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Introduction

The hip joint provides an articulation that allows relative angulation between the upper body and the lower limbs. This is critical for achieving both an upright posture and normal ambulation. Most of the weight of the body is supported by the hip joint, especially in single leg support, and the muscles controlling the hip act relatively close to the center of the joint. Thus, the forces acting on the femoral head and the acetabulum are considerable and have significant consequences in terms of skeletal health and musculoskeletal function [1, 2]. Over the last hundred years, this has led to the emergence of a formal field of study, Musculoskeletal Biomechanics, which has made valuable contributions to our understanding of the hip. These have included new methods

for evaluating joint function and understanding pathologic conditions, alternative surgical approaches for hip preservation and reconstruction, and the development of methods for measuring joint forces and moments developed in vivo. The application of scientific principles to the study of the hip has also provided insight into morphologic factors compromising hip motion, including acquired abnormalities (e.g., post-traumatic deformities, Perthes disease, slipped capital femoral epiphysis [SCFE]), developmental pathologies (e.g., congenital dislocation of the hip [CDH] and developmental dysplasia of the hip [DDH]), and abnormalities of unknown origin (e.g., cam deformity of the femoral head-neck junction and pincer deformities of the acetabular margin). Ongoing investigations of the biomechanics of the capsule, labrum, and femoroacetabular impingement (FAI) should further reduce the proportion of patients that are labeled as having “idiopathic” coxarthrosis.

Much of our recent knowledge derived from clinical and radiographic studies has confirmed that hip symptoms and even joint degeneration are not automatically predestined by abnormalities of anatomy or bony morphology [3–6]. Indeed, detailed analysis of cadaveric specimens confirms that dysmorphic conditions and soft tissue lesions are far more common than symptomatic impairment of hip function [7–9]. This observation leads us to study the interaction between the demands that function places on the hip, whether through sport, vocation, or lifestyle, and the capacity of the joint to function asymptotically. In this chapter we will discuss the external demands placed upon the hip during function and the mechanisms for dissipating the loads developed across the joint, in both the normal joint and in the presence of structural and pathological abnormalities.

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Forces Acting Across the Hip Joint

During functional activities, the magnitude and direction of the contact force developed between the head of the femur and the weight-bearing surface of the acetabulum is primar-

ily dictated by the muscle forces needed to stabilize the hip and generate motion. Data collected using instrumented hip prostheses show that hip joint forces vary extensively, ranging from 2.1 to 4.3 times body weight (BW) during gait [10–13] and 2.3–5.5 BW during stair-climbing [10–12], to values in excess of 8 BW during accidental incidents of stumbling (Figs. 7.1 and 7.2) (Table 7.1) [11, 14].

The net (resultant) force applied to the head of the femur is directed both laterally and inferiorly during the stance

phase of the gait cycle and changes direction from posterior at heel strike to anterior at toe off. Although the predominant component of the joint reaction force is directed down the shaft of the femur during gait, with peak values ranging from 1.4 to 4.1 BW, substantial forces are also observed in both the mediolateral (0.4–1.7 BW) and anterior-posterior directions (0.2–1.0 BW), especially at points of greatest flexion during the gait cycle or during the transitions between single- and double-limb support. The torsional and shear forces

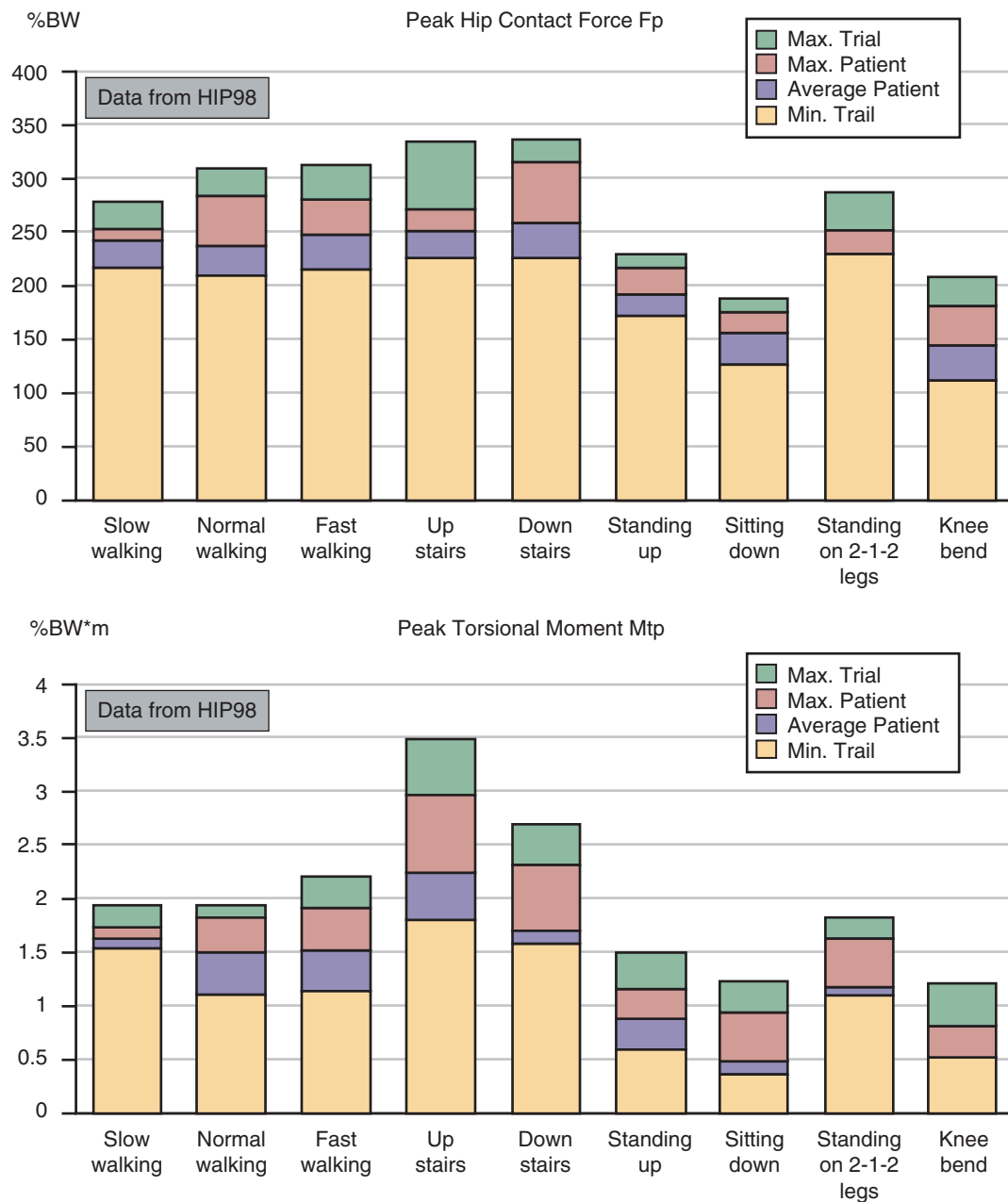


Fig. 7.1 Hip contact force data (*top*) and torsional moments (*bottom*) for a range of nine different activities recorded using instrumented hip prostheses. Hip forces are plotted in units of percentage of body weight (%BW), while moments are plotted in units of %BW×height of each

subject in meters [%BW.m]. From Stal et al., *Biomechanics of the Natural Hip Joint*, In: *Surgery of the Hip*, D.J. Berry and J.R. Lieberman, eds. Volume 2. 2013, pp 5–13. Copyright Elsevier 2013, reprinted with permission

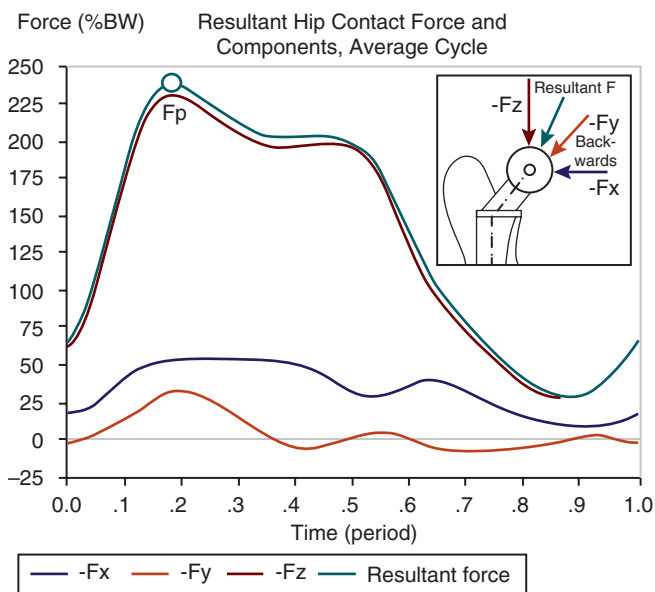


Fig. 7.2 Typical variation in the hip contact force and its components during an average gait cycle, commencing with heel strike. From Stal et al., *Biomechanics of the Natural Hip Joint*, In: *Surgery of the Hip*, D.J. Berry and J.R. Lieberman, eds. Volume 2. 2013, pp 5–13. Copyright Elsevier 2013, reprinted with permission

