Hip Instability in the Athlete

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Amit Nathani and Marc Safran

10.1 Introduction

The etiology of hip pain is often elusive. While the injuries associated with macro-instability of the hip following significant trauma have been well documented, the concept of more subtle instability patterns has only recently been described. Generally, hip instability is defined as extraphysiologic hip motion that causes pain with or without symptoms of hip joint unsteadiness [1]. In contrast to gross instability of the hip, microinstability is more poorly defined, has a far less dramatic clinical presentation, and lacks standardized objective evaluative criteria. The proposed pathomechanism (Fig. 10.1) of microinstability begins with subtle anatomic abnormalities in the presence of repetitive hip rotation and axial loading. This pattern of microinstability has been observed in athletes who participate in sports such as gymnastics, golf, martial arts, tennis, ballet, skating, football, and baseball [1, 2]. Alternatively, microinstability can result from inherent ligamentous laxity (i.e., Ehlers-Danlos or other hypermobility syndromes) and/or periarticular muscle weakness. Regardless of the etiology, microinstability leads to supraphysiologic motion of the femoral head relative to the acetabulum. This resultant increase in motion can lead to damage to the native labrum, now recognized as an important hip stabilizer, injury or attenuation to the capsuloligamentous complex (CLC) of the

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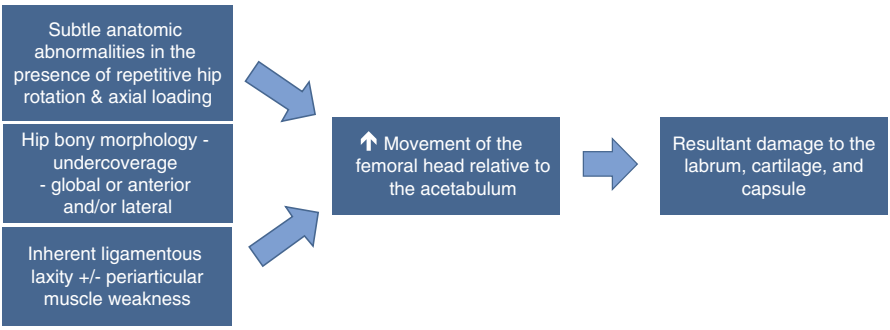

Fig. 10.1 Pathomechanics of hip microinstability

hip, or bony and chondral injury. Similar to other etiologies of labral-chondral injury, this raises significant concerns for the development of early hip degeneration and its associated morbidities.

10.2 Anatomy

The relative contributions of the bone and soft-tissue structures contribute to normal hip joint stability (Fig. 10.2). Traditionally, the hip has been considered highly constrained, relying heavily on assumed concentric bony congruence between the femoral head and acetabulum as the primary contributor to stability in all directions. More recent anatomic studies utilizing gross anatomic, imaging and finite element analysis have demonstrated more incongruence and asphericity than was previously assumed, suggesting that the role of soft-tissue structures to hip joint stability may previously have been underestimated or overlooked [3, 4]. Even under physiologic loads, dynamic MRI studies show there can be as much as 2–5 mm of translation of the hip joint center and flattening and widening of the weight-bearing surface [4, 5]. More specifically, Safran et al. demonstrated in a cadaveric model the center of the femoral head moves 3.4 mm in the medial-lateral plane, 1.5 mm in the anteroposterior plane, and 1.5 mm in the proximal-distal plane relative to the center of the acetabulum as the hip is taken through terminal motion [4]. The hip center likely translates even further during supraphysiologic motion. A recent motion capture study of 11 professional ballet dancers with morphologically normal hips showed that 4 typical ballet movements (développéá la seconde, grandé cart facial, grandé cart latéral, grand plié) caused hip center subluxation of up to 6.35 mm [6]. Recent studies have attempted to quantify the importance of the labrum, capsule, and ligamentous structures to joint stability, particularly as the hip moves through physiologic and supraphysiologic motion. It is clear that the interplay of bone and soft-tissue restraints to hip instability is complex, similar to other less congruous joints in the body like the shoulder, where microinstability is a well-established concept and damage to the bony and soft-tissue restraints is an accepted cause.

