Abstract

A normal hip has a natural tendency toward stability because of both osseous and soft tissue structures. Hip motion is primarily rotational around a center of rotation. When the femoral head and its center of rotation translate, with or without rotation, the inherent stability of the femoroacetabular articulation may be lost. The spectrum of hip instability ranges from subtle microinstability to traumatic dislocation. Microinstability may be the cause or the effect of several other hip pathologies. Soft tissue contributions to stability include the static capsule, dynamic musculotendinous units, and underlying generalized connective tissue (eg, Ehlers-Danlos). Osseous contributions include multiple femoral and acetabular radiographic coverage parameters. Iatrogenic contributions include an unoperated capsulotomy, overresection of the acetabular rim (iatrogenic dysplasia), overresection of cam osteochondroplasty, iliopsoas tenotomy, labral debridement, and ligamentum teres debridement. Patients with hip microinstability often have deep groin pain, exhibited by a C sign. These patients frequently participate in flexibility sports and activities, such as ballet, gymnastics, figure skating, and martial arts. On physical examination, generalized hypermobility syndromes should be assessed, as should loss of log-roll external rotation recoil, excessive abduction, trochanteric-pelvic impingement, and abductor fatigue. Standard imaging, including plain radiographs, magnetic resonance imaging, and computed tomography, should be analyzed for all causes of hip pain. A new plain radiograph, the splits radiograph is introduced here, consistently showing lateral femoral head translation and creation of a vacuum sign, showing hip microinstability. The splits radiograph is illustrated in a 22-year-old female dancer who presented with bilateral deep anterolateral groin pain. [Orthopedics. 2016; 39(1):e169-e175.]

The patient had symptoms for more than 18 months. Initially, dancing was the only instigating activity, especially with the splits position, during “turnout,” with deep hip flexion and rotation, and with extension and external rotation. Physical examination showed 9 of 9 Beighton criteria and symmetric range of motion in both hips, with 150° flexion, 155° hip abduction, and 20° internal and 70° external rotation (at 90° flexion). On hip abduction to a firm endpoint, the extremity was internally rotated and exacerbation of pain was noted (trochanteric-pelvic impingement test). Importantly, these signs on physical examination were the same symptoms that the patient experienced while dancing. Over 6 months, the pain progressed and the patient became weaker and was unable to perform a single-leg stance without a pelvic dip (Trendelenburg) or a single-leg squat. Radiographs showed Tönnis grade 0, normal lateral and anterior-center edge and Tönnis angles, especially with the splits position, during “turnout,” with deep hip flexion and rotation, and with extension and external rotation. Physical examination showed 9 of 9 Beighton criteria and symmetric range of motion in both hips, with 150° flexion, 155° hip abduction, and 20° internal and 70° external rotation (at 90° flexion). On hip abduction to a firm endpoint, the extremity was internally rotated and exacerbation of pain was noted (trochanteric-pelvic impingement test). Importantly, these signs on physical examination were the same symptoms that the patient experienced while dancing. Over 6 months, the pain progressed and the patient became weaker and was unable to perform a single-leg stance without a pelvic dip (Trendelenburg) or a single-leg squat. Radiographs showed Tönnis grade 0, normal lateral and anterior-center edge and Tönnis angles,
and normal alpha angles (Dunn 45° and 90°). However, the tip of the greater trochanter was above the center of the femoral head, and there was coxa profunda, a type 2 anterior inferior iliac spine, and evidence of head-neck junction cortical sclerosis and distal anterolateral neck impingement cyst formation. Splits radiograph showed a vacuum sign and trochanteric-pelvic extra-articular impingement. Magnetic resonance imaging showed no labral injury. The findings of evaluation strongly suggested intra-articular pathology. A preliminary diagnosis of hip microinstability was made, with soft tissue (elevated Beighton) and osseous (trochanteric-pelvic impingement, subspine impingement, acetabular rim impingement [pincer]) contributions. Ultrasound-guided diagnostic and therapeutic bilateral intra-articular injections of local anesthetic and corticosteroid were performed, with complete resolution of pain 5 minutes after injection. The patient began a core, hips, and pelvis strengthening protocol to focus on dynamic stabilization of microinstability. Within 6 weeks, the patient had returned to dancing 5 days per week without symptoms. One year after injection, the patient was continuing a strengthening program in addition to dancing, without and hip or groin symptoms.