Table 7.1 Summary of values of the peak joint reaction force reported by Bergmann and coworkers during different investigations using instrumented hip prostheses

Activity	Typical peak force (BW)	Number of patients	Months post-op	References
Walking, slow	1.6–4.1	9	1–30	[10, 11]
Walking, normal	2.1–3.3	6	1–31	[10]
Walking, fast	1.8–4.3	7	2–30	[10, 11]
Jogging/running	4.3–5.0	2	6–30	[10, 11, 21]
Ascending stairs	1.5–5.5	8	6–33	[11]
Descending stairs	1.6–5.1	7	6–30	[11]
Standing up	1.8–2.2	4	11–31	[10]
Sitting down	1.5–2.0	4	11–31	[10]
Standing/2-1-1 legs	2.2–3.7	3	11–14	[10]
Knee bend	1.2–1.8	3	11–14	[10]
Stumbling	7.2–8.7	2	4–18	[12]

generated within the proximal femur during many routine functional activities are also significant and vary with activity [11–13, 15, 16]. For example, during stair-climbing the anterior-posterior component of the joint force reaches 20–25% of the frontal plane load [12], whereas, the peak twisting moment and first peak contact force are decreased by 18% and 14%, respectively [16]. In contrast, the axial torques recorded during descending stairs and walking are of similar magnitude [11, 12, 17].

The magnitude of the joint reaction force and its direction with respect to the skeleton is affected by numerous factors,

including the specific activity, the age, gender, height, body weight and gait velocity of the individual subject [17–21], and environmental factors such as the nature of any footwear and the surface upon which the activity is performed [22, 23]. In this chapter we will present a summary of the latest information on the mechanical environment of the hip joint, the kinematics of the joint during normal function, the role of tissues controlling joint motion, and the mechanics of pathological conditions, such as FAI, hip dysplasia, and chronic hip laxity.

Load Transfer Across the Hip Joint

The congruency of the joint surfaces allows for more even distribution of contact forces during loading [24, 25]. Under light loads, contact between the joint surfaces is limited to the anterior and posterior lunate surfaces. Increasing load results in a greater contact which extends to the superior, anterior, and posterior regions of the joint [25, 26]. Studies have shown that the area of joint contact varies greatly between individuals and changes in response to joint angle and load [24–26]. During gait, contact area is least during flexion when the joint load is low, as observed during terminal swing and heel strike. The area of joint contact increases as the hip moves through the stance phase of gait, peaking at terminal extension [24]. Maximum acetabular pressures of 3.3 MPa occur during midstance of gait and are located on the superior and posterior lateral roof. Activities which require higher ranges of motion reduce the amount of contact area and result in greater acetabular pressures. The greatest pressures occur during standing and sitting where pressures of 9–15 MPa have been recorded over the apex of the femoral head and the superior and posterior aspects of the acetabulum, which are sites of degenerative changes commonly reported in cadaveric studies [26, 27].

The Kinematics of the Normal Hip

The hip joint is a multiaxial joint formed by the articulation between the concave acetabulum and convex femoral head. Despite its sturdy joint capsule which limits extreme motion and prevents frank dislocation, the hip joint allows a great deal of mobility [28]. Joint motion is greatest in the sagittal plane, as the femur flexes and extends around a left-right axis [29]. Active hip flexion is greatest with the knee flexed (110° – 120°) rather than extended (90°), due to the effects of hamstring tension [2, 29–31]. For Caucasian populations, typical values for single-plane motions are 120° for flexion, 20° for extension, 45° for abduction, 30° for adduction, and 40° for internal and external rotation [32–35] (Tables 7.2 and 7.3). In non-Western subjects accustomed to squatting for

Table 7.2 Difference in mean active range of motion (in degrees) for ages 25–39 years compared with ages 60–74 years by sex-race groups

Motion	All subjects	White men	White women	Black men	Black women
<i>Hip flexion</i>					
Ages 25–39 years	122°	123°	123°	115°	116°
Ages 60–74 years	118°	118°	119°	118°	106°
<i>Hip extension</i>					
Ages 25–39 years	22°	22°	22°	19°	17°
Ages 60–74 years	17°	17°	16°	16°	12°
<i>Hip abduction</i>					
Ages 25–39 years	44°	46°	44°	41°	38°
Ages 60–74 years	39°	39°	40°	38°	37°
<i>Hip internal rotation</i>					
Ages 25–39 years	33°	34°	33°	32°	27°
Ages 60–74 years	30°	31°	29°	27°	25°
<i>Hip external rotation</i>					
Ages 25–39 years	34°	33°	36°	32°	32°
Ages 60–74 years	29°	27°	32°	27°	28°

From Roach et al. [34]

Table 7.3 Range of motion of the hip during functional activities

Motion	Squatting (heels down)	Squatting (heels up)	Kneeling (dorsiflexed)	Kneeling (plantar-flexed)	Sitting cross-legged
Flexion	95.4±26.2	91.3±17.1	73.9±29.4	58.8±9.7	85.4±34.2
Abduction	28.2±13.9	31.7±11.2	25.3±15.3	27.6±12.5	36.5±15
External rotation	25.7±11.8	33.7±12.7	28.1±12.8	34±14.9	40.3±18.4

All values are expressed as the average±standard deviation; units are degrees. From Hemmerich et al. [36]

long periods, 130° of hip flexion is observed during a full squat and 90°–100° in a cross-legged position; hip external rotation has ranged from 5° to 36° for a full squat and 35°–60° while sitting cross-legged, and hip abduction has ranged from 10 to 30° for a full squat and 40°–45° while sitting cross-legged [36–39].

Tissues Controlling the Range of Motion of the Hip Joint

The limits of three-dimensional joint motion are determined by a complex interplay of impingement between the osseous and soft tissues of the joint and the passive restraint of structures connecting the femur to the pelvis [40]. The capsular ligaments of the hip make the largest contribution in limiting the range of motion of the hip, with a secondary contribution from the acetabular labrum. The muscles surrounding the joint contribute to the passive restraint of motion [40]. Computer modeling suggests that osseous impingement (e.g., abutting of the anterolateral head-neck junction against the acetabular rim) contributes primarily to restricting flexion, adduction, flexion combined with adduction, and flexion combined with adduction and internal rotation. Soft tissue impingement restricts abduction and abduction combined with flexion, while soft tissue restraint restricts extension and adduction through the midrange of joint flexion [41].

The Articular Surfaces

The osseocartilaginous surfaces of the femoral head and acetabulum are often thought of as being truly spherical, while in reality, both are more elongated in the direction of the neck axis, leading to an “egg-shaped” morphology (often termed “conchoidal”) [28]. The femoral head forms two-thirds of a sphere, becoming flatter where the acetabulum applies its largest load [42, 43], while the acetabulum possesses a slightly smaller diameter than the femoral head [44]. The acetabulum covers approximately 170° of the femoral head, being incomplete in the inferior portion [45]. The lack of a truly congruent articulation permits rolling and gliding between the joints surfaces, allowing for a great deal of mobility while maintaining the inherent stability of the joint [28]. This morphologic configuration also allows the lower extremity to be placed in positions requiring simultaneous flexion-extension, abduction-adduction, and internal-external rotation.

The Hip Capsule and Ligaments

During abduction and adduction, the stability of the joint is maintained by the passive restraint of the hip capsule (the capsular ligaments and the zona orbicularis) which also

keeps the hip from dislocating when loaded at the extremes of motion [41, 46, 47]. The capsule is a complex structure formed by three discrete ligaments: the iliofemoral, femoral arcuate (pubofemoral), and ischiofemoral ligaments. The iliofemoral ligament, also known as the “Y” ligament of Bigelow, is located anteriorly and restricts extension and external rotation of the joint [48–51]. The femoral arcuate ligament helps to limit abduction and external rotation. Lastly, the ischiofemoral ligament acts during flexion to limit internal rotation and adduction [48, 49]. The ischiofemoral ligament is the weakest of the capsular ligaments, which makes the joint susceptible to posterior dislocation [52]. Additionally, the zona orbicularis plays a role in preventing frank dislocation by stabilizing the axial position of the femur in the socket. The zona orbicularis is a circular condensation of the capsule which surrounds the femoral neck and appears to prevent distraction of the femoral head out of the joint [53, 54].