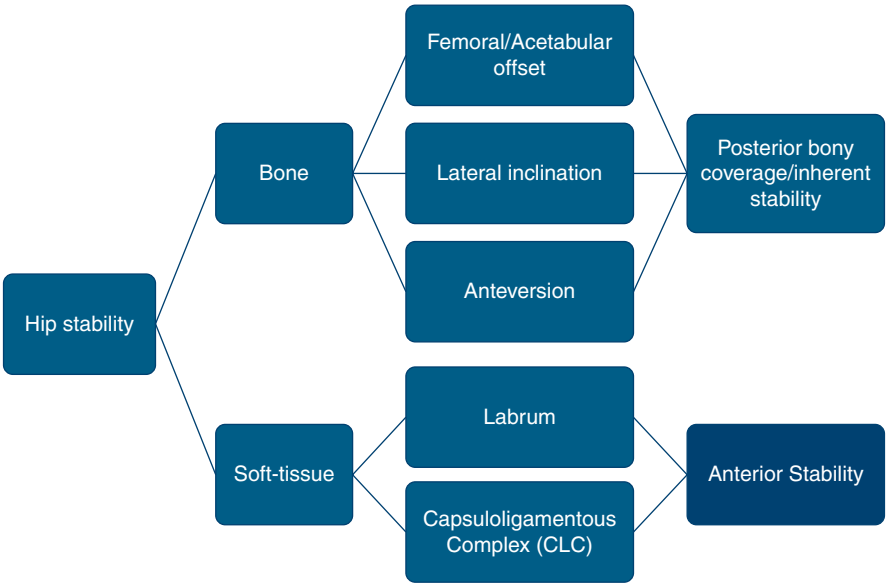

Fig. 10.2 The relative contributions of bone and soft-tissue structures to normal hip joint stability

10.2.1 Bone

Native bony morphology of the acetabulum and proximal femur contributes significantly to inherent hip joint stability, particularly in the posterior plane. The quasi-hemispherical shape of the acetabulum covers approximately 170° of the femoral head [7]. In normal individuals, the acetabulum is oriented in the pelvis in an anteverted position, with about 15–20° of anterior tilt and roughly 45° of lateral tilt, while the proximal femur is superiorly inclined on average nearly 130° (neck-shaft angle) and in about 10° of anteversion [8]. Overall, the combination of femoral and acetabular offset, anteversion, and lateral inclination results in greater posterior bony coverage. The inherent stability provided by posterior bony constraint afforded by the bony anatomy explains the ability for relative increased hip flexion and abduction compared to hip adduction and extension in normal individuals. As a consequence of inherent posterior stability, stability in the anterior plane relies more heavily on soft-tissue restraints, particularly when the hip is in positions of extension, adduction, and external rotation.

10.2.2 Ligamentum Teres

The role of the ligamentum teres in hip stability is controversial. The pyramidal structure takes origin from the transverse acetabular ligament and posterior inferior acetabular fossa and inserts onto the femoral head at the fovea capitis [1]. This non-capsular ligament tightens in a position of flexion, adduction, and external rotation and

therefore has been hypothesized to potentially add to posterior hip stability [9, 10]. Traumatic rupture has been identified and treated as a source of hip pain, but the exact contribution of the ligamentum teres to hip stability remains poorly defined [11].

10.2.3 Labrum

As the field of hip arthroscopy advanced from its infancy to current state of the art, the importance of the labrum to hip joint stresses and stability became appreciated. Early arthroscopic procedures focused on labral resection to improve mechanical symptoms associated with tearing, while current practice emphasizes and supports labral preservation (repair/reconstruction) when possible [12–15]. The labrum is in circumferential continuity with the bony acetabular rim, measuring approximately 3–8 mm in width, increasing the acetabular surface area by roughly 25% and the acetabular volume by approximately 20% [16]. The tissue characteristics and increased surface area function to distribute joint stresses during loading. Additionally, the labrum functions to maintain negative intra-articular pressure by forming a suction seal between the central and peripheral compartments. Crawford and colleagues showed 60% less force was required to distract the hip in the presence of a labral tear, demonstrating its importance to native hip stability [16–20].

10.2.4 Capsuloligamentous Complex (CLC)

The capsuloligamentous complex of the hip joint plays an important role in hip stability. Approximately 60% of the hip capsule is comprised of and reinforced by named ligaments, which represent discreet capsular thickenings. The circular zona orbicularis wraps around the femoral neck at the narrowest point of the capsule [21]. It can be visualized intra-articularly as a band of tissue encircling the femoral neck and has been shown to be an important restraint to femoral head distraction in the axial plane [22]. Three longitudinal capsular ligaments—the iliofemoral ligament (ILFL), pubofemoral ligament (PFL), and ischiofemoral ligament (ISFL)—form a helical structure around the femoral head and attach onto the acetabulum just proximal to the labrum (Fig. 10.3). The ILFL is the strongest of the longitudinal ligaments and forms an inverted Y position with a single proximal attachment at the base of the anterior inferior iliac spine and two distal attachments. The medial arm runs to the level of the lesser trochanter, while the lateral arm inserts on the anterior prominence of the greater trochanter. The ILFL limits external rotation with the hip in flexion and limits both internal and external rotation with the hip in extension [23, 24]. Additionally, Myers showed that anterior femoral head translation is primarily limited by the ILFL, with the labrum acting as a secondary restraint [25]. The PFL functions as a sling, originating on the anterior acetabular rim and wrapping posteroinferiorly around the femoral head. Its distal insertion blends with the medial arm of the ILFL and distal ISFL. It similarly limits external rotation with the hip in extension. The ISFL originates on the ischial acetabular margin and runs superolaterally to the base of the greater trochanter, serving two functions: limiting internal

