursor” to hip OA
Capsular Responses to Impingement?
Rodeo, J Orthop Res, 1997

Cytokine driven inflammation & hyperplasia of synovium
Changes in hyaluronic acid metabolism
Formation of adhesions within the ECM
Capsular Thickening & Fibrosis

Anterior-Superior Capsular Thickening in FAI
Inflammation & Arthrogenic Muscle Inhibition
Hopkins & Ingersoll, J Sport Rehabil, 2002

Intra-articular inflammation & pain

\[ \downarrow \]

Altered afferent neuron activity

\[ \downarrow \]

Decreased motorneuron recruitment & muscular force production

The Presence of Intra-articular Fluid Results in Gluteal Muscle Inhibition

Hip Muscle Force Production Deficits in Persons with Symptomatic FAI

- Diminished hip flexion, abduction, adduction and external rotation strength (Casartelli et al., Osteoarthritis & Cartilage 2012)

- Diminished hip flexion and abduction strength (Nepplle et al, JAAOS, 2015)
**Movement Impairments Contributing to FAI**

- Excessive anterior tilt (contributes to impingement)
- Limited posterior tilt (limits ability to avoid impingement)
- Hip adduction & internal rotation with a flexed hip (>60 degrees)

**Persons with FAI Have Decreased Posterior Pelvic Tilt During Squatting**

Abnormal Hip Motions Associated with Acetabular Labral Pathology
Austin et al. JOSPT, 2009

Protective Posterior Pelvic Tilt and Femur External Rotation are Coupled Motions

Capsular Fibrosis: Loss of Protective Hip External Rotation and Posterior Pelvic Tilt?
Capsular Fibrosis: Loss of Protective Hip External Rotation and Posterior Pelvic Tilt?

Gluteal Inhibition: Loss of Protective Hip External Rotation and Abduction

Gluteal Inhibition: Loss of Protective Posterior Pelvic Tilt?
Persons with FAI Have Decreased Hip Extensor Moments During Squatting

**Gluteus Maximus:**
“Protects against Impingment”
- Posterior tilts the pelvis
- Abductor
- External Rotator

**Gluteus Medius:**
“The Frontal Plane Muscle”
Neuromuscular Compensations Associated With Glut Max Inhibition

- Extensors
- Hamstrings
- Adductor Magnus

Neuromuscular Compensations Associated With Glut Med Inhibition

- Abductors
- TFL
- GMAX

Hip Adduction and Internal Rotation

- Weakness/Inhibition?
  - Abductors
  - External rotators

- Compensatory muscle activity?
  - Adductors
  - Internal rotators
Femoroacetabular Impingement Syndrome (FAIS): Treatment Decisions

Alexander E. Weber, MD
Assistant Professor of Orthopaedic Surgery
Section of Sports Medicine
Department of Orthopaedic Surgery

Orthopedic Surgery is a Commodity

It is all about shared decision-making

Often, there is no right or wrong...just options!

Factors Affecting Treatment Decisions

<table>
<thead>
<tr>
<th>Patient-related</th>
<th>Surgeon-related</th>
<th>Healthcare-related</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preferences/Expectations</td>
<td>Patient-related factors</td>
<td>Standard of care</td>
</tr>
<tr>
<td>Demographics – age, sex, race</td>
<td>Pathology/complexity</td>
<td>Regional variation</td>
</tr>
<tr>
<td>Education-level/Socioeconomic status</td>
<td>Skill/training</td>
<td>Insurance treatment algorithm - cost</td>
</tr>
<tr>
<td>Comorbidities – obesity, smoking status</td>
<td>Prior experiences – anecdotal success/failure</td>
<td></td>
</tr>
<tr>
<td>Ease/Recovery</td>
<td>Outcomes literature</td>
<td></td>
</tr>
<tr>
<td>Out-of-pocket cost</td>
<td>Practice and reimbursement model</td>
<td></td>
</tr>
</tbody>
</table>
### Treatment Decisions for FAIS

- Length of symptoms
- Prior treatment history
  - No treatment vs. exhaustive nonoperative modalities
  - Treatment modalities to-date
- Activity level
- Athletic timeline
  - In-season vs. out-of-season
  - Contract year

### Doc, if I don’t get treated will I develop hip arthritis?

- Prospective study of 1002 early OA patients at 5 years:
  - Alpha > 60°: OR 3.67 developing end-stage OA
  - Alpha > 80°: OR 9.66 developing end-stage OA
  - Alpha > 80° and Limited IR: OR 25.21 end-stage OA

- Systematic review of the natural history of FAIS:
  - Good short- and mid-term improvement in pain and function after surgical correction of FAIS
  - Insufficient evidence to prove casual relationship

### Doc, then why should we do surgery?

- Greater improvements for:
  - Younger patients
  - Less joint damage at the time of surgery

- Predictably decrease pain and increase function
- Return athletes to a high level of play (87-95%)
Cam Decompression and Dynamic Examination

FAIS Treatment

Thank You!
Treatment Implications from the Physical Therapist’s Perspective

Breaking the Cycle....