The Acetabular Labrum

The acetabular labrum is a fibrocartilaginous extension of the bony edge of the acetabular rim with specialized structural properties, deepening the effective depth of the acetabular socket and dramatically increasing the resistance of the joint to dislocation. In mechanical terms, labral tissue is highly anisotropic, with a preferential stiffness in the circumferential direction [55] and significant variation as a function of gender, anatomic location, and the degenerative state of the hip [56, 57].

In the normal hip, the labrum only makes a small contribution to direct mechanical support of the femoral head; though with increasing degrees of acetabular dysplasia, weight is shifted more peripherally on the articular surface and the contributions of the labrum and capsule become far more significant [58, 59]. Studies performed over the past decade strongly suggest that the primary function of the labrum is not to increase the weight-bearing surface of the hip, but rather to form a compliant seal with the articular surface of the femoral head [60, 61, 62]. The ability of the labrum to seal off the central compartment of the hip is easily seen intraoperatively during distraction or dislocation of the hip (Fig. 7.3). Moreover, laboratory studies have shown that this suction phenomenon increases joint stability and allows the joint load to be distributed more uniformly over the articulating surfaces (Figs. 7.4 and 7.5) [63–65]. Computer simulations and *in vitro* experiments [62, 64, 66] have shown that the labrum controls the rate of egress of synovial fluid during weight-bearing which allows retention of a layer of synovial fluid between the femur and the acetabulum, thus preventing direct contact of the articulating surfaces during dynamic loading. If the labrum fails to perform this function, strains

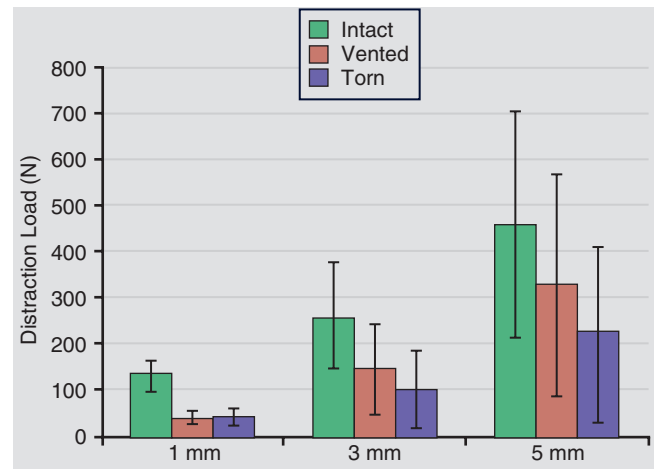


Fig. 7.3 Average load required to generate distraction of the femur with respect to the acetabular margin by distances of 1, 3, and 5 mm with the labrum intact, vented, and incised to simulate a full-thickness tear. From Stal et al., *Biomechanics of the Natural Hip Joint*, In: *Surgery of the Hip*, D.J. Berry and J.R. Lieberman, eds. Volume 2. 2013, pp 5–13. Copyright Elsevier 2013, reprinted with permission

within the cartilage matrix are substantially increased. The rate of consolidation of cartilage under load, with displacement of internal synovial fluid, has been shown to increase by up to 40 % following excision of the labrum [62]. This leads to a dramatic rise in the internal stresses within the cartilage layers and subchondral shear stresses at the bone-cartilage junction, increasing the risk of delamination [62].

Damage to the labrum through injury or pathology can compromise its sealing function, leading to displacement of the head from its normal physiologic position. This leads to increase in peak articular stresses with a shift of loading to the acetabular rim, potentially leading to accelerated erosion of cartilage and early onset of osteoarthritis. Through the combination of these factors, labral tears occur predominantly in the anterior quadrant, leading to hip instability, as well as watershed labral lesions, which ultimately can lead to degenerative joint disease [60, 61, 67, 68].

In middle-aged to elderly individuals, labral pathology arises primarily through degenerative changes accompanying aging, as evidenced by the high incidence of lesions observed in cadaveric specimens [68, 69]. In younger individuals, primarily those engaged in sporting activities, labral pathology has been attributed to repetitive microtrauma, especially during high impact sports or those involving loading of the extremity at the extremes of the motion arc [70–72]. A common observation in patients with labral injury is enlargement of the anterior junction of the femoral head-neck junction, leading to cam-type FAI [73–75]. In these cases, it has been hypothesized that the mechanism of injury is forced passage of the enlarged area of the anterior head-neck junction beneath the labrum at the extremes of the natural range of joint motion. Repetition of the abnormal loading

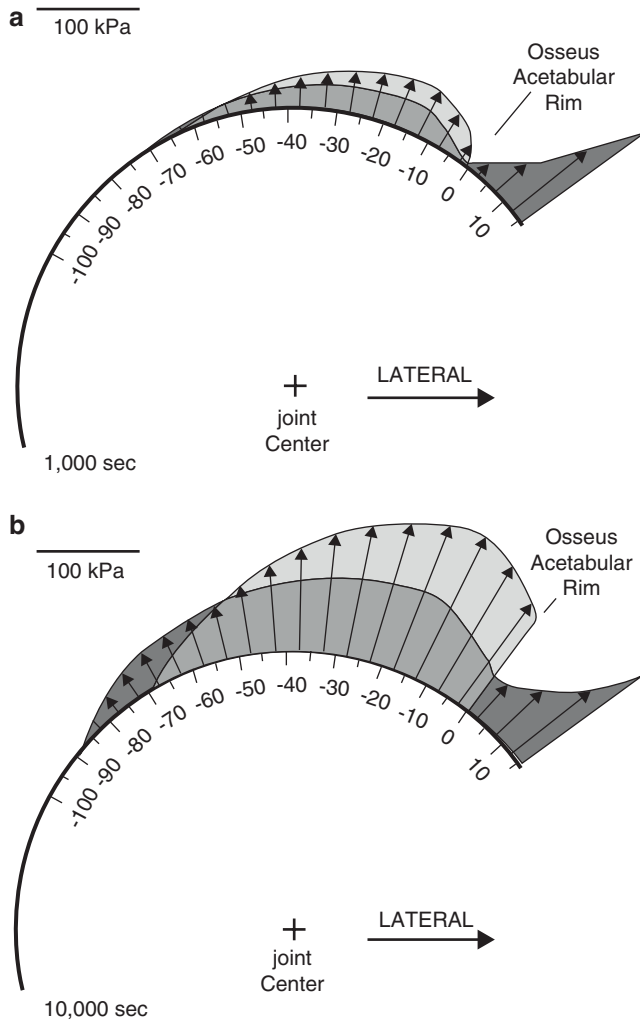


Fig. 7.4 Predicted distribution of cartilage contact stresses (solid on solid) at 1000 and 10,000 s after application of load to the hip joint in the presence of an intact labrum (dark gray) and without a labrum (light gray). From Ferguson SJ, et al. The influence of the acetabular labrum on hip joint cartilage consolidation: a poroelastic finite element model. *Journal of Biomechanics*. 2000; 33(8):953–960. Reprinted with permission from Elsevier

caused by this event is thought to lead to tensile loading of the chondro-labral junction and formation of a “watershed” lesion.

An alternate hypothesis arises from the work of Dy et al. [76] who showed that substantial tensile strains can be developed within the anterior labrum without the occurrence of impingement. In this novel combination of experimental loading and finite element analysis, maximum strains were predicted at the junction between the labrum and the acetabular rim during activities involving twisting or pivoting of the hip with tightening of the joint capsule.

Labral injury may also occur secondary to compression and shear forces imposed by the iliopsoas tendon [77]. In this situation, the site of injury is often located more anteromedially

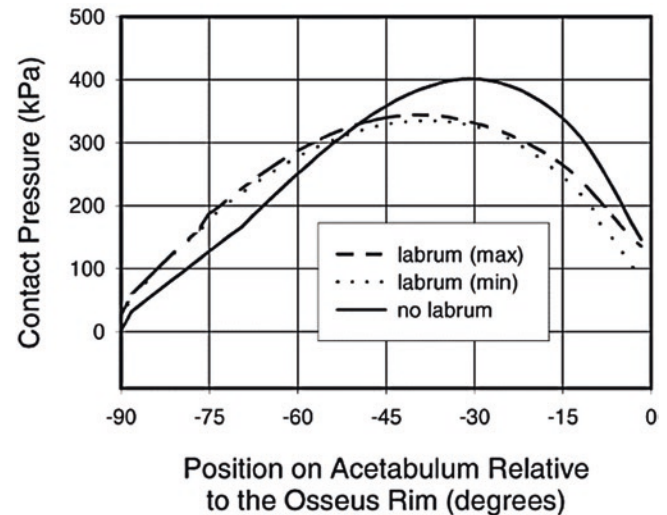


Fig. 7.5 Predicted variation in total contact pressure (interstitial fluid pressure plus solid contact stress) at 10,000 s after application of load to the hip joint, with and without an intact labrum. The dashed and dotted lines depict the maximum and minimum values of contact pressure corresponding to the extremes in assumed mechanical properties of the labrum within the finite element model. From Ferguson SJ, et al. The influence of the acetabular labrum on hip joint cartilage consolidation: a poroelastic finite element model. *Journal of Biomechanics*. 2000; 33(8):953–960. Reprinted with permission from Elsevier

ally than is seen in cases of femoroacetabular impingement [78]. Recognition of iliopsoas impingement has prompted hip preservation surgeons to repair these non-femoroacetabular impingement-induced labral injuries and to address the inciting cause, either the iliopsoas tendon or anterior inferior iliac spine and psoas groove [77, 79].