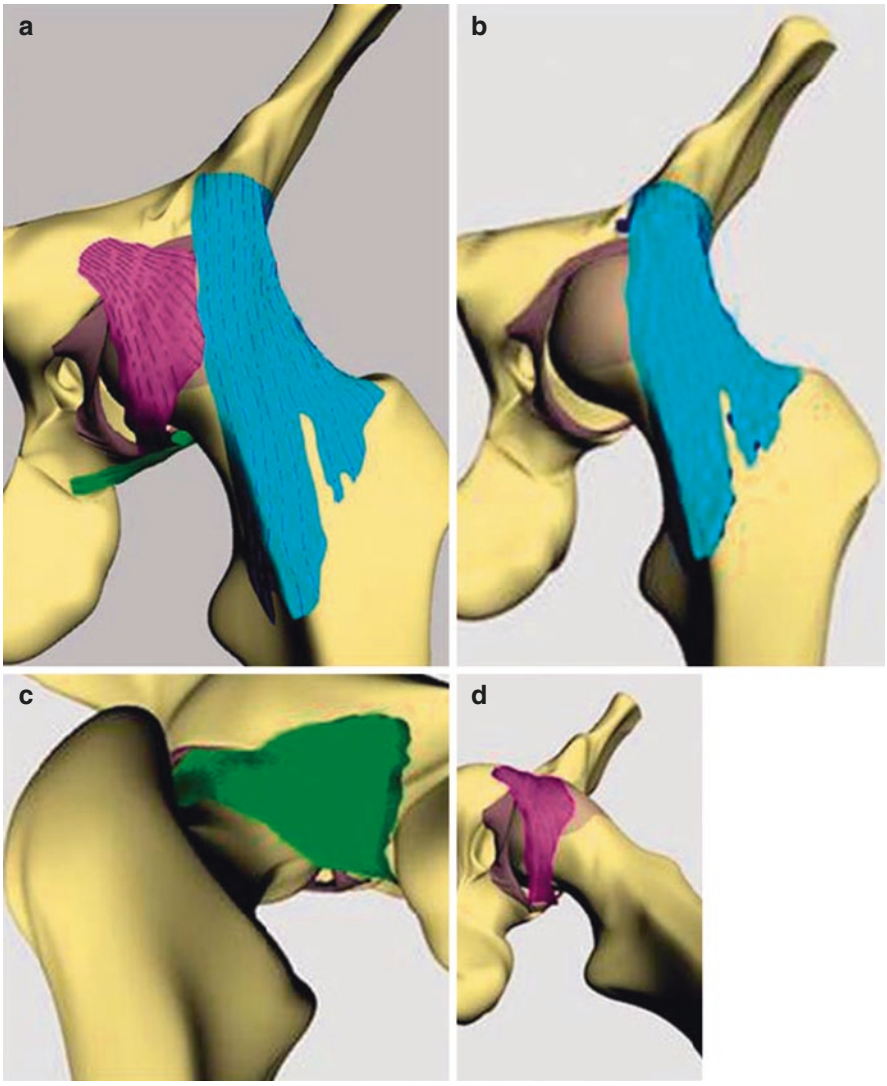

Fig. 10.3 The capsuloligamentous complex (CLC) of the hip joint. (**a**) The helical structure of the CLC, (**b**) ILFL, (**c**) PFL, (**d**) ISFL. Reprinted with permission of [59]

rotation in both hip flexion and extension as well as limiting posterior femoral head translation. The combination of the ILFL, PFL, ISFL, and zona orbicularis forms the so-called hip "screw-home" mechanism. In the potentially unstable position of hip extension, the CLC twists, tightens, and compresses the femoral head into the acetabulum. During flexion, adduction, and internal rotation, the CLC untwists and loosens, providing less soft-tissue constraint in the more inherently stable position [24]. Attenuation of the CLC due to underlying collagen disease, repetitive microtrauma during supraphysiologic motion, or iatrogenic injury has serious implications to hip stability.

10.2.5 Dynamic Stabilizing Factors

Several dynamic factors contribute to hip stability including adhesion-cohesion, negative intra-articular pressure, and periarticular muscle contraction. Seventeen muscles cross the hip joint and during contraction increase joint reactive forces and thus hip stability, by compressing the femoral head into the acetabulum. Anterior femoral head translation is also additionally resisted by the anatomic location of the iliopsoas musculotendinous unit.

10.3 Etiology

The etiology of hip microinstability is diverse, but can generally be divided into six categories: (1) bony abnormalities, (2) connective tissue disorders, (3) post-traumatic, (4) idiopathic, (5) repetitive microtrauma (athletics), and (6) iatrogenic (Fig. 10.4). Bony abnormalities such as acetabular dysplasia and, more recently, certain patterns of FAI can predispose to microinstability. A spectrum of anatomic changes are typical in developmental dysplasia of the hip (DDH), including a shallow acetabulum with lack of anterolateral coverage, increased acetabular tilt, and increased femoral and acetabular anteversion. In mild cases, this type of morphology predisposes to anterior microinstability while in severe cases can lead to hip subluxation and even frank dislocation [26]. The aforementioned soft-tissues responsible for anterior stability see increased stress, with subsequent labral and CLC damage over time [27]. In FAI, both Cam- and Pincer-type morphologies can lead to microinstability. Impingement of a CAM deformity against the acetabular rim during terminal motion can lever the head out of the socket (Fig. 10.5). While