Hip Microinstability

The normal hip has a natural tendency to stability because of the depth of the acetabulum, congruency of the femoral head and acetabulum, and surrounding contractile musculotendinous (dynamic) and capsuloligamentous inert (static) tissues. The spectrum of hip instability ranges from subtle microinstability to traumatic dislocation. Microinstability may be either a cause or an effect of several other hip pathologies. In the native hip, these include osseous, chondralabral, capsuloligamentous, and musculotendinous causes. Further, generalized hyperlaxity (eg, connective tissue disease) may contribute to hip microinstability. Postoperatively, especially in hips undergoing arthroscopy, these include unrepaired capsulotomy (or capsulectomy), iliotibial tenotomy, labral debridement, ligamentum teres debridement, debridement of the iliocapsularis or gluteus minimus, overresection of the acetabular rim (iatrogenic dysplasia), overresection of the femoral head-neck junction, and lack of recognition of femoroacetabular or other sources of extra-articular impingement.

Some patients may be at increased risk because of their activities of daily living or participation in sports and activities, primarily those that require high degrees of strength and power as well as flexibility and motion. Dance, gymnastics, figure skating, yoga, and cheerleading are among the sports and activities that may predispose to microinstability (symptomatic) over simple hyperlaxity or hypermobility (asymptomatic). Patients

<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>Journal</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerezal et al (2012)</td>
<td>European Journal of Radiology</td>
<td>Inability to keep the femoral head centered within the acetabular fossa without complete luxation or marked subluxation of the joint</td>
</tr>
<tr>
<td>Domb et al (2013)</td>
<td>Arthroscopy</td>
<td>Microinstability may lead to ligamentum teres tears, large labral tears, and advanced acetabular chondral damage</td>
</tr>
<tr>
<td>Amenabar and O’Donnell (2012)</td>
<td>Arthroscopy Techniques</td>
<td>Idiopathic instability occurs in the absence of trauma, dysplasia, overuse, or a connective tissue disorder</td>
</tr>
<tr>
<td>Guanche and Sikka (2005)</td>
<td>Arthroscopy</td>
<td>Ligamentum teres has a role as a hip stabilizer and should be included in the evaluation of stability</td>
</tr>
<tr>
<td>Kolo et al (2013)</td>
<td>Skeletal Radiology</td>
<td>Eight elite runners (mean age, 36 years) underwent hip arthroscopy for a labral tear</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Repetitive hyperextension during stride leads to subtle instability or recurrent subluxation and attritional stress at the chondrolabral junction and ligamentum teres (3 subjects)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Joint subluxation occurred in all hips of professional ballet dancers in the splits position, with only 1 of 59 hips showing cam or pincer femoroacetabular impingement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Compared with control subjects, ballet dancers had significantly more acetabular chondral lesions, labral tears, and herniation pits</td>
</tr>
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</table>
with large cam deformities may be at risk for impingement-induced instability.

Thorough subjective and objective evaluations, combined with plain radiographic imaging and occasionally advanced imaging (magnetic resonance imaging or computed tomography), are necessary to diagnose hip microinstability accurately. Initial management of microinstability is nonoperative. Without femoroacetabular impingement or labral pathology in patients who have undergone unsuccessful conservative treatment, diagnostic and therapeutic arthroscopic evaluation may be performed. Examination under anesthesia may better show subtle loss or excessive asymmetry in motion. Addressing fixable bony and soft tissue pathology in combination with capsulorrhaphy (plication), with or without management of ligamentum teres, may lead to successful clinical outcomes. Although these patients have shown successful short-term results, mid- and long-term outcomes have not been reported.