The Ligamentum Teres

The potential role of the ligamentum teres in stabilizing the hip remains a topic of debate [80, 81]. Anecdotal reports indicate that some patients obtain relief of hip symptoms following repair of acute ruptures of the ligamentum teres, although scientific data corroborating the role of this structure in enhancing hip stability is scarce [82]. It is known that the ligamentum teres is taut during hip adduction, flexion, and external rotation, positions in which the joint is least stable, which demonstrates the potential contribution of the ligamentum teres to hip stability [83, 84] (Figs. 7.6 and 7.7). In patients with developmental hip dysplasia and low lateral head coverage (lateral center-edge angle—acetabular inclination), the risk of ligamentum teres tears is significantly increased. Thus, tears of the ligamentum teres may occur secondary to subtle instability induced by acetabular undercoverage. In patients with atraumatic microinstability (e.g. ligamentous laxity due to hypermobility syndrome[s]), the ligamentum teres may be at increased risk of tear [85].

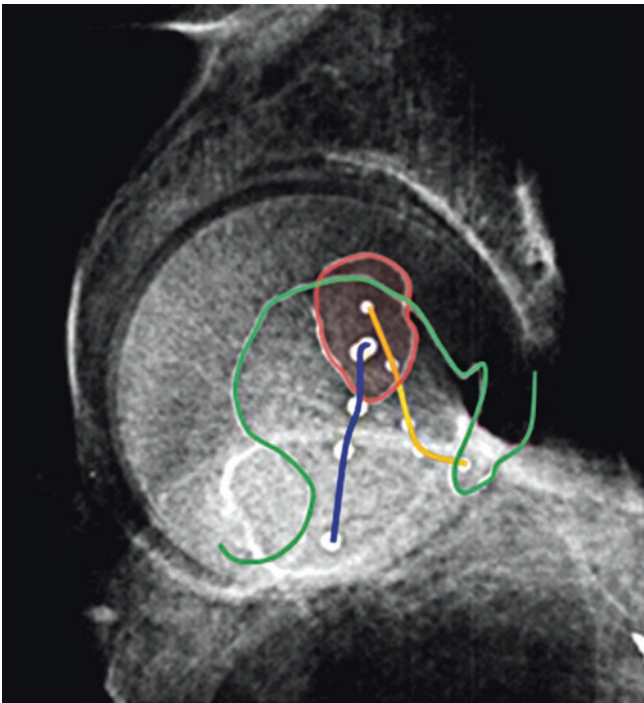


Fig. 7.6 Experimental radiograph of a hip joint in which radiopaque spheres have been implanted in the anterior and posterior bundles of the ligamentum teres. Implanted wires also define the inner edge of the articular surface of the acetabulum (*green*) and the fovea of the femoral head (*red*). In this case the hip has been placed in maximum external rotation in 90 degrees of flexion causing the posterior bundle to be loaded in tension

However, additionally, a tear of the ligamentum teres may also induce microinstability. Surgical treatment of ligamentum teres pathology includes both debridement and reconstruction [86–88]. Both techniques may be performed arthroscopically, with short-term clinical outcomes demonstrating success via improved pain, function, and return to sport [87, 88]. Further clinical and biomechanical studies are needed to conclusively demonstrate the magnitude of the stabilizing effect of the ligamentum teres in adults.

Pathological Impediments to Joint Motion

Femoroacetabular Impingement

Like any other joint, the hip is free to rotate in any direction (i.e., abduction-adduction, flexion-extension, and internal-external rotation) until constrained by either:

- Tightening of soft tissues crossing the joint (primarily the joint capsule and extracapsular ligaments, but also the surrounding musculature)
- Direct contact between the opposing articular surfaces (i.e. of the femur and the pelvis).

In the normal hip, the relative contributions of these limiting factors vary with the relative position of the femur and the pelvis. However, for most motions, joint motion is primarily determined by the laxity of the joint capsule and comes to an end once the ligamentous structures become taut [48, 89]. With some motions, for example, flexion and internal rotation, resistance to motion occurs before ligamentous tightness becomes significant. In these motions, contact either occurs between the bony surfaces (i.e., through bone-on-bone impingement) or the motion arc brings the femoral head to the edge of its spherical surface where the radius of curvature increases causing a cam braking effect if further rotation is attempted.

While these interactions are important for understanding the kinematics of the normal hip, they are of critical importance to our understanding of the role of pathologic bony morphology on chondro-labral degeneration and the development of osteoarthritis. Characteristic deformities of the hip joint that are known to affect joint motion, especially flexion and internal rotation, include SCFE in the pediatric hip [90]; asphericity of the femoral head, with a flattening or extension of the articular surface at the anterolateral head-neck junction (“pistol grip deformity” [91]); and the presence of a deep acetabulum with overgrowth of the acetabular margin. It has long been appreciated that the mechanism of reduced flexion/internal rotation in patients with SCFE is posterior displacement (“slip”) of the femoral head with respect to the neck leading to reduced offset at the anterior head-neck junction. This brings the femoral neck closer to the anterior rim of the acetabulum during flexion and further away during extension, leading to a shift in the functional motion arc and damage to the acetabular margin with repetitive impingement [92, 93].

In hips with morphologic changes in the shape of the head or the depth of the acetabulum, two distinctly different forms of FAI have been popularized, respectively termed “cam-type” and “pincer-type” impingement [53] (Figs. 7.8, 7.9, and 7.10). The first form (“cam-type”) occurs when the femoral head rotates within the acetabulum until the point that an enlarged portion of at the head-neck junction encounters the acetabular rim and is forced beneath the labrum [94–96]. The forced expansion of the labrum causes a stress concentration at the chondro-labral junction where the relatively stiff subchondral plate with its overlaying cartilage surface meets the more flexible labrum. With repetitive engagement of the oversized cross-section of the head and head-neck junction with the anterior acetabulum, pathological changes may occur, including subchondral para-acetabular cysts, fibrillation and splitting of the chondral surface, and delamination of cartilage from underlying bone [97]. Ultimately cysts may also develop within the femoral head or the head-neck junction.

The reduction in the functional range of hip motion arising from the abnormalities of the head-neck junction present