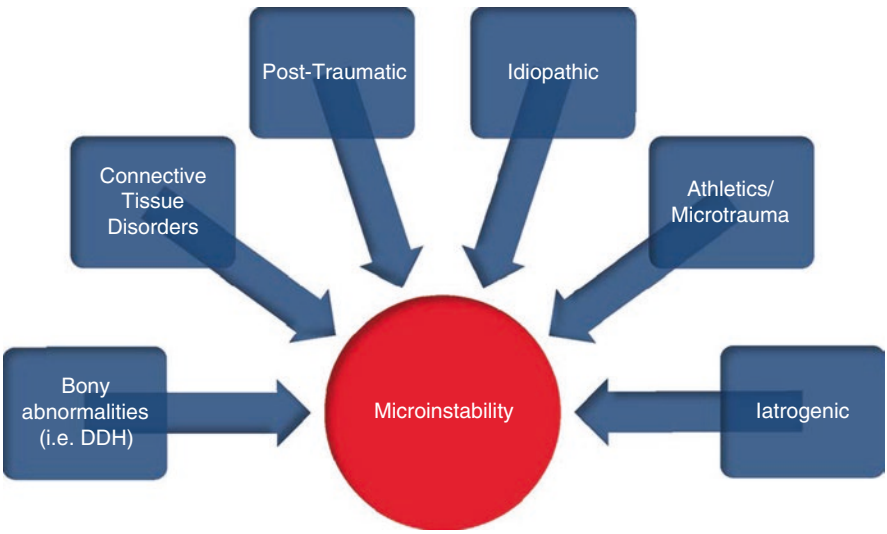

Fig. 10.4 Etiology of hip microinstability

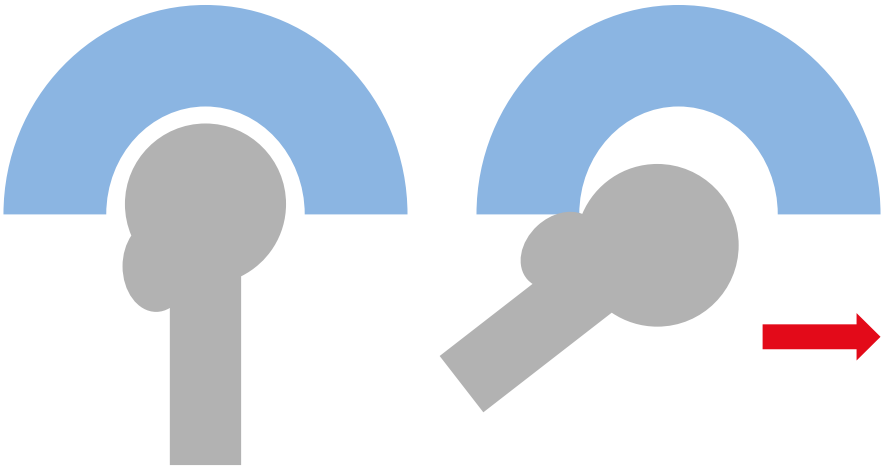

Fig. 10.5 FAI (Cam lesion) showing mechanism of microinstability

dysplasia is commonly thought of as a problem stemming from "undercoverage" of the femoral head, a Pincer-type deformity or "overcovered" head from deformities such as coxa profunda or severe acetabular retroversion can similarly lead to symptomatic posterior instability through a common lever-type mechanism. A recent systematic review showed high rates of FAI morphologic characteristics (74% CAM, 64% Pincer) in patients with symptomatic hip microinstability [28]. While the quality of literature included only Level III and Level IV research, three of the four included studies utilized real-time visualization (dynamic confirmation) of FAI-induced hip subluxation.

The second category of hip microinstability stems from underlying connective tissue disease such as Ehlers-Danlos syndrome, Marfan's syndrome, Down's syndrome, or other hypermobility syndromes. Regardless of the etiology, these disorders typically present with a spectrum from extreme to subtle joint laxity. The CLC and other soft tissues typically are attenuated, allowing for microinstability in hip positions less reliant on inherent bony stability.

Post-traumatic microinstability of the hip can occur following a high-energy mechanism (MVA) or sports injury [29]. While frank dislocation and macroinstability are well-described events in the trauma literature, post-traumatic microinstability is less clearly defined. Generally speaking, hip subluxation/dislocation events are considered stable when they occur in the absence of an associated fracture. While immediate surgical stabilization is often not necessary, residual laxity of the hip joint can occur due to soft-tissue injury to the ILFL, ligamentum teres, and chondral-labral surfaces. Often, instability in this acute setting is only observed during fluoroscopic examinations under anesthesia.

Idiopathic microinstability represents a subset of patients without a clear etiology. Often, these patients present with several possible contributing factors, including mild acetabular dysplasia not meeting radiographic criteria, subclinical ligamentous laxity (Beighton 4–5), or subtle FAI morphology [1].