### Soft Tissue Contributions

The hip capsule is primarily composed of 4 discrete ligamentous structures, the iliofemoral (Y ligament of Bigelow), ischiofemoral, pubofemoral, and zona orbicularis. The iliofemoral ligament is the strongest of these and is disrupted during interportal capsulotomy (anterolateral to midanterior) in hip arthroscopy. Several biomechanical studies have shown the importance of the iliofemoral ligament for retention of normal hip kinematics. Iliofemoral ligament sectioning (unrepaired capsulotomy) leads to increased external rotation, extension, and anterior and distal translation.

Clinically, during hip arthroscopy with a T capsulotomy, significantly better outcomes are noted in patients who undergo complete capsular repair vs partial repair (unrepaired interportal capsulotomy) (Table 2). The iliofemoral ligament was much stronger than the ischiofemoral ligaments. The iliofemoral ligament had greater stiffness than the ischiofemoral ligaments. The iliofemoral ligament had greater tensile load to failure than the ischiofemoral ligaments.

### Table 2: Role of the Hip Capsule in Microinstability

<table>
<thead>
<tr>
<th>Study (Year)</th>
<th>Study Design</th>
<th>Role</th>
</tr>
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<tbody>
<tr>
<td>Myers et al (2011)</td>
<td>Cadaveric biomechanical: fluoroscopy</td>
<td>Increased external rotation with iliofemoral ligament sectioning (increased 12.9° (P&lt;.0001))</td>
</tr>
<tr>
<td>Martin et al (2008)</td>
<td>Cadaveric biomechanical: motion tracking</td>
<td>Release of the medial and lateral arms of the iliofemoral ligament gave the greatest increase in external rotation</td>
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<tr>
<td>Hewitt et al (2002)</td>
<td>Cadaveric biomechanical: load to failure</td>
<td>The iliofemoral ligament was much stronger than the ischiofemoral ligaments</td>
</tr>
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</table>

In addition to the capsule and ligamentum teres, other soft tissue deficiencies have been considered contributors to iatrogenic hip instability (Table 3). These include iliopsoas tenotomy, with or without capsular repair, especially in patients with excessive femoral antversion. Additionally, labral debridement negates the acetabular suction seal and may lead to instability. Resection of the iliopsoas origin of the indirect or direct head of the rectus femoris, and

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**Table 3**

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the gluteus minimus may predispose to instability. Patients with generalized hypermobility as a result of connective tissue disorders (e.g., Ehlers-Danlos, Marfan, and Down syndromes) may also have elevated risk of hip instability as a result of soft tissue laxity. Although there is no single sine qua non evaluation for microinstability, a 9-point Beighton score may be measured to assess soft tissue laxity. Further history and examination may assess for Brighton’s criteria to make a diagnosis of benign joint hypermobility syndrome.

### Osseous Contributions

Acetabular undercoverage may contribute to varying degrees of hip instability. Based on an asymptomatic population of 409 hips undergoing computed tomography, the normal acetabulum globally covers 40% of the femoral head (61% superiorly, 40% anteriorly, and 48% posteriorly). The mean lateral center-edge angle was 31°, with 15% showing a crossover sign and 30% showing a posterior wall sign. Based on plain radiographs of hips with dysplasia, normal acetabular coverage, overcoverage, and severe overcoverage (protrusio), several reference values were determined for “normal” coverage: lateral center-edge angle of 23° to 33°, femoral head extrusion index of 17% to 27%, negative crossover sign, anterior coverage of 15% to 26%, cranial coverage of 70% to 83%, and posterior coverage of 36% to 47%. Thus, patients with undercoverage that correlates with the defined reference values in these 2 studies may be at risk for microinstability or