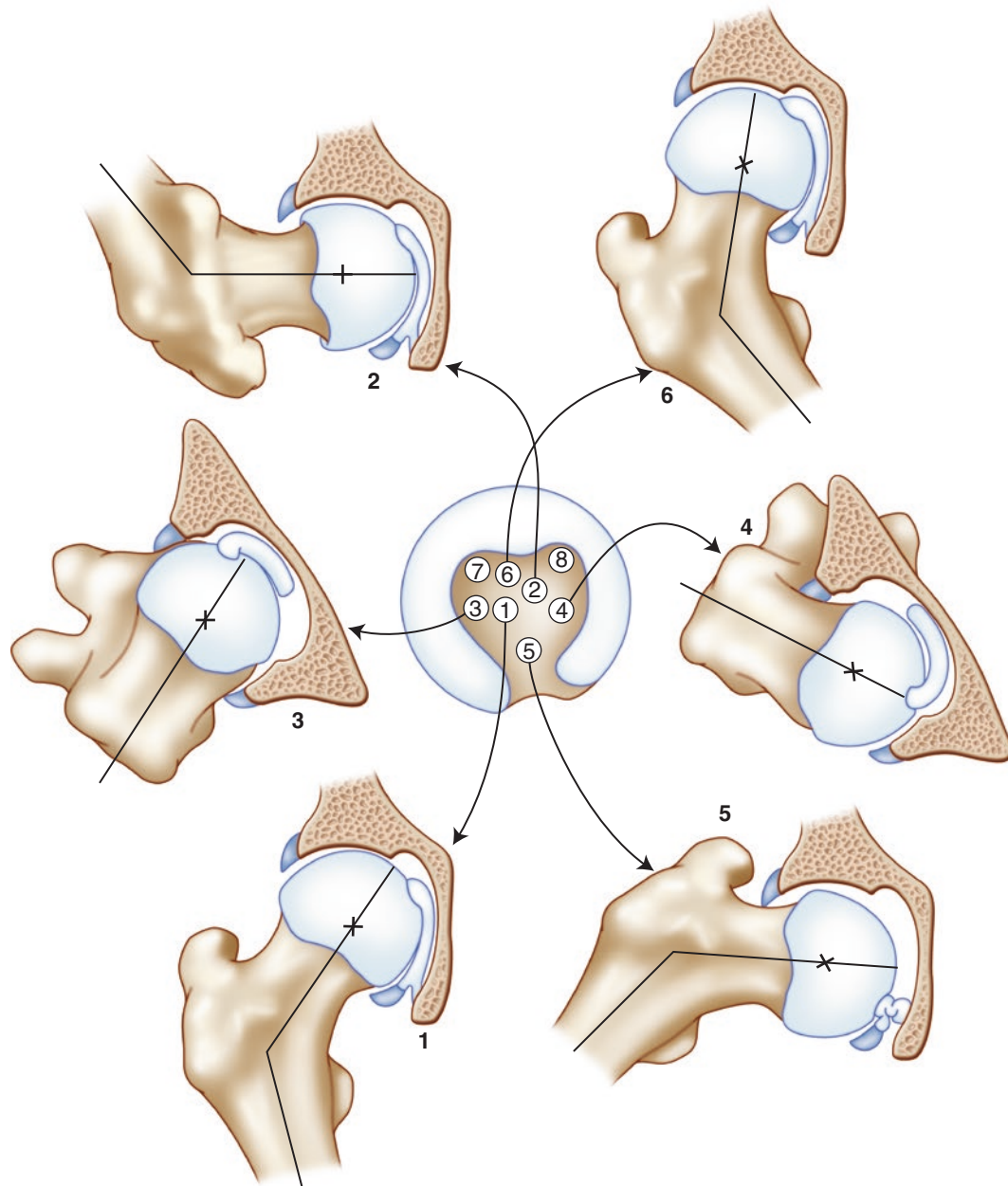


Fig. 7.7 Diagrammatic representation of the relative positions of the femur, ligamentum teres, and the floor of the acetabulum with placement of the hip joint in eight different positions encompassing the range of motion of the joint

in FAI has been extensively studied by Kubiak-Langer et al. [98], using computer simulation of hip motion. Using patient-specific models of the hip derived from CT reconstructions, significant loss of hip motion was demonstrated in FAI cases during flexion, abduction, and internal rotation in 90 degrees of flexion (Table 7.4). The pathomechanics of FAI at the tissue level has been investigated by Ferguson and coworkers using three-dimensional computational models of normal and pathological joints incorporating morphologic variations in the head-neck junction (alpha angle) and femoral head coverage (center-edge angle) [99] (Figs. 7.11 and

7.12). In this study, the effect of functional loading of the hip on stresses developed within the soft tissues of the joint was modeled for walking and sit-to-stand activities. Quite different stress distributions were predicted as a function of activity. During walking, von Mises stresses within the joint cartilage increased gradually as the center-edge angle decreased, but were not affected by the presence of a Cam-FAI (Fig. 7.12). Moreover, peak stresses only exceeded 4 MPa in simulations with a CE angle of less than 23°. Conversely, during a stand-to-sit activity, peak stresses in the normal hip were less than 2.5 MPa and changed little with

Fig. 7.8 Diagrammatic representation of the pathomechanics of joint damage associated with femoroacetabular impingement, as proposed by Ganz and coworkers [71, 90, 92–95]. “Pincer impingement” (a, b) is observed in cases over “over-coverage” of the femoral head by the acetabular margin, leading to direct impact at the extremes of motion and labral and articular damage. “Cam impingement” (c, d) occurs when an enlarged area of the femoral head at the head-neck junction is too large to fit into the acetabulum, typically during flexion and internal rotation. Attempts to force the hip to flex or rotate further cause overstuffing of the acetabulum and chondro-labral separation [92]. From Stal et al., *Biomechanics of the Natural Hip Joint*, In: *Surgery of the Hip*, D.J. Berry and J.R. Lieberman, eds. Volume 2. 2013, pp 5–13. Copyright Elsevier 2013, reprinted with permission

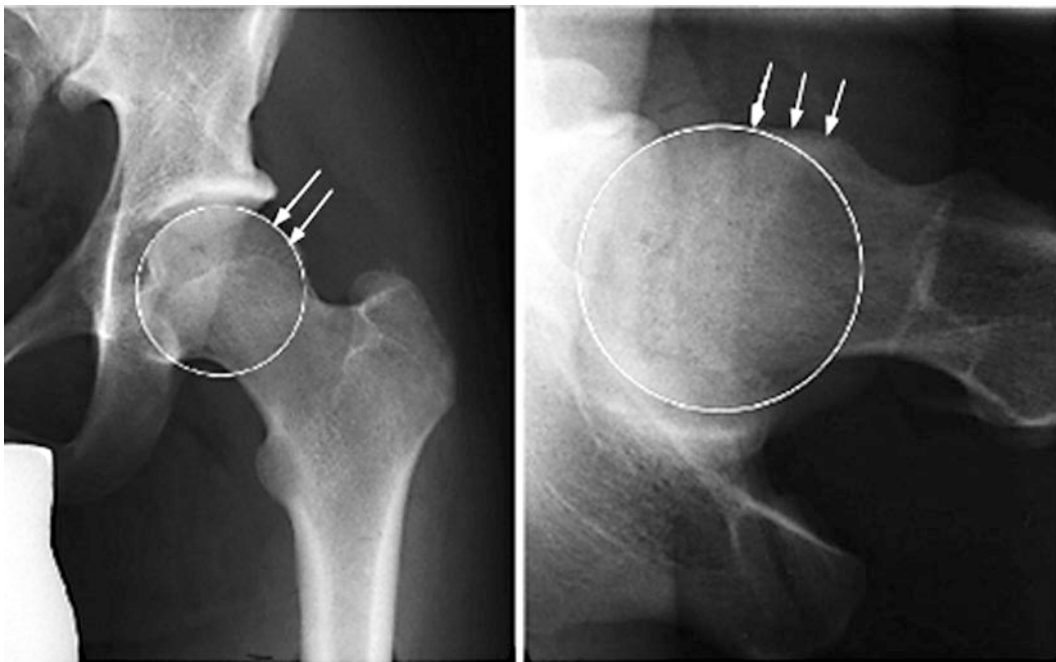
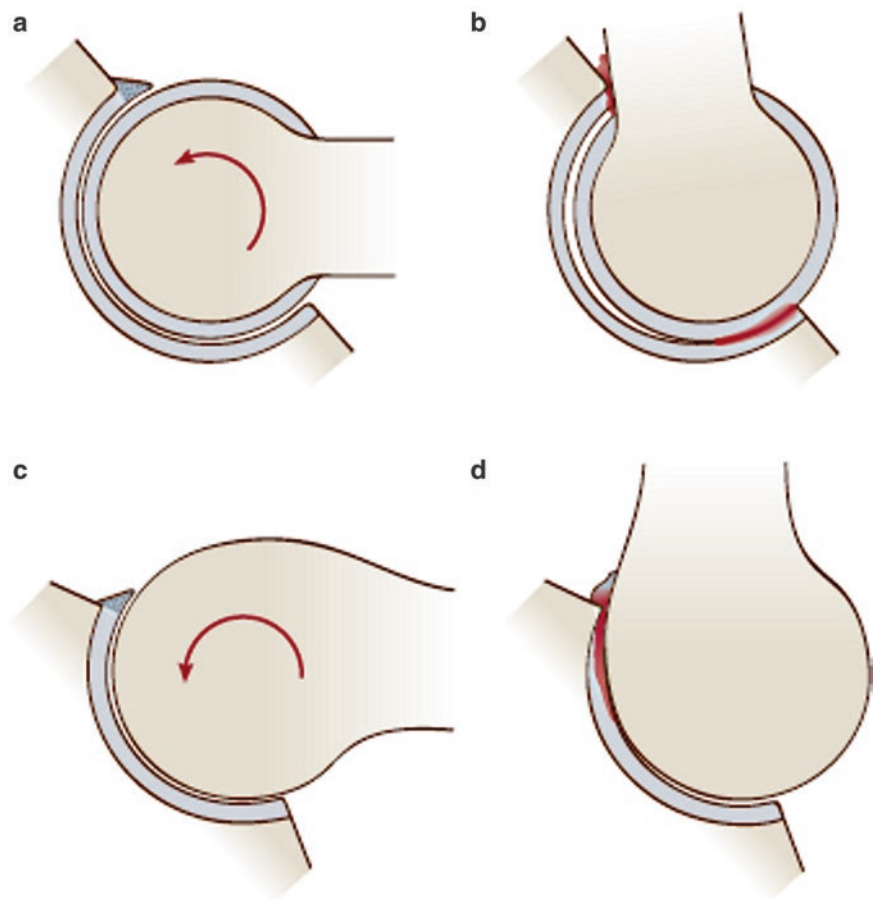