While an athlete is subject to microinstability from any etiology, microtrauma represents a specific subset of patients in which the repetitive motion of sport causes underlying soft-tissue damage or attenuation leading to instability. This pattern of microinstability has been observed at the extremes of motion in sports that require repetitive hip rotation and axial loading, such as dance, golf, and gymnastics [1]. Often, subclinical laxity is advantageous for the athlete, allowing supraphysiologic hip motion; however it can also place them at higher risk for hip injury and instability. Impingement and hip subluxation are observed frequently in ballet movements, even in morphologically normal hips. Charbonnier et al. showed that four specific ballet movements were associated with a high frequency of impingement and hip subluxation. Furthermore, the locations of impingement correlated with radiologically diagnosed damaged zones in the labrum [6, 30].

Iatrogenic hip microinstability is an evolving and controversial topic. Catastrophic failure (dislocation) following hip arthroscopy has been reported in the literature several times; however given variable arthroscopic techniques for capsular management, identifying and generalizing the etiology is challenging [31–33]. Some have postulated that partial division of the ILFL during routine hip arthroscopy without repair may be the cause of hip subluxation due to increased femoral head translation in neutral flexion-extension and rotation [25, 34, 35]. During routine arthroscopy, the anterior and anterolateral portals are often connected (interportal capsulotomy). The interportal capsulotomy cuts the ILFL by necessity, and by extending the capsulotomy distally in a "T" fashion, more of the ILFL is divided [21]. Several biomechanical studies have investigated the consequence of ILFL division and have shown increased hip external rotation and distraction as more of the ligament is sectioned [36, 37]. Recently, Frank and colleagues showed improved outcomes after hip arthroscopic surgery in patients undergoing T-capsulotomy with complete repair versus partial repair (T-capsulotomy repaired and interportal capsulotomy left open) [38].

10.4 Diagnosis

In the absence of significant bony abnormalities or underlying connective tissue disease, the diagnosis of hip microinstability can be challenging. In contrast to macroinstability of the hip, microinstability is more poorly defined and lacks standardized objective evaluative criteria. Often, the presentation is quite subtle, and the treating clinician must have a strong clinical suspicion based on history, physical examination, and imaging. Ultimately, examination under anesthesia and other intraoperative findings can help confirm the diagnosis.

10.4.1 History

Pain is the primary complaint of patients with hip microinstability, though less commonly apprehension or a sense of giving way is also reported. Exacerbating factors, such as axial loading, rotation, and other sport-specific activities, can give the

examiner clues to the underlying diagnosis. Most patients do not report a specific trauma or inciting event, instead describing an insidious onset with gradually worsening discomfort. It is very important to elicit any previous hip injuries or previous surgical procedures. Iatrogenic instability is becoming a more commonly recognized etiology. In patients requiring revision hip arthroscopy, McCormick and colleagues reported 78% had radiographic evidence of capsular and ILFL defects on magnetic resonance arthrography [39]. Another study by Philippon et al. similarly showed that one in three patients undergoing revision hip arthroscopy required capsulorrhaphy at the time of revision, suggesting undiagnosed hip microinstability as a potential cause for revision [40].

10.4.2 Physical Examination

A thorough physical exam is crucial in the diagnosis of suspected hip microinstability. Intra-articular pain is typically described as groin, buttock, thigh, or in the classic C-distribution. It is important to rule out other sources of pain that can be referred to the hip from common dermatomal/myotomal origins and thus confused with hip pain, including those from the lumbar spine, abdomen, and knee. Palpation, strength, and range of motion testing should be a part of every hip examination, as well as a general screening for ligamentous laxity using the Beighton scoring system. More specific laxity testing for the hip is observation of internal or external rotation of the hip greater than 60° in either direction and hip flexion greater than 150° and/or observation that if the leg is placed in a figure-of-four position, the knee joint line falls to less than 3 in. from the examination table.

The goal of diagnosing hip microinstability is to reproduce pain or apprehension the patient feels utilizing a specific set of provocative testing. Six specific maneuvers have been described to evaluate hip stability: (1) hyperextension anterior apprehension test, (2) abduction-extension-external rotation test, (3) prone external rotation test, (4) log roll test, (5) axial distraction test, and (6) posterior apprehension test. Hoppe et al. recently published on the diagnostic accuracy of the first three of the aforementioned tests and showed a sensitivity of 71% and specificity of 85% for the hyperextension anterior apprehension test, a sensitivity of 81% and specificity of 89% for the abduction-extension-external rotation test, and a sensitivity of 33% and specificity of 98% for the prone external rotation test. When all three tests were positive, the likelihood of intraoperative confirmation of hip microinstability was 95% [41]. Those three tests are described in Fig. 10.6. The log roll test is performed while the patient is supine and the knee is in extension. The examiner first fully internally rotates the foot, then removes their hand, and allows the foot to passively fall back into external rotation. Asymmetric external rotation compared to the contralateral side suggests anterior hip laxity (especially if the foot-table angle <20°). The axial distraction test is similarly performed in a supine position with the knee of the examiner abutting the ischium of the extremity being examined. The hip and knee are flexed to about 30° while an axial force is placed on the hip. Any sense of hip "toggling" or reproduction of apprehension or pain is considered a positive