<table>
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<tr>
<th>Study (Year)</th>
<th>Subject Age/sex</th>
<th>Time to Instability After Surgery</th>
<th>Direction of Instability</th>
<th>Generalized Ligamentous Laxity</th>
<th>Osseous Morphology</th>
</tr>
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<tbody>
<tr>
<td>Austin et al (2014)</td>
<td>19/F</td>
<td>5 mo</td>
<td>Anterior</td>
<td>Yes</td>
<td>Tenotomy</td>
</tr>
<tr>
<td>Rosenbaum et al (2014)</td>
<td>24/M</td>
<td>19 d, 26 d</td>
<td>Posterior</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Sansone et al (2013)</td>
<td>26/M</td>
<td>3 mo</td>
<td>Anterior</td>
<td>No</td>
<td>Tenotomy</td>
</tr>
<tr>
<td>Mei-Dan et al (2012)</td>
<td>42/F</td>
<td>14 mo</td>
<td>Superior lateral</td>
<td>Yes</td>
<td>Yes (&lt;1 mm bone)</td>
</tr>
<tr>
<td>Schroeder e Souza (2012)</td>
<td>NR</td>
<td>1 d</td>
<td>Anterior</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Ranawat et al (2009)</td>
<td>49 F</td>
<td>2 mo</td>
<td>Anterior</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Benali and Kattah (2009)</td>
<td>39 M</td>
<td>Recovery room</td>
<td>Anterior</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
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**Abbreviations:** F, female; M, male; NR, not reported.

In addition, Ilizaliturri (2014) reported 2 cases of anterior hip dislocation after excessive arthroscopic rim trimming.

Table 3

Instability After Hip Arthroscopy

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**Abbreviations:** F, female; M, male; NR, not reported.

In addition, Ilizaliturri (2014) reported 2 cases of anterior hip dislocation after excessive arthroscopic rim trimming.
frank instability. Assessment of coverage by measuring acetabular version combined with femoral version (McKibbin index) also may help to identify hips that are at risk for microinstability.16

Femoroacetabular impingement may induce instability in the following 4 ways: (1) excessive acetabular anteversion can result in posterior acetabular rim impingement and anterior hip instability; (2) excessive acetabular retroversion can result in anterior impingement and posterior instability; (3) excessive femoral anteversion can result in posterior acetabular rim impingement and anterior hip instability; and (4) excessive femoral retroversion can result in anterior impingement and posterior instability.

Cam impingement prohibits true ball-and-socket mechanics, causing anterior levering over the rim (fulcrum), with subsequent posterior instability.17 With traumatic posterior hip dislocation, there is a greater prevalence of anterior cam and femoral retroversion than in normal hips.18 In an exclusively athletic population with low-energy subluxation or dislocation and posterior acetabular rim fracture, cam and/or pincer impingement has been identified in 64% to 82% of subjects.19-21 Athletes with larger degrees of motion (eg, dancers, gymnasts) (Figure 1) may have impingement-induced instability without abnormal cam or pincer deformities. In a cohort of 59 professional ballet dancers, only 1 hip had evidence of a cam deformity, whereas several other abnormalities were identified on magnetic resonance imaging because of a dynamic “pincer” mechanism from the extreme motion involved with their activity (or extra-articular impingement).22 Further, while in the splits position, all hips subluxated (mean, 2.1 mm). Compared with a control group, the abnormalities in ballet dancers included significantly more acetabular cartilage lesions (mostly superior), more labral tears (mostly posterosuperior to anterosuperior), and more superior hemiation pits. Despite the prevalence of imaging abnormalities in the latter study,22 fewer than two thirds of subjects had symptoms at the time imaging was performed in a follow-up study.23 Further, when certain ballet positions were examined, mean translation was as high as 4.6 mm.24 Whether the microinstability observed in these studies (with the associated intra-articular pathologies) will lead to early osteoarthritis is yet to be determined (Figure 2).

Figure 1: Anteroposterior pelvis radiograph in a 22-year-old female ballet dancer with bilateral deep anterolateral groin pain showing Tönnis grade 0, bilaterally negative ischial spine, posterior wall, and crossover signs with coxa profunda. Although the tip of the greater trochanter is above the center of the femoral head, the neck shaft angle is normal (131° bilaterally). The femoral head extrusion index is normal (16% left; 18% right); the lateral center edge angle is 25° left and 23° right; the Tönnis angle is 12° left and 9° right; the anterior center edge angle is 26° left and 30° right (false profile not shown); and the hip center position is 3.0 mm left and 5.9 mm right (A). Dunn 45° radiograph in the same patient showing an alpha angle of 39° left and 40° right; head-neck offset of 7.6 mm left and 6.3 mm right; and head-neck offset ratio of 17% left and 14% right (B). Splits radiograph in the same patient showing a vacuum sign with hip subluxation as a result of lateral and inferior femoral head translation. Hip center position is 11.0 mm left and 11.2 mm right. Greater trochanteric-pelvic impingement is seen at the posterior acetabular rim (C).