Fig. 7.9 Radiographic appearance of a cam-impinging hip with a “pistol grip” appearance. An aspherical section of the femoral head is seen extending beyond the circle of best fit to the head profile on both the AP and lateral radiographs (see *arrows*). From Stal et al., *Biomechanics of*

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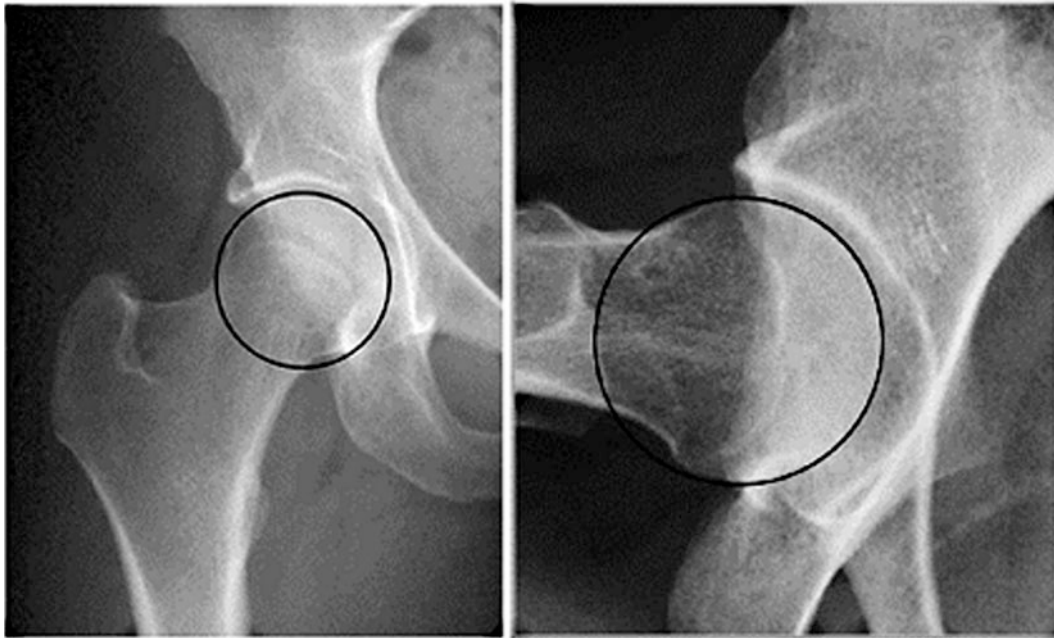


Fig. 7.10 Radiographic appearance of a hip with coxa profunda combined with pincer impinging appearance secondary to ossification of the acetabular labrum. The femoral head is spherical in both the AP and

lateral views. From Stal et al., *Biomechanics of the Natural Hip Joint*, In: *Surgery of the Hip*, D.J. Berry and J.R. Lieberman, eds. Volume 2. 2013, pp 5–13. Copyright Elsevier 2013, reprinted with permission

Table 7.4 Hip motion (in degrees) predicted by computer simulation for a population of normals compared to patients with femoroacetabular impingement, before and after osteochondroplasty

Parameter	Normal hips	FAI hips (pre-op)	<i>p</i> value (normal vs. FAI)	FAI hips (post-osteoplasty)	<i>p</i> value (pre vs. post)
Flexion	122.0° ± 16.3°	105.2° ± 12.2°	<0.0001	125.4° ± 9.7°	<0.0001
Extension	56.5° ± 20.1°	61.1° ± 31.8°	0.751	71.1° ± 26.4°	0.051
Abduction	63.3° ± 10.9°	51.7° ± 12.2°	<0.0001	63.6° ± 7.5°	0.0001
Adduction	32.7° ± 12.3°	34.6° ± 12.3°	0.927	35.8° ± 15.3°	0.262
Internal rotation (90° flexion)	35.2° ± 6.9°	11.1° ± 6.9°	<0.001	35.8° ± 15.3°	0.002
External rotation (90° flexion)	102.5° ± 14.2°	83.0° ± 33.7°	0.194	93.9° ± 32.7°	0.327

All values are expressed as the average ± standard deviation. From Kubiak-Langer et al. [98]

CE angle, except in cases of extreme femoral containment (CE angle = 40°). Much higher stresses were calculated for the FAI cases, where peak values exceeding 5 MPa were predicted whenever the sum of the alpha and CE angles exceeded 90°, with stresses exceeding 10 MPa in almost all simulations with a CE angle of 30° or greater. These predictions correlate well with the clinical observations seen during the progression of joint degeneration in the face of FAI, including the development of chondral defects at the acetabular margin, full-thickness delamination of the acetabular cartilage, and detachment of the labrum at the chondro-labral junction [100] (Fig. 7.11).

Femoral and Acetabular Dysplasia

Finite element analysis has shown that subtle variations in the morphology and orientation of the femur and acetabulum may generate substantial perturbations in the distribution of load between the bony, cartilaginous, and soft tissues. This could explain the occurrence of secondary remodeling, deviations in joint kinematics, “microinstability,” and, ultimately, osteoarthritis [101]. This patho-mechanical progression is exemplified by the dysplastic hip, in which the shallow acetabulum provides reduced coverage of the femoral head, combined with a more vertically inclined weight-bearing

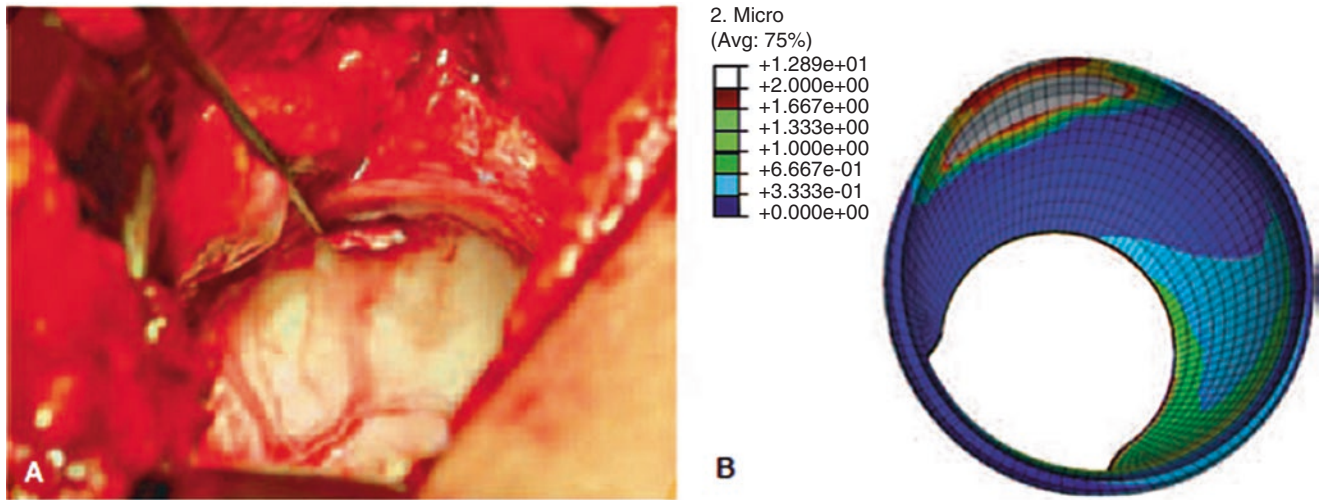


Fig. 7.11 (a) Intraoperative appearance of an anterior-superior labral tear in cases of cam impingement. (b) Distribution of von Mises stresses at the acetabular rim predicted by computer modeling of a severe cam-impinging joint ($\alpha=80^\circ$) during deep flexion (standing to sitting, ante-

rior=*left*). From Chegini, S., et al. The effects of impingement and dysplasia on stress distributions in the hip joint during sitting and walking: a finite element analysis. *J Orthop Res*, 2009. 27(2): p. 195–201. Reprinted with permission from John Wiley and Sons