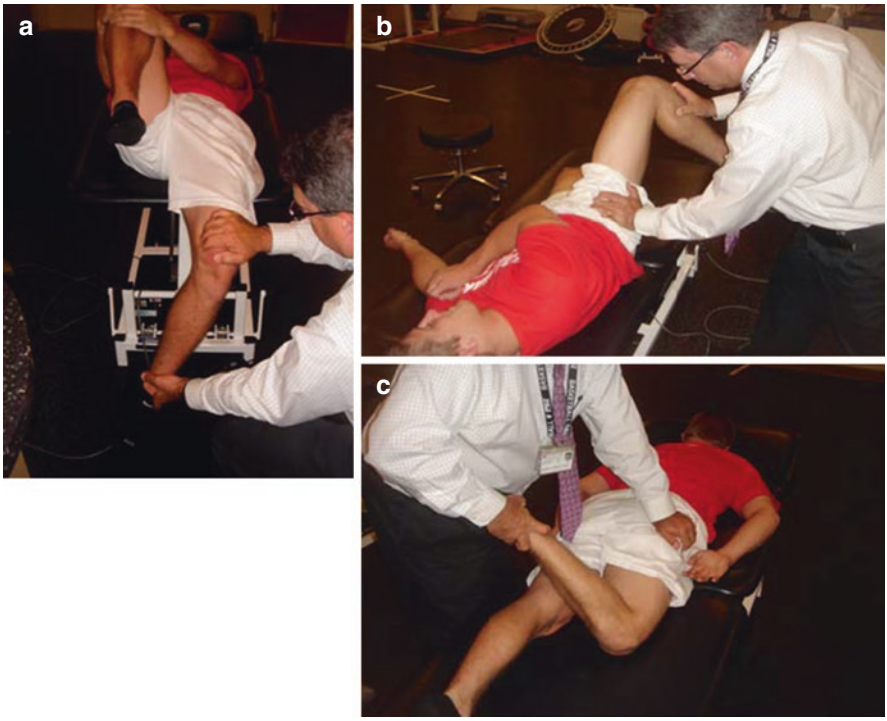

Fig. 10.6 Provocative maneuvers for hip instability: (**a**) anterior apprehension test, (**b**) abduction-extension-external rotation test, (**c**) prone external rotation test

result. The posterior apprehension test is also performed in a supine position. The examiner flexes the patient's hip to 90° while also adducting and internally rotating the hip and placing a posteriorly directed force on the knee. Any sense of pain or apprehension is considered a positive result.

10.4.3 Imaging

Imaging begins with high-quality plain radiographs, including a supine AP pelvis, lateral radiograph (cross-table or Dunn view), and false-profile views. X-rays should be scrutinized for degenerative changes, trauma, prior surgery, FAI, and morphological clues about the shape and orientation of the proximal femur and acetabulum. A lateral center-edge angle of less than 20–25° on the AP pelvis and on the false-profile view is highly suggestive of acetabular dysplasia, as does an acetabular roof inclination (Tonnis angle) of greater than 10° upsloping. Acetabular version is important to assess as it is associated with hip pathology, with anteversion correlated with DDH and retroversion related to Pincer-type FAI. However, assessing version on an AP radiograph relies upon the relationship between the anterior and

posterior walls, but cannot capture the volume (quantitative) of the socket, which is often abnormal in cases of dysplasia or global overcoverage [42]. While normal version lies between 12 and 20° of anteversion, this parameter changes with both pelvic tilt (increased tilt reduces version) and changes in the supine to standing position (pelvic tilt decreases in the standing position) [43, 44]. A crossover sign, posterior wall sign, and ischial spine sign are qualitative indicators of acetabular version. On an AP radiograph, when the contour of the anterior wall lies lateral to the corresponding point of the posterior wall, the presence of retroversion or focal anterosuperior overcoverage is present. The sensitivity and specificity of the crossover sign are 96% and 95%, respectively [45]. The posterior wall sign is seen when the posterior wall of the acetabulum lies medial to the center of the femoral head, indicated acetabular retroversion or global acetabular dysplasia [46]. If the posterior wall sign is present, but the crossover sign is absent, this denotes a so-called "low-volume" acetabulum without version abnormalities [47]. The ischial spine sign is seen on an AP radiograph when the ischial spine lies medial to the iliopectineal line [48]. When all three (crossover, posterior wall, and ischial spine) signs are present, the acetabulum is globally retroverted [47, 49]. More recently, Safran and Packer described the "cliff sign"—a radiographic finding where there is a steep drop-off of the lateral femoral head-neck junction and its strong correlation to microinstability. On an AP pelvic XR, a perfect circle is created around the center of the femoral head. If the lateral femoral head does not completely fill the perfect circle, a positive "cliff sign" was denoted (Fig. 10.7). In their study of 96 patients, 74% of those with a positive cliff sign had microinstability, while only 7% with microinstability did not demonstrate a cliff sign (unpublished, presented at ISAKOS 2017). Given the