Figure 2: Spectrum of osseous and soft tissue contributions to hip microinstability. Abbreviation: FAI, femoroacetabular impingement. (Image courtesy of Brayden Gerrie.)

An additional osseous reason for microinstability caused by extra-articular impingement is greater trochanteric-pelvic impingement.25 This may be evident at 2 separate locations: (1) with the limb internally rotated, the trochanter may impinge on the superior rim at approximately 12 o’clock (Figure 3); (2) with
Patients with microinstability Frequently, the pain is deep 

Figure 1C 

Additional objective evidence in pa-
tients with hip pain and suspected micro-
instability should include radiographs, with 
or without magnetic resonance imaging 
(earthography) and/or 3-dimensional com-
puted tomography (if indicated for bony 
morphology). Plain radiographs should 
include the standing anteroposterior pelvis
view, standing false profile view, supine 
Dunn (45°, 90°) or frog-leg lateral view, 
and hip splits view. The splits view requires 
a bed or table for the patient to be appro-
priately positioned. A vertical cassette is 
placed behind the patient’s pelvis, and the 
beam is aimed anterior to posterior. 

Considerations for hip splits radiographs 
include the following: The anteroposterior 
view is used. The cassette is placed as close 
as possible to the patient’s pelvis and low 
back. The patient sits upright as straight as 
possible (both legs and torso parallel in the 
sagittal plane). Limb internal rotation (toes 
pointing forward) may engage the tip of 
the greater trochanter above the 12 o’clock 
position on the acetabulum. Greater degrees 
(>0°) of hip flexion (limbs out of parallel 
vs the torso or sagittal plane) permit greater 
external rotation and posterior positioning 
of the greater trochanter. Advanced imaging 
is performed to assess for labral abnor-
malities, subchondral edema, paralabral cysts, 
tenosynovitis, bursitis, effusion, loose bod-
ies, and stress fractures.

TREATMENT
Symptomatic patients with suspected microinstability should initially undergo conservative, nonsurgical management. Physical therapy with core, pelvic, hip 
abductor, gluteus maximus, and quadri-
ceps/hamstring strengthening should be 
initiated if muscular weakness is a com-
ponent of instability. The tendency to-
ward instability (rotation and translation) 
requires increased dynamic stabilization 
(muscle strengthening) if static structures 
are the cause. This is especially true with 
abductor fatigue in patients with dysplas-
ia. Musculotendinous units (eg, iliotibial 
band, hamstring, rectus femoris) should 
be stretched if they are abnormally tight. 
Oral nonsteroidal anti-inflammatory drugs 
may be used liberally if inflammation is 
causing pain. A trial of rest and activity 
modification should also be attempted. 
If these measures are unsuccessful, re-
sponse to intra-articular injection of local 
esthetic (with or without corticosteroid) 
may help to differentiate between intra-
and extra-articular disorders.

If nonoperative treatment is unsuc-
cessful, then diagnostic arthroscopy with 
possible capsular plication to address cap-
sular redundancy may be undertaken. 
However, this should be a final option for 
patients who have capsular laxity as the 
source of microinstability. The clinician 
must be aware of all possible intra- and 
extra-articular sources of pain and ensure 
that these are addressed concurrently. 
Preoperative imaging should elucidate 
common findings, such as cam or pincer 
femoroacetabular impingement, labral 
tear, extra-articular impingement, osteo-
arthritis, capsular defect (if the patient 
had previous surgery), effusion, bursitis, 
or tenosynovitis. The technique of cap-
sular plication depends on the type of 
capsulotomy. The degree of hip flexion 
in which the interportal capsulotomy is 
closed may determine the amount of pli-
cation achieved. Although the goal is cap-
sular tightening, the surgeon must avoid 
overtightening the iliofemoral ligament. A 
double-loaded suture anchor-based com-
bined labral and capsular repair may not 
only repair the interportal capsulotomy
but also overtighten because the proximal side of the capsulotomy is not included.  

**References**