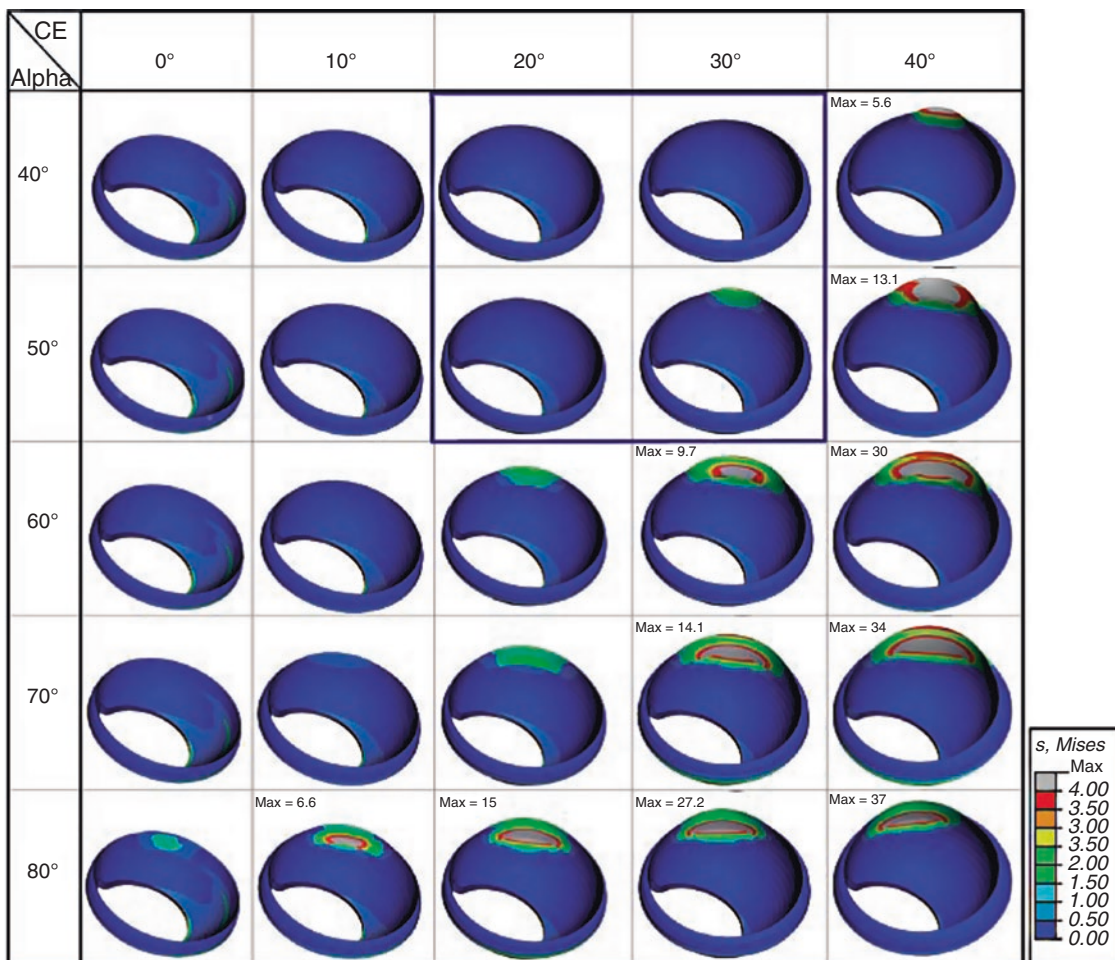


Fig. 7.12 Distribution of von Mises stresses (MPa) within the acetabular cartilage during standing to sitting for all simulated joint geometries. The joints considered normal are surrounded by the blue rectangle. From Chegini, S., et al. The effects of impingement and dysplasia on

stress distributions in the hip joint during sitting and walking: a finite element analysis. *J Orthop Res*, 2009. 27(2): p. 195–201. Reprinted with permission from John Wiley and Sons

surface. This results in high contact stresses, overloading of the labrum and hip capsule, and premature joint degeneration [100, 102, 103].

The influence of acetabular dysplasia on contact pressure has been studied using a variety of computer simulation and experimental methods. One approach, presented by Genda and coworkers [19], utilized a 3D parametric model of the hip joint to explore the impact of classic anatomic parameters on the calculated value of the hip joint reaction force. These parameters included the center-edge (CE) angle, the Sharp angle, the head diameter, the direction of pull of the abductors, and the greater trochanter and the femoral head center. An interesting finding was that under conditions typical of the dysplastic hip (i.e., low CE angle), the model predicted that peak articular pressures would exceed 6 MPa when the abductor line of action was within 10° of vertical. This corresponds to four times peak pressures predicted for the normal female hip using the same method of analysis. Similar results were reported by Tsumura et al. [104] using patient-specific CT reconstructions. In this case, peak pressures of 2.5 MPa were predicted at the acetabular rim of normal hips, compared to 5.3 MPa for dysplastic cases.

Other authors, including Hadley et al. [105] and Russell et al. [102], have studied the effect on articular cartilage of cumulative pressure exposure over a simulated lifetime of loading. Substantial differences in cumulative pressure were demonstrated between normal and dysplastic hips, suggesting a patho-mechanical mechanism for cartilage degeneration in osteoarthritis based on chronic overload. Russell and coworkers [102] also highlighted that, beyond gross morphological differences, small bone irregularities can cause localized pressure elevations. Subsequent computational models have provided further insight into the relationship between

joint morphology, daily loading, and cartilage contact pressures and stresses [106–108] (Figs. 7.13 and 7.14).

Soft tissue damage and degeneration are unavoidable consequences of the focal overload of the acetabular rim that occurs as part of the natural history of hip dysplasia [92, 94, 103]. As luxation of the femoral head progresses, high shearing and tensile stresses develop within the labrum as this peripheral structure takes over some of the load-bearing function of the acetabulum, leading to hyperplasia and ultimately damage to the labrum, typically within the anterior-superior quadrant [103], as predicted by computer models [98]. Clinical observations have provided confirmation of the causal link between focal overload of the acetabular rim and cartilage degeneration and labral rupture [103, 109, 110]. Thinning of the anterior cartilage has been observed in 80% of dysplasia patients, in association with forward and upward displacement of the femoral head [111]. Ultimately, the peripheral soft tissues of the joint are an inadequate substitute for the stability afforded by a congruent acetabulum with good lateral coverage; hence, acetabular reorientation surgery is often performed in an attempt to increase the longevity of the dysplastic hip [112, 113].

Microinstability

Some patients with radiographically normal hips and increased range-of-motion experience a sensation of joint instability without radiographic evidence of femoroacetabular impingement. In these cases, hypermobility of the joints is common syndrome with elevated Beighton and/or Brighton scores [114, 115]. Dancers, ballet dancers, figure skaters, yogi and yogini, cheerleaders, martial artists, and gymnasts

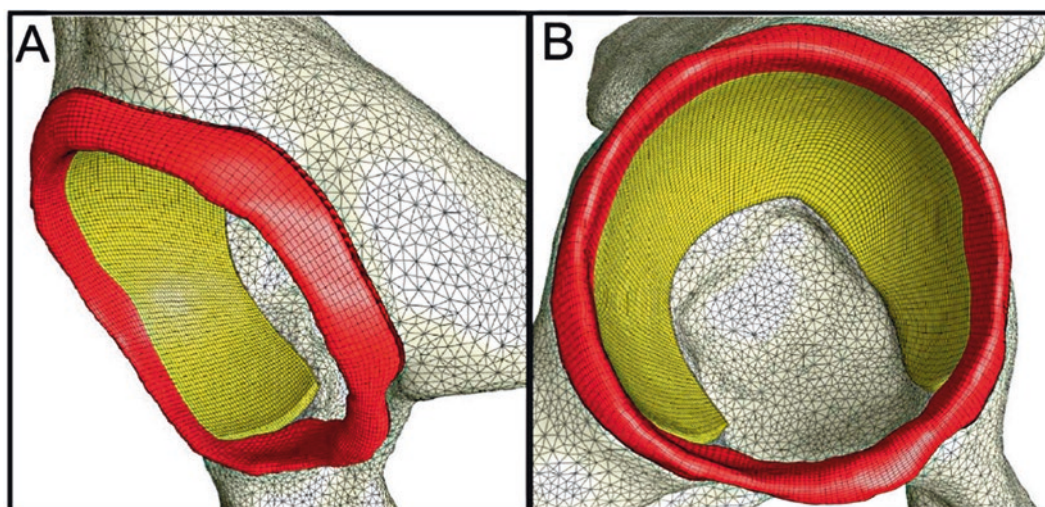
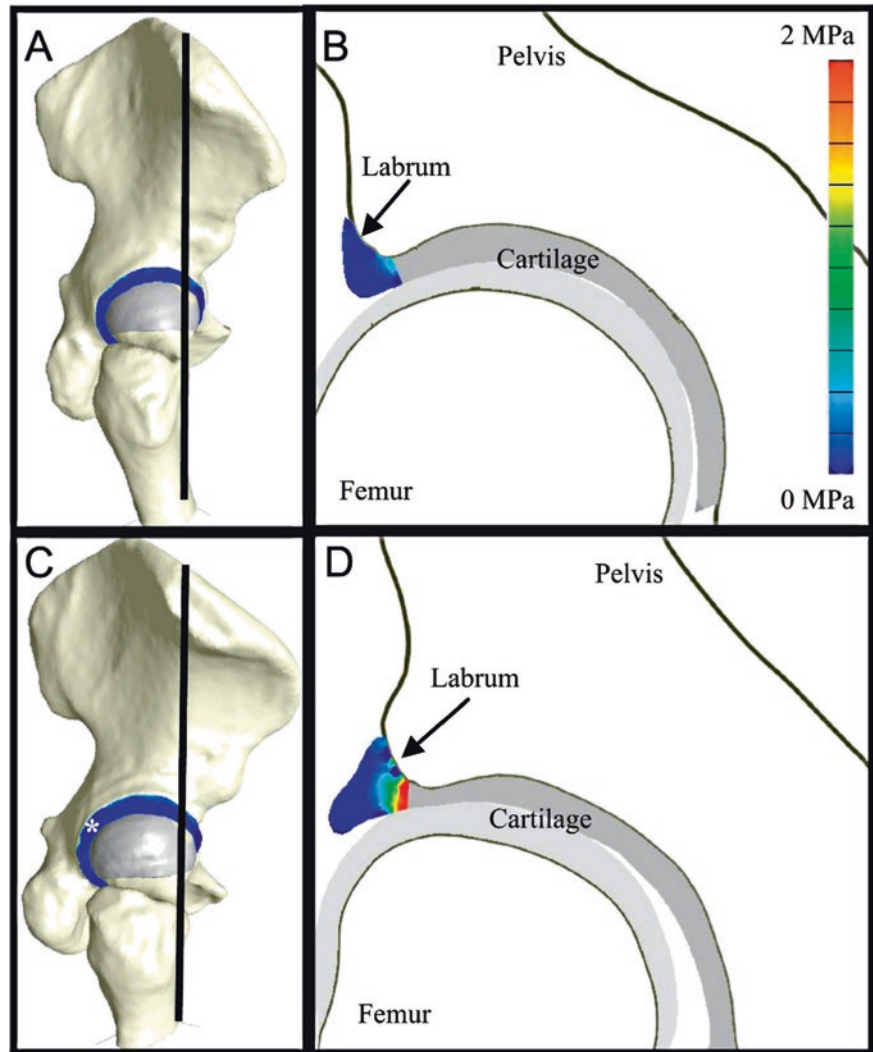