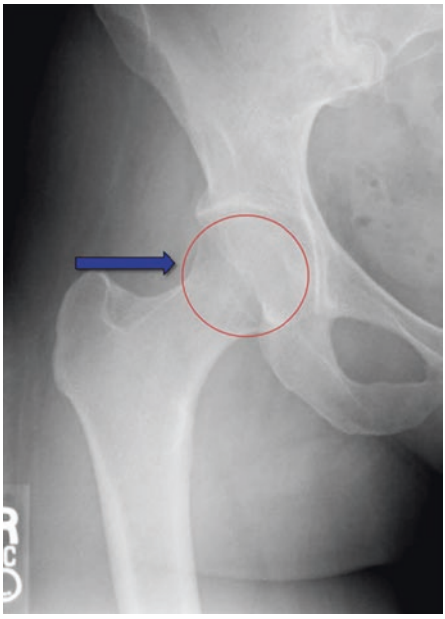

Fig. 10.7 "Cliff" sign

difficulty in diagnosis, another study recently reported an additional radiographic parameter associated with hip instability in borderline dysplasia patients. Wyatt and colleagues described the FEAR (femoral-epiphyseal acetabular roof) index—formed by two lines that connect the acetabular roof inclination and the femoral head physeal scar. This angle, when positive (acetabular roof inclination steeper than physeal scar), was associated with instability [50].

MRI is often utilized in cases of intra-articular hip pain to evaluate the soft-tissue structures, including the chondral surfaces, labrum, and capsule. Magnetic resonance arthrogram (MRA) can be even more useful in the work-up of suspected hip microinstability, as the gadolinium dye distends the hip capsule. McCormick et al. utilized MRA and showed a high prevalence of capsular defects following hip arthroscopy [39]. Magerkurth and colleagues retrospectively reviewed preoperative MRAs and noted that hip joint laxity was associated with a widened hip joint recess of >5 mm and a thinned lateral capsule <3 mm adjacent to the zona orbicularis [51].

Ultimately, the patient's intraoperative findings provide confirmation of the diagnosis of hip microinstability, beginning before the arthroscope is even introduced. The ease of distractibility is a very reliable indicator of instability. Often, body weight traction alone provides significant distraction. After removal of negative intra-articular pressure, traction is released, and the hip joint is reassessed fluoroscopically and often will show a femoral head that remains incompletely reduced or lateralized relative to the acetabulum (Fig. 10.8a, b). Intraoperative findings consistent with microinstability are direct anterior or direct lateral labral injury compared with the classic anterolateral labral damage commonly associated with FAI [52]. Other frequent findings include adjacent chondral wear that is typically shallow (1–3 mm) and worn rather than delaminated, central femoral head chondral injury, and tearing and hypertrophy of the ligamentum teres.

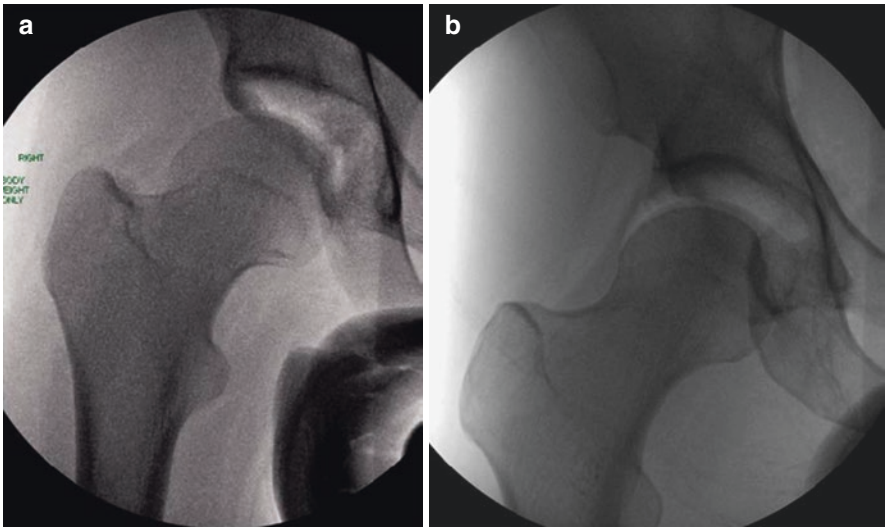

Fig. 10.8 Intra-operative fluoroscopy of (**a**) hip distracted only using body weight to remove gross traction and (**b**) after removal of negative intra-articular pressure and traction, the hip is incompletely reduced

10.5 Treatment

The treatment of hip microinstability is controversial and not yet well defined. It is becoming more accepted as a cause of hip disability in the athlete, but management algorithms backed by long-term data are nonexistent. Instead, at this time there is a reliance upon some of the pioneers and experts of the field who treat this entity more commonly. Parallels are drawn from the shoulder instability literature, and typically non-operative management is attempted first. Conservative care focuses on modifiable factors, including activity modification, anti-inflammatories, and especially periarticular hip muscle (dynamic) and core strengthening. While there is no literature to date documenting or comparing operative and non-operative treatment, it is the author's (M.S.) experience that a percentage of patients do improve with therapy alone and are able to return to regular activities. However, a significant number of patients do require surgical intervention.