Rehabilitation Goals

1. Improve hip mobility
2. Improve gluteal activation
3. Address gluteal strength deficits
4. Address underlying movement impairments
Improve Hip Mobility

Scientific Rationale for “Hip Activation” Exercises

Pathway to Gluteal Muscle Weakness

Gluteal Muscle Inhibition
\[\downarrow\]
Diminished use during functional activities
\[\downarrow\]
Learned non-use (glut amnesia)
\[\downarrow\]
Gluteal Muscle Weakness
The Big Challenge

- The hip is a redundant muscular system: it is easy to compensate!
- Gluteus Maximus is a postural muscle with poor representational area in the primary motor cortex
How do you Strengthen Gluteus Maximus if you Cannot Access (Activate) it?

Neuroplasticity Associated with Gluteus Maximus Activation Training

Quantifying Corticomotor Function: *Transcranial Magnetic Stimulation (TMS)*

- Safe, noninvasive & painless way to stimulate the human motor cortex through electromagnetic induction to evaluate the evoked motor response
- TMS can be used to assess the integrity and responsiveness of the corticomotor pathways
TMS Data Acquisition

TMS can be used to identify areas of representation in the primary motor cortex.

TMS Data Acquisition for Glut Max
Fisher et al., JOSPT, 2013
Why Static Holds??

• Static holds facilitate the “encoding” phase for cognitive processes which are thought to play an important role in helping the learner create a motor memory.

• Static holds require prolonged focus and concentration, thus strengthening the corticomotor pathway. Armer create a motor

Hip Activation Exercises

Gluteal Strengthening
Squat with Theraband

Standing Fire Hydrant

Monster Walks (Forward)
Identification and Correction of Abnormal Hip Kinematics
Identification and Correction of Abnormal Hip Kinematics

Breaking the Cycle....
Post Operative Hip Arthroscopy Rehabilitation Protocol for Dr. Alexander Weber
Labral Repair With or Without FAI Component

Date of Surgery:

**ROM Restrictions:**
-Perform PROM in patient’s PAIN FREE Range

<table>
<thead>
<tr>
<th>FLEXION</th>
<th>EXTENSION</th>
<th>EXTERNAL ROTATION</th>
<th>INTERNAL ROTATION</th>
<th>ABDUCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited to: 90 degrees x 2 weeks (may go higher in the CPM)</td>
<td>Limited to: 0 degrees x 3 weeks</td>
<td>Limited to: *30 degrees @ 90 degrees of hip flexion x 3 weeks *20 degrees in prone x 3 weeks</td>
<td>Limited to: *20 degrees @ 90 degrees of hip flexion x 3 weeks *No limitation in prone</td>
<td>Limited to: 30 degrees x 2 weeks</td>
</tr>
</tbody>
</table>

**Weight Bearing Restrictions:**

<table>
<thead>
<tr>
<th>20# FOOT FLAT Weight Bearing</th>
<th>Gait Progression:</th>
</tr>
</thead>
<tbody>
<tr>
<td>-for 3 weeks (non-Micro-fracture)</td>
<td>Begin to D/C crutches at 3 weeks (6 wks if MicroFracture is performed).</td>
</tr>
<tr>
<td>-for 6 weeks (with Microfracture)</td>
<td>Patient may be fully off crutches and brace once gait is PAIN FREE and NON-</td>
</tr>
<tr>
<td></td>
<td>COMPENSATORY</td>
</tr>
</tbody>
</table>

**PATIENT PRECAUTIONS:**
-NO Active lifting of the surgical leg (use a family member/care taker for assistance/utilization of the non-operative leg) for approximately 4 weeks  
-NO sitting greater than 30 minutes at a time for the first 3 weeks  
-DO NOT push through pain

**POST-OP DAY 1/INITIAL PHYSICAL THERAPY VISIT:**

☑️ Check List:

<table>
<thead>
<tr>
<th>Activity/Instruction</th>
<th>Frequency</th>
<th>Completed ?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Instructed in ambulation and stairs with crutches and 20# FFWB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upright Stationary bike no resistance</td>
<td>20 minutes daily</td>
<td></td>
</tr>
<tr>
<td>CPM usage</td>
<td>4 hours/day (decrease to 3 hours if stationary bike used for 20’)</td>
<td></td>
</tr>
<tr>
<td>Instruction on brace application/usage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PROM (circumduction, abduction, log rolls) instructed to the family/caregiver</td>
<td>20 minutes; 2 times each day</td>
<td></td>
</tr>
<tr>
<td>*maintain restrictions for 3 weeks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prone lying</td>
<td>2-3 hours/day</td>
<td></td>
</tr>
<tr>
<td>Isometrics (quad sets, glut sets, TA activation)</td>
<td>Hold each 5 seconds, 20 times each, 2x/day</td>
<td></td>
</tr>
</tbody>
</table>
**PHASE 1**