Fig. 7.13 The finite element mesh used by Henak and coworkers [107] to simulate load transfer across the hip joint in the presence of varying degrees of hip dysplasia and FAI morphology. The bony hemipelvis (white), the acetabular cartilage (yellow), and the labrum (red) are each

represented. From Henak, C. R., et al. Role of the acetabular labrum in load support across the hip joint. *Journal of Biomechanics*, 2011;44(12):2201–2206. Reprinted with permission from Elsevier

Fig. 7.14 Predicted values of internals stresses within the anterosuperior labrum developed in response to load transfer during walking (heel strike). This analysis was performed for the normal hip (a, b) and the dysplastic hip (c, d). The stresses depicted in the (b, d) were calculated for the vertical planes taken along the black lines in (a, c) corresponding to the approximate location of maximum deflection of the labrum. From Henak, C. R., et al. Role of the acetabular labrum in load support across the hip joint. *Journal of Biomechanics*, 2011;44(12):2201–2206. Reprinted with permission from Elsevier



may have a high prevalence of increased motion and potential microinstability, often prerequisite to the activity performed [116]. In a cohort of 59 professional ballet dancers, only one hip had evidence of a cam deformity, while several other abnormalities were identified on magnetic resonance imaging due to a dynamic “pincer” mechanism from the extreme motion involved with their dance activities [116]. Further, while in the “splits” position, all hips subluxed (mean: 2.1 mm). In comparison to a control group, the musculoskeletal abnormalities in the ballet group included a higher incidence of acetabular cartilage lesions (mostly superior), labral tears (mostly posterosuperior to anterosuperior), and a larger number of herniation pits in the superior femoral neck. Despite the relatively low incidence of bony deformities, 60% of dancers reported groin pain, but only when dancing [117]. Further, when each dancer assumed the most demanding positions of classical ballet, the mean translation of the femoral head within the acetabulum ranged from 0.93 to 6.35 mm [118].

Iatrogenic microinstability may be induced in patients who have undergone hip arthroscopy with capsulotomy without capsular closure [119, 120]. This conclusion is consistent with the findings of four cadaveric biomechanical studies (Table 7.5) demonstrating the importance of the ilio-femoral ligament to the structural integrity of normal hip joint mechanics. In each of these studies, sectioning the ilio-femoral ligament increased external rotation, extension, and anterior translation of the hip with no difference between the intact and repaired state. Discrete frank hip dislocation is at the end of the microinstability spectrum. In the postoperative setting, instability may occur along this spectrum to variable degrees. Nine cases of post-arthroscopic iatrogenic hip dislocation have been presented in the literature [121–128]. Due to publication bias, this is likely a significant underestimate of the true incidence of instability following hip arthroscopy. Thus, to avoid micro- or macro-instability during hip preservation surgery, most authors recommend avoiding the following: extended capsulectomy without repair, labral

Table 7.5 Cadaveric studies examining the effect of the ligaments of the hip capsule on the stability and range of motion of the hip

Study	Study design	Role
Bayne et.al. (2014)[128]	Cadaveric biomechanical— motion tracking	• After capsulotomy, in neutral, increased distal, lateral, and anterior translation
		• After capsulotomy, in flexion, increased distal, medial, and posterior translation
		• After capsulotomy, more translation in neutral, more rotation in flexion
		• In maximal flexion, hips with capsulotomy rotated more than intact capsules
Myers et al., (2011) [50]	Cadaveric biomechanical— fluoroscopy	• Increased external rotation with IFL sectioning (increased 12.9°) ($p < .0001$)
		• Increased anterior translation with IFL sectioning (increased 1.8 mm) ($p < .001$)
		• No difference in external rotation or anterior translation between intact/repared state
Martin et al. (2008) [48]	Cadaveric biomechanical— motion tracking	• Release of medial, lateral arms IFL gave greatest increase of external rotation
		• Lateral arm release provides more motion in flexion and neutral
		• Lateral arm release also provides more internal rotation, primarily in extension
Hewitt et al. (2002) [46]	Cadaveric biomechanical— load to failure	• IFL much stronger than the ischiofemoral ligaments
		• IFL greater stiffness than ischiofemoral ligaments
		• IFL greater tensile load to failure than ischiofemoral ligaments

resection (versus repair or refixation), aggressive acetabuloplasty (rim resection) in dysplastic configurations, and overall capsular laxity.

The presence of microinstability does not automatically preclude other concomitant intra- and extra-articular disorders. In fact, it is likely that subtle instability increases the loading of some of the structures of the hip including the labrum, the articular cartilage and underlying subchondral bone, the ligamentum teres, the iliopsoas tendon, the iliotibial band, the hip abductors, and the remaining normal capsule. Even patients with traditional femoroacetabular impingement may have subtle underlying microinstability due to preclusion of true ball-and-socket mechanics, thus causing anterior levering over the rim (fulcrum) with subsequent posterior instability. Further:

- Excessive acetabular anteversion can result in anterior hip instability and posterior acetabular rim impingement.
- Excessive acetabular retroversion can result in anterior impingement and posterior instability.
- Excessive femoral anteversion can result in anterior hip instability and posterior acetabular rim impingement.
- Excessive femoral retroversion can result in anterior impingement and posterior instability.

Conclusions

We believe that this chapter demonstrates how much Musculoskeletal Biomechanics has contributed to our understanding of the hip over the past half-century. The progress we have documented in this work has now delivered us to the dawn of Translational Biomechanics. This transformation has been made possible through the intersection of many advances in computing and imaging technology, combined with a deepening understanding of hip pathology. The focus of this field has shifted from aca-

demical predictions of hip joint forces to their direct measurement and from speculation as to possible relevance of stresses to OA to the prediction of tissue stresses in individual hips using patient-specific finite element models.

Meanwhile, the intense investigation by clinicians and scientists of common conditions affecting the health of the joint, most notably FAI, has led to the utilization of research tools in the study of the pathomechanics of hip degeneration and strategies for joint preservation. This approach has the potential for use in creating individual treatment plans in much the same manner as Medical Physics transformed the practice of radiotherapy in decades past.

The next steps in the emergence of “Translational Biomechanics” are already in motion. These include:

• Patient- and activity-specific modeling of the mechanical response of the hip

• Prediction of the risk of individual patients in developing hip OA on the basis of biomechanical risk factors

• The role of instability and tissue laxity in the etiology of osteoarthritis of the hip.

• The interaction of hard and soft tissues as determinants of joint stability and their effect on the mechano-biology of joint health and degeneration

The authors invite every reader to participate in this journey and contribute to its ultimate success!

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