Surgical treatment should focus on the underlying etiology for instability. In the case of severe bony abnormalities (dysplasia, acetabular retroversion), redirectional osteotomies should be considered. In the absence of significant bony abnormalities, the focus of treatment is on the soft tissues responsible for secondary stability, including the labrum and CLC. Both open and arthroscopic techniques to reduce capsular volume have been described, but comparative studies are lacking [53–55]. As hip arthroscopy technique has advanced, arthroscopic management is the primary method of addressing hip microinstability. During treatment of microinstability, the treating surgeon should address associated intra-articular pathology, including labral repair when possible, and consideration for labral reconstruction when non-salvageable, as the labrum is an important hip stabilizer [56]. Arthroscopic capsular repair or plication is recommended for patients with capsular redundancy, symptomatic capsular laxity, and any patient with underlying connective tissue disorder or generalized ligamentous laxity undergoing arthroscopic treatment for any other reason (FAI, labral tear, chondral injury). Philippon reported on the use of arthroscopic thermal capsulorrhaphy in 12 patients with hip instability [7]. While he reported no complications and good results, concern over possible thermal capsular necrosis and chondrolysis, as seen in the shoulder, has led to the development of alternative procedures. Arthroscopic suture capsular plication is a technically demanding procedure, but several authors have no reported good results with a variety of techniques. Larson and colleagues showed 90% good to excellent results in 16 hips of patients with Ehlers-Danlos syndrome with suture capsular plication [57]. Domb et al. published on a series of patients with hip microinstability and borderline hip dysplasia. His technique involved shifting the inferior capsule proximally (shortening the ILFL) and showed favorable results at 2 years post-op; however patients in this cohort lost approximately 10° of hip external rotation [58]. Most recently, Kalisvaart and Safran reported on 31 consecutive patients treated for microinstability of the hip with labral surgery and suture capsular plication. In their series, all patients were women, 71% did not have dysplasia, 29% had mild dysplasia (CEA 18–24°), and no bony work was done. Their technique utilized suture plication through the capsular "bare area" between the ILFL anteriorly and the ISFL posteriorly (Fig. 10.9). All patients had improvement in symptoms, with 100% return to sport in the athletes treated and without significant loss of motion.

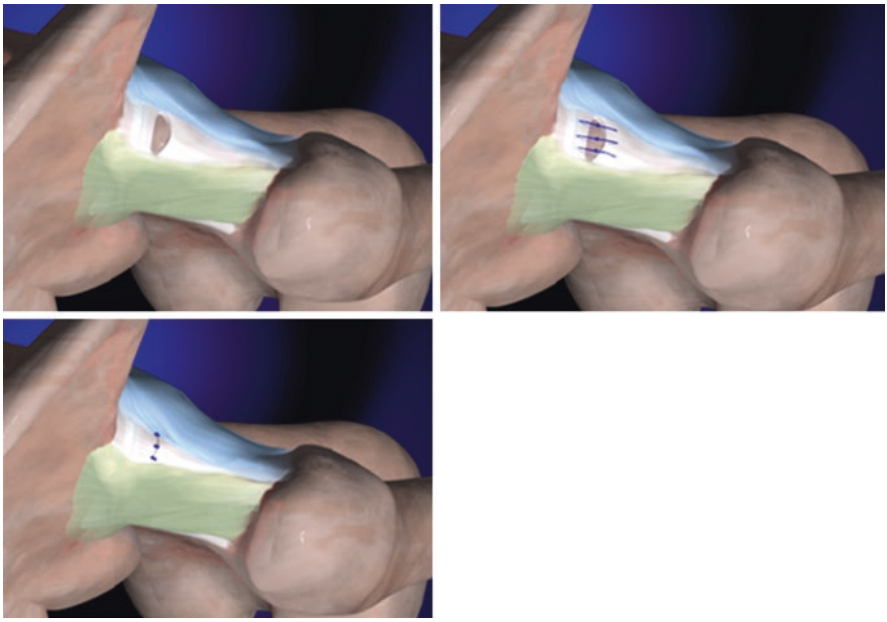

Fig. 10.9 Arthroscopic capsular plication technique

10.6 Summary

Hip microinstability is an emerging concept that is gaining acceptance as a cause for hip disability. While there are many etiologies, in the absence of significant bony abnormalities or underlying connective tissue disorders, athletes with repetitive motion can cause microtrauma or attenuation to the secondary stabilizers of the hip (labrum and CLC). Specific sports with rotation and axial hip loading are at higher risk, as well as athletes that attain supraphysiologic motion despite normal bony morphology. Microinstability is a difficult diagnosis as it lacks standardized evaluative criteria. Its clinical presentation is less dramatic than macroinstability; thus the treating clinician should have a high index of suspicion. Several well-described provocative maneuvers help identify instability reliably, but confirmation of the diagnosis is an intraoperative finding. Once identified, both nonsurgical and surgical management can be effective. If conservative management fails, arthroscopic capsular plication techniques have shown promising short- and midterm outcomes.

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