Goal: Protect the Joint and Avoid Irritation

**PT Pointers:**
- Goal is symmetric ROM by 6-8 weeks
- NO Active open chain hip flexor activation
- Emphasize Proximal Control
- Manual Therapy to be provided 20-30 minutes/PT session

<table>
<thead>
<tr>
<th>Date of surgery:</th>
<th>Week</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
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</thead>
<tbody>
<tr>
<td>Stationary bike (20 min, increase time at week 3 as patient tolerates)</td>
<td>Daily</td>
<td>✓</td>
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<tr>
<td>Soft tissue mobilization (specific focus to the adductors, TFL, lliopsoas, QL and inguinal ligament)</td>
<td>Daily (20-30 minutes each session)</td>
<td>✓</td>
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<tr>
<td>Isometrics -quad, glutes, TA</td>
<td>daily</td>
<td>✓</td>
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<td></td>
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<tr>
<td>Diaphragmatic breathing</td>
<td>daily</td>
<td>✓</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Isometrics -rocking, pelvic tilts, arm lifts</td>
<td>daily</td>
<td>✓</td>
<td>✓</td>
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</table>

**PHASE 2**

Goal: Non-Compensatory Gait and Progression

**PT Pointers:**
- Advance ambulation slowly without crutches/brace as patient tolerates and avoid any compensatory patterns
- Provide tactile and verbal cueing to enable non-compensatory gait patterning
- Advance exercises only as patient exhibits good control (proximally and distally) with previous exercises
- If MicroFracture was performed, Hold all weight bearing exercises until week 6

<table>
<thead>
<tr>
<th>Date of Surgery:</th>
<th>Week</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<th>7</th>
<th>8</th>
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<tbody>
<tr>
<td>Progress off crutches starting week 3</td>
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<tr>
<td>Continuation of soft tissue mobilization to treat specific restrictions</td>
<td>2x/week</td>
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<tr>
<td>Joint Mobilizations posterior/inferior glides</td>
<td>2x/week</td>
<td>✓</td>
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<tr>
<td>Joint Mobilizations anterior glides</td>
<td>2x/week</td>
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<tr>
<td>Prone hip extension</td>
<td>5x/week</td>
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<tr>
<td>Tall kneeling and ½ kneeling w/ core and shoulder girdle strengthening</td>
<td>5x/week</td>
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<tr>
<td>Standing weight shifts: side/side and anterior/posterior</td>
<td>5x/week</td>
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<tr>
<td>Backward and lateral walking no resistance</td>
<td>5x/week</td>
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<tr>
<td>Standing double leg ½ knee bends</td>
<td>5x/week</td>
<td>✓</td>
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<tr>
<td>Advance double leg squat</td>
<td>5x/week</td>
<td>✓</td>
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<tr>
<td>Forward step ups</td>
<td>5x/week</td>
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<tr>
<td>Modified planks and modified side planks</td>
<td>5x/week</td>
<td>✓</td>
<td>✓</td>
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<tr>
<td>Elliptical (begin 3 min, ↑ as tolerated)</td>
<td>3x/week</td>
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</table>
Phase 3
Goal: Return the Patient to Their Pre-Injury Level

PT Pointers:
- Focus on more FUNCTIONAL exercises in all planes
- Advance exercises only as patient exhibits good control (proximally and distally) with previous exercises
- More individualized, if the patients demand is higher than the rehab will be longer

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<tr>
<th>Date of surgery</th>
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<tr>
<td>Continue soft tissue and joint mobilizations PRN</td>
<td>2x/week</td>
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<tr>
<td>Lunge forward, lateral, split squats</td>
<td>3x/week</td>
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<tr>
<td>Side steps and retro walks w/ resistance (begin w/ resistance more proximal)</td>
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<tr>
<td>Single leg balance activities: balance, squat, trunk rotation</td>
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<tr>
<td>Planks and side planks (advance as tolerated)</td>
<td>3x/week</td>
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<td>Single leg bridges (advance hold duration)</td>
<td>3x/week</td>
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<td>Slide board exercises</td>
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<tr>
<td>Agility drills (if pain free)</td>
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<tr>
<td>Hip rotational activities (if pain free)</td>
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</table>

Phase 4
Goal: Return to Sport

PT Pointers:
- It typically takes 4-6 months to return to sport, possible 1 year for maximal recovery
- Perform a running analysis prior to running/cutting/agility
- Assess functional strength and obtain proximal control prior to advancement of phase 4

<table>
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<tr>
<th>Date of surgery</th>
<th>Week</th>
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<tr>
<td>Running</td>
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<tr>
<td>Return to sport specifics</td>